TURKANA COUNTY

VETERINARY HANDBOOK

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TURKANA COUNTY GOVERNMENT

FOR PRIORITY LIVESTOCK DISEASES

Diagnosis, Treatment and Control Guidelines
For Animal Health Service Providers

Acknowledgements

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Disclaimer

THIS HANDBOOK IS INTENDED TO GUIDE ANIMAL HEALTH PRACTITIONERS WORKING IN TURKANA COUNTY MANAGE ENDEMIC LIVESTOCK DISEASES. THE HANDBOOK INTEGRATES CONVENTIONAL VETERINARY KNOWLEDGE WITH ETHNOVETERINARY KNOWLEDGE & PRACTICE. PRACTITIONERS NEED TO BE AWARE OF THE LAWS AND REGULATIONS THAT APPLY TO THE PRACTICE OF VETERINARY MEDICINE IN KENYA AS THE HANDBOOK DOES NOT EXPLICITLY COVER VETERINARY JURISPRUDENCE. THIS HANDBOOK IS NOT INTENDED TO SUBSTITUTE FOR THE MANDATED PHYSICAL EXAMINATION OF ANIMALS BEFORE PRESCRIBING MEDICATION. IN ADDITION, THE DRUG PACKAGE INSERT OF INSTRUCTIONS FROM MANUFACTURERS SHOULD BE READ AND UNDERSTOOD BEFORE ANY DRUG IS ADMINISTERED OR PRESCRIBED. THE PRACTITIONERS SHOULD ALWAYS ENSURE THAT LIVESTOCK KEEPERS UNDERSTAND THE LENGTH OF THE WITHDRAWAL PERIOD FOR MILK, MEAT AND EGGS. THE HANDBOOK MENTIONS VARIOUS VETERINARY MEDICINAL PRODUCTS TRADE NAMES. THIS SHOULD NOT BE TAKEN AS AN ENDORSEMENT OF THE PRODUCT.

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Preface

The Turkana County Veterinary Handbook provides a summarised description of the priority infectious diseases and conditions known to occur in camels, cattle, goats, sheep, donkeys and chickens in the county. The compilation of the Handbook builds on information sourced from existing veterinary medicine textbooks and peer-reviewed articles as well as the ethnoveterinary information gathered from the county veterinary staff and livestock keepers. The Handbook has incorporated the ethnoveterinary livestock disease names and practices of the Turkana community. The incorporation of ethnoveterinary knowledge is based on the understanding that working in pastoralists communities necessitates that animal health workers not only acquire updated livestock disease knowledge but should also possess an understanding of the social, economic and cultural context in which they operate.

The Handbook is written for users with formal training in animal health and production. The handbook is divided into nine chapters as detailed below:
Chapter one begins with a glossary of terminologies and gives a brief description of microbes that cause infectious diseases.
Chapter two highlights guidelines on how to conduct general and physical clinical examination.
Chapter three to nine details specie specific priority diseases information that includes aetiology, epidemiology, clinical presentation, post-mortem lesions, diagnosis, treatment and control guidelines. The chapters also include local dialect names of the diseases and associated clinical signs as well as ethnoveterinary treatment and control practices.
Annex section- The handbook book has 5 Annex sections: Annex 1 details the Turkana county vaccination schedule for identified priority livestock diseases; Annex 2 summarises information of four priority Zoonotic Diseases in Kenya; Annex 3 Discusses one health approach and antimicrobial resistance; Annex 4 describes how to calculate drug dosages in livestock using weight estimates and Annex 5 is a pictorial guide of some medicinal and poisonous plants identified by livestock keepers in Turkana County.

It is the author's sincere expectation that the handbook will prove to be a practical field companion for animal health practitioners in Turkana county.

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Chapter 1:

Introduction to Infectious Livestock Diseases

1.1 Glossary of Terminologies

Abortion	Premature expulsion of the foetus and foetal membranes. The termination of pregnancy. Abortion is one of the indicators of infertility in livestock.
Abscess	Localised lesion comprising of pus, surrounded by inflamed tissue.
Acute	A disease characterised by sudden/rapid onset, short course (3-14 days), severe symptoms and either causes death quickly or leads to a speedy recovery.
Aetiology / Etiology	Study of disease causative agents
Alopecia	Loss of hair, wool or feathers
Anaemia	A reduction in the number and/or size of the red blood cells or the haemoglobin in the blood
Anorexia	Loss of appetite
Anuria	Ceasation of urination
Antibody (Ab)	A substance in the blood serum or other body fluids formed to exert a specific restrictive or destructive action on bacteria, their toxins, viruses, or any foreign proteins
Antibiotic	Chemical compound derived from living organisms or synthesised from biological material that is capable, in small concentration, of inhibiting the life process of micro-organisms particularly bacteria
Antigen	A toxin or other foreign substance which induces an immune response in the body especially the production of antibodies
Ascites	Accumulation of fluid in the abdominal cavity/ peritoneal sac
Ataxia	Incoordination of gait
Attenuated	Term used to describe a reduction in the virulence of a micro-organism, particularly applied to those incorporated in Vaccines.
Bacteria	One celled microscopic organism that don't require a host to survive, they are free living, they reproduce/multiply by binary fission and do not have an organized nuclei or any other membrane-bound organelles.
Bacteraemia	Presence of non-multiplying bacteria in blood
Carcass	Body of a dead animal or bird
CDR	Community Disease Reporters are livestock keepers selected by the community to identify certain syndromes indicating occurrence of notifiable diseases and report these syndromes to the county veterinary staff.
Chronic	A disease whose course is more than 4 weeks and signs are not severe
Coma	State of unconsciousness in an animal or bird

Table 1: Glossary of terminologies

1.1 Glossary of Terminologies

Colic	Abdominal pain
Congestion	Accumulation of blood in a body part due to overfilling of blood vessels.
Contagious/ infectious	The spread of contagion or infection from animal to animal by direct or indirect contact.
Cystitis	Inflammation of urinary bladder
Dandruff/ Ptyriasis	Condition characterised by presence of bran like scales over the skin surface.
Dermatitis	Inflammation of skin tissue
Dehydration	Excessive loss of water or other fluids from the body. Typically through either vomiting and/or diarrhoea or inappropriate intake of water into the body (decreased thirst)
Diagnosis	An art of confirming a disease with the help of case history, symptoms and laboratory tests
Diarrhoea	Frequent passage of loose faeces
Disease	Deviation from a normal state or function in animals that produces specific symptoms or that affects a specific body organ or system and is not directly due to physical injury.
Dysentery	Frequent passage of loose faces mixed with blood, mucus and accompanied by straining
Dysphagia	Difficulty is swallowing
Dysuria	Difficulty in urination
Ectoparasites	Live on the skin of animals and include Ticks, Mites, lice, fleas and to some extent biting flies. Some of them are visible and can be seen with the naked eye while others are microscopic.
EDTA	Ethylene Diamine Tetra Acetic acid (EDTA) is an anticoagulant that is coated into purple top blood collection tubes. EDTA tubes are used to collect blood samples that should not clot.
ELISA	Enzyme-linked immunosorbent assay (ELISA) is a laboratory technique used to measure the antigen-antibody concentration in a sample.
Emaciation	Excessive loss of body condition / weight due to disease
Endemic / Enzootic	Endemic refers to a disease that is present in a livestock population at all times while Enzootic refers to an outbreak of an endemic disease among livestock populations in a particular locality (Village or Ward)
Epidemiology	Study of disease in populations and of factors that determine their occurrence
Epidemic / Epizootic	A disease that spreads rapidly to affect a large population of livestock in a large geographical area (sub-county, county, country) at the same time.
Erythema	Reddish discolouration of the skin
Fever	Rise in body temperature associated with infection

1.1 Glossary of Terminologies

Fomites	Inanimate objects or materials such as clothes, utensils, and furniture which are likely to be contaminated with infectious agents
Fungi	Fungi (plural Fungus) are single celled or multicellular organisms that are free living and include moulds and yeasts
Healthy	Describes the physical, physiological and mental wellbeing of an animal
Haematuria	Blood in urine
Haemoglobinuria	Haemoglobin in urine
Hepatitis	Inflammation of the hepatic (liver) cells
Host	Living organism that is susceptible to or harbours an infectious agent under natural conditions. host factor is an intrinsic factor (such as age, species, breed or) that influences exposure, susceptibility or response to a pathogenic agent. Reservoir host
Hyperaesthesia	Increased sensitivity / response to external stimuli like touch, noise and light
Hypersalivation	Excessive production of saliva due to disease condition
Hypothermia	Decrease of the normal body temperature associated with terminal disease state
Incoordination	In ability to move the limbs in normal sequence
Incubation period	The time that elapses between infection or exposure to a pathogenic agent and appearance of signs of a disease
Incidence (Attack rate)	Number of newly diagnosed cases of a disease. An incidence rate is the number of new cases of a disease divided by the number of animals at risk for the disease.
Infection	Invasion of body tissues by pathogenic organisms resulting in disease
Inflammation	Physiological response to injury or infection where the part of the body becomes reddened, swollen, hot and painful. Inflammatory means causing inflammation.
Immunology	Study of the body's reaction to the presence of foreign and pathogenic substances.
Immunisation	Process of artificially producing resistance to a given infection generally by means of a vaccine or an antiserum or antitoxin
Immunity	The power of the body to resist infection or the action of certain poisons.
Jaundice	Yellowish discolouration of the visible mucous membranes of the body
KEVEVAPI	The Kenya Veterinary Vaccines Production Institute (KEVEVAPI) is a government institution mandated to manufacture and supply of affordable veterinary vaccines in Kenya
Laboured breathing / Dyspnoea	Difficulty in breathing due to disease condition
Lacrimation	Excessive production of ocular discharge due to a disease condition
Lesion	Visible changes in size, shape, colour or structure of a body organ
Microbes	Microscopic organisms that cause disease. They can be bacteria, viruses, protozoa and fungi. Microbes are also called disease causative or pathogenic agents

1.1 Glossary of Terminologies

Mode of transmission	Manner in which a pathogenic agent is transmitted from its reservoir to a susceptible host
Morbidity	State of being symptomatic or unhealthy due to disease or condition.
Mortality	Means death. Mortality rate is a measure of the frequency of occurrence of death among a defined population during a specified time interval.
Necrosis	Death of body tissue, occurs when too little blood flows to the tissue. This can be due to physical injury, infection, radiation, or chemical exposure
Nephritis	Inflammation of kidney
Nodule	Palpable, solid swelling or aggregation of inflammatory cells that is greater than 1 cm in diameter. Nodules are usually found in the dermal or subcutaneous tissue, and the lesion may be above, level with, or below the skin surface.
Notifiable	A disease that, by law, must be reported to public health authorities upon diagnosis
Nystagmus	Involuntary constant jerky movements of the eye ball
Oedema	Abnormal accumulation of fluid in the intercellular spaces /tissues
Opisthotonus	Dramatic abnormal posture due to spastic contraction of the extensor muscles of the neck, trunk and extremities. This results in an upward deviation of the neck that causes the head to be held backwards and facing the sky.
Pathophysiology	the body function changes that accompany a particular syndrome or disease
Pandemic / Panzootic	Global spread of an epidemic disease
Parasite	Organism that benefits by nourishing itself at the expense of the host
Paralysis	Incomplete or complete loss of nervous control over any bodily function
Pallor	Unhealthy pale (white) colour of the visible mucous membranes
Pathogenic	Causing or capable of causing disease pathogenic microorganisms
Per-acute	Disease characterised by a very short course of a few hours to 48 hours with very severe symptoms
Pica	Depraved/ Abnormal appetite
Pharyngitis	Inflammation of pharyngeal mucosa
Post-mortem	Examination of a carcass
Polydipsia	Excessive thirst
Predisposing factor	Anything that makes the animal more prone to infection. Stress lowers immunity hence predisposes animals to infection
Prevalence	Number of disease cases or events among a given population. Prevalence rate is the proportion of a population that has a particular disease at a specified point in time.
Pot-belly	Pendulous abdomen that causes the lower part of the abdomen to assume a convex shape
Prognosis	Forecast regarding to outcome of the disease

1.1 Glossary of Terminologies

Protozoa	Microscopic single celled free living parasitic organisms
Pruritus	Sensation that gives rise to the desire of scratching /itching
Purulent	Containing pus
Pyuria	Pus in urine
Recumbency	Abnormal state of animal lying down associated with difficulty in rising
RT-PCR	Reverse Transcriptase Polymerase Chain Reaction (RT- PCR) is a laboratory confirmatory diagnostic test used to detect specific genetic material of antigens in samples. The test converts the antigen RNA molecule in the sample into its complementary DNA (cDNA) sequence by reverse transcriptase.
Scab	Dried inflammatory exudate such as blood, pus and skin debris
Septicaemia	Presence of multiplying bacteria and toxins in blood
Shock	State in which there is generalised acute and serious reduction in the perfusion of tissue due to reduced blood circulation and pressure.
Spasm	Involuntary contraction of group of muscles
Sporadic	Disease that affects a few (one or two) animals in a herd and shows little or no tendency to spread within the herd
Stomatitis	Inflammation of the oral mucosa
Sudden death	Unexpected death of an apparently healthy animal
Symptom	Outward clinical manifestation of disease
Transmission	Passing of a disease causing agent from an infected individual or group to previously uninfected individual or group.
Treatment	Curative drug and symptomatic treatments are drugs to get rid of symptoms
Tenesmus	Straining to pass faeces
Tetany	Tonic symmetrical muscular contractions
Tremor	Jerky involuntary contractions of muscles
Trismus	Lock-jaw
Toxaemia	Presence of toxins in blood
Vaccine	Suspension of attenuated or killed disease causing bacteria, virus or rickettsia administered for prevention of infectious diseases
Vaccination	Protective inoculation with a vaccine to induce immunity
Virus	Viruses are the smallest of all microbes. They are unable to exist outside the body of the host. They are sometimes referred to as microscopic particles.
World Organisation for Animal Health (WOAH)	World Organisation for Animal Health (WOAH) formerly known as OIE, is an inter-governmental organisation responsible for improving global animal health. WOAH publishes health standards that are used by the World Trade Organisation (WTO).
Zoonotic	Disease transmitted from animal to humans and vice versa.

1.2 General Appearance of Heathy and Diseased Livestock

Table 2: Parameters of healthy and diseased livestock

Parameter	Healthy	Diseased
1. Feed and water intake	Normal	Reduced or absent
2. Rumination	Frequent	Less frequent or stops
3. Body condition and hair coat	Good body condition with smooth and glossy hair coat	Emaciated with rough and dull hair coat
4. Demeanour	Alert with head facing forward and raised	Dull with head facing downwards or facing the side if recumbent
5. Gait	Coordinated movement and ambulates with ease	Difficult slow movement or recumbency
6. Eyes	Bright, moist and normally open	Dry or have profuse lacrimation and partially or completely closed
7. Ears and tail movement	Ears are erect and both ears and tail move frequently	Dropping ears and both ears and tail move less.
8. Mouth and Muzzle	Muzzle is moist and mouth is wet with no odour	Dry muzzle, mouth with hypersalivation and has a bad odour
9. Nose	No discharge	Discharge present
10. Dung and Urine	Dung is semi-solid. Urine is slightly yellow	Hard or loose dung. Urine is dark yellow, coffee coloured or pinkish

1.3 Aetiology and Epidemiology of infectious livestock diseases

The success of any livestock operation is closely related to the disease level of the animals. Losses due to disease outbreaks can be classified as direct such as death, medication costs, and restriction to markets or indirect due to poor growth, low production levels, poor feed conversion rates and downgrading of livestock products. Disease is defined as any change in the state of an animal or its organs that affect the proper performance or its normal function.

How Infectious Diseases Are Spread

Common ways infectious diseases are introduced into and spread within herds or flocks are listed below;

I. Introduction of diseased animals or birds.
II. Introduction of healthy animals or birds that have recovered from disease but are still carriers and can still transmit the disease to others.
III. Contact with inanimate objects (fomites) that are contaminated with disease organisms (feeds, vehicles, clothing, feeding and watering troughs).
IV. Contact with carcasses of dead animals that have not been properly disposed.
V. Contaminated surface water, pastures or soils.
VI. Rodents and free-flying birds.
VII. Arthropod vectors like Mosquitoes.
VIII. Airborne organisms through aerosol droplets especially in heavily populated livestock areas.

General measures for prevention of infectious diseases

I. Identification and isolation of infected and incontact animals or birds
II. Treatment of affected animals
III. Slaughter of animal suffering from incurable disease
IV. Disposal of dead animals either through burning or deep burial
V. Regular disinfection of contaminated areas like feeding and watering troughs and chicken houses.
VI. During outbreaks restrict movement of infected animals into clean areas (Quarantine and close livestock markets)
VII. Control access of infected animals to communal pasture and watering areas. Infected animals can be watered last and troughs disinfected after use.
VIII. Regular use of anthelmintics and acaricides to control ecto and endoparasites.
IX. Ensure access to balanced nutrition
X. Develop a vaccination schedule and ensure annual vaccination of animals and birds before an outbreak occurs and when animals or birds are in good body condition.

Diseases can be classified into 5 classes or groups based on;

A. Mode of Origin

i. Hereditary diseases- transmitted from parents to the offspring
ii. Congenital diseases- acquired during intra-uterine life
iii. Acquired diseases- transmitted after birth

B. Causative agent

i. Infectious diseases

Infectious diseases are caused by pathogenic microscopic pathogens also known as microbes

that include bacteria, rickettsia, viruses, protozoa and fungi or macroscopic agents such as helminths (worms), liver or rumen flukes, lice and ticks.

ii. Non-infectious diseases

Non-infectious disease is caused by physical, chemical or poisonous agents, nutritional deficiency or disturbed metabolism.

iii. Non-specific diseases

Diseases whose cause is indeterminate or has multiple causes.

C. Mode of spread

i. Contagious disease

Transmitted by means of direct or indirect contact with diseases animal or birds. All infectious diseases may or may not be contagious but all contagious diseases are infectious.

ii. Non-contagious disease

Do not spread by means of contact for example tick borne infections

D. Duration and severity

i. Peracute disease

Characterised by short course (less than 24 hours) and associated with severe symptoms that are often fatal for example Anthrax.

ii. Acute disease

Characterised by sudden onset and a short course of 2 to 14 days with severe symptoms that may be fatal if early treatment intervention is not given for example Haemorrhagic Septicaemia

iii. Sub-acute disease

Characterised by symptoms that are less severe and a course of 2 to 4 weeks, for example Foot and Mouth Disease in Bos Taurus breeds or Sub-Acute Mastitis

iv. Chronic disease

Characterised by a course of more than 4 weeks with signs that are not severe for example Fascioliasis, Trypanosomiasis.

E. Geographical area of spread

i. Sporadic disease

Affects one or two animals in a herd or flock and has no or little tendency to spread for example Johne's disease or Rabies

ii. Enzootic/ Endemic disease

Endemic refers to a disease that is present in a livestock population at all times while Enzootic refers to an outbreak of an endemic disease among livestock populations in a particular locality (village or ward). Example is Haemorrhagic Septicaemia in Camels

iii. Epizootic/ Epidemic disease

Refers to a disease that affects a large population of animals in a large area at the same time and spreads rapidly. Examples, Sheep and Goat Pox, Foot and Mouth disease and New castle disease.

iv. Panzootic/ Pandemic disease

Wide spread epidemic disease with world-wide distribution such as bird flu.

Detailed description of Five disease causative agents

I. Viruses

Viruses are the smallest of all microbes and cannot be observed with a light microscope. Viruses are unable to reproduce and function outside the body of living things hence the reason they are sometimes called microscopic particles instead of single celled organism. Due to their very small size they can easily be carried in dust, water and air. Viruses exist everywhere on earth. They are present in animals, plants, and other living organisms. Unlike bacteria which affect multiple livestock species most viruses are species specific. This explains why a virus that causes illness in sheep and goats may not affect a cow. Viruses have a core of genetic material, that is either DNA or RNA. The core is covered with a capsid, a protective coat made of protein. Around the capsid, there may be a spiky covering known as the envelope. These spikes are proteins that enable viruses to bind to and enter host cells. Viruses hijack the host cell and commandeer it

to replicate virus particles. This causes the cell to die and release viral particles that infect other cells and the multiplication process continues damaging the tissue whose cells have been infected. As they multiply viruses especially RNA have "copying errors" resulting in genetic changes known as mutations. This is a natural process that results in one virus having many antigenically distinct strains. Sometimes there is no cross-immunity among the different strains and animals that recover from infection with a specific strain are susceptible to the other strains.

Viruses come in different shapes as shown in the figure below.

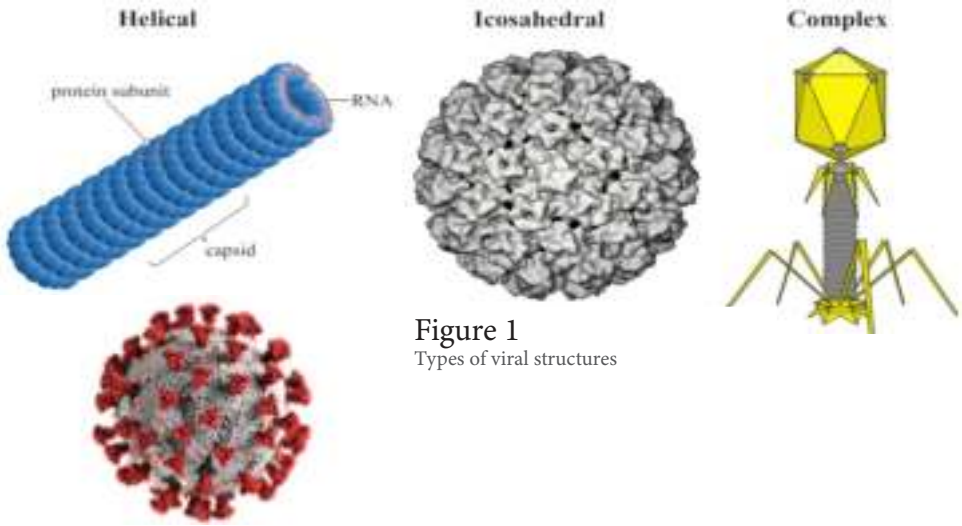


Figure 1
Types of viral structures

Field based diagnosis of viral infections

Viruses are too small to be visualised by light microscopy. The most common tests used in the field to support clinical diagnosis of viral infections are rapid tests. Rapid diagnostic tests (RDTs) are easy-to-use tests as they provide quick results, usually in 20 minutes or less. Unlike most standard tests, which have to be sent to a lab, rapid tests are done and provide results at the point of care (POC). The use of rapid diagnostic tests in the field can greatly enhance disease surveillance, confirm clinical case and facilitate rapid response activities, especially in resource poor settings. The RDTs are mainly lateral flow cassettes based on immunoassay chromatography technology. These tests involve the interaction of a fixed reagent of either target antigen or antibody that is linked to some type of visible detector (Colour dye), that reacts with a patient sample. An example is the Foot and Mouth Disease simple lateral-flow assay (LFA) based on a monoclonal antibody (MAb 70-17) that detects FMD virus under non-laboratory conditions. The LFA uses epithelial suspensions from FMD lesions as the test sample.

Figure 2
FMD Lateral rapid Test

Picture source: Bovine Foot-and-Mouth Disease NSP Antibody Rapid Test
http://www.lifebioscience.com.au/Life_bio/documents/InsertsheetofAnigenRapidFMDNSPAb.pdf

II. Bacteria

Bacteria are bigger than viruses and unlike viruses they are free living (meaning they exist and multiply on their own outside a host). Bacteria are microscopic single-celled organisms and found everywhere on Earth and are vital to the planet's ecosystems. Bacteria are the most abundant free-living organisms on earth the animal and human body is full of bacteria. It is estimated that the body has more bacterial cells than animal/human cells. Some bacteria are harmful while others are useful. For example, the normal bacteria of the cow's rumen and human large intestine help in digestion of cellulose and fibre respectively. Bacteria in unfavourable environments produce resistant bodies called spores. Spores can remain viable for many years in soil and cause disease outbreak as is seen in Anthrax and Black Quarter. Some bacteria produce toxins and impair body functions by destroying tissues for example, Clostridium bacteria that causes Enterotoxaemia, Tetanus and botulism. Bacteria that enter the bloodstream are rapidly removed by white blood cells. However, if there are too many bacteria to be removed easily an infection develops. An infection that is widespread throughout the bloodstream is called sepsis. Bacteria are classified into five groups according to their shapes: Spherical (cocci), rod (bacilli), spiral (spirilla), comma (vibrios) or corkscrew (spirochaetes). The bacteria occur as single cells, in pairs, chains or clusters.

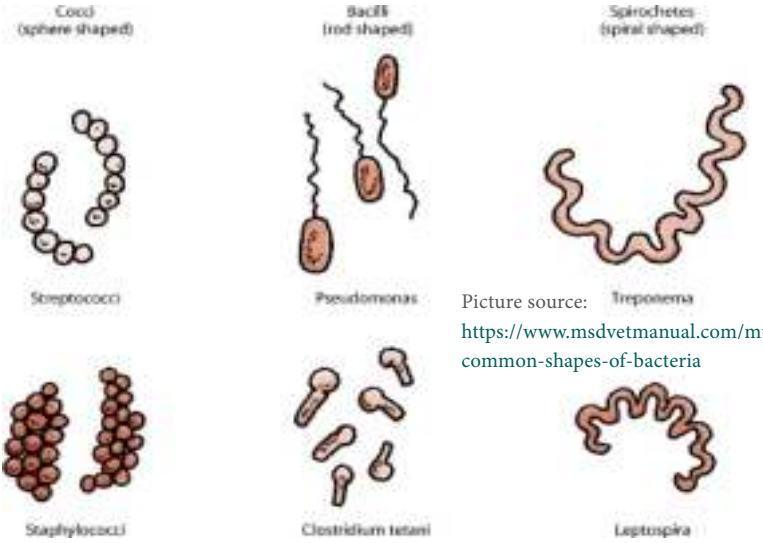


Figure 3
Common shapes of bacteria

Picture source:
https://www.msdvetmanual.com/multimedia/figure/some-common-shapes-of-bacteria

Bacteria can also be classified based on the Gram Staining properties. The two key features that lead to the differing visualization properties of Gram positive (+) and Gram negative (-) bacterial species is the thickness of the peptidoglycan layer and presence or absence of the outer lipid membrane. The bacterial wall structure affects the cell's ability to retain the crystal violet stain used in the Gram staining procedure which can then be visualized under a light microscope. Gram positive bacteria have a thick peptidoglycan layer and no outer lipid membrane while Gram negative bacteria have a thin peptidoglycan layer and have an outer lipid membrane. Gram positive bacteria have a distinctive purple appearance when observed under a light microscope. This is due to retention of the purple crystal violet stain in the thick peptidoglycan layer of the cell wall. Examples of Gram positive bacteria include Staphylococci and Streptococci species. Gram negative bacteria appear as pale reddish/pink colour when observed under a light microscope. This is because the structure of the cell wall is unable to retain the crystal violet stain and is only coloured by the safranin counterstain. Examples of Gram negative bacteria include enterococci, salmonella and pseudomonas species.

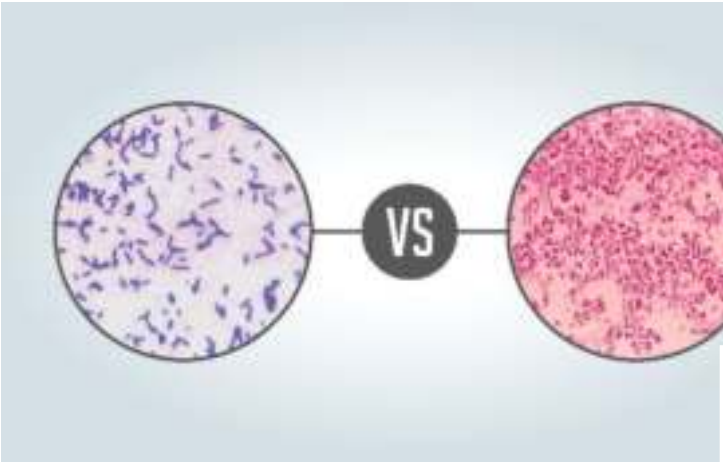


Figure 4
Gram Positive (left) verses Gram Negative (right) as observed under a light microscope

Field Based Diagnosis of Bacterial infections

Stained smears made from lesions can yield information that is inexpensive and quick to diagnose. The animal health worker only needs access to Gram stain and a light microscope. Readers should look at the reference section at the end of this chapter to access links for further reading on how to prepare and stain bacterial smears. The table below highlight some infectious diseases that can be diagnosed through stained smears.

Table 3: Infectious bacterial diseases diagnosed through Gram stained smears

Disease and Species affected	Specimen to collect	Pathogen	Appearance in Gram stained smears
Abscesses or suppurative Conditions in all livestock species Pus or exudate		Staphylococcus species	Gram + cocci, often in clumps
		Streptococcus species	Gram + cocci, usually in chains
		Trueperella pyogenes	Gram + rods, pleomorphic
		Corynebacterium pseudotuberculosis	Gram + rods
		Pseudomonas aeruginosa	Gram - rods
		Pasteurella multocida	Gram - rods
		Fusobacterium necrophorum	Gram - long, slender, filaments, often staining irregularly
Strangles- Donkeys	Nasal swab with pus	Streptococcus equi	Gram + cocci, often in chains
Dermatophilosis (Streptothricosis) Many animals, mainly in sheep, cattle and donkeys	Scabs	Dermatophilus congolensis	Gram +, filamentous and branching with coccal zoospores arranged two or more across
Clostridial enterotoxaemia Sheep and calves mainly	Scrapings from small intestine of recently dead animal (< 2 hours after death)	Clostridium perfringens	Gram +, fat rods. Large numbers suggestive of enterotoxaemia
Disease and Species affected	**Specimen to collect**	**Pathogen**	**Appearance in Giemsa stained smears**
Anthrax in cattle, sheep, goats, camels, donkeys	Blood smear from ear vein	Bacillus anthracis	Purplish, square-ended rods in short chains surrounded by a reddish-mauve capsule
Dermatophilosis (Streptothricosis) Many animals, mainly in sheep, cattle and donkeys	Scabs	Dermatophilus congolensis	Blue, filamentous and branching with coccal zoospores arranged two or more across

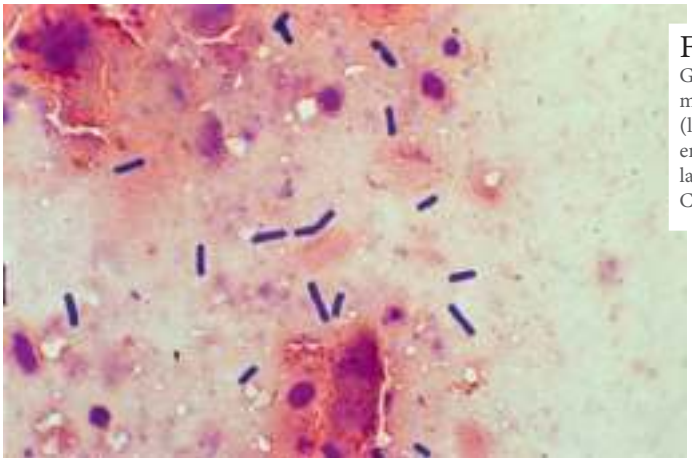


Figure 5
Gram-stained smear from the mucosa of the small intestine (lamb recently dead from enterotoxaemia):
large Gram-positive rods of Clostridium perfringens. (×1000)

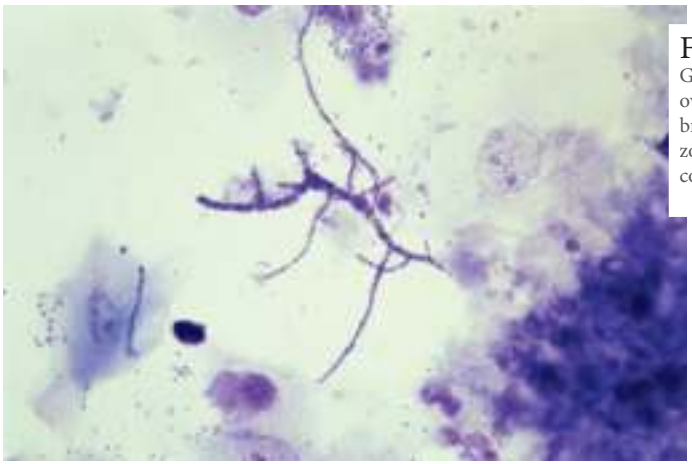


Figure 6
Giemsa-stained smear from ovine scab material showing branching filaments and zoospores of Dermatophilus congolensis. (×1000)

Picture source:
Quinn, P.J., Carter, M.E., Markey, B. and Carter, G.R. (2013). Clinical Veterinary Microbiology. Wolf/Mosby, London, 1994. (Textbook)

III. Protozoa

Protozoa are microscopic single celled organisms that are free living and found in land and water. However, pathogenic protozoa are obligate intracellular parasites and require to be in a host cell to survive. Ruminants harbour large numbers of protozoa in their fore- stomachs to help in digestion. Most protozoa are harmless. Some species of protozoa, however, are significant as they cause disease in livestock. Most pathogenic haemoprotozoan parasites are intracellular and are found in erythrocytes (red blood cells) or white blood cells.

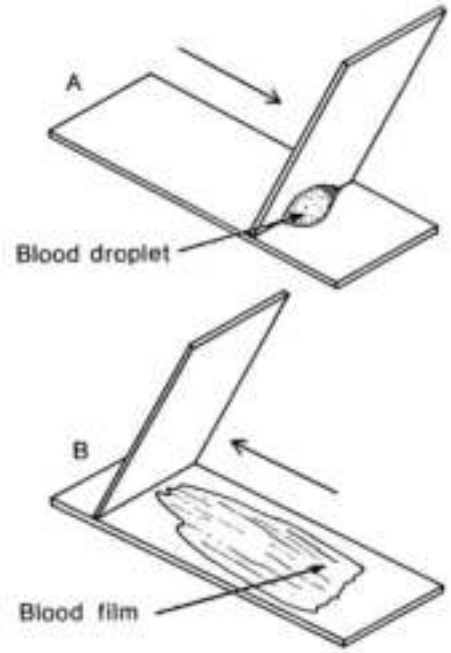
Field Based Diagnosis of Haemoprotozoan infections

A routine thin blood smear useful for assessing erythrocyte abnormalities and for detecting the presence of haemoprotozoan parasites. Parasites are most likely to be detected in blood smears during acute infection when the animal has a fever. Once infections become chronic, immunologic diagnostic techniques are more sensitive. Microscopic examination of blood smears is best achieved with Giemsa staining. However, Wright's stain can also be used, it should be noted that commercial stain kits used in many veterinary practices such as Dip Quick Stain, Jorgensen Laboratories, Loveland, CO, www.jorvet.com) will also stain haemoprotozoan parasites when used as directed, but the staining will be of poorer quality.

The following procedure can be followed when Giemsa stain is being used.

Figure 7

Thin blood smear preparation for haemoprotozoan parasite filed based diagnosis



Picture source:
Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Text book)

To prepare a thin blood smear, place a drop of blood on one end of a microscope slide and draw the blood into a thin film as shown in Figure 7 above.

Giemsa Stain procedure

1. Air-dry the blood film, protecting it from flies and other insects if it is not to be stained
2. immediately.
3. Fix in absolute methanol for 5 minutes and air-dry.
4. Dilute stock Giemsa stain 1: 20 with distilled water and flood the film (or place slide in staining jar). Fresh stain should be prepared every 2 days.
5. Stain for 30 minutes.
6. Wash stain away gently with tap water.
7. Air-dry; parasite cytoplasm will stain blue, and nuclei will stain magenta.
8. The stained blood film can be scanned using the 40× objective of the microscope and with the oil immersion lens for greater detail when suspected parasites are found.

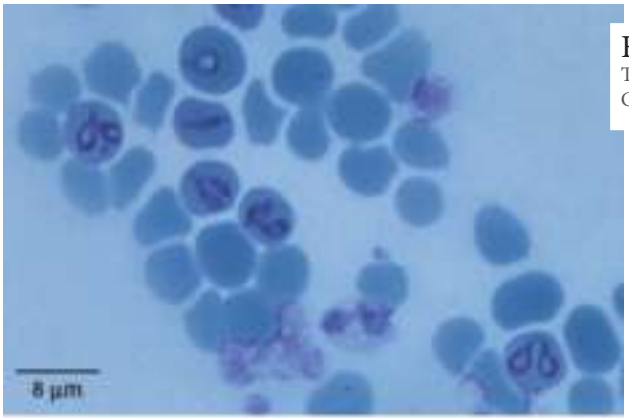
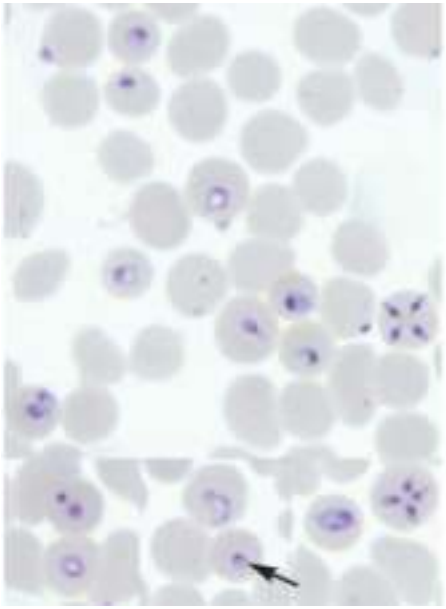
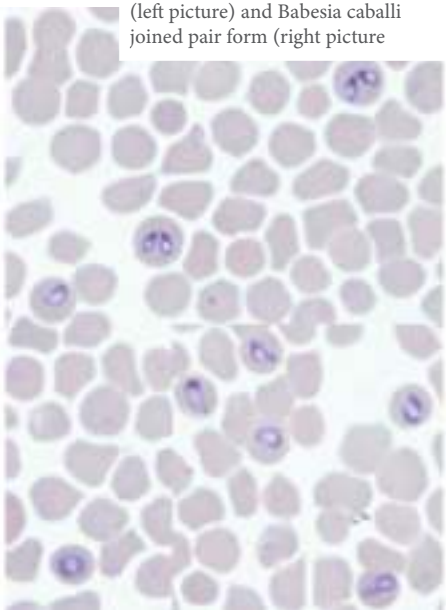


Figure 8
Tear drop shaped Babesia bigemina in a Giemsa stained bovine blood smear

Figure 9
Giemsa stained Equine blood smear showing Tetrad and small ring (Black arrow) forms of Babesia equi (left picture) and Babesia caballi joined pair form (right picture





Picture source:
Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell (Textbooks)

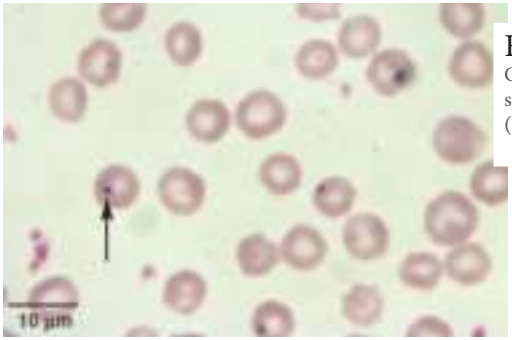


Figure 10
Giemsa stained bovine blood smear showing Theileria parva RBC forms (Piroplasms)

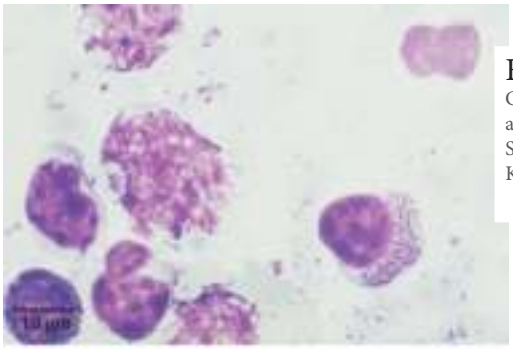


Figure 11
Giemsa stained bovine lymph node aspirate smear showing Theileria parva Schizont forms (Koch's Blue Bodies-KBBs) in the lymphocyte cytoplasm.

Figure 12
Giemsa stained bovine blood smear showing Trypanosome brucei with prominent undulating membrane (U) and sub terminally located kinetoplast (K)

Picture source:
Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)

IV. Fungi

The most common pathogenic fungi that cause disease in livestock are known as dermatophytes. Dermatophytes are a group of closely related, septate fungi that require and use keratin for growth. They are confined to the superficial integument including the outer stratum corneum of the skin, claws and hair of animals. They infection manifests as the classical circular lesion known as ringworm. There over 30 species of dermatophytes, however, those affecting animals are mainly from genera Microsporum or Trichophyton. The Infective arthrospores are found in soil or on infected animals they germinate within six hours of adhering to keratinized structures. Minor trauma of the skin and dampness facilitate infection. The dermatophytes are able to hydrolyse keratin thus causing damage to the epidermis, hair shafts, hair follicles and feathers. The nature of the lesions is affected by the virulence of the fungus and the immunological response of the host. Young, old and immunosuppressed animals are susceptible to severe infection. The inflammatory response of the infected animal is harmful to the fungus; this makes the dermatophyte move peripherally towards normal skin. The result is the commonly seen circular lesions (ringworm) of alopecia with healing at the centre and inflammation at the edge. The manifestations of dermatophyte infections can vary and may be summarized as:

- Subclinical or inapparent infections
- Classical round ringworm lesions
- Serious generalized lesions that may

be complicated by mange mites or by secondary bacterial infection, in particular by Staphylococcus aureus or S. pseudintermedius

Field based diagnosis of pathogenic fungi

Hair should be plucked from the lesion and the edge of the lesion scraped with a blunt scalpel blade until blood begins to ooze. Plucked hair, skin scrapings (including the scalpel blade) and any scab material should be submitted for microscopic examination.

Preparation of skin scrapping sample

1. Place one to two drops of 10–20% Potassium hydroxide (KOH) on a microscope slide and add a small amount of the specimen to the drop of KOH and mix well.
2. Gently pass the underside of the slide through a low flame of a Bunsen burner /cigarette lighter/match (do not boil or over-heat). This is an optional step
3. Place a cover slip on top of the preparation and press down gently and Allow to stand for one to two hours, or even overnight. The time required will depend on the density of the specimen.
4. The KOH will partially digest the proteinaceous debris.
5. Examine under phase contrast or under the low- and high-dry objectives of a light microscope. Microsporum and Trichophyton species appear as chains of refractile, round arthrospores on hairs.

V. Mites

The bodies of mites and ticks are divided into two parts: the gnathosoma, which contains the mouthparts on a false head (basis capituli), and the idiosoma, which comprises the remainder of the animal and to which the legs are attached. After hatching from the egg, mites and ticks pass through larval and nymphal stages before becoming adults. Larvae of ticks and mites have six legs; while nymphs and adults have eight legs. Parasitic mites are usually microscopic, rarely exceeding 1 mm in length. Female mites are larger than males.

Field based diagnosis of parasitic mites

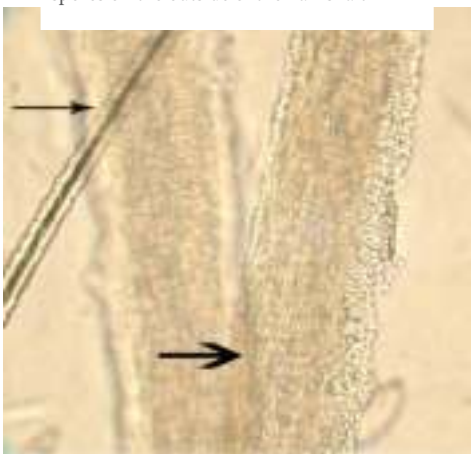
Diagnosis is through deep or superficial skin scrapings. For a deep skin scraping, a dulled, rounded scalpel blade (#10) and the area of skin to be scraped are coated with mineral oil. The site selected for scraping should be at the periphery of a lesion or the predilection site of the suspected parasite. The blade should be scraped back and forth over the skin until capillary bleeding is evident.

However, sample collection for the Demodex mite which is a follicle mite, one needs to form a skin fold by squeezing the skin between the fore

and thumb fingers to express the mites before the scraping is done. The debris collected on the scalpel blade is then placed on a microscope slide, the cover slip is then applied and the microscope slide with the sample is examined using the 10× microscope objective. Several slides may need to be examined before mites are found, especially in cases of Sarcoptic infestation. To recover surface mites, such as Cheyletiella, a superficial scraping that does not result in bleeding is made with a scalpel blade coated in mineral oil. Alternatively, a clear acetate tape can be used to collect material. The sticky side of the tape is pressed to the hair coat in an affected area and then placed on a microscope slide, trapping skin debris and mites against the slide and allowing microscopic examination. Brushings from the hair coat may also be examined. If sample is to be examined by a another laboratory for identification, they skin scrapings should be stored in 70% alcohol.

Figure 13

A 40X image of a normal hair (thin arrow) and a dermatophyte infected hair (thick arrow). The infected hair has ectothrix spores on the outside of the hair shaft



Picture source:
Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)

Figure 14

Sarcoptes mites are round bodied, the third and fourth pairs of legs are short and do not project beyond the margin of the body

Figure 15

Psoroptic mites have an oval body shape with longer legs that project beyond the body margins

Figure 16

Demodex mite with the 'cigar' body shape and short legs that do not project beyond the body margin

Picture source:
Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)

Chapter 2:

Clinical Examination Guidelines

2.1 Purpose of Clinical Examination

- Identify clinical abnormalities
- Determine risk factors that resulted in the disease
- Establish cause/aetiology of the disease
- Deduce what organ or system is involved and the location, type of lesion present,
- Understand the pathophysiologicaL processes occurring and
- Gauge the severity of the disease

2.2 Clinical examination steps

Clinical examination proceeds through a number of steps

1. Owner's complaint
2. Signalment (age, breed, sex, colour)
3. History of the animal/s affected
4. History of the herd
5. Observation of the environment
6. Observation of the sick animal/s at a distance
7. Detailed general physical examination of the animal
8. Further investigation- Laboratory

General Physical examination of animal using a body region approach (Thermometer and stethoscope are basic and essential diagnostic tools to have)

- Head and neck
- Left thorax and abdomen
- Right thorax and abdomen
- Rear/ Tail end

-Rectal examination,

- External genital -udder, vagina and Scrotum and Penis

Record after general physical examination;

- Respiratory rate and temperature
- Mucous membranes colour
- Hair/skin coat condition
- Body condition score
- Any abnormalities like lameness, lymph node enlargement, or distension of the abdomen or wounds. Note the location of abnormality (the body region affected and what side-left or right)

2.3 Techniques used to conduct a physical examination

Clinical examination proceeds through a number of steps

1. Palpation (touching)
2. Auscultation (listening)
3. Percussion (tapping)
4. Manipulation (moving)
5. Ballottement (rebound).
6. Visual inspection
7. Olfactory inspection

2.4 Normal body parameters

Resting Heart rate

Specie	Range (beats per minute)
Camel	32-50
Cattle beef	40-70
Cattle dairy	48-84
Goat	70-80
Sheep	70-80
Donkey	36-52

Resting Respiratory rate

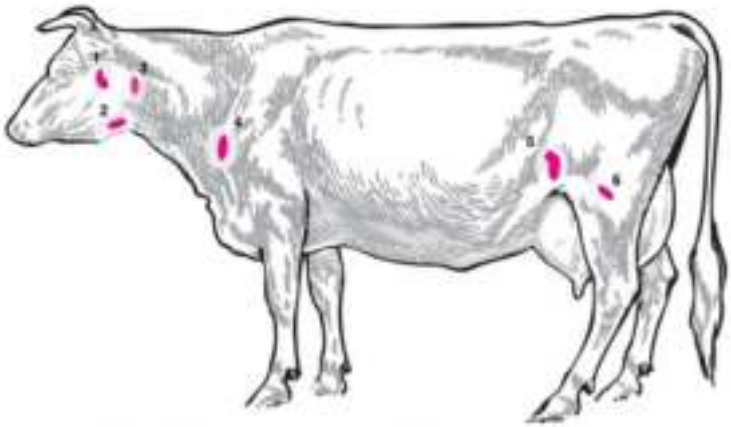
Specie	Range (breaths per minute)
Camel	5-12
Cattle beef	10-30
Cattle dairy	26-50
Goat	12-24
Sheep	16-34
Donkey	12–28

Rectal Temperature

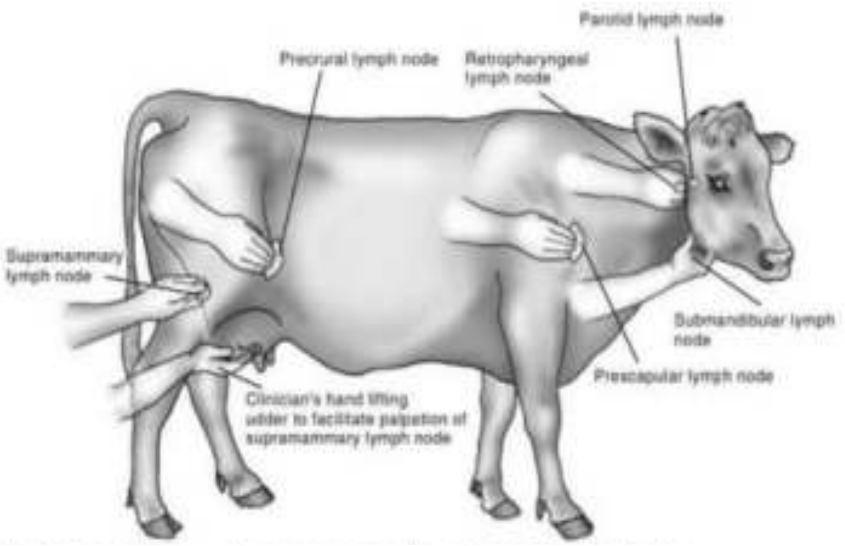
Specie	Range (°C)
Camel*	34.0- 41.0
Cattle- Beef	36.7- 39.1
Cattle-Dairy	38.0-39.3
Goat	38.5-39.7
Sheep	38.3- 39.9
Donkey	36.5 - 37.8

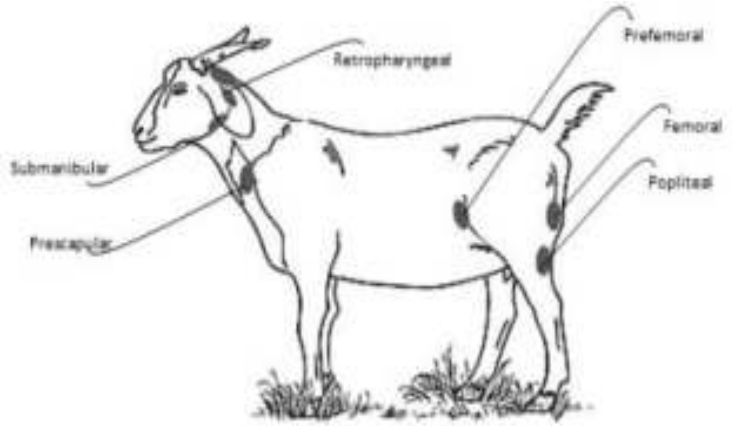
* A fully hydrated camel has a diurnal body temperature range of 36 to 38°C. However, a dehydrated camel temperature may fluctuate to 34 to 41°C

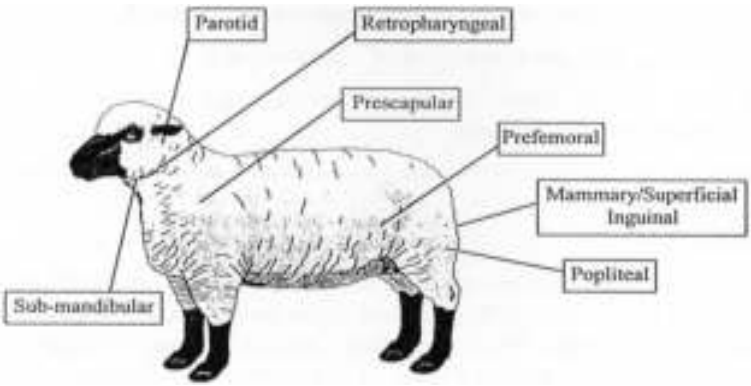
2.5 Location of palpable superficial lymphnodes of ruminant livestock

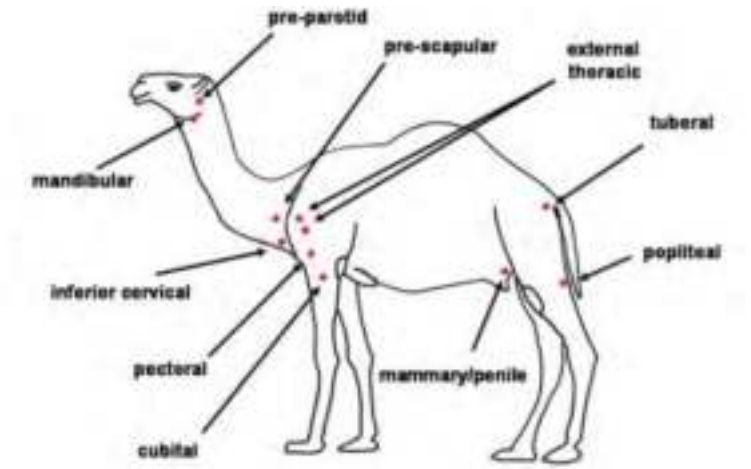


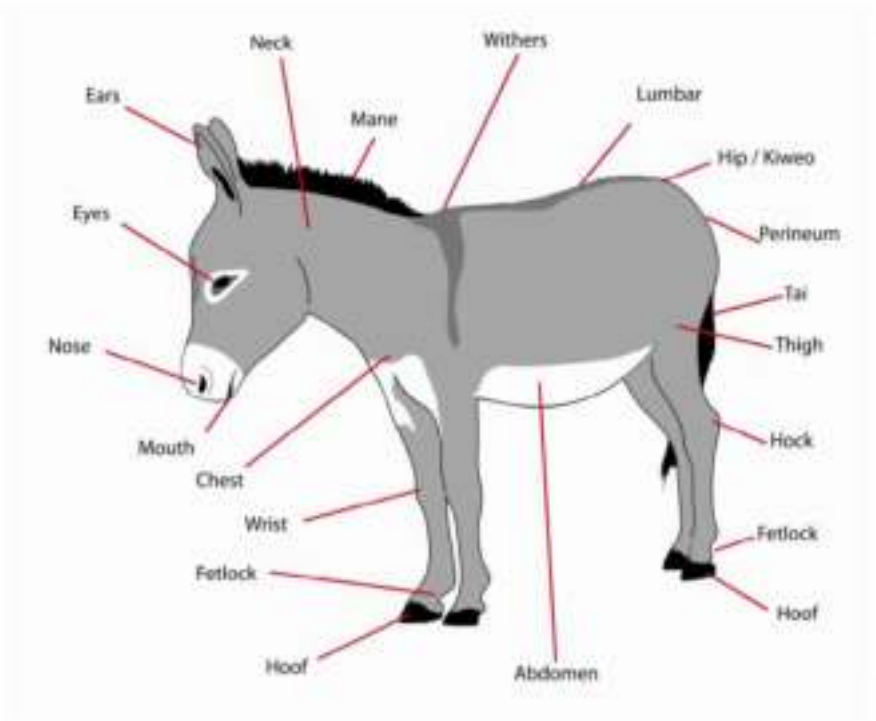
(1) Parotid
(2) Submandibular
(3) Retropharyngeal
(4) Prescapular
(5) Prefemoral
(6) Supramammary




Retropharyngeal
Prefemoral
Femoral
Popliteal
Submanibular
Prescapular


Parotid
Retropharyngeal
Prescapular
Prefemoral
Mammary/Superficial Inguinal
Popliteal
Sub-mandibular


pre-parotid
pre-scapular
external thoracic
tuberal
mandibular
popliteal
inferior cervical
pectoral
mammary/penile
cubital



Reference

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4. The clinical companion of the donkey (2021) 2nd edition Produced by The Donkey Sanctuary UK.

https://www.thedonkeysanctuary.org.uk/what-we-do/for-professionals/resources/clinical-companion

Chapter 3:

Priority Livestock Diseases in Turkana County

3.1 Type of livestock species kept in Turkana County

Table 4: Local dialect name of livestock species

Type of livestock species	Local dialect name (Singular)	Local dialect name (Plural)
Camel	EKAAL – Male AKAAL - Female	NG'IKAALA Female -NG'AKAALA
Cattle	AITE	NG'AATUK
Goats	AKINE	NG'AKINEI
Sheep	AMESEK	NG'AMESEKIN
Large Ruminants (Camel, Donkeys, Cattle)	NG'IBAREN LUAPOLOK	
Shoats / Small ruminants	NG'IBAREN LUUCIK	
Donkey	ESIKIRIA	NGISIKIRIA
Chicken	IKUKUT-Chicks EKOKOROIT- Cock AKUKUT- Hen	NGIKUKUI

3.2 Turkana Community Seasonal Calendar

The Turkana seasonal calendar has two seasons in a year, the first is the wet season followed by the dry season. Months are named according to the prevailing weather condition, environmental conditions, socio-cultural and socio-economic activities that take place during the month. The Turkana 'year' begins at the beginning of long rains; the wet and dry seasons have six months each. The Turkana seasonal calendar has been harmonised with the Gregorian as detailed below.

Month of the Year	Local dialect name	Origin of the name
January	LOMARUK	Derived from the word 'AKIMARUK', literal meaning is the formation of clouds. This indicates that rain is imminent and women begin to renovate huts and kraals.
February	LOCOTO	Derived from the word 'ECOTO', literal meaning is mud, this is the month of lots of rain, every place is muddy. Thus restricting movement of people and livestock.
March	TITIMA	Derived from the word "AKITITIMARE' that describes the process of pasture germination. During this month there is plenty of grass for livestock. Herders graze the animals close to the homestead.
April	ELE-EL	Derived from the word 'AKIELAR' meaning to spread or to blossom. This is the month when wild fruits ripen and are ready for harvest.
Month of the Year	**Local dialect name**	**Origin of the name**
May	LOSUBAN	Derived from the word "AKISUB" that means to make, do or make. This is the month of festivities and ritual celebrations.
June	LOTIAK	Derived from the word 'AKITIAK' meaning to divide or separate. This is the month that marks the end of the wet season and beginning of dry season. All festivities end, grass to dry up, livestock begin to migrate to look for better pasture.
July	LOLONG'U	Derived from the word 'ALONG'U' meaning an arid or dry land. This is the month where people and livestock experience a lot of heat, vegetation has completely dried up, and most livestock and are in constant movement in search of pasture and water.
August	LOPOO	Derived from the word 'AKIPORE' meaning to cook. During this month people draw blood from animals to drink or cook, they also gather and cook wild fruits and berries (NG'IBEYO, EDAPAL an EDUNG).

Month of the Year	Local dialect name	Origin of the name
September	LORARA	Derived from the word 'ARARAUN' or 'ARARAAR' meaning to shed, drop or fall something. This is the when trees begin to shed leaves (ABSCISSION) due to hot and dry weather. It is the month of extreme hardship where people use hooked sticks (ESEGER) to shake acacia trees to get dry leaves(NGATUR) and dry seeds (NGITIT) for their livestock.
October	LOMUK	Derived from the word 'AKIMUK' that means to cover. This is the month when the sky is covered with scattered clouds, sometimes short rains are experienced.
November	LOKWANG	Derived from the word 'EKWANG' meaning white/bright, this is the month of sun and wind.
December	LODUNG'E	Derived from the word 'ADUDUNG'IAR' meaning fall down completely. This month marks the fall of end of dry season and beginning of the wet season.

To allow for data collection on the seasonal occurrence of diseases it was agreed that the calendar year be divided into 4 seasons as shown in table 6 below. Due to the changing climate the seasons do not have calendar months assigned to them rather they focus on prevailing weather condition.

Table 6: Turkana Community Seasonal Calendar

Turkana name	AIT / NAIT	AKIPORO	AKAMU	AKICHERES*
Conventional name	Short Dry with grass	Long rains	Long Dry	Light rain (Showers)

**This season is also called ERUPE which is the grass that sprouts after light showers that occur in December. The grass sustains the livestock through the short dry season before the long rains.*

3.3 Local dialect name for presenting clinical symptoms in livestock

Table 7: Local dialect name of presenting clinical signs associated with priority livestock diseases

Conventional name	Turkana name
Sudden death	ANG'EDAR
Haemorrhages of internal organs	ARENG'IKIN ANG'IBORO AKOK
Haemorrhagic carcass	ARENG'IKIN AKIRING
General nasal discharge	NG'ISURUMA
Nasal discharge -Mucoid	NG'ISURUMA NAIBULONIT
Nasal discharge- Watery	ALELERE ANG'ISURUMA
Yellow ocular discharge	NG'AKADOMELA NANYANG'AYEK
Watery ocular discharge	ALELERE ANG'AKIYO
Green nasal discharge	NG'ISURUMA LUPUSIEK
Yellow nasal discharge	NGI'SURUMA LUNYANG'AYEK
Laboured breathing or dyspnoea	AKIYANG'A NA AKUNUWA
Fast breathing	AKIYANG'AYANG'A
Enlarged lymph nodes	ABUORE ANG'ARUREI / LONG'ARUREI / ABUORE ANGA'RUREI
Fever	ARIRARE AKWAAN/ ARIRAUN
Brisket oedema	AKIWALWAL AUKIT
Emaciation/ Wasting	AKIJIWAKIN / AKIJUIKIN
Alopecia	ACHUDAR ANG'AJUL / LOIR
Pruritus/Itchiness and scratching	AKIPIYA / AKOO
Rough hair coat	AKIWO ANG'AJUL / APUKIARIT ANG'AJUL
Elephant like skin	LOTOME
Nodular skin lesions	ENAARU
Pox like skin lesions	ETUNE
Coughing	AWALA
Foul smelling watery diarrhoea	AREMOR NAKAUROK (FOUL SMELL-ABOSIS)
Scratching on objects	AKOO / AKIRIGA/ ANG'ING'ARE
Wart like lip lesions	NGI'BWORUOK/ NG'IBUORUOK
Necrotic oral lesions	NG'AJEMEI AKITUK
Jaundice of Mucous Membranes	LONYANG'
Icterus/ Jaundiced carcass (meat)	ANYANG'ANUT AKIRING
Diarrhoea	AKIREM / AREMOR
Aggressive behaviour Biting everything	ADEDENG'U AKONYANUT
Persistent passing of gas	AWORE
Distended abdomen	ATEBUSUS
Stiff muscles	ETEREGEGE
Unsteady gait Staggering	AKIRANG'ARANG'A AKITEROTERO
Blood oozing from body orifices -Mouth -Nose -Ears -Anus Blood that does not clot	ALELERE ANG'AKOT ANAKITUK ALOKUMES NG'AKI ANABOR NG'AKOT NAPENYIDIKETE

Conventional name	Turkana name
Pale mucous membranes	AKWANG'IAR ANG'AKONYEN / AKWANG'IS ANG'AKONYEN / AKWANG'UUN ANG'AKONYEN
Lethargy	AKILOIKIN / NG'ILOE
Hard dry faeces	ANG'ONG'ONG ANG'ACIN/KORIANG
Enlarged gall bladder	ABUORE APID
Foul smell from the mouth	ABOSIS AKITUK
Mouth lesions- wound	NG'AJEMEI AKITUK
Foot lesions-wound	NG'AJEMEI AKEJU
Bottle jaw/ submandibular oedema	LOBOLIBOLIO
Adhesions of lungs to ribcage	ATAPAKINA ANG'IUKOI NAMARAN
High fever	ARIRARE AKWAAN
Shade seeking behaviour	AKISAK ETOLIM
Gelatinous subcutaneous fat	ERAKIRAK
Red urine Brown urine	NG'AKUL NAARENG'AK NG'AKUL NAANG'ORA
Foul smell - interdigital space (Foul smell)	ABOSIS ANG'ANG'ICHETA ANG'IMOLOKONY (ABOSIS)
State of a sick animal – described as a depressed or dull animal	EKITOWO
Limping Lameness	AKICHODO ANG'UOL
Change of behaviour	AKILOKONYIKIN EPITE
Hypersalivation	AJEELEL

Conventional name	Turkana name
Difficulty in swallowing	AGOGONGU AKILIKOR
Bloating	AKUUKIN
Difficulty in passing stool (Tenesmus)	AKIDING ANG'ACIN
Difficulty in passing urine	AKIKID ANG'AKUL
Lock jaw	AGOGONGU AKITUK ANG'AAR
Wound	AJEME
Reluctance to move	AWOUN AROTOKIN
Flared nostrils	APUKOR ANG'IKUMES
Convulsions	AKIRIDANAKIN
Lack of rigor mortis	AMAMAU ETEREGEGE NE ETONIA
Rapid decomposition	AKIBOS ATIPEI
Recumbency	ARABAN
BQ Swelling of muscles Crepitation (crackling sounds)	LOKICHUMA/ LOKICHUM-sharp pain ABUORE AKIRING AKALOKIDING AKIWAIWA AKIRING AKALOKIDING
Clots in milk Blood in milk	NG'ING'OLE ANAKILE NG'AAKOT ANAKILE
Water around the heart	NG'AKIPI ALOTAU
CNS sign-Circling	AKIRIM
CNS sign- Peddling	AKIPETAPETA
CNS sign- Excessive bleating CNS sign- Excessive bleating	ARUORE LOKIJON/ LOKOJOKON
Cyst in the brain/ spinal cord	NG'IKAPESPES LU ANG'ADAM
Abortion	AKIYEC

Table 8: Local dialect name of presenting clinical signs associated with priority chicken diseases

Conventional name	Turkana name
Swollen foot Scaly legs	ABUORE ANG'IMOLOKONY ANG'ARABAB ANG'AKEJEN
Greenish diarrhoea	EREMONU LOPUS
High Mortality	ALALAU AKITU ANG'IKIENY
Wattle or Comb paleness	AKWANG'IAR ANG'ITELTEL
Pruritus/Itching	AKIPIA
Tracheal rales Snoring	AKIRURUMARE EPOROTO AKING'ORARE
Head swelling	AKILILING'IAR AKOU
Pox like skin lesions	ETUNE ANG'IKUKUI
Lose of sight	AMUDUKANUT
Blood stained faeces	NG'AKOT ANACIN

Conventional name	Turkana name
gasping	ANG'AMIANAR
Ruffled feathers	APUKIARIT ANG'AKOPIR
Drooping wings	ACHAKUNA ANG'ABEBEN
Drooping head	AKIJURORE AKOU
Severe depression	EKITOWO
Paralysis	ALILIMIYOR ANG'AKEJEN
Laying of misshapen eggs/ immature eggs	AKITETERE NG'ABEYEI ANA EKUWOM
Nasal discharge	ALAKAKIN NG'ISURUMA
Presence of worms in faeces	AYAKAU ANG'IPELEI NACIN

3.4 Local dialect name for endo and ectoparasites affecting livestock

Table 9: Local dialect name of common ectoparasites affecting livestock

Type of Ectoparasite	Turkana name
Mites	ASASA
Fleas	NG'IKADESDES
Ticks	EMADANG'
Lice	ELACHIT
Tsetse fly (Glossina species)	EDIIT / ECHUT
Helminths	NG'IPELEI / NG'IRITAN

3.5 Community knowledge and perception on livestock disease control strategies

During focus group discussion with livestock keepers, the community was asked to explain what control strategies they used to prevent the priority disease in their herds. They remarked that there were no control measures used as the diseases were endemic and most did not know the cause (aetiological agent) hence, they could not prevent them. Further probing allowed the livestock keepers to realise some strategies used during disease outbreaks were actually control measures. It was noted the livestock keepers relied on strategies that are in line with the recommended animal husbandry practices to prevent disease spread during outbreaks. The community were aware of mass vaccination and treatment initiatives conducted by the government. They however, noted that the initiatives always arrived late when most animals were sick or during drought periods when animals were nutritionally compromised. They requested the county government to offer vaccination services when they animals are in good body condition and roads are passable (not muddy as seen in March after long rains). Best vaccination time is between the months of November to January (after the short rains).

Table 10: Control strategies used by livestock keepers to prevent priority livestock diseases in Turkana county

Control Strategy	Turkana name
Migration out of an area with disease outbreak or offending vector (Ticks or Tsetse flies)	AKIWOT
Migration to new area that is disease/ vector free	ARAMAKIN / AWESIT
Isolate or separate sick animals/birds	ATIAKTIAK / ACHADAKIN
Avoidance of known disease/vector hot spot areas	ACHADAKIN
Proper carcass disposal	AKINUK- (Burying) AKINOM- (Burning)
Access to salt water or salty mineral rocky/spring areas / salty water baths	AKITAMAT NG'AKIPI NA AMAKAT
Provide water regularly especially in faecal impaction in donkeys	AKITAMAT NG'AKIPI JIIK
Restrict water especially for HS in camels	AKITORON NG'IKAALA
Burning of pasture to control ticks	AKICHUNY NG'INYAA KIRATAKINERE NG'IMADANY
Burning of livestock dung/droppings	AKINOM NG'ASIKE
Good hygienic practice- like regular clearing of dung/droppings from boma/chicken house	APIAR NG'ANOKIN
Construction of new boma/chicken house	AIYATAKIN/ AKIDUK NG'IDORIN
Bath calves with camel urine that has been stored for one week to control ticks	AKILAT NG'ITAK ANG'ACHOTO

Table 10: Control strategies used by livestock keepers to prevent priority livestock diseases in Turkana county

Control Strategy	Turkana name
Topical application of pounded seeds from the tree EDUNG (Boscia coriacea) for tick control	EDUNG- crushed seeds topical application
Raising chicken house to prevent ectoparasite infestation and predation	AKIKEUN AKAI ANGIKUKUI
To keep birds healthy regularly put in water plants perceived to have medicinal value like sap/latex from Aloe and Emus species	ECHUCHUKAR (Aloe Turkanensis)
Chicken- ectoparasite control	Dust birds ash - epuru
Prayer for divine intervention to protect livestock	EMURON
Regular use of acaricides	ADIP / ADIP NA NG'IGURAI
Regular use of anthelmintics	NAOSIN / EDAWA LOA ANG'IPELEI / EDAWA LOA LOPELEI
Vaccination that needs to be timely (preventive) not when outbreaks are occurring	ECHANJO

Chapter 4:

Cattle Diseases

Table 11: Priority cattle diseases ranked in order of importance by livestock keepers in Turkana county

Conventional name	Turkana name
Contagious Bovine Pleuropneumonia (CBPP)	LOUKOI / LOUKOI ANG'ATUK
Trypanosomiasis	LOKIPI / EKALICH
Foot rot	EBAIBAI / EKICHODONU
Babesiosis	LONYANG / LORENG'EKUL
Mange	LOTOME / EKOIKOI / LOIR
Anaplasmosis	LOPID / APID / LONYANG
Lumpy Skin Disease (LSD)	LOMERI / EDEKE LOMERI
East Coast Fever (ECF)	LOKIT
Foot and Mouth Disease (FMD)	LOJAA / EDEKE AKITUK KA NG'AKEJEN
Mastitis	LOEKETA
Black Quarter (BQ)	LOKICHUMA
Anthrax (see Annex 2: Zoonotic diseases)	ENOMOKERE
Heartwater	AMIL
Helminthiasis	NG'IRITAN / NG'IPELEI
Haemorrhagic septicaemia (HS)	LOOKOT
Bovine Ephemeral Fever (BEF)	EYALA

Table 12: Medicinal plants and traditional practices used by livestock keepers to manage priority cattle diseases in Turkana County

Medicinal plant botanical name	Turkana name for medicinal plant and traditional practice	Target Cattle Disease
Aloe Turkanensis- sap collected from succulent leaves and given as an oral drench	ECHUCHUKA	CBPP, HS, Helminthiasis,
Euphorbia kalisina or uhligiana- the spines/ thorns are removed and the succulent leaves pounded and soaked in cold water and given as an oral drench	EMUS	CBPP
Azadirachta indica- Neem tree leaves are boiled or soaked in water and given as an oral drench when cool	MWARUBAINI	CBPP
Cadaba rotundifolia- leaves are pounded and soaked in cold water and given as an oral drench. Leaves can also be burnt to chase away biting flies	EPUU	CBPP LSD

Table 12: Medicinal plants and traditional practices used by livestock keepers to manage priority cattle diseases in Turkana County

Medicinal plant botanical name	Turkana name for medicinal plant and traditional practice	Target Cattle Disease
Cissus quadrangularis – succulent part of the plant is pounded and mixed with cold water and given as an oral drench	EGIS	CBPP LSD
Traditional practice	Turkana name	Target Disease
Hot iron or stone cauterization of affected body part	AKIMAD EMACHAR KORI AMORU	Anaplasmosis, CBPP LSD, BQ and HS
Jugular bleeding	AKIGUM NG'AKOT	HS, Trypanosomiasis
Take animals to salty water springs to drink the water	EDOOT	Trypanosomiasis

**Text in red highlights practices that go against animal welfare principles*

Table 13: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat priority cattle diseases in Turkana county

Type of Veterinary Medicinal Product (VMP)	Active ingredient	Turkana name	Target disease
Antibiotic	Oxytetracycline Adamycin®	Edawa lomung' Edawa Aloukoi	Anplasmosis, CBPP, HS, LSD
	Tylosin	Edawa la ng'iukoi	Anplasmosis, CBPP, HS, LSD
	Procaine Penicillin G and Dihydrostreptomycin (Penstrep®)	Edawa lokakoun lo akwaan	Anplasmosis, CBPP, HS, LSD, Anthrax
Antiprotozoal	Homidium Chloride (Novidium®)	Edawa loareng'an (Red tablet)	Trypanosomiasis, Babesiosis HS, Heart water
Antiparasitic / Anthelmintic	Ivermectin	Edawa alotome / Edawa lokirion / Edawa lomoo	Trypanosomiasis, CBPP, LSD, FMD
	Albendazole	Naosin Edawa lo ang'ipeelei	Helminthiasis
Acaricide	Amitraz Synthetic Pyrethroids (Ectopor®) Organophosphate	Adip na ng'igurai Adip	Heart water, Babesiosis and Anaplasmosis

** The drug of choice used by livestock keepers to treat the diseases in red do not have any therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance*

Cattle Diseases:

Viral Diseases

4.1 Foot and Mouth Disease (FMD) - LOJAA / EDEKE AKITUK KA NG'AKEJEN

Definition

- Highly contagious viral disease of cattle, swine, sheep, goats and other cloven-hoofed ruminants.
- Characterised by sudden lameness, hypersalivation and appearance of blister-like sores on the tongue, muzzle and between the hooves.

Distribution (Epidemiology)

- The most important viral diseases of livestock in the world.
- FMD is endemic in Africa, Middle East, Asia and in a few countries in South America.
- North America and Western European countries have eradicated the disease and are currently free of FMD without vaccination.
- Cloven-hoofed wildlife, Bactrian camels and new world camelids are susceptible to FMD.
- The African buffalo is the main reservoir host for FMD virus.
- Based on WOAH estimates, FMD's morbidity rate can be as high as 100% in susceptible populations. Mortality is low in adult animals (1–5%), but higher in young calves and lambs (20%) due to myocardium lesions.
- In Turkana county, livestock keepers in the year 2022 estimated that FMD has an apparent annual morbidity rate of 2%, mortality rate of 1% and case fatality rate of 50%.

Causative agent/ Aetiology

- Foot-and-mouth disease virus (FMDV) is a small, enveloped and single-stranded RNA virus. RNA viruses are prone to mutation errors during replication.
- FMDV belongs to the family Picornaviridae and genus Aphthovirus. The family includes human rhinovirus that causes common cold and poliovirus that causes polio.
- FMDV occurs as seven major distinct serotypes: A, O, C, Southern African Territories (SAT) 1, SAT 2, SAT 3, and Asia 1.
- Each serotype has multiple subtypes with varying antigenicity and degrees of virulence, especially within the A and O types.
- There is no cross-immunity between serotypes-this means that immunity to one type does not confer protection against the others.
- FMDV can persist in contaminated fodder and the environment for up to 1 month, depending on the temperature and pH conditions.
- The virus survives in milk and milk products during regular pasteurisation, but is inactivated by ultra-high-temperature (UHT) pasteurisation.
- Quickly inactivated by conditions that have a pH of more than 6.0.

Transmission

Source of infection

- Virus is shed by incubating animals before clinical signs begin.
- Aerosol droplets, saliva, faeces, urine, milk and semen have high infective dose of virus.
- The virus is also found in meat and meat by-products that have a pH of below 6.0.
- Carrier, recovered or vaccinated animals can harbour the virus in the oropharynx for more than 28 days. Carrier state in cattle varies from 15–50%.

Modes of Transmission

- Direct contact between infected and susceptible animals through Inhalation of infectious aerosol droplets- FMDV is small and airborne, especially in temperate zones (virus can be carries by wind up to 60 kms overland and 300 kms by sea)
- Indirect contact with contaminated inanimate objects fomites.
- Ingestion of contaminated milk (by calves)
- Artificial insemination with contaminated semen
- Humans can harbour FMDV in their respiratory tract for 24–48 hours and can transmit it to cattle.

Pathophysiology

- Primary site of replication of FMD virus is the mucosa of the pharynx. The virus then enters the lymphatic system and is spread throughout the body where it replicates in the epithelium of the mouth, muzzle, teats and feet.
- The replication in the epithelium causes vesicles to form that after rupture after 48 hours.
- Once the vesicles rupture the healing process begins.
- Carrier animals can harbour the virus for n more than 4 years in cattle, 9 months in sheep, and over 5 years in the African buffalo.
- The African buffalo is the main maintenance host for FMD SAT serotypes.

Affected age group

- All age groups and all breeds of cattle are susceptible.

Clinical signs

- Incubation period is variable and depends on the host, environment, route of exposure, and virus strain. The average incubation period for sheep and goats is 3–8 days and 2–14 days for cattle.
- Animals develop a high fever of 40° to 41° C.
- Animals make a smacking sound with their mouth and exhibit hypersalivation. The saliva hangs in form of rope-like strings.
- Animals careful chew the feed and or often seen grinding their teeth due to stomatitis.
- Mouth lesions appear as vesicles that are 1 to 2 cm in diameter. The vesicles are found on the buccal mucosa, dental pad and tongue.
- Vesicles are thin walled and easily rupture after 24 hours releasing fluid.
- The rupture leaves a raw painful surface that heals in 1 week.
- Vesicular feet lesions also appear in the interdigital space, heel of the claws and on the coronet band. The vesicle rupture causing severe lameness and recumbency.
- Secondary bacterial infection interferes with healing especially of foot lesions.
- Mastitis due to vesicles on the teats may also occur.
- Calves under 3 months may suffer heavy mortality due to myocardial damage. Death in calves may occur even when there are no vesicular lesions in the mouth or feet.
- Recovery is often seen after 8 to 15 days.

FMD Complications

- The animal can develop serious bacterial infection of lesions especially if animal is kept in a muddy dung filled environment.
- Animals may develop hoof deformation.
- FMD caused mastitis can cause permanent impairment of milk production, abortion and permanent loss of weight.
- An unusual symptom in cattle is linked to endocrine damage and is characterised by a chronic syndrome of dyspnoea, anaemia, overgrowth of hair, and lack of heat tolerance. Affected cattle are described as “hairy panters.”

Post-mortem lesions

- Necrosis of heart muscle (tiger heart), usually only in young acutely infected animals.
- Ulcerative lesions on tongue, palate, gums, pillars of the rumen and feet.

Season of occurrence

- FMD outbreaks occur in any season due to introduction of sick animals, presence of naïve

(not immune) animals. In pastoralist settings shared pasture and water are a key driver of FMD outbreaks.
- In Turkana county, FMD is endemic, however outbreaks mainly occur during the long dry season AKAMU.

Diagnosis

Field based diagnosis
- FMD Point of Care Rapid Diagnosis test kit that is available in Kenya.
- Clinical signs can be used to make a field based presumptive diagnosis. Animal health workers should age lesions as this is an important aspect of outbreak investigation that determines what control intervention measures will be adopted.

Laboratory diagnosis
- Laboratory confirmation is essential for diagnosis of FMD and should be performed in specialized laboratories that meet WOAH requirements for Containing Group 4 pathogens.
- In Kenya, all samples are sent to the WOAH FMD reference laboratory located in Embakasi, Nairobi county.
- Samples to be taken should include- vesicular epithelium or fluid. At least 1 g of epithelium should be placed in a phosphate-buffered saline (PBS) transport medium or glycerol.
- Samples should be refrigerated or transported on ice.
- If vesicles are not present, oropharyngeal fluid can be collected via probang cup or pharyngeal swab.
- Clotted and unclotted blood should be collected with red topped and purple topped EDTA tubes respectively.
- Laboratory tests to be conducted include – RT- PCR for antigen serotype or ELISA for antibody level analysis. ELISA antibody serum tests are not useful in areas with vaccinated animals that are experiencing new FMD outbreaks.

Differential Diagnosis (DDx)
- Clinically indistinguishable from vesicular stomatitis

Other differential diagnosis:
- Bovine viral diarrhoea and Mucosal disease
- Infectious bovine rhinotracheitis
- Bluetongue
- Bovine mammillitis
- Malignant catarrhal fever

Treatment
- Viral diseases have no treatment.
- Supportive treatment with broad spectrum antibiotics to manage secondary bacterial infections - observe drug label instructions for dosage and delivery route.

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

** mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)*

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

Prevention/control

- FMDV is quickly inactivated by pH ranges of 6.0 and 9.0. Disinfectant products that achieve this pH range include; sodium hydroxide (2%), sodium carbonate (4%), citric acid (0.2%), acetic acid (2%) and sodium hypochlorite (3%). These products should be used to disinfect contaminated areas;
- Humans exposed to herds with FMD can harbour the virus in their respiratory tract for 24–48 hours and should be prevented from coming into contact with livestock for 1 week.
- Isolate sick animals and impose movement quarantine in affected areas
- Use vaccines after receiving laboratory results of the circulating serotype. KEVEVAPI has two types of vaccines the Purified oil based FOTIVAX® vaccine that requires one annual vaccination. The second vaccine called FOTIVAX™ requires 2 or 4 annual boosters to be effective.
- Livestock keepers should be advised not to purchase new animals during FMD outbreak (they should wait 6 months after the outbreak).

Zoonotic potential

- Yes
- FMD is not readily transmissible to humans and is not a public health risk. However, humans are thought to be slightly susceptible and may develop vesicles in the mouth or hands.

Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Ethnoveterinary treatment practices

- Refer to tables 12 and 13

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where FMD outbreaks are occurring
- Present animals for vaccination
- Use of Amulet (sacrifice a squirrel cut it into half and put each half at either entrance of the cattle kraal)
- Request for religious blessing to safeguard the herd from disease. The blessing ritual is done by specific people who have being given the divine ability to protect livestock and property from ills like diseases.

Further Reading

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Figure 17
Profuse salivation with saliva hanging in long ropy strings

Figure 18
FMD point of care Rapid Test Kit

Picture source:
https://agritech.tnau.ac.in/expert-system/cattlebuffalo/Foot%20and%20Mouth%20Disease.html

Figure 19
A pictorial guide to estimate the age of FMD lesions

Day 1 –Intact fluid filled vesicles with overlying skin becoming blanched.
Day 2 - Vesicle rupture but the blanched epithelium is still intact with sharp edges (arrowed), and where detached, raw red underlying dermis can be seen.
Days 3-4 - Vesicular epithelium is lost, with subsequent fibrin deposition evident on the exposed dermis. Epithelium starts to re-grow at lesion borders.
Day 5-10 - Epithelial regrowth is marked, with loss of fibrin infilling, and subsequent scarification
Day 7 onwards - Fibrin infilling has disappeared, with new epithelium covering the dermis. Scar formation progresses.

Picture source:
http://www.fao.org/fileadmin/user-upload/eufmd/docs/training/EnglLesionageing.pdf

4.2 Lumpy Skin Disease (LSD) - LOMERI / EDEKE LOMERI

Definition
- Lumpy skin disease (LSD) is a viral vector borne disease of cattle and buffaloes that is characterised by sudden appearance of nodular skin eruptions.
- The skin nodules are also known as pox lesions.

Distribution (Epidemiology)
- LSD is endemic in Africa since 1929.
- In 2012, LSD spread to the Middle East and South Eastern countries of Europe. Vaccination has stopped further spread in Europe.
- In 2019, LSD spread to Asian countries of Bangladesh, India, China, Chinese Taipei, Vietnam, Bhutan, Hong Kong, Nepal, Sri Lanka, Myanmar and Thailand.
- Disease outbreaks are sporadic and depend on animal movements, animal immune status, wind and rainfall patterns that affect vector populations.
- Based on WOAH estimates, LSD has a varied morbidity rate of 10 to 20% and a mortality rate of 1 to 5%.
- In Turkana county, livestock keepers in the year 2022 estimated the disease to have an apparent annual morbidity rate of 3% with a mortality rate of 1% and case fatality rate of 33%.

Causative agent/ Aetiology
- Lumpy skin disease virus (LSDV) is from the family Poxviridae and genus Capri poxvirus (CaPV).
- It is a DNA virus and is genetically similar to goat and sheep pox virus. LSDV is stable and is resistant to inactivation. LSDV can survive for long periods at ambient temperatures and can remain viable for up to 35 days in dried scabs.

Transmission
- Mechanical transmission – most common mode - blood feeding vectors like biting flies, mosquitoes and ticks transmit the virus to susceptible animals. The vector species involved in transmission include; Fly species like Stomoxys, Glossina, Muscidae and Tabanidae. Mosquitoes species like Aedes and Tick species like Rhipicephalus and Amblyomma.
- Sexual transmission- the virus is found in semen. Natural mating or artificial insemination is a source of infection for adult female animals.
- Vertical transmission - pregnant cows can transmit the virus to the foetus and deliver calves with characteristic nodular skin lesions.
- Direct transmission - virus is also found in milk or teat skin lesions and can be directly transmitted to suckling calves.
- Animals are immune for only 3 months after natural infection.
- There is no carrier state and all animals clear the virus form their body after infection.

Affected age group
- All age groups are affected.
- Exotic breeds (Bos Taurus), calves and lactating cows are more susceptible to severe infection.

Clinical signs

- LSD has an incubation period of 2 to 4 weeks.
- Animal has sudden eruption of nodules of various sizes on the skin and mucosa.
- Nodules range from a few to several hundred and are spread all over the body.
- Nodules affect the full skin thickness and involve the epidermis, dermis, subcutaneous tissue and sometimes the underlying muscle.
- Animal develops a persistent High fever of over 40.5°C for 1 week.
- The superficial lymph nodes especially the pre-scapular and pre-crural become enlarged.
- Animal have serous nasal discharge and hypersalivation due to the inflammatory nodules developing in the nasal buccal (mouth) mucosa.
- Skin nodules either resolve rapidly, or indurate and persist as hard lumps ('sitfasts').

Post-mortem lesions

- At necropsy, the carcass has nodular/pox lesions throughout the entire digestive and respiratory tracts and on the surface of almost all internal organ.

Season of occurrence

- LSD is endemic in Turkana county and cases are reported throughout the year
- However, epidemic outbreaks are reported during the long rainy season AKIPORO.

Diagnosis

Field based diagnosis

- There is no point of care rapid diagnosis test kit developed.
- Characteristic clinical signs are used to make a field based presumptive diagnosis.

Laboratory diagnosis

- Samples to be collected include; skin lesions and scabs, saliva, nasal swabs and clotted and unclotted blood.
- Polymerase Chain Reaction (PCR) is the main confirmatory test used to detect LSDV antigen
- ELISA is used to detect antibodies but it does not differentiate antibodies due to other Capri pox virus (sheep and goat pox virus).

Differential Diagnosis (DDx)

Disease conditions that have superficially located nodules but have less severe signs:

- Pseudo lumpy skin disease/Bovine herpes mammillitis;

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20-30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

** mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)*

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

- Insect bites/stings, urticaria, and photosensitisation;
- Pseudo cowpox (Para poxvirus) (lesions occur only on the teats and udder);
- Dermatophytosis (fungus infection);
- Demodicosis (Mite infection)- lesions predominantly over withers, neck, back, and flanks, with alopecia present.
- Bovine papular stomatitis (Para poxvirus) lesions occur only in the mucous membranes of the mouth.
- Dermatophilosis (bacterial infection) lesions associated with prolonged wetting. Skin lesions are raised with matted tufts of hair and spread over the head, dorsal surfaces of the neck and body, and upper lateral surfaces of the neck and chest.

Treatment

- Viral diseases have no treatment.
- Supportive treatment with broad spectrum antibiotics to manage secondary bacterial infections - observe drug label instructions for dosage and delivery route.

Prevention and Control

- Movement restrictions during outbreaks.
- Annual vaccination before outbreak season. KEVEVAPI produces a vaccine called LUMPIVAX™ this is a freeze-dried, live attenuated vaccine that is available in Kenya.
- Vaccine confers immunity for 3 years but annual vaccination is encouraged due to young immune naïve animals in the herd.
- Conduct regular (quarterly) active surveillance using PDS techniques.

Zoonotic potential

- None, does not affect man

Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Difficult to control due to high population of arthropod vectors, some use pour-on acaricides to control biting flies.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where LSD outbreaks are occurring
- Present animals for vaccination

Figure 20
Generalised LSD skin nodules of various sizes with enlargement of retropharyngeal lymph node (black arrow)

Picture source:
Diba Denge, Saku Animal Health Assistant, Marsabit County

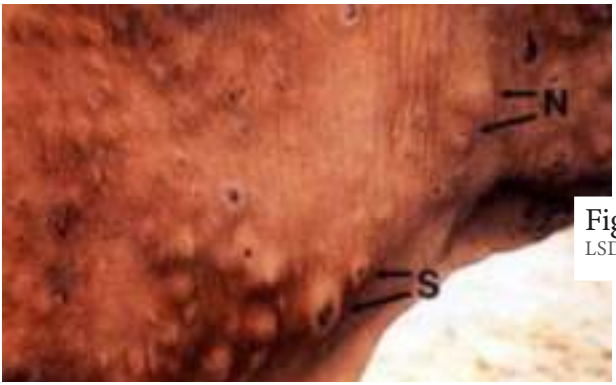


Figure 21
LSD nodules (N) and sit fast (S) lesions

Picture source:
Tuppurainen, and Beltrán-Alcrudo 2017

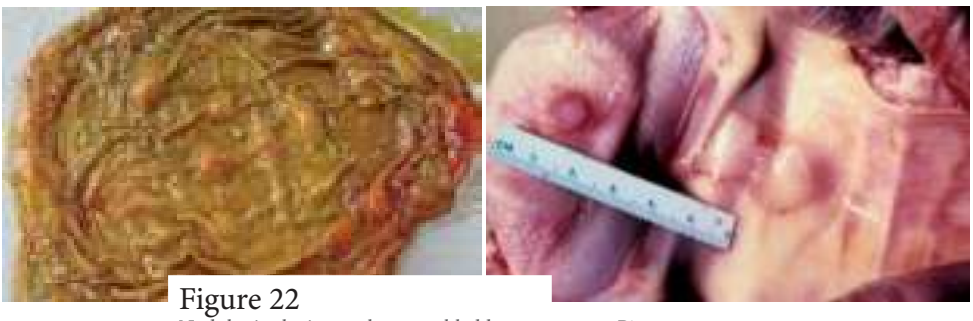


Figure 22
Nodules in the internal organs, bladder (Left) and epiglottis and trachea (Right)

Picture source:
Tuppurainen, and Beltrán-Alcrudo 2017

Further Reading

1. Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. WOAH (World Organisation for Animal Health) ISBN 978-92-9044-804-4. https://rr-asia.WOAH.int/wp-content/uploads/2020/04/WOAH-atlas-of-transboundary-animal-disease_ahs-chapter.pdf
2. Kiplagat SK, Kitala PM, Onono JO, Beard PM and Lyons NA (2020) Risk Factors for Outbreaks of Lumpy Skin Disease and the Economic Impact in Cattle Farms of Nakuru County, Kenya. Front. Vet. Sci. 7:259. https://doi.org/10.3389/fvets.2020.00259
3. WOAH (2017). Technical Disease Card: Lumpy Skin Disease Terrestrial Manual, Chapter 2.4.14. https://test.WOAH.int/fileadmin/Home/eng/Health_standards/tahm/2.04.13_LSD.pdf
4. Tuppurainen, E., Alexandrov, T. & Beltrán-Alcrudo, D.(2017). Lumpy skin disease field manual –A manual for veterinarians. FAO Animal Production and Health Manual No. 20. Rome. Food and Agriculture Organization of the United Nations (FAO). 60 pages.
http://www.fao.org/documents/card/en/c/1fcf63b0-80e9-4f8e-825f-10ea6e998479/

4.3 Bovine Ephemeral Fever (BEF) - EYALA

Other name: Three-day sickness/ fever/stiffness, bovine epizootic fever or dengue of cattle

Definition

- BEF is an arthropod-borne viral disease of cattle and water buffalo
- BEF is a non-contagious disease that manifests as an acute febrile condition that is usually mild in young calves. Adults manifest more severe clinical signs that include; Sudden drop in milk production, bi-phasic fever, hypersalivation, lameness, muscle stiffness and recumbency. The disease rarely causes death.
- Dairy cows can manifest a more severe disease characterised by general depression, muscle tremors, hind limb paralysis that cause prolonged sternal recumbency and death if supportive care is not instituted early. In dairy cows, BEF is also associated with reduced fertility and abortions.

Distribution (Epidemiology)

- BEF geographical range includes Africa, Middle East, Australia, and Asia. Inapparent infections (antibodies in serum without overt clinical signs) has been demonstrated in goats, sheep, Cape buffalo, hartebeest, waterbuck, wildebeest, deer and gazelles.
- Seasonal outbreaks, particularly following periods of high rainfall, are indicative of a vector-borne transmission. However, research has failed to identify the range of vectors with most studies implicating biting midges (Culicoides species).
- Based on WOAH, BEF in infected countries occurs throughout the year either as an enzootic or sporadic forms. During epizootic outbreaks morbidity rates are between 50 and 100%) but mortality is low at 1 to 3%.

Causative agent/ Aetiology

- Bovine ephemeral fever virus (BEFV) is a member of the genus Ephemerovirus in the family Rhabdoviridae. It is a single-stranded RNA virus.
- BEVF was mainly considered to exist as a single serotype but recent research have demonstrated at least 5 different antigenic strains.
- The virus is readily inactivated at pH levels below 5 and above 10 and is therefore inactivated by the meat maturation process in which the pH falls below 5. The virus is also sensitive to disinfectant chemicals and to lipid solvents.

Transmission

- BEFV can be transmitted from infected to susceptible cattle by IV inoculation; as little as 0.005 mL of blood collected during the febrile stage is infective.
- To date, infection by virus obtained from virus culture has not been successful. The virus has been recovered from several Culicoides species and from Anopheles and Culex mosquito species collected in the field. The identity of the whole range of vectors involved in transmission has not been proven.
- Transmission by contact or fomites does not occur.
- The virus does cause a carrier state and recovered cattle clear the virus within a week of infection.
- Infection results in long term immunity.

Pathophysiology

- On entry into the body, the virus causes an acute inflammatory reaction that results in neutrophils being released into circulation within 24 hours.

- The virus also infects the endothelial cells resulting in hypocalcaemia.

Affected age group
- Disease of adult cattle and water buffalo. All breeds of cattle are susceptible.

Clinical signs
- Signs occur suddenly and vary in severity and include sudden drop in milk production, biphasic to poly phasic fever (40°– 42°C). Shivering, in appetence, lacrimation, serous nasal discharge, drooling saliva, pulmonary emphysema (auscultated as harsh lung sounds), increased heart rate and dyspnoea.
- Cessation of rumen movements.
- Clinical signs are mild in water buffalo.
- Affected cattle may become recumbent and paralyzed for 8 hours to more than 1 week.
- Bulls, heavy cattle, and high-lactating dairy cows are the most severely affected, but spontaneous recovery usually occurs within a few days. Hidden losses may result from decreased muscle mass and lowered fertility in bulls.

Post-mortem lesions

Bovine ephemeral fever is an inflammatory disease. The most common lesions include:
- Sero fibrinous poly serositis, of varying degrees, in the articular synovial membranes and in the thoracic and peritoneal cavities.
- Serous surfaces may also show signs of bleeding and oedema to varying degrees. The oedema fluid in the thoracic or abdominal cavity contains fibrin.
- In the joints, this peri articular inflammatory fluid is yellow or brown and gelatinous in appearance a Giemsa stain of the fluid reveals extravascular neutrophils.
- Other lesions include pulmonary and lymph node oedema, inflammation of the parietal and visceral pleura, pericarditis (especially at the base of the heart), necrosis at certain points of the skeletal muscles, and, sometimes, emphysematous lesions of the lungs.

Season of occurrence
- BEP occurs as an epizootic and is associated with high rainfall seasons that favour the arthropod vector.

Diagnosis

Field based diagnosis
- To date there is no rapid diagnostic test developed. Presumptive diagnosis is based on epidemiological factors of proliferation of arthropod vectors and clinical signs of several animals in an area experiencing sudden episode of fever accompanied by arthritis, stiffness of the limbs and lameness followed by rapid recovery.
- A Giemsa stained blood smear can be used as a field based diagnosis test- observing many immature neutrophils (band cells) in a blood smear can be used as a field based presumptive diagnostic test.

Laboratory diagnosis
- Samples to be taken should include whole blood from sick and apparently healthy cattle in affected herds in heparin anticoagulant (not EDTA).
- A blood smear can be tested using Immunofluorescence that identifies the virus or its antigens using immune serum that marks the virus-specific antibodies with fluorochrome. Confirmation can also be made by detecting the viral genome using RT-PCR or a real-time loop mediated isothermal amplification (RT-LAMP) assay.

Differential Diagnosis (DDx)
- BEF should be differentiated from conditions that cause fever, such as Rift Valley fever and bluetongue as they are similar due to link to with arthropod vectors (arboviruses), and both occur during the rainy season.
- BEF should also be differentiated with diseases that cause nervous and respiratory symptoms like ehrlichiosis of ruminants, intoxication by poisonous plants, hypocalcaemia and acute pneumonia that causes pulmonary oedema

Treatment

- Viral diseases have no treatment.
- Treatment includes administration of Non-steroidal anti-inflammatory drugs (NSAIDs) so as to pain, reduce inflammation, and bring down the high temperature. Anti-inflammatory drugs should be given early and in repeated doses for 2–3 days so as to be effective.
- Oral dosing of water or drugs should be avoided unless the swallowing reflex is functional.
- Signs of hypocalcaemia should be treated with calcium borogluconate are treated as for milk fever. Antibiotic treatment to control secondary infection and rehydration with isotonic fluids may be warranted.
- Use of antibiotics is discouraged, unless warranted. Use of tetracycline based antibiotics should not be used as it will worsen the hypocalcaemia.
- Use of NSAIDs should be accompanied by supportive care to recumbent cows.

Active ingredient	Indication	Dosage* and Route**
Calcium borogluconate	Hypocalcaemia	Slow IV or SC Cattle & Buffalo: 250 - 300 ml per day as a single dose. Monitor heart function if giving IV.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

** mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)*

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

Prevention/control

- Commercially available attenuated or inactivated BEFV vaccines are used in Australia but their effectiveness to induce long term immunity similar to natural infection is highly variable.
- Kenya does not have a BEFV vaccine
- The efficacy of vector control remains uncertain, because the insect vectors have not been fully identified.

Zoonotic potential - None.
Notifiable disease - No

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Burn dung at the cattle boma to reduce the number of arthropod vectors

Further Reading

1. Akakpo A.J. (2015). Three-day fever. Rev. Sci. Tech. Off. Int. Epiz., 2015, 34 (2), 533-538. http://boutique.WOAH.int/extrait/19akakpo533538ang.pdf
2. MSD Veterinary Manual https://www.msdvetmanual.com/
3. Stokes, J.E., Darpel, K.E., Gubbins, S. et al. Investigation of bovine ephemeral fever virus transmission by putative dipteran vectors under experimental conditions. Parasites Vectors 13, 597 (2020). https://doi.org/10.1186/s13071-020-04485-5

Figure 23
A dairy cows in sternal recumbency (left) and cow in lateral recumbency with respiratory distress (dyspnoea)-Right Picture

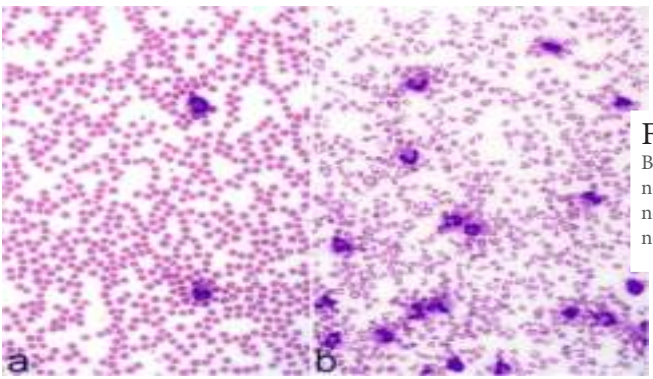


Figure 24
Bovine blood smear (a) shows normal neutrophils (b) shows neutrophilia with some immature neutrophils (band cells)

Picture source:
Albehwar, A.M., Wafaa, M., El-Neshwy, Hiam, M., Fakhry and Hemmat, S. El- Emam (2018). Molecular Characterization of Recent Isolates of BEF Virus in Egypt. Global Journal of Medical Research: Veterinary Science and Veterinary Medicine Volume 18 Issue 2 Version 1.0.
https://globaljournals.org/GJMR/Volume18/E-Journal/GJMR/(G)/Vol/18/Issue/2.pdf

4.4 Heart Water (HW) - AMIL

Definition

- Heartwater is an infectious, non-contagious, tick borne rickettsial disease of ruminants.
- HW only occurs in areas infested by ticks of the genus Amblyomma.

Distribution (Epidemiology)

- Heartwater occurs in nearly all the sub-Saharan countries of Africa where Amblyomma ticks are present and in the surrounding islands: Madagascar, Reunion, Mauritius, Zanzibar, the Comoros Islands and Sao Tomé.
- Morbidity rate is highly variable and depends on the degree of tick infestation, previous exposure to infected ticks, and level of acaricide protection. can be as high as in susceptible populations.
- Once signs of the disease have developed, prognosis is poor for exotic breeds of cattle the mortality rate varies from 60 to 80%.
- Recovery from heart water infection usually results in complete immunity against homologous strains, although animals remain carriers of infection.
- In Marsabit county, livestock keepers in the year 2022, estimated an apparent annual morbidity and mortality rate of 4% and case fatality rate of 100%.

Causative agent/ Aetiology

- Heartwater is caused by Ehrlichia ruminantium (formerly Cowdria ruminantium), it is a s a small Gram negative, pleomorphic coccus.
- Ehrlichia ruminantium belongs to the order Rickettsiales and the family Anaplasmataceae.
- Ehrlichia ruminantium is an obligate intracellular bacterium that does not survive outside the host for more than a few hours. It is heat labile and loses its viability and infectivity in less than 4 hours at room temperature.
- HW can only be transmitted through a tick vector, therefore, parameters associated
- With resistance to physical and chemical actions (i.e. temperature, chemical/ disinfectants, and environmental survival) are not applicable.

Transmission and Pathophysiology

- E. ruminantium is transmitted by the bite of the Amblyomma variegatum (tropical bont tick) which is a three host tick that requires each stage (larvae, nymph, adult) to feed on a different animal.
- Tick infects the host via bacterial organisms that are found in the tick saliva or regurgitated gut contents.
- Once in the host, the organisms replicate first within the regional lymph nodes with subsequent dissemination via the bloodstream to invade the endothelial cells of blood vessels. In domestic ruminants, there is a predilection for the bacteria to infect endothelial cells of the brain.
- The tick gets infected when it feeds on infected or carrier animals and then transmits the

disease to other susceptible animals
- Wild animals play a role as reservoir hosts.
- HW can be transmitted vertically and through colostrum of carrier dams

Affected age group
- All ages of domestic and wild ruminants are susceptible

Clinical signs
- Heart water occurs in four clinical forms that are determined by strain virulence, infective dose and host susceptibility.
- Clinical signs are dramatic in the peracute and acute forms. In peracute cases, animals may drop dead within a few hours of developing a fever (41°C or more), sometimes without any apparent clinical signs others display an exaggerated respiratory distress and/or paroxysmal convulsions.
- Cattle that develop the acute disease cases have persistent high fever (41°C) that remains high for 4 to 5 weeks and drops shortly before death. Other signs include anorexia and depression along with congested mucous membranes. Respiratory distress slowly develops along with nervous signs such as a hyperaesthesia, high-stepping stiff gait, exaggerated blinking, and chewing movements. Terminally, prostration with bouts of opisthotonus; "pedalling," "thrashing," or stiffening of the limbs and convulsions before death.
- Diarrhoea is seen mainly in cattle.
- In subacute cases, signs are less marked and CNS involvement is absent or present.
- Recovery from heart water infection usually results in complete immunity against homologous strains.
- Animals remain carriers for long periods.

Post-mortem lesions
- Heart water derives its name from the hydropericardium prominent lesions observed.
- Other common lesions are hydrothorax, pulmonary oedema, intestinal congestion, oedema of the mediastinal and bronchial lymph nodes, petechiae on the epicardium and endocardium. Congestion of the brain and moderate splenomegaly.

Season of occurrence
- Heart water can occur all year round in areas infested by the tick.
- Epizootic outbreaks can occur if naïve animals are brought into a tick infested area.
- In Turkana county, livestock keepers in the year 2022 indicated that heart water was endemic and cases occur throughout the year. However, more cases may be seen during AKIPORO season in cattle and AKAMU season in small ruminants.

Diagnosis
- To data there is no rapid diagnosis test kit developed.
- Clinical signs can be used to make a field based presumptive diagnosis.
- Samples to be collected include clotted blood and in dead animal's the brains tissue from well vascularised section of the brain
- Polymerase chain reaction (PCR) tests can identify E.ruminantium in tissues or in the blood of live animals.
- Heartwater can also be diagnosed by observing E. ruminantium colonies in Giemsa stained brain squash smears from the brain or the intima of large blood vessels.
- The best samples to collect from the brain are well-vascularized portions such as the cerebrum, cerebellum or hippocampus. E. ruminantium occurs as clumps of reddish-purple to blue, coccoid to pleomorphic organisms inside capillary endothelial cells. These organisms are often found close to the nucleus of the endothelia cell and may be in a ring or horseshoe shape.
- Demonstration of colonies of organisms in the cytoplasm of capillary endothelial cells is necessary for a definitive diagnosis.

Differential Diagnosis (DDx)
- The peracute form of heart water can be confused with anthrax.

- Acute form may resemble rabies, tetanus, bacterial meningitis or encephalitis due to babesiosis, anaplasmosis, cerebral trypanosomiasis, or theileriosis
- HW must also be differentiated from plant poisoning

Treatment

- Antibiotic treatments should start early in the course of the disease for them to be effective.
- Oxytetracycline is the first choice of antibiotics as it effects good cure rates. Given that most cases in pastoralists set up are identified late. The first oxytetracycline treatment should be given slowly IV. A minimum of three daily doses should be given regardless of temperature. If fever persists, oxytetracycline treatment should be continued for a fourth and fifth day.
- If the fever still does not decrease, after 5 days of Oxytetracycline use. Switch to a bactericidal antibiotic like potentiated sulphonamide that has been shown to be successful in clearing the fever.
- Ensure owners adheres to the withdrawal times for milk and meat after treatment with antibiotics.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting Most effective for acute cases	Broad spectrum Bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV first dose then deep IM for subsequent doses. Give once a day for 3 days. If fever persists, continue for 4 to 5 days.
Sulfamethoxazole Trimethoprim	Broad Spectrum Bacteriocidal antibiotic	15 mg/kg/day/IM twice daily for 3 days

** mL- Millilitre, body weight-BWT, kg- kilograms, Intramuscular (IM)*

Prevention/control

- Ticks are the main reservoir of E. ruminantium, and infection can persist in them for 15 months. Amblyomma ticks are three-host ticks whose life cycles take 5 months to 4 years to complete. In general, adult ticks prefer to feed on large ruminants (cattle) and nymphs on small ruminants (sheep and goats).
- Control of tick infestation is a useful preventive measure in some instances but may be difficult and expensive to maintain in pastoralists setting. In addition, excessive reduction of tick numbers, interferes with the maintenance of adequate immunity through regular field challenge in endemic areas and may periodically result in heavy losses.
- It is recommended that in endemic areas a preventive dosing with long acting oxytetracycline injections be done if new and immune naïve animals are introduced into an endemic area. This will protect susceptible animals from contracting heart water while allowing them time to develop a natural immunity.
- Regular acaricide use through spraying or dipping and hand-dressing followed by inspection to ensure the absence of ticks is recommended for animals in transit to heart water free areas.

Vaccination Challenge

- The infection and Treatment method" (IM) for immunization is in use in southern Africa, where infected sheep blood containing fully virulent organisms of the Ball 3 stock is used for infection, followed by monitoring of rectal temperature and antibiotic therapy after a

fever develops. In certain circumstances, the "controlled" infection is followed by preventive "block treatment" without temperature recording for cattle on day 14 [susceptible B taurus breeds] or day 16 [for the more resistant B indicus breeds], sheep and Angora goats on day 11, and Boer and crossbreed goats on day 12).

- The IM vaccine method has several drawbacks such as cold chain constraints and intravenous inoculation with daily follow-up of temperature.
- Young calves (<4 weeks old), lambs, and kids (<1 week old) have an innate age-related resistance to heartwater, so if challenged by natural or induced infections within this time period, most recover spontaneously and develop a reasonable immunity.
- The IM vaccine does not protect animals from all field strains, and revaccination is risky due to the possibility of anaphylactic reactions.
- Research is ongoing to develop a better vaccine using attenuated or inactivated organisms. Field trials have revealed that antigenic diversity is important in formulating effective vaccines, and further investigations are critical for the delivery of any vaccine in the field.

Zoonotic potential

- None

Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- As it is tick borne and ticks are endemic in the area, livestock keepers if the resources are available ensure once a month spraying with acaricides.
- Some livestock keepers also hand pick the bont ticks from animals.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/
2. World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header

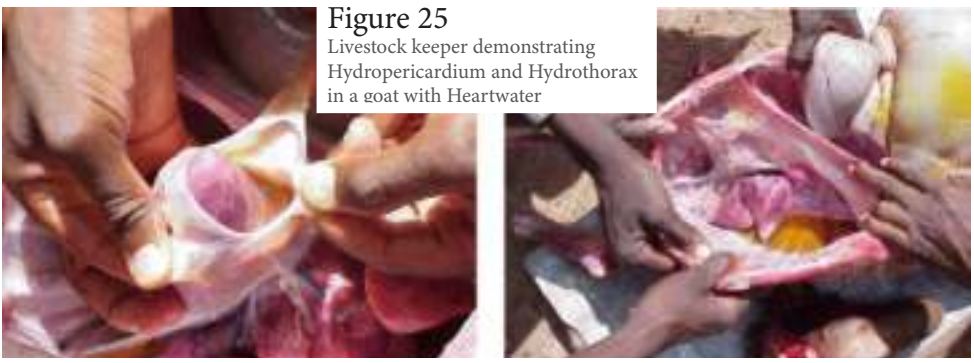
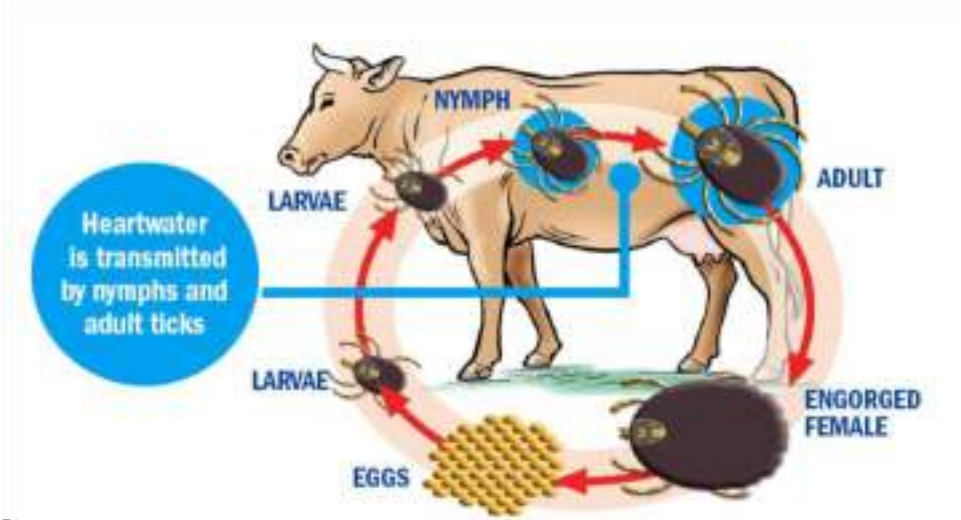


Figure 25
Livestock keeper demonstrating Hydropericardium and Hydrothorax in a goat with Heartwater

NB: Gloves should always be worn during a post-mortem and if not available ensure thorough hand washing after conducting the post mortem.

Figure 26
Role of Amblyomma species tick in Heart water transmission cycle



Picture source:
https://za.virbac.com/home/every-health-care/pagecontent/every-advices/heartwater-disease-in-livestock.html

Figure 27
Hydrothorax in cattle due to heartwater infection

Picture source:
https://repository.up.ac.za/handle/2263/6957

Figure 28
Opisthotonus and "pedalling" of the limbs due to heart water infections in cattle

Picture source:
https://www.msdvetmanual.com/generalized-conditions/heartwater/overview-of-heartwater

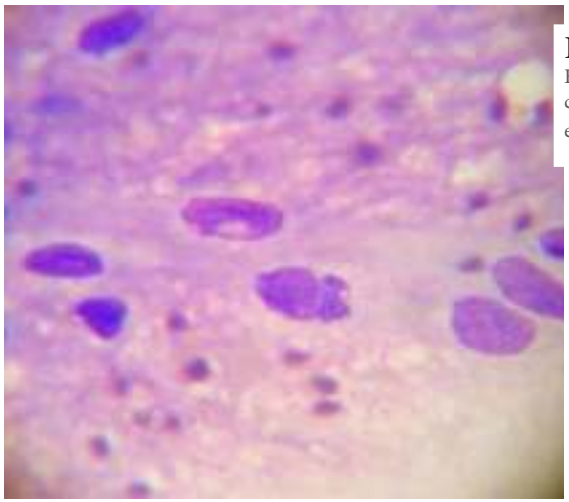


Figure 29
Ehrlichia ruminantium parasite colonies (Purplish staining) near the endothelial cell nucleus

4.5 Bovine Mastitis – LOEKETA

Case definition
- Mastitis is an inflammation of one or more quarters of the udder.
- Occurs most commonly in dairy cows, in beef cows' mastitis is common after calving when bacteria enter the teat canal if cows calve in dirty areas or lie in mud or manure.
- Traumatic injury and heavy tick infestation of the udder can also result in mastitis in beef cows.

Epidemiology
- Mastitis affects both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus), but dairy breeds are more prone to mastitis.
- Mastitis incidences are more common during the rainy season due to the muddy environment.

Causative agent/ Aetiology
- Most infections are caused by various species of bacteria, the main bacterial species include; Gram positive- Streptococci, Staphylococci, and Gram-negative rods, especially lactose-fermenting organisms of enteric origin, commonly termed as coliforms.
- Except for Mycoplasma species, which may spread from cow to cow through aerosol transmission and invade the udder subsequent to bacteraemia. All the other bacteria require direct or indirect contact with udder of the cow.

Mode of transmission and Pathophysiology
Transmission and Pathophysiology
- Dairy cows are more prone to mastitis because of the larger mammary gland tissue and the frequent handling of their udders.
- Mastitis is an immune response to bacterial invasion of the teat canal by variety of bacterial species.
- Mastitis is a multifactorial disease, closely related to the production system and environment that cows are kept in. Mastitis risk factors or disease determinants can be classified into three groups: host, pathogen and environmental determinants.
- Most mastitis cases are subclinical and the cows generally get better on their own, but might have a drop in milk production from that quarter due to scar tissue.

Affected group
- All breeds affected however dairy breeds and older beef breeds are more susceptible.

This is due to their higher milk production and loosening of the suspensory udder ligaments that makes the udder more pendulous hence prone to mastitis.

Clinical signs
- Main symptom is udder swelling, the affected quarter or udder is hot to the touch, there may be hardness, redness or pain.
- Beef cows refuse the calf to suckle.
- Milk from affected quarter or udder has a watery appearance or has flakes, clots, blood or pus. The milk may also have a foul odour.
- There may also be reduced milk yield and increase in body temperature if infection is severe.

Post mortem lesions
- There are few gross lesions in animals that die of mastitis, main lesions are linked to septicaemia such as petechial or ecchymotic haemorrhages in the lymph nodes, parenchymatous organs (liver, heart and kidneys), mucous and serous membranes.
- The most common change is a slight mottling of the cut surface of the gland or abscessation.

Diagnosis
- Milk sample for culture and antibiotic sensitivity profile should be collected where possible.

Aseptic Milk Sampling Procedure

1. Clean the udder of all visible dirt
2. Wash hands
3. Clean the teat end with 3 clean swabs dipped in 70% alcohol disinfectant
4. Open the milk collection tube keeping the top held near the mouth of the tube
5. Collect the milk sample keeping the tube in horizontal position.
6. Close the top immediately
7. Label the tube with cow number, quarter and date of collection.
8. Submit to laboratory ensure sample is kept in a cool place away from direct light.

NB: Each quarter should have separate milk samples taken.

Mastitis diagnostic tests and their test principles are shown in the table below.

Type of test	Test principle	Preferred sample
California Mastitis Test (CMT)	Detergent lysis white blood cell (leucocytes) in milk resulting in viscosity of the fluid sample. The level of viscosity is a measure of mastitis severity.	Fresh milk
Somatic Cell Count (SCC)	Count of leucocytes in a milk sample either under a microscope or using an automated system (flow cytometry).	Fresh milk
Enzyme Linked immunosorbent assay (ELISA)	Detects antibody levels in milk	Fresh or Frozen milk
Bacterial culture	Milk sample streaked on culture plates and growth of colonies counted and species of bacteria identified through biochemical tests.	Fresh milk
Multiplex Polymerase Chain Reaction (PCR)	Amplification and detection of nucleic acid of pathogens. Many pathogens can be identified and they don't need to be viable.	Fresh or Frozen milk

Differential Diagnosis (DDx)

- Differentiate between infectious or traumatic mastitis

Treatment

- If more than 2 quarters are affected opt for systemic antibiotic preparations
- Most commonly used antibiotics include amoxicillin, penicillin, or cephalosporins. Antibiotic of choice should be informed by culture and sensitivity test.
- Non-steroidal anti-inflammatory drugs (NSAID) like flunixin meglumine and phenyl butazone are widely used for the treatment of acute mastitis
- Where possible milk the affected quarter frequently to remove the mastitic milk

Prevention/control

- Ensure clean calving environment
- Washing of hands between cows when milking
- Post milking teat dip or application of milking salve
- Introduce dry cow therapy for cows that had mastitis in the lactation cycle

Zoonotic potential

- Yes, consumption of milk with bacteria can cause food poisoning.
- In addition, bacteria that cause systemic disease and fever are excreted in milk for example Brucella abortus, Coxiella burnetii and Mycoplasma bovis are important zoonotic diseases transmitted through milk.

Notifiable disease

- No

Further Reading

- Christine M. Mbindyo, George C. Gitao and Charles M. Mulei (2020). Veterinary Medicine International Volume 2020, Article ID 8831172, 12 pages. https://doi.org/10.1155/2020/8831172
- FAO. 2014. Impact of mastitis in small scale dairy production systems. Animal Production and Health Working Paper. No. 13. Rome http://www.fao.org/3/i3377e/i3377e.pdf
- MSD Veterinary Manual https://www.msdvetmanual.com/

Figure 30
Sequential milk samples collected from 6 to 36 hours after E coli infection in a quarter of a cow.

Peak bacterial numbers in milk occurred at 12 hours after infection (second vial from the left) when milk still appeared normal and the cow was asymptomatic. Photo Courtesy of Dr. Ronald Erskine.
https://www.msdvetmanual.com/reproductive-system/mastitis-in-large-animals/mastitis-in-cattle

Figure 31
Stripping the quarter to remove mastitic milk before infusion with the Intramammary tube

4.6 Black Quarter (BQ)- LOKICHUMA

Other names- Clostridial myositis; Black leg; Quarter evil

Case definition

- Blackleg is an acute, febrile, highly fatal, disease of cattle and sheep caused by Clostridium chauvoei and characterized by emphysematous swelling and necrotizing myositis that commonly affects large muscles.

Distribution (Epidemiology)

- Global distribution

Causative agent/ Aetiology

- C. chauvoei is found naturally in the intestinal tract of animals.
- It is a Gram-positive, anaerobic bacterium which produces endospores and toxins in favourable conditions.

Mode of transmission and Pathophysiology

- Cattle and sheep are susceptible as they are grazers and come into contact with C. chauvoei spores that remain viable in the soil for years. Spores are the main source of infection.
- Outbreaks of blackleg has occurred in farms which had recent excavation activities or had flooded.
- The organisms once ingested passes through the wall of the GI tract, and, after gaining access to the bloodstream, are deposited in muscle and other tissues (spleen, liver, and GI tract) and may remain dormant or cause disease.
- In cattle, blackleg infection is endogenous. This means that lesions develop without any history of wounds, although bruising or excessive exercise/trekking may precipitate disease in some cases.

Affected group

- Animals that contract blackleg are mainly beef breeds that are in excellent body condition.
- Cases are sporadic and the most affected age groups are cattle 6–24 months old.
- However, there are report of calves in good body condition as young as 6 weeks and cattle as old as 10–12 years being affected.

Clinical signs

- Sudden death with affected limb sticking out of the carcass.
- Sudden onset of acute, severe lameness, more commonly affecting the hind legs,
- There is a fever, but by the time clinical signs are obvious, body temperature may be normal or subnormal.
- Affected large muscle group are oedematous and swollen and when palpated make a crepitant sound. The most common muscles involved are hip, shoulder, chest, back and neck.
- At first, the swelling is hot, and painful. As the disease rapidly progresses, the swelling enlarges, there is crepitation on palpation, and the skin over the affected muscle group becomes cold and insensitive due to muscle necrosis as there is decreased blood supply to the affected muscle.

Post mortem lesions
- Red–brown water leaks from the swellings that has a rancid smell.
- The affected muscle when is cut open, looks like a black sponge.

Morbidity and Mortality Rates
- Based on WOAH reports, BQ has an estimated morbidity and mortality of local indigenous breeds of 17.9 % and 3.6 % respectively.

Seasonality of occurrence
- BQ occurs in sporadic outbreaks affecting 1 or 2 animals in a herd. The disease can occur in any season as it is associated with events that disrupt the soil covering like excavations, drought and floods.

Diagnosis
- Characteristic lesions of emphysematous swelling of the musculature.
- Standard culture techniques-anaerobic culture and biochemical identification of muscle tissue samples that are collected as soon as possible after death.
- Fluorescent antibody test for C chauvoei is rapid and reliable.
- PCR assay is available and has been reported to be reliable for clinical samples.

Differential Diagnosis (DDx)
- Anthrax
- Trauma
- Lightning struck
- Bloat

Treatment
- The rapid fatal progression prevents treatment success in clinically sick animals.
- In an outbreak, all susceptible cattle should be vaccinated and treated prophylactically with administration of penicillin to prevent new cases.
- Cattle should be moved from affected pastures.
- Bury or burn the carcass to prevent the disease from spreading to other animals.

Prevention/control
- Calves over 2 months old should be vaccinated twice, 4 weeks apart, followed by annual boosters before the anticipated outbreak period.
- Blanthrax™ vaccine available at KEVEVAPI is used to control black quarter and anthrax diseases in Kenya.
- In South Africa, there is a popular management practice that has seen livestock keepers vaccinate beef cattle breeds every year until the animals are 3 years' old as they believe a lifelong immunity sets in adult animals.

Zoonotic potential
- None, but due to toxaemia the carcass meat is susceptible to rapid decomposition and can be a source of food poisoning to humans if consumed.

Notifiable disease
- No

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid grazing animals in past outbreak areas
- Migrate out of an area if there are reports of a BQ case
- Present animals for vaccination

Further Reading

1. Birhanu Ayele, Worku Tigre, and Benti Deressa (2016). Epidemiology and financial loss estimation of blackleg on smallholder cattle herders in Kembata Tambaro zone, Southern Ethiopia. SpringerPlus 5:1822. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5074928/#__ffn_sectitle
2. MSD Veterinary Manual https://www.msdvetmanual.com/

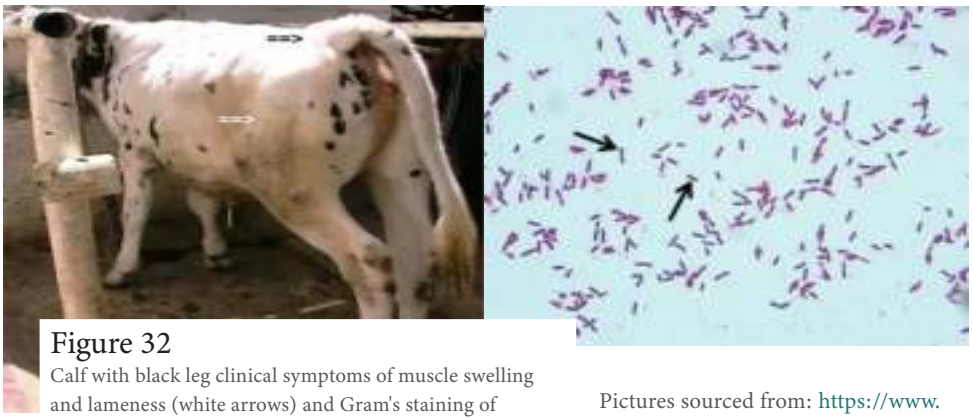


Figure 32

Calf with black leg clinical symptoms of muscle swelling and lameness (white arrows) and Gram's staining of Clostridium chauvoei showing Gram positive, short, thick, straight, round ended rods (black arrows)

Pictures sourced from: https://www.banglajol.info/index.php/BJVM/article/view/28824

4.7 Haemorrhagic septicaemia (HS)- LOOKOT

Case definition

- Haemorrhagic septicaemia is an acute disease of cattle and water buffalo characterised by sudden onset, fever, anorexia, depression, oedematous swelling of the throat, brisket and upper dewlap regions, dyspnoea, nasal discharge and salivation.
- WOAH defines HS as a highly fatal disease of cattle and buffaloes caused by specific serotypes of Pasteurella multocida designated B:2 (Asian serotype) and E:2 (African serotype). The WOAH Terrestrial code revised HS description in January 2021, they revised HS serotypes to 6: B and 6: E. However, literature is still using B:2 and E:2 naming.
- The disease has an incubation period of 90 days and has active and latent carriers.
- Pasteurella multocida is a Gram negative bacteria.
- Animals can develop a Peracute form characterised by asymptomatic pneumonia, endo toxaemia, severe dyspnoea and sudden death that has high mortality in the herd.
- Antimicrobial treatment very early in disease can reduce mortality.
- Definitive diagnosis requires the isolation of P multocida serotypes B:2 or E:2.

Distribution (Epidemiology)

- HS is a major disease of cattle and buffaloes characterised by an acute, highly fatal septicaemia with high morbidity and mortality
- HS is endemic in Asia particularly India, Africa and Middle East. Sporadic outbreaks are reported in Southern Europe.
- HS outbreaks in India occur during the monsoon season due to high humidity and high temperatures. However outside Asia incidences of disease occur any month of the year.
- Natural HS occurs rarely in pigs, sheep, and goats and has been reported in camels, elephants, rhinoceros, horses, donkeys, yaks, and various species of deer and other wild ruminants.
- It is estimated that 5% of healthy cattle are colonized by small numbers of P multocida serotype B:2 or E:2 that are shed during periods of stress.
- Common stressors associated with outbreaks include high ambient temperature, humidity, concurrent infection with blood parasites or foot and mouth disease, poor nutrition, or work stress.
- Outbreaks can occur at any time but disease is most prevalent during the rainy season. Increased outbreaks associated with high rainfall are most likely due to the multiple stressors present during this time and the moist conditions, which prolong the survival time of the organism in the environment.
- Based on WOAH reports, the disease in endemic areas affects older calves and young adults with variable morbidity and mortality rates. In non-endemic areas, epizootics results in high morbidity and mortality that can reach 100%.
- In Turkana county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 4%, mortality rate of 2% and case fatality rate of 50%.
- Recovery stimulates acquired immunity to

homologous and often heterologous strains of P multocida. Recovered animals become healthy carriers that can be a source of infection for future outbreaks.

Causative agent/ Aetiology

- HS is caused by Pasteurella multocida serotypes B:2 and E:2. The letter denotes the capsular antigen and the number stands for the somatic antigen.
- P. multocida is, a Gram negative coccobacillus residing mostly as a commensal in the nasopharynx of animals. Pasteurella multocida has five types of capsular serotype namely; A, B, D, E and F.
- The bacteria are susceptible to mild heat (55°C) and most disinfectants.
- P. multocida does not remain viable for long periods in the environment.
- However, it can survive for hours or days in damp soil or water. Rainy conditions and high humidity facilitate transmission. Biting arthropods are not significant vectors.

Transmission and Pathophysiology

- Infection occurs through direct contact with infected oral or nasal secretions from either healthy carrier animals or animals with clinical disease.
- Indirect contact occurs through ingestion of contaminated feed or water.
- Infection begins in the tonsil and adjacent nasopharyngeal tissues that subsequently, lead to a bacteraemia that leads to dissemination and rapid growth of bacteria in various body organs,
- The host cytokine response and release of killed bacteria lipopolysaccharides results in a rapidly progressing endo toxaemia.

Affected age group

- All age groups and all breeds of cattle are susceptible. However older calves and juvenile adults are most affected. Buffaloes are three times more susceptible when compared to cattle.

Clinical signs

- Clinical signs appear 1 to 3 days after infection with death occurring within 8–24 hours after the first clinical signs develop.
- Peracute cases result in death within 8 to 24 hours after animals shows signs of fever, hypersalivation, nasal discharge, and laboured respiration. However, clinical signs are rarely observed before death in the peracute form.
- Acute disease lasts for 3 to 5 days and is characterized by fever of 40°–41.1°C, dyspnoea, reluctance to move, hypersalivation, lacrimation, nasal discharge that begins as serous and progresses to mucopurulent. Acute infections develop subcutaneous swelling in the pharyngeal region that extends to the ventral neck, brisket and sometimes the forelimbs. Terminally, the animals develop severe respiratory distress, cyanosis, terminal recumbency and sometimes abdominal pain with diarrhoea.

Post-mortem lesions

- The carcass has widespread haemorrhages, oedema, and hyperaemia that is consistent with severe sepsis. Oedema fluid consists of coagulated sero-fibrinous mass with straw-coloured or blood stained fluid.
- In most cases, the carcass has swellings of the of the head, neck, and brisket.
- Petechial haemorrhages occur throughout the body.
- Thoracic and abdominal cavities contain blood-tinged fluid.
- The lungs tissue is congested and oedematous, sometimes with interstitial pneumonia.
- Gastroenteritis may also be seen.

Season of occurrence

- HS cases are reported throughout the year but outbreaks are associated with humid and heavy rainfall season.
- In Turkana county, livestock keepers in the year 2022 indicated that HS was endemic in the county with serious outbreaks common during the long rainy season AKIPORO.

Diagnosis

Field based diagnosis

- To date there is no field based Point of Care Rapid Diagnosis test kit developed.
- Clinical signs are used to make a field based presumptive diagnosis in endemic areas.
- There are no microscopic features that are specific for haemorrhagic septicaemia – all lesions are consistent with severe endotoxic shock and massive capillary damage.

Laboratory diagnosis is based on culture and blood tests

- A definitive diagnosis of HS is based on isolation of P multocida serotype B:2 or E:2 (or other less common serotypes recognized by the WOAH as causing HS) from the blood and tissues of a patient with typical clinical signs. Various other P multocida serotypes can cause HS-like disease in cattle and water buffalo, which must be differentiated from classical HS.
- Pasteurella multocida is not always found in blood samples before the terminal stage of the disease, and is not consistently present in nasal secretions or body fluids of sick animals or recently dead animals.
- Samples include heparinised blood sample or swabs collected from the heart within a few hours of death. Nasal swabs or swabs of other visceral organs can also be taken.
- If a complete necropsy examination is not feasible, blood samples can be taken from the jugular vein by aspiration or incision.
- Blood samples should be placed in a standard transport medium and transported on ice packs
- In animals that have been dead for more than 12 hours, spleen and bone marrow samples provide excellent samples for the laboratory, as these are contaminated relatively late in the post-mortem process by other bacteria.
- In animals dead for a long time, a long bone (femur/humerus) should be taken from its carcass
- In live animals, Nasal swabs and clotted and unclotted blood samples are the main samples collected.
- Definitive diagnosis of HS depends on the isolation of the causative organism, P. multocida, from the blood or bone marrow of a dead animal by cultural and biological methods, and the identification of the organism by biochemical, serological and molecular methods.
- Blood smears from affected animals can be stained with Gram, Leishman's or methylene blue stains. The organisms appear as Gram-negative, bipolar-staining short bacilli. However, no conclusive diagnosis can be made on direct microscopic examinations of blood smears alone.

Differential Diagnosis (DDx)

- Shipping fever is often mistakenly confused for HS, but has a multifactorial aetiology that also involves Mannheimia haemolytica. Shipping fever does not cause septicaemia, and does not also cause multisystem petechial haemorrhages.
- The peracute nature of the disease and the extensive oedema and haemorrhage make it difficult to differentiate HS from blackleg and anthrax.
- Other differential diagnosis includes acute salmonellosis, mycoplasmosis, lightning strike, snake bites, poisoning and pneumonic Pasteurellosis.

Treatment

- Antimicrobials are effective against HS if administered very early in the disease. However, because HS progresses rapidly, treatment is often unsuccessful.
- During outbreaks, any patient with a fever should be treated with intravenous antimicrobials (10% Oxytetracycline and as soon as possible to quickly obtain systemic bactericidal antimicrobial concentrations.
- Various antibiotics have been used to treat HS effectively and they include; potentiated sulphonamides, tetracyclines, penicillin, gentamicin, ceftiofur and enrofloxacin. However, plasmid- and chromosomal-mediated multidrug resistance seems to be increasing for some strains of P multocida, and resistance to tetracyclines and penicillin has been reported for serotype B:2 (Asian serotype).
- Antimicrobial susceptibility testing (AST) is necessary for P. multocida.

The table below highlights dosage of antibiotics commonly used to treat HS.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline Short acting	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT given IV daily for 3 to 5 days.
Penicillin G and Dihydrostreptomycin (Penstrep)	Penicillin G has bactericidal action against mainly Gram-positive bacteria and has narrow spectrum	1 ml per 10 kg body weight every 48 hours three times or until fever subsides. Given IM
The synergistic action makes the combination have a broader spectrum of activity	Dihydrostreptomycin has narrow spectrum of bactericidal action against Gram-negative bacteria	Shake bottle well before use and do not administer more than 20 ml in cattle and 5 ml in calves per injection site.
Trimethoprim and sulfamethoxazole (potentiated sulphonamide)	Antibiotic with bactericidal broad spectrum activity against Gram-positive and Gram-negative bacteria.	Twice daily 1 ml per 10 - 20 kg body weight for 3 - 5 days. Given IM
10% Gentamycin	Bactericidal against mainly Gram-negative bacteria. Has a narrow spectrum of activity	Given IM, Twice daily at a dosage rate of 1 ml per 20 - 40 kg body weight for 3 days.
Enrofloxacin 10%	Bactericidal against mainly Gram-negative bacteria. Has a narrow spectrum of activity	Given IM, Twice daily at a dosage rate of 1 ml per 20 - 40 kg body weight for 3-5 days.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

** mL- Milliliter, body weight-BWT, kg- kilograms, Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC) NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage*

Prevention/control

- Commercial vaccines include, killed vaccines such as bacterins that have alum-precipitated, aluminium hydroxide gel and oil-adjuvants.
- The recommended vaccination schedule is animals less than (>3) years old includes; an initial two doses given 1 to 3 months apart, followed by booster vaccinations once or twice yearly. The oil-adjuvant vaccine provides protection for 9–12 months and is administered annually. It is most effective when administered 1 month before the outbreak monsoon or rainy season. Although it provides the strongest immunity, it is unpopular in the field because of its viscosity and difficulty of administration.
- Commercial vaccines are not readily available in Kenya. KEVEVAPI does not produce HS vaccine based on the website information accessed in May 2022.
- Animals that acquire natural infections with Pasteurella multocida serotypes 6: B and 6: E and survive develop a solid immunity.
- There is a need for a vaccine that has longer duration of immunity, easier to administer and offers cross-protection as some African countries, have both strains circulating.

Zoonotic potential

- None reported to date
- However, other P. Multocida serotypes have been reported to cause human infections and precautions should be taken to avoid exposure

Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals and ensure they do not share same grazing land and watering points.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where HS outbreaks are occurring.
- Migrate out of area that has HS outbreaks.

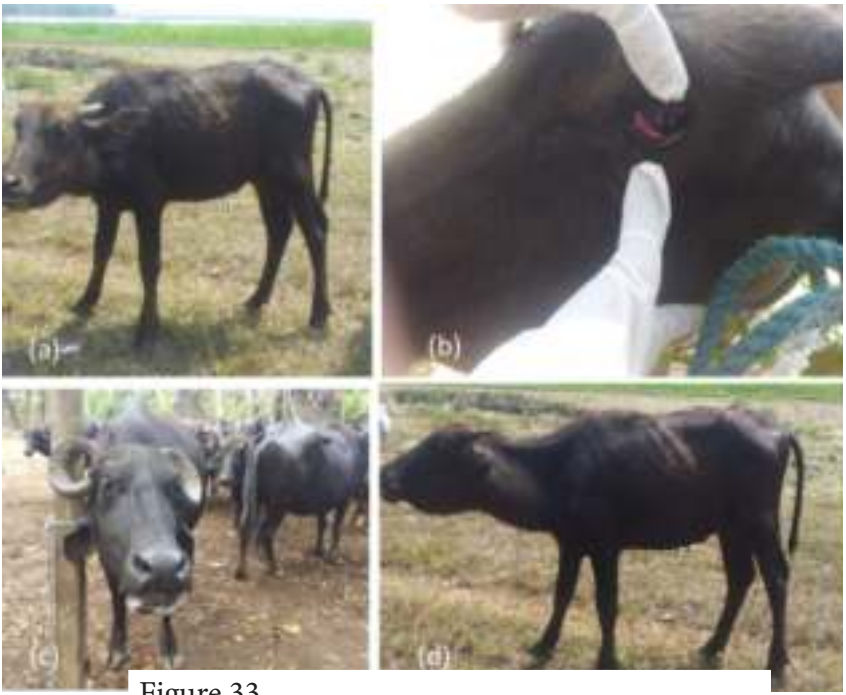


Figure 33

Clinical cases of HS affected buffaloes (a, depressed animal; b, congested mucous membrane; c, hypersalivation and d, oedema in submandibular, cervical region and brisket

Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/
2. WOAH (2021). Technical Disease Card https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
3. Weerathunga M.W.D.C., Ubeyratne , J.K.H. and Nadheer M.A. (2017). An outbreak of haemorrhagic septicaemia in the Navithanveli veterinary range in Ampara district,Sri lanka. S.L.Vet.J. 64(2):7-12. http://doi.org/10.4038/slvj.v64i2.22

4.8 Contagions Bovine Pleuropneumonia (CBPP)- LOUKOI / LOUKOI ANG'ATUK

Other names- Lung plague

Case definition

- Contagious bovine pleuropneumonia (CBPP) is a highly contagious disease of cattle and water buffaloes. Affected animals have difficulty in breathing due to damage to the lungs, the disease in calves occurs as a polyarthritis with painful swelling of all joints.

Distribution (Epidemiology)

- CBPP is endemic in Africa, with minor outbreaks occurring in the Middle East. North America has been free of the disease since 1892, UK since 1898 and Australia since 1973. The last outbreak of CBPP in Europe was seen in Portugal in 1999.
- Little is known about the disease in Asia, China reports its last outbreak was in 1995 but this is not WOAH confirmed.
- CBPP is endemic in Turkana county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus). Livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 17%, mortality rate of 11% and case fatality rate of 64%.
- CBPP incidences in Turkana county occur throughout the year but more cases are reported during the long rainy season (AKIPORO).

Causative agent/ Aetiology

- Bacteria called Mycoplasma mycoides mycoides, small-colony type (MmmSC)
- The Mycoplasma bacteria lacks a cell wall it is pleomorphic and resistant to antibiotics of the betalactamine group, such as penicillin.
- Mycoplasma do not survive for long in the environment and transmission requires close contact.
- The causative agent belongs to the Mycoplasma mycoides cluster. The cluster consists of five mycoplasma species or subspecies from bovines and goats that share serological and genetic characteristics.
- A number of Mmm strain genomes have been fully sequenced. Molecular typing showed that all European Mmm strains groups belong to a single lineage.
- Growth of mycoplasma is fastidious and requires special media rich in cholesterol (addition of horse serum).

Mode of transmission and Pathophysiology

Source of infection

- Aerosol droplets from cough.
- Organism can also be spread through saliva, urine, foetal membranes, uterine discharges and semen.
- Fomites are not a major source of transmission as the organism does not survive outside the host.
- Chronically infected and asymptomatic animals play an important role in the persistence and spread of the disease. Pastoralists herds may contain chronically infected animals. Disease avoidance strategy of migrating from a focus of disease with apparently healthy animals has been known to spread the disease widely.

Transmission and Pathophysiology

- Direct, close, repeated contacts between diseased and healthy animals in shared night bomas, water holes, dip tanks, markets, common grazing and gathering places for mass vaccination campaigns. Air-borne transmission can occur over distances of up to 200 metres.
- Transplacental infection of the unborn calf.
- Indirect transmission is rare and is not an important transmission route of CBPP as the organisms is killed rapidly in hot, dry environments.
- Outbreaks are common and extensive when groups of cattle from different herds are housed or have been transported by truck or trekked on foot.

Affected group

- All age and breeds of cattle are susceptible.

Clinical signs

- The incubation period is one to six months based on WOAH Terrestrial Code guidelines.
- Clinical diagnosis of CBPP is unreliable as initial signs may be slight or non-existent and may be indistinguishable from any severe pneumonia.
- The disease occurs in peracute, acute, subclinical and chronical forms.
- In peracute form few cattle die with no symptoms other than fever (41.5°C). This form occurs in only 10% of infected animals.
- The acute form occurs in 20% of infected animal. The first sign is fever (of up to 41.5°C); anorexia; and painful, difficult breathing. In hot periods of the day, the animal stands on its own in the shade with its head lowered and extended, its back slightly arched, and its elbows turned out, elbows are said to be abducted. Percussion of the chest is painful; respiration is rapid, shallow, and abdominal. If the animal is forced to move quickly, the breathing becomes more distressed and a soft, moist cough results. The disease progresses rapidly and animals have a poor body condition, and breathing becomes more laboured, with a grunt at expiration. The animal then becomes recumbent and dies after 1 to 3 weeks. There may be nasal discharge, sometimes streaked with blood, and frothy saliva that accumulates around the mouth. Some animals develop swellings of the throat and dewlap. Pregnant cows and heifers may abort and diarrhoea has also been recorded.
- If animal survives the acute form they become chronically affected in which the lesions gradually resolve and the animals appears to recover.
- The subclinical form is the most common and occurs in 40 to 50% of the animals affected. The symptoms resemble those of the acute form but are less severe; fever is intermittent. Most of the animals suffering this form move to the chronic form.
- The chronic form is a natural evolution of both acute and sub-acute forms but in some animals it can develop directly. The clinical signs regress but cattle can still have intermittent fever together with loss of weight.
- Infected calves under 6 months present primarily with polyarthritis that is seen as swelling of joints and lameness. Infected calves present with poly-arthritis with swelling of carpal and tarsal joints.
- Lesions in the lungs take a long time to heal fully and inside them the causative agent can survive for up to two years. Up to 25% of affected cattle can become chronic carriers of infection. These chronic carriers are called 'lungers' and are believed to play a role in initiating new outbreaks when they are moved into susceptible herds.

Post mortem lesions

Lesions confined to chest cavity

- The thoracic cavity may contain 10 to 30 litres of yellow fluid that is mixed with fibrin flakes or large clots.
- The lungs are covered with thick deposits of yellow coloured fibrin and do not collapse as is the normal when the chest cavity is open. The lung tissue is firm and resembles the liver tissue.
- Lung lesions in 80 to 90% of cases are unilateral (affect only 1 lung) and in most cases only the diaphragmatic lobes are involved.
- Lung lesions adhere to the chest wall and on cut surface the cut surface of the lung tissue has a marbled appearance because of the widened interlobular septa.
- In chronic cases there may be areas of dead lung tissue that become surrounded by a capsule of fibrous connective tissue. This structure is called a sequestrum.

Diagnosis

Field based diagnosis

- A field based rapid latex agglutination test (LAT) that gives results in less than two minutes, using sera or whole blood, has been developed for screening purposes.
- Post mortem lesions are characteristic and slaughterhouse monitoring is a powerful tool to use in CBPP surveillance.
- Co-existence of pulmonary signs in adults and arthritis in young animals should alert the
- o clinician to a tentative diagnosis of CBPP.
- Samples from live animals include nasal swabs and/or broncho-alveolar washings, or pleural fluid obtained by puncture. Collection of blood in anticoagulant and clot activator tubes.
- Samples taken at necropsy are lung lesions, lymph nodes, pleural fluid and synovial fluid from calves with arthritis.
- Samples should be shipped in a cool box but may be frozen if transport to the laboratory is delayed.

Laboratory diagnosis

- Confirmation is by isolation of the mycoplasma followed by growth inhibition or immunofluorescence test using hyper immune rabbit sera against the mycoplasma, or use of molecular PCR techniques.

Differential Diagnosis (DDx)

- Haemorrhagic septicaemia (HS).
- Bacterial or viral Broncho-pneumonia.
- East Coast Fever.
- Traumatic Reticulo-pericarditis (TRP)/ Foreign body pericarditis,
- On post mortem- Abscesses or Tuberculosis lesions.

Treatment

- In disease free countries immediate slaughter of infected and in contact animals is practiced this is called stamping out.
- Treatment is recommended only in endemic areas because the organisms may not be eliminated, and carriers may develop.
- Drug label dosage should be followed. Three major classes of antibiotics are effective against mycoplasmas, namely tetracyclines, fluoroquinolones and macrolides. The most common antibiotic used is Tylosin at 10 mg per kilogram body weight twice a day for six consecutive days. This is a cumbersome regime for pastoralists settings but animal health providers need to encourage adherence through frequent extension messaging.
- In Turkana county livestock keepers indicated they used the following antibiotics to treat CBPP, Penstrep® (Penicillin and Streptomycin), Tylosin and Oxytetracycline. Based on the nature of the pathogen the penicillin antibiotic is not effective and animal health providers need to discourage its use in managing CBPP.

Prevention/control

- In endemic areas, during outbreaks cattle movement should be restricted through quarantine and ring vaccination.
- Where cattle cannot be confined as is the case of pastoralist set ups, the spread of infection can be limited by immunization with attenuated vaccine (T1/44 strain). In Kenya, KEVEVAPI manufactures CBPP vaccine traded under the name CONTAVAXTM.
- The vaccine is effective only if herd coverage within a county is high (> 60%) of animals covered.

Zoonotic potential

- No

Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS using mobile surveillance app (Kenya Animal Bio Surveillance System (KABS).

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where CBPP outbreaks are occurring
- Present animals for vaccination
- Migrate out of an area when CBPP outbreak are occurring.

Figure 34
Typical Elbow abducted stance of an animal affected by CBPP

Figure 35
Post-mortem finding of yellow turbid fluid in the thoracic cavity with thick yellow fibrin

Figure 36

Typical mottled appearance of cut surface of the lung tissue of an animal infected with CBPP

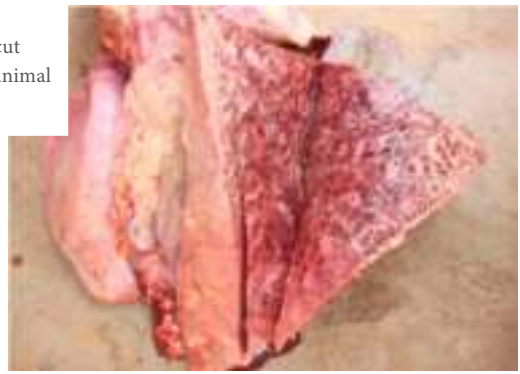
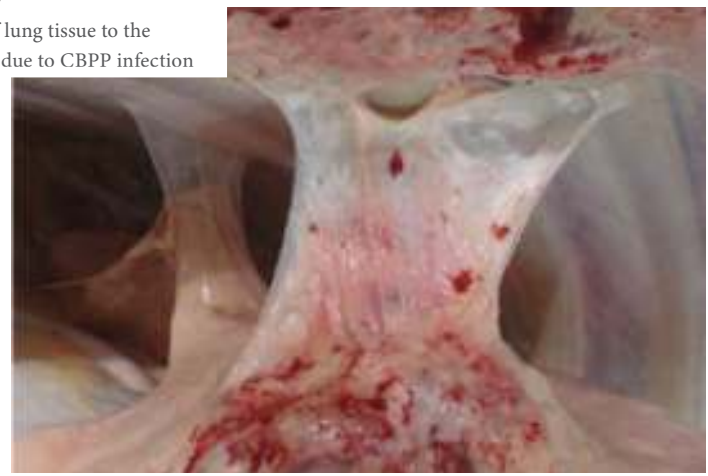


Figure 37

Adherence of lung tissue to the thoracic wall due to CBPP infection



Pictures sourced from: https://vdocuments.mx/contagious-bovine-pleuropneumonia-cbpp-contagious-bovine-pleuropneumonia.html

4.9 Anaplasmosis- LOPID / APID / LONYANG

Other name: Gall sickness – due to gall bladder distention on post mortem

Case definition

- Anaplasmosis is a disease of ruminants caused by an obligate intraerythrocytic bacteria called Anaplasma.
- The Anaplasma bacteria infects red blood cells of cattle, sheep, goats, buffalo, and some wild ruminants.
- Anaplasmosis is the second most important tick borne disease in Kenya after East Coast Fever.

Distribution (Epidemiology)

- Endemic in Turkana county.
- Occurs in tropical and subtropical regions including South and Central America, USA, southern Europe, Africa, Asia, and Australia.

Causative agent/ Aetiology

- Anaplasma bacteria is from the order Rickettsiales, family Anaplasmataceae, genus Anaplasma.
- Bovine Anaplasmosis is caused by Anaplasma marginale.
- A marginale has a strain that has an appendage, formerly known as A caudatum before genome analysis.
- Cattle are also infected with A centrale, which causes mild disease.
- The Anaplasma genus also includes species that were previously known as Ehrlichia like A. bovis (formerly E bovis), and A. platys (formerly E platys). These can invade erythrocytes of other mammalian hosts and therefore are potentially zoonotic.
- In small ruminants, anaplasmosis is caused by Anaplasma Ovis.

Mode of transmission and Pathophysiology

Source of infection

- In Kenya infections are due to the bite of the blue tick, Boophilus decoloratus and possibly also biting flies. Boophilus ticks are one-host ticks and only the engorged adult females drop off the host to lay eggs in the environment.
- Infections can also be due iatrogenic transmission through re-use of hypodermic needles or surgical instruments that have not been cleaned between animals for example during castration or dehorning procedures.

Transmission lifecycle and Pathophysiology

- Close to 17 tick species can transmit A. marginale including Dermacentor, Rhipicephalus, Ixodes, Hyalomma, and Argas.
- Transplacental transmission has been reported and is usually associated with acute infection of the dam in the second or third trimester of gestation.
- There is a strong correlation between age of

cattle and severity of disease. Calves are more resistant to disease (although not infection) than older cattle. This resistance is not due to colostrum antibody from immune dams but it is from the fact that calves born in endemic areas become infected with A. marginale early in life and gain carrier status without manifesting any signs. This situation is called enzootic stability.

- Studies have shown that cattle are capable of developing certain natural immunity to ticks and tick-borne diseases when exposed to them.
- Animals that are infected with the parasite remain lifelong carriers and serve as sources of future infections. Serious losses occur when mature cattle with no previous exposure are moved into endemic areas.

Affected group

- All age groups and cattle breeds are susceptible to infection especially if they are not from endemic areas. However, calves and indigenous cattle breeds (Bos indicus) are less susceptible to severe clinical diseases.

Clinical signs

- Steadily increasing temperatures but does not exceed 41°C like in ECF.
- Anaemia, weakness and respiratory distress particularly after exercise.
- Jaundice and marked loss of body condition.
- Urine is often brown due to the presence of bilirubin.
- Constipation, faeces are dark and firm and sometimes covered with mucus.
- Severely affected animals may die.
- The urine brown colour in Anaplasmosis contrasts with the bloody red urine seen in Babesiosis infections. This is because the infected red blood cells in Anaplasmosis are being destroyed in the spleen while in Babesiosis, the red blood cells rupture/ haemolyse while in circulation hence the bloody urine.
- Anaplasmosis frequently occurs at the same time with ECF. The reason is that ECF causes a depression of the animal's immune responses. This then allows an Anaplasma infection which was already present in the carrier state in the animal to flare up as clinical Anaplasmosis.
- Animals with mixed ECF and Anaplasma infection are very sick with poor prognosis. Research at KALRO Muguga in Kenya indicates that even low Anaplasma parasitaemia (2%) can cause severe symptoms if there is co-infection with ECF.
- Animals with high Anaplasmosis parasitaemia of between 10% and 70% may show milder disease than those with co-infection.

Post mortem lesions

Lesions are typical of those found in animals with anaemia due to erythrophagocytosis.

- Blood is thin and watery.
- Entire carcass is generally markedly anaemic and jaundiced.
- Liver is enlarged with a yellow-orange discolouration.
- Gall bladder is distended and contains thick brown or green bile.
- There are fluid (serous) effusions in the body cavities.
- Petechiae haemorrhages occur in the epi and endocardium.
- Spleen is enlarged with reddish-brown pulp and enlarged splenic follicles.

Morbidity and Mortality Rates Estimates

- A recent molecular study in Kenya, estimated the prevalence of Anaplasma in dairy cows in Nairobi county and its environs to be for A. platys (44.8%), A. marginale (31%) and A. bovis (13.8%). Another study found the seroprevalence of Babesiosis, Anaplasmosis

and ECF to be 0.42%, 4.64% and 4.92% and 1.45%, 32.11% and 39.05% in the rural and peri-urban areas of Western Kenya respectively.
- Molecular based Anaplasma ovis prevalence studies conducted in 2018 and 2019 found a 40.8% prevalence in sheep and goats from central and western Kenya, 19.5% prevalence in small ruminants from North-eastern Uganda, a 60.1% prevalence in sheep and goats from Sudan and in Tunisia the prevalence was higher in sheep at 93.8% when compared to goats at 65.3%.
- Turkana county livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 3%, mortality rate of 2 % and case fatality rate of 67% in cattle and small ruminants.

Seasonality of occurrence
- The presence of active and infected tick vectors determines season of occurrence. reports indicate it can occur in all seasons.
- In Turkana county, Anaplasmosis occurrence in cattle is during the short dry season (AKAMU) and in small ruminants the disease is common during the long rains season (AKIPORO).

Diagnosis

Field based diagnosis
- Microscopic examination is used as a rapid and confirmatory test.
- Samples to be collected include unclotted blood and lymphnodes smear if the nodes are enlarged so as to rule out mixed infections with ECF.
- The best blood smear should be taken from the tip of either the ear or the tail of the sick animal (peripheral blood smear).
- Thin blood smears stained in Giemsa show Anaplasma species appearing as dense, homogeneously staining blue-purple inclusions in the red blood cells that are located towards the margin of the infected erythrocyte.
- At necropsy, thin blood films of liver, kidney, spleen, lungs, and peripheral blood should be prepared for microscopic examination

Differential Diagnosis (DDx)
- Babesiosis
- Leptospirosis
- Bacillary haemoglobinuria
- Eperythrozoonosis

Treatment
- Always check if animal has a mixed infection with ECF before deciding on course of treatment. ECF treatment with Parvaquone or Buparvaquone causes Theileria piroplasms to become round and dark-staining thus resembling Anaplasma parasites.
- The carrier state may be eliminated by administration of a long-acting oxytetracycline preparation (20 mg/kg, IM, at least two injections with a 1-week interval). Injection into the neck muscle rather than the rump is preferred.
- Imidocarb dipropionate is also highly efficacious against A marginale as a single injection at 3 mg/kg. Elimination of the carrier state requires the use of higher repeated doses of imidocarb (5 mg/kg, IM or SC, two weeks apart). Imidocarb is a suspected carcinogen with long withholding periods and is not approved for use in the USA or Europe.

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT IM neck muscles q48 hours PRD IM
Imidocarb dipropionate	Antiprotozoal	6.6 mg/kg BWT (2.5mL per 100kg BWT) Repeat dose in two (2) weeks, for a total of two (2) treatments
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

** Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q and mL- Milliliter, body weight-BWT , kg- kilograms** By Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)*

Prevention/control

- Vaccine is not available in Kenya.
- Vaccine is available in South Africa, Australia, Israel, and South America using a live vaccine derived from Anaplasma centrale (originating from South Africa). There is a killed vaccine in USA used as an experimental vaccine. It requires two doses (28 days apart) and booster doses every 1 to 2 years depending on herd history
- Attempts to use live or attenuated A. marginale have results in fatal disease.
- Sustained and stringent tick control using acaricides is the most common strategy, however it is not practical in pastoralist settings and a balance needs to be made to ensure young animals acquire endemic stability this become immune to infections when they are adults.

Zoonotic potential

- No for A. Marginale. Yes, for Anaplasma species that are adapted to infect more than one mammalian host like A. bovis and A. platys.

Notifiable disease

- No

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Hand pick ticks from animal,

Further Reading

1. Mbogo S.K., Kariuki D.P., McHardy N., Payne R., Ndungu S.G., Wesonga F.D., Olum M.O. and Maichomo M.W. (2016). Training manual for veterinary staff on immunisation against East Coast Fever. A Kenya Agricultural and Livestock Research Organisation (KARO) publication supported by GALVmed, Bill & Melinda Gates Foundation and UK aid. https://assets.publishing.service.gov.uk/media/5aa8e9a040f0b66b625e2bfa/66_Training_Manual_for_Veterinary_Staff_on_Immunisation_against_ECF.pdf
2. MSD Veterinary Manual https://www.msdvetmanual.com/
3. Okuthe OS, Buyu GE. Prevalence and incidence of tick-borne diseases in smallholder farming systems in the western-Kenya highlands. Vet Parasitol. 2006 Nov 5;141(3-4):307-12. https://doi.org/10.1016/j.vetpar.2006.05.016
4. Peter, S.G., Aboge, G.O., Kariuki, H.W. et al. Molecular prevalence of emerging Anaplasma and Ehrlichia pathogens in apparently healthy dairy cattle in peri-urban Nairobi, Kenya. BMC Vet Res 16, 364 (2020). https://doi.org/10.1186/s12917-020-02584-0

Figure 38
Jaundiced liver with distended gall bladder as seen in anaplasmosis in a sheep

Figure 39
Post mortem Anaplasmosis lesions: Enlarged and jaundices liver (right) and spleen (left) in a beef animal

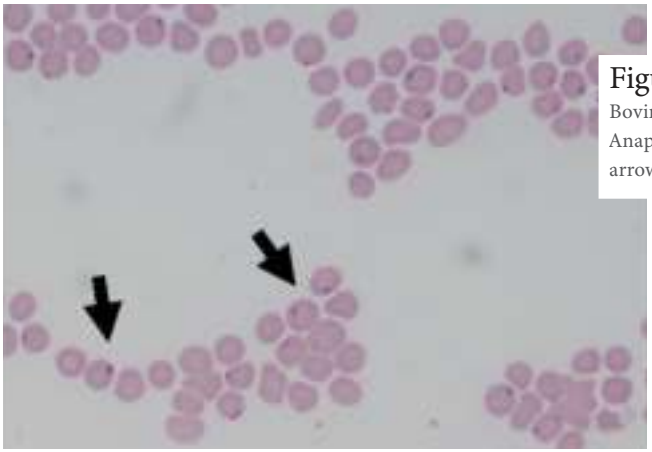


Figure 40
Bovine blood smear showing Anaplasma marginale parasite (black arrows) in the red blood

Pictures sourced from: : http://www.ndvsu.org/images/StudyMaterials/Parasitology/Anaplasmosis.pdf

4.10 Foot rot- EBAIBAI / EKICHODONU

Other names: Interdigital Phlegmon and Foul in the foot

Case definition and Epidemiology

- Foot rot is a sub-acute or acute necrotic (decaying) infectious disease of cattle, causing swelling and lameness in at least one foot. This disease can cause severe lameness and decreased weight gain or milk production.
- Lame bulls and females will be reluctant to breed. If treatment is delayed, deeper structures of the foot may become affected, leading to chronic disease and a poor recovery prognosis. Severely affected animals may need to be culled from the herd.
- Incidence of foot rot varies according to the weather, season of the year, grazing periods and housing system. Foot rot is usually random in occurrence, approximately 20% of all diagnosed of lameness in cattle is foot rot.

Aetiology

- Injury to the interdigital skin provides a portal of entry for infection. Maceration of the skin by water, faeces, and urine facilitate bacteria entry.
- Bacteria organisms that cause foot rot are anaerobic and thrive in environments without oxygen. Both organisms are considered to be normal flora of the bovine gastrointestinal tract and are readily isolated from the rumen and, to a lesser extent, faeces of cattle.
- Two gram negative anaerobic bacteria implicated in foot rot are Fusobacterium necrophorum and Dichlobacter nodosus (formerly Bacteroides nodosus). They are symbiotic in that they work together. One of them enters the break in the skin and then the other one comes along and helps perpetuate the infection.
- Other bacteria isolated in foot rot infection in cattle include, Porphyromonas levii, Trueperella pyogenes (formerly Arcanobacterium pyogenes), and Prevotella melaninogenica (formerly Bacteroides melaninogenicus).
- Most research links occurrence of the disease to injury or abrasion of the interdigital skin. Lesions of the interdigital skin.

Clinical signs

- Foot rot occurs in all ages of cattle, with increased incidences during wet, humid conditions.
- The first signs of foot rot include: Anorexia, extreme pain leading to sudden onset of lameness, which increases in severity as the disease progresses.
- Acute swelling and redness of interdigital tissues and adjacent coronary band.
- Lesions in the interdigital space are often necrotic along its edges and have a characteristic foul odour.
- Severe cases cause swelling around both digits and the hairline of the hoof, leading to separation of the claws and invasion of deeper structures of the foot, such as the navicular bone, coffin joint, coffin bone and tendons.

Diagnosis and Differential diagnosis

- Field based diagnosis can be made by a thorough examination of the foot to identify the characteristic signs of sudden onset of lameness (usually in one limb), interdigital swelling and separation of the interdigital skin.
- Other foot conditions causing lameness that may be confused with foot rot are: FMD lesions, interdigital dermatitis, sole ulcers, sole abscesses, sole abrasions, infected corns, fractures, septic arthritis and inflammation

or infection of tendons and tendon sheaths, all of which often involve one claw of the foot. Swelling attributable to foot rot involves both claws.

Treatment

- Treatment should be administered as soon as signs are observed.
- Most treated animals recover in a few days. Good results are obtained with penicillin G, IM, for 3 days. Early cases respond well to single doses of long-acting oxytetracycline.
- Sodium sulfadimidine solution IV or trimethoprim/sulfadoxine, IV or IM, bid for 3 days, can also be used.

Prevention and Control

- Animals actively shedding infectious organisms should be isolated until signs of lameness have resolved.
- Preventive use of a footbath with an antiseptic and astringent solution (such as copper or zinc sulphate [7%–10% in water]) and regular hoof trimming.
- Vaccines against F necrophorum have failed because of the weak immune response to the bacterium.
- High levels of zinc fed as a supplement have a beneficial effect by improving epidermal resistance to bacterial invaders.

Further Reading

1. Kontturi, M., Junni, R., Simojoki, H. et al. Bacterial species associated with interdigital phlegmon outbreaks in Finnish dairy herds. BMC Vet Res 15, 44 (2019). https://doi.org/10.1186/s12917-019-1788-x
2. MSD Vet Manual. https://www.msdvetmanual.com/musculoskeletal-system/lameness-in-cattle/interdigital-phlegmon-in-cattle

Figure 41
Bull with arched back which is a sign of pain (Left) and swollen hoof (right)

Pictures sourced from: https://www.progressivecattle.com/topics/herd-health/observe-cattle-closely-for-foot-rot

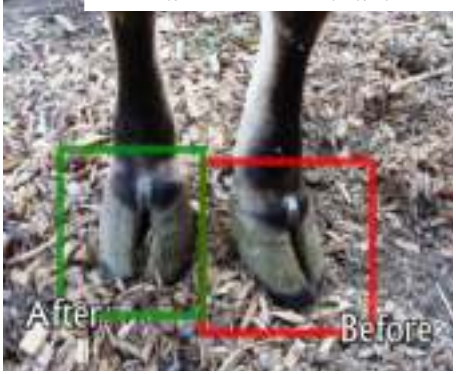


Figure 42
Wound at the interdigital space due to foot rot (Left) and proper hoof trimming of claws in cattle (Right)

4.11 East Coast Fever (ECF)- LOKIT

Other names: Corridor disease, Fortuna disease, January disease, and Theileriosis.

Case definition:

- ECF is an acute disease of cattle, characterized by high fever, swelling of the superficial lymph nodes, dyspnoea and high mortality.
- It is estimated that annually, ECF causes the death of 100,000 cattle of all ages in Kenya. This makes ECF the most important disease of cattle in the country.

Distribution (Epidemiology)

- ECF occurs in 13 countries in Sub-Saharan Africa.
- The disease is a new incursion in Turkana county with few sporadic cases being reported in cattle coming from Uganda.

Causative agent/ Aetiology

- ECF is cause by a protozoan parasite called Theileria parva.
- Theileria parva is an obligate intracellular protozoan parasite that has many strains that vary in virulence. Most mild strains do not result in the animal developing the disease.

Mode of transmission and Pathophysiology

Source of infection

- The bite of an infected tick called Rhipicepalus appendiculatus, the brown ear tick. The saliva of the tick carries the infective form of the parasite called Sporozoites.
- The brown ear tick is a three-host tick. This means that the three stages in its life cycle, the larva, nymph and adult attach to different hosts.
- Eggs of the brown ear tick are not infected with Theileria parasites, so the larvae which hatch from them cannot transmit the disease. Only nymphs and adults are infective.
- About 2-10% of ticks in an ECF endemic area are infected with Theileria parasites. This means that the presence of brown ear ticks on a susceptible animal does not necessarily mean that the animal will develop ECF. However, only 1 tick is needed to transmit the disease to a susceptible animal.
- Animals that recover are immune to subsequent challenge with the same strains but may be susceptible to heterologous strains. However, most recovered or immunized animals remain carriers.

Affected group

- All age groups and all breeds of cattle are affected.
- Calves rarely develop fatal disease like adults.
- Exotic breeds (Bos Taurus) are more susceptible when compared to indigenous Bos indicus breeds.

Clinical signs

- ECF incubation period is 8 to 12 days.
- Fever occurs and continues throughout the course of infection and may be as high as 41 to 42 °C.
- Marked enlargement of all palpable and superficial lymph nodes, particularly, the ones in front of the shoulder (pre-scapulars) and below the ears (parotids) and the on the flanks (pre-crurals).

- Lacrimation, increased nasal discharge and coughing also occurs.
- Terminally there is marked dyspnoea. In the advanced stages of ECF the lungs become full of fluid, breathing becomes difficult and finally froth fills the airways and the animal dies.
- Just before death, there is a sharp decrease in body temperature that is accompanied by recumbency (at this point treatment should not be attempted).
- After death pulmonary exudate pours out of the nostrils.
- Death occurs in 18–24 days after infection.

Other important clinical signs

- Corneal opacity due to infiltration of the cornea by infected lymphoblast cells. This may resolve with treatment but sometimes recovered animals can have permanent loss of eyesight.
- Diarrhoea.
- Infected cells sometimes block capillaries of the central nervous system and result in neurological signs called the 'turning syndrome'.
- Petechiae and ecchymosis haemorrhages may be found on the conjunctiva, oral, lingual (underside of the tongue) and vaginal mucous membranes.
- Mixed infection with other tick-borne parasites due to destruction of lymphoid immune. Cells. Animals may also be infected with Babesiosis (red water) and Anaplasmosis (gall sickness). In endemic tick borne areas, mixed infection with the three parasites is common. This makes correct diagnosis and treatment difficult and may be the reason for failure of some ECF treatments strategies.
- Other diseases, particularly bacterial infections, may also become more apparent in cattle suffering from ECF.

Post mortem lesions

- Marked external and internal lymph node enlargement.
- Extensive pulmonary oedema and hyperaemia, the lung tissue does not collapse and remain distended with fluid after death.
- Haemorrhages are common on the serosa and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Anaemia is not a major diagnostic sign (as it is in Babesiosis) because there is minimal division of the parasites in RBCs and thus no massive destruction of them.
- The mucosa of the digestive tract has lesions that resemble "cigarette burn" these are ulcers in the rumen. In addition, the colon, may show "zebra striping" due to destruction of the cells of the gut wall.
- Surfaces of the kidneys may show white spots (lymphoid infarcts), which extend down into the kidney tissue.

Morbidity and Mortality Rates

- The fatality rate for untreated ECF is 100% in cattle from non–endemic areas especially exotic breeds (Bos Taurus).
- In endemic areas, the morbidity rate is high at 100% even amongst indigenous cattle breeds. However, the mortality for untreated ECF in indigenous breeds is lower and is estimated to range from 20% to 50%.
- Cross breed cattle show an intermediate degree of susceptibility.

Seasonality of occurrence

- Occurrence of disease is dependent on availability of an infected tick, susceptible animals and effectiveness of tick control strategies. Disease incidence can therefore occur all year round.

Diagnosis

- Presumptive diagnosis is based on clinical signs.
- Laboratory samples include lymph node smear, peripheral blood smear, unclotted blood in EDTA and clotted blood in Red-top tubes with clot activator.
- In resource constrained settings like the county laboratories, the main confirmatory tests done are Giemsa staining of lymph node

and blood smears to identify Macroschizont (Koch's blue bodies, KBBs) in lymphoblast and piroplasms in red blood cells.

Differential Diagnosis (DDx)

- Haemorrhagic septicaemia
- Babesiosis
- Malignant Catarrhal Fever (MCF)
- Trypanosomiasis
- Heart water
- Bovine leucosis

Treatment

- Tetracycline though widely used by some livestock keepers in Turkana County has limited value in treating clinical ECF cases. Tetracycline is mainly used during ECF vaccination to suppress development of overt disease.

Active ingredient	Indication	Dosage* and Route**
Parvaquone or Buparvaquone 20- 30%	Antiprotozoal drug	1mL per 20kg bodyweight. (2.5mg / kg BWT) IM into the neck muscles q48-72 hours in severe or advanced cases
Tripelennamine hydrochloride	Anti-inflammatory (anti-histamine)	0.5 – 1.1mg per kg BWT (2.5 – 5ml / 100kg)
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

** Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q mL- Milliliter, body weight-BWT, kg- kilograms. **By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC). NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage*

Prevention/control

- Ticks can survive for up to 2 years on the ground without feeding on cattle. This means it can take more than two years to eliminate ECF infection. This fact makes tick control in pastoralists rangelands a daunting task.
- Chemical control of ticks with acaricides is still the most practical and widely used method for the control of ECF. However, acaricide resistance, high cost of acaricides and improper acaricide use hinder the effectiveness of acaricides.
- Tick control strategy aims at reducing the tick burden but not to eliminate the ticks as cattle acquire resistance to most tick borne disease if they are exposed to a low burden of tick infestation when young.
- ECF vaccination is done through the infection and treatment Method (ITM) in which cattle are given a subcutaneous dose of tick-derived Sporozoites and a simultaneous treatment with a long-acting tetracycline formulation. This treatment results in a mild or inapparent ECF reaction followed by recovery.
- Recovered animals demonstrate a robust immunity that lasts for the lifetime of the animal.
- The most widely used and studied ITM vaccine is the Muguga cocktail, developed in Kenya and comprising of 3 stocks (T. parva Muguga and T. parva Kiambu 5 and buffalo-derived T. parva Serengeti transformed).

Zoonotic potential

- No

Notifiable disease

- No

Further Reading

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2. Mbogo S.K., Kariuki D.P., McHardy N., Payne R., Ndungu S.G., Wesonga F.D., Olum M.O. and Maichomo M.W. (2016). Training manual for veterinary staff on immunisation against East Coast Fever. A Kenya Agricultural and Livestock Research Organisation (KARO) publication supported by GALVmed, Bill & Melinda Gates Foundation and UK aid. https://assets.publishing.service.gov.uk/media/5aa8e9a040f0b66b625e2bfa/66_Training_Manual_for_Veterinary_Staff_on_Immunisation_against_ECF.pdf

3. MSD Veterinary Manual https://www.msdvetmanual.com/

Figure 43

Picture of a cow with heavy tick infestation in the ear photo courtesy of Diba Denge, Animal Health Assistant in Saku, Marsabit County

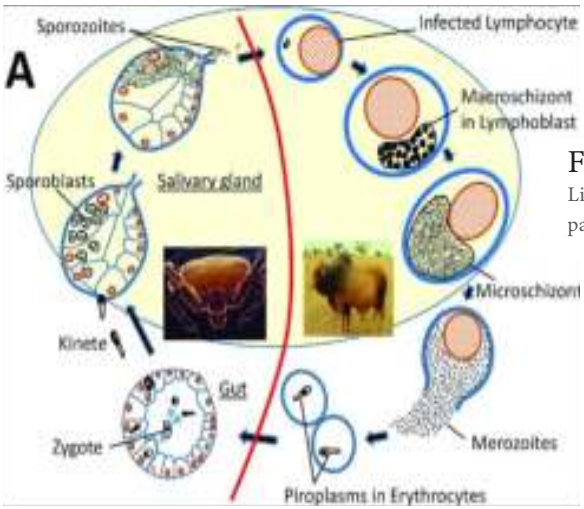

Figure 44

Lifecycle of Theileria parva protozoan parasite in the tick and cattle

Pictures sourced from: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf

Figure 45

Black arrow showing enlarged parotid and prescapular lymphnodes of a cow with ECF

Pictures sourced from: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf

Figure 46

Post-mortem lesion showing frothy lung exudate, the lung tissue has not collapsed

Pictures sourced from: https://assets.publishing.service.gov.uk/media/5aa8e9a040f0b66b625e2bfa/66_Training_Manual_for_Veterinary_Staff_on_Immunisation_against_ECF.pdf

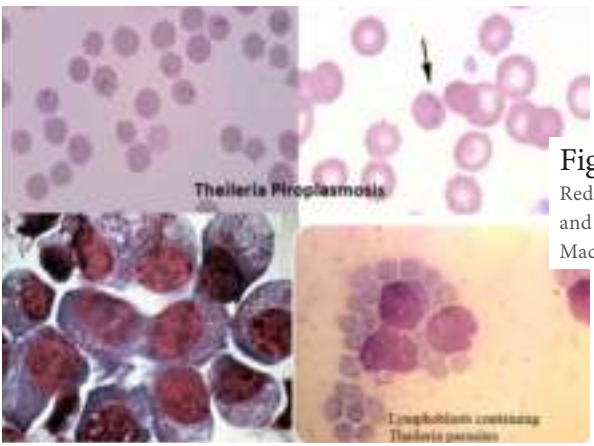

Figure 47

Red blood cells with Theileria piroplasms and lymphoblasts with Theileria Macroschizonts (KBBs)

Pictures sourced from: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf

4.12 Babesiosis- LONYANG / LORENG'EKUL

Other names: Red water, Piroplasmosis, Tick fever, Texas Fever

Case definition

- An acute disease characterised by, fever, intravascular haemolysis leading to progressive anaemia, haemoglobinuria, and jaundice.
- Diagnosis is principally by light microscopic evaluation of blood smears. The most common treatments are imidocarb dipropionate and diminazene aceturate.
- Babesiosis is caused by an intraerythrocytic protozoan parasites of the genus Babesia.
- It is a tick borne infection and affects a wide range of domestic and wild animals and occasionally people.

Aetiology and Epidemiology

- Babesiosis occurs where the arthropod vector is distributed, especially tropical and subtropical climates.
- The main Babesia species that cause disease in cattle are Babesia bovis and B. bigemina in Africa, Asia, Australia, and Central and South America. Babesia divergens occurs in some parts of Europe and northern Africa.
- In small ruminants, Babesia species that cause the disease include; B ovis, B motasi and B. crassa. The disease is more severe in sheep than in goats. Ovine babesiosis Infection are important in the Middle East, southern Europe, and some African and Asian countries.
- A 2018 meta-analysis study estimated the global bovine babesiosis prevalence to be 29% with B. bigemina having a prevalence rate of 22% and B. divergens prevalence of 12 %. An Ethiopian study in 2018 found a prevalence of 11.7% babesiosis and 6.1% anaplasmosis.

Mode of transmission and Pathophysiology

Source of infection in cattle

- Transmitted by tick vectors Rhipicephalus microplus (formerly Boophilus microplus) and Rhipicephalus annulatus (formerly Boophilus annulatus) where transmission occurs transovarially.
- Intrauterine infection has also been reported but it is rare.

Source of infection in small ruminants

- Babesia ovis and B. motasi. They are transmitted vertically by ticks of the genera Rhipicephalus, Dermatocenter, Ixodes and Haemophysalis.

Transmission and Pathophysiology

In endemic areas, two features are important in determining the risk of clinical disease:

- Calves, lambs and kids have a degree of immunity (related both to colostrum-derived antibodies and to age-specific factors that persists for 6 months to 2 years) animals that recover from Babesia infections are immune for their commercial life (4 years),
- Susceptibility of breeds to ticks and Babesia infections varies; Bos indicus cattle are more resistant to ticks and the effects of B bovis and B bigemina infection than Bos taurus breeds. In small ruminants' indigenous breeds like the black head Persian and Galla goats are less susceptible when compared to exotic breeds like Dorper and Toggenburg.

Clinical signs

- Incubation period is between 2 to 3 weeks or longer after tick infestation.
- B bovis is a much more virulent organism than B bigemina in cattle while B. ovis is more virulent in small ruminants especially in sheep.
- B bovis, causes hypotensive shock syndrome, combined with generalized nonspecific inflammation, coagulation disturbances, and erythrocyte stasis in capillaries.

Babesia bovis clinical signs

- High fever (41°C) that persists throughout the clinical course of the disease.
- There is anaemia, jaundice, and weight loss.
- Haemoglobinemia and haemoglobinuria occur in the final terminal stages of the disease.
- CNS signs may be seen due to adhesion of parasitized erythrocytes in brain capillaries can occur with B bovis infections.
- Either constipation or diarrhoea may be present.
- Late-term pregnant cows may abort, and temporary infertility due to transient fever may be seen in bulls.
- Animals that recover from the acute disease remain infected for a number of years with B bovis and for a few months in the case of B bigemina. No clinical signs are apparent during this carrier state.
- Haemoglobinuria is the production of dark red or brown-coloured urine

B. ovis infections

- acute form of the disease is characterized by fever, progressive anaemia and icterus. Haemoglobinuria is rare but may occur in the late stage of the disease. Pregnant animals may abort. Susceptible flocks may suffer a high rate of mortality. Recovered animals that are latently infected are usually immune for a certain period. There is no cross-immunity between the parasites.

Post mortem lesions

- intravascular haemolytic condition -Pale or icteric mucous membranes; blood may appear thin and watery
- Subcutaneous tissues, abdominal fat and omentum appear icteric
- Swollen liver with an orange-brown or paler coloration; enlarged gall bladder containing thick, granular bile
- Enlarged, dark, friable spleen
- Kidneys appear darker than normal with possible petechial haemorrhages
- Bladder may contain dark red or brown-coloured urine
- Possible oedema of lungs
- Petechiae or ecchymosis on surface of heart and brain

Morbidity and Mortality Rates

- Morbidity and mortality vary greatly and are influenced by several factors, such as tick control, previous exposure to a species/strain of parasite, age and breed.
- In endemic areas, cattle become infected at a young age and develop a long-term immunity. However, outbreaks can occur in these endemic areas if exposure to ticks by young animals is interrupted or immuno-naïve cattle are introduced. The introduction of Babesia infected ticks into previously tick-free areas may also lead to outbreaks of disease.
- In Turkana county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 5%, mortality rate 3% and case fatality rate of 60% in cattle while in sheep and goats they estimated an apparent morbidity rate of 4%, mortality rate of 3% and case fatality rate of 75%.

Seasonality of occurrence

- Disease occurs throughout the year as it depends on tick vector presence and availability of susceptible host.
- In Turkana county, the disease occurs during the long rainy season (AKIPORO).

Diagnosis

Field based diagnosis

- Microscopic examination of Giemsa-stained blood or organ smears. From the live animal, thick and thin blood smears should be prepared, preferably from capillaries from the ear or tail tip. Smears of heart muscle, kidney, liver, lung, brain, and from a blood vessel in an extremity (lower leg) should be taken at necropsy.
- Babesia are found within RBCs, and all divisional stages – ring (annular) stages, pear-shaped (pyriform) also known as trophozoites are found as single or in pairs.

Confirmatory test

- Serologic tests such as indirect fluorescent antibody test and ELISA for detection of antibodies to Babesia especially in animals with low parasitaemia or carriers. PCR and real-time PCR assays are also done to detect extremely low parasitaemia, as occurs in carrier animals.

Differential Diagnosis (DDx)

- Anaplasmosis
- Trypanosomiasis
- Theileriosis
- Bacillary haemoglobinuria
- Leptospirosis
- Eperythrozoonosis
- Rapeseed poisoning
- Chronic copper poisoning
- Rabies- Central nervous system signs

Treatment

Early treatment before animals become severely anaemic gives a better prognosis. In both cattle and small ruminants, the following drugs should be used. Always use lower range in very sick animals. Supportive treatment with multivitamin should also be done.

- Diminazene aceturate at 3.5 mg/kg, IM, once.
- Imidocarb dipropionate at 1.2 mg/kg, SC, once. At a dosage of 3 mg/kg, imidocarb provides protection from babesiosis for approximately 4 weeks and may also eliminate B bovis and B bigemina from carrier animals.

Prevention/control

- Vaccination using live, attenuated strains of the parasites has been used successfully in Argentina, Australia, Brazil, Israel, South Africa, and Uruguay. The vaccine is provided in either as chilled or frozen form. One vaccination produces adequate immunity for the commercial life of the animal (4 years).
- Vaccine breakdowns have been reported.
- Controlling or complete eradication of the tick vector can break the transmission cycle, this approach is rarely feasible in the long term and can lead to large, susceptible populations in endemic areas with consequent risk of outbreaks of disease in naive animals.

Zoonotic potential

- Yes, Human babesiosis has been reported with the rodent parasite B microti and the cattle parasite B divergens in North America and Europe, respectively.
- Human Babesia infections are acquired via bites from infected ticks or through contaminated blood from an infected transfusion donor. Disease can be fatal in splenectomised or immunocompromised individuals.

Notifiable disease

- Yes, Bovine Babesiosis is listed by WOAH and should be reported to county and national director of veterinary services through the mobile app Kenya Animal Biosurveillance System(KABS).

Further reading

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2. Keneth Iceland Kasozi, Susan Christina Welburn, Gaber El-Saber Batiha, Najat Marraiki, David Paul Nalumenya, Monica Namayanja, Kevin Matama, Kelly Katenta Zalwango, Wycliff Matovu, Gerald Zirintunda, Justine Ekou, Stellamaris Kembabazi, Claire Mack Mugasa, Annah Kitibwa, Dickson Stuart Tayebwa, Simon Peter Musinguzi, Michael Mahero, Ibrahim Ssengendo, Anne Nanteza, Enock Matovu, Ewan Thomas MacLeod,Molecular epidemiology of anaplasmosis in small ruminants along a human-livestock-wildlife interface in Uganda, Heliyon,Volume 7, Issue 1,2021,. https://doi.org/10.1016/j.heliyon.2020.e05688

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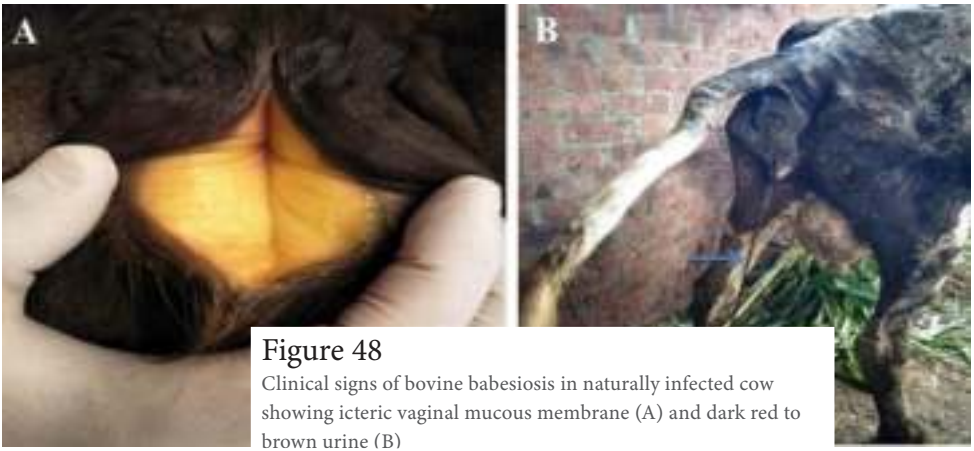

Figure 48

Clinical signs of bovine babesiosis in naturally infected cow showing icteric vaginal mucous membrane (A) and dark red to brown urine (B)

Pictures sourced from: https://www.researchgate.net/publication/329416657_A_study_on_bovine_babesiosis_and_treatment_with_reference_to_hematobiochemical_and_molecular_diagnosis/figures?lo=1

Figure 48 shows various B. motasi intracellular stages. a-c Trophozoites in the periphery of infected RBCs (arrowheads). b-d Different schizonts due to multiplication of Babesia motasi merozoites inside RBCs (arrows).

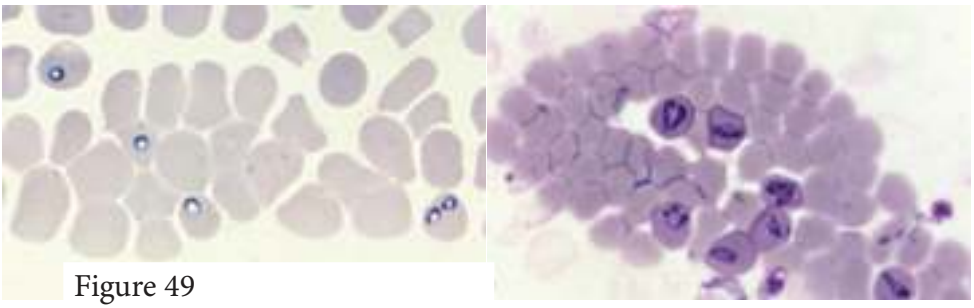

Figure 49

Blood smear showing Babesia Bovis (Right) and Babesia bigemina (Left)

Pictures sourced from: Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. WOAH (World Organisation for Animal Health) ISBN 978-92-9044-804-4.

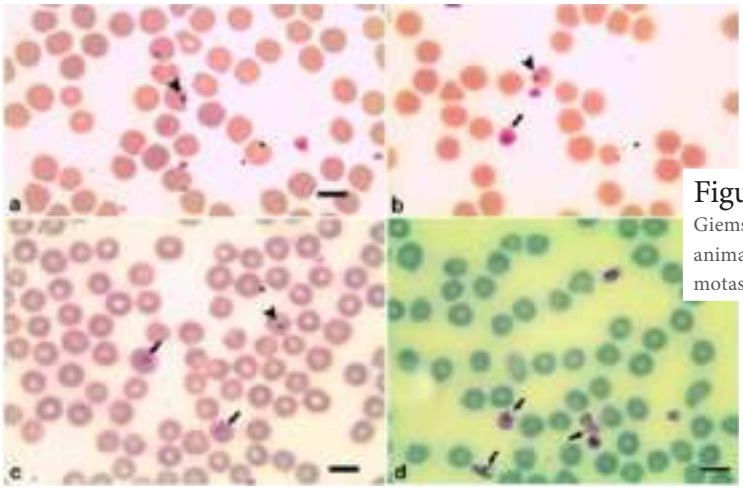

Figure 50
Giemsa-stained smears from animals infected with Babesia motasi

Pictures sourced from: Hussein, Nermean & Mohammed, Eman & Hassan, Amal & El-Dakhly, Khaled. (2017). Distribution Pattern of Babesia and Theileria Species in Sheep in Qena Province, Upper Egypt. Archives of Parasitology.

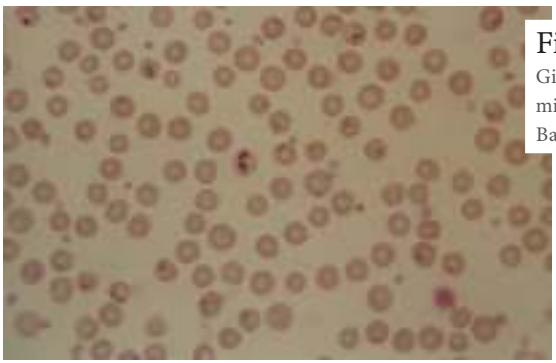

Figure 51
Giemsa stained blood smear showing mixed infection of Anaplasma spp. and Babesia ovis.

Pictures sourced from: Pieragostini, Elisa & Ciani, Elena & Rubino, Giuseppe & Petazzi, Ferruccio. (2011). Tolerance to Tick-Borne Diseases in Sheep: Highlights of a Twenty-Year Experience in a Mediterranean Environment. 10.13140/2.1.2385.6968.

Urine before treatment (i) and progressive improvement (ii to iv) to normal (v) after prompt treatment.

Figure 52
Serial collection of urine from a dairy cow affected by Babesiosis

Pictures sourced from: https://www.dairyknowledge.in/article/babesiosis

4.13 Trypanosomiasis – LOKIPI / EKALICH

Other Names: Surra, Nagana, Tsetse Fly Disease, African Animal Trypanosomosis

Case definition

- Protozoan disease of animals and humans caused by parasites of the genus Trypanosoma, which are found in blood plasma, various body tissues and fluids. The disease causes serious economic losses in livestock.

Trypanosoma spp	Animals Mainly Affected	Major Geographic Distribution
T congolense	Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals	South and eastern Africa
T vivax	Cattle, sheep, goats, camels, horses, various wild animals	Africa, Central and South America, West Indiesa
T brucei brucei	All domestic and various wild animals; most severe in dogs, horses, cats	South and eastern Africa
T brucei rhodesiense	Cattle, wild hooved stock, people	South and eastern Africa

Causative agent/ Aetiology

- The major veterinary species are T congolense, T vivax, T brucei brucei, and T simiae. T brucei rhodesiense and T brucei gambiense are zoonotic, with people as the predominant host. Cattle, sheep, and goats are infected, in order of importance, by T congolense, T vivax, and T brucei brucei. T vivax is found outside tsetse-infested areas of sub-Saharan Africa, carried mechanically by biting flies. There are three variants of T.congolense, called the savannah, forest and kilifi (or Kenya Coast) types.
- Trypanosomes do not survive for long periods outside the host.

Mode of transmission and Pathophysiology

Source of infection

- Tsetse flies (Glossina spp.) are biological vectors for the trypanosomes that cause African animal trypanosomiasis and transmit these organisms in their saliva. Trypanosomes must develop for one to a few weeks in the fly before they reach the infective stage. T. vivax has the shortest cycle.
- Mechanical transmission is significant for T. vivax, which is transmitted primarily by this route through biting flies like Stomoxys, Tabanus and Chrysops species.
- Animals infected with trypanosomes can become chronic carriers, and inapparent infections can be reactivated if the animal is stressed.
- Transplacental and venereal transmission has been reported.

Transmission and Pathophysiology

- Infected tsetse inoculates metacyclic trypanosomes into the skin of animals, where the trypanosomes reside for a few days and cause localized inflammation (chancres). They enter the lymph and lymph nodes, then the

bloodstream, where they divide rapidly by binary fission.
- In T congolense infection, the organisms attach to endothelial cells and localize in capillaries and small blood vessels. T brucei species and T vivax invade tissues and cause tissue damage in several organs.
- The immune response is vigorous, and immune complexes cause inflammation, which contributes to fever and other signs and lesions of the disease. Antibodies against the surface-coat glycoproteins kill the trypanosomes. However, trypanosomes have a large family of genes that code for variable surface-coat glycoproteins that are switched in response to the antibody response, thus enabling them to evade animal's immunity. This antigenic variation results in persistence of the organism. Antigenic variation has prevented development of a protective vaccine and permits reinfections when animals are exposed to a new antigenic type.

Affected group and season of occurrence
- All breeds affected but indigenous breeds less susceptible and seem to resist clinical disease if they are in good nutritional status.
- All ages but studies have shown that cattle less than 2 years are not affected by the disease. This may be because older cattle graze in tsetse fly infested areas whereas younger animals graze close to homesteads, hence lowering their exposure to tsetse bites.
- In Turkana county, livestock keepers in the year 2022 indicated that the disease occurs mostly during the short dry season (AIT/ NAIT).

Morbidity and Mortality rates
- Study estimate a morbidity rate of between 30 and 50% and mortality rate of 6 to 30%.
- In Turkana county livestock keepers in the year 2022, estimated an apparent annual morbidity rate of 3% with 1% mortality rate and 33% case fatality rate.

Clinical signs
- The incubation period is usually 1 to 4 weeks. The first sign is a localized swelling (chancre) at the site of the fly bite, but this usually remains unnoticed.

Acute form
- An acute haemorrhagic syndrome has been seen sporadically in cattle infected with some isolates of T. vivax, mostly in East Africa. This condition might be mediated by antibodies directed against red blood cells. Affected animals have enlarged lymph nodes and signs of severe anaemia, and they develop widespread visceral and mucosal haemorrhages, particularly in the gastrointestinal tract. In one outbreak, the main haemorrhagic sign was bleeding from the ears. This form can be rapidly fatal.

Chronic form
- The primary clinical signs are an intermittent fever, signs of anaemia (e.g., pale mucous membranes, lethargy), lymphadenopathy and weight loss.
- Progressive emaciation leads to concurrent signs such as decreases in milk yield, decreased appetite, and may develop neurological signs, dependent oedema (including o submandibular oedema), cardiac lesions, diarrhoea and keratitis/ corneal opacity.
- There may also be abortions, premature births, perinatal losses and damage to the male reproductive organs (e.g., orchitis, epididymitis), with reduced semen quality.
- Trypanosomes can cause immunosuppression, and concurrent infections may complicate this disease. Sudden deaths have been reported
- Animals that recover clinically may relapse when stressed.

Post mortem lesions
- Necropsy findings vary and are nonspecific. In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen. In chronic cases,

swollen lymph nodes, serous atrophy of fat, and anaemia are seen.

Filed based diagnosis

- A presumptive diagnosis is based on finding an anaemic animal in poor condition in an endemic area.
- Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. The most sensitive rapid method is to examine a wet mount of the buffy coat area of a PCV tube after centrifugation, looking for motile parasites. This is called the Buffy coat concentration technique
- Rapid agglutination tests to detect circulating trypanosome species-specific antigens in peripheral blood are available for both individual and herd diagnosis, although their reliability remains varied.

Differential Diagnosis (DDx)

- Infections that cause anaemia and weight loss, such as babesiosis, anaplasmosis, theileriosis, should be excluded by examining a stained blood smear.

Treatment

- Most have a narrow therapeutic index, which makes administration of the correct dose essential. Drug resistance occurs and should be considered in refractory cases
- Drugs are normally sold in powder form or compressed tablets, and are stable over long periods when kept dry. Some become unstable when dissolved and must be used within a short period. The manufacturer's instructions should always be followed and it is a good practice to use solutions only on the day they are made. Some are a sterile solution diluent while some ask you to dilute in hot/warm or cold clean boiled water. (allow hot water preparations to cool before administering)

Prevention/control

- Control is ideally achieved by combining methods to reduce the tsetse/biting fly challenge and by enhancing host resistance with prophylactic drugs.
- Control can be exercised at several levels, including eradication of tsetse flies and use of prophylactic drugs. Tsetse flies can be partially controlled by frequent spraying and dipping of animals, aerial and ground spraying

Drug	Effective against Trypanosoma	Main Action	Dosage and route
Diminazene aceturate (7%)	vivax, congolense, brucei	Curative	Deep IM or SC in the neck muscles 5-10 ml/100 kg BWT
Homidium chloride or bromide (2.5%)	vivax, congolense, brucei	Curative and some Prophylactic	Deep IM of neck muscles only 1 mg/kg BWT (1 ml/25 kg)
Isometamidium chloride (1% or 2%)	vivax, congolense	Curative and Prophylactic For preventive repeat every 10 to 12 weeks	Deep IM of neck muscles only 50 kg- 1.25 to 2.5 ML 100kg-2.5 – 5 ML 150kg-3.75-7.5 ML 200kg-5-10ML

Body weight-BWT, kg- kilograms; Intramuscular (IM), and Subcutaneous (SC)

of insecticides on fly-breeding areas, use of insecticide-impregnated screens and targets, bush clearing, and other habitat removal methods. The Sterile Insect Technique (SIT) has been used with success in Zanzibar and may be used in other area-wide control operations after suppression of tsetse populations by insecticides. There is renewed international interest in large-scale tsetse eradication through the Pan African Tsetse and Trypanosomiasis Eradication Campaign (PATTEC) supported by the African Union. Animals can be given drugs prophylactically in areas with a high population of trypanosome-infected tsetse. Drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

- Several breeds of cattle and water buffalo have been identified that have innate resistance to trypanosomiasis and could play a valuable role in reducing the impact of the disease in these areas. However, resistance may be lost because of poor nutrition or heavy tsetse challenge.

Zoonotic potential

- No
- Humans are normally not susceptible to the trypanosomes that cause African animal Trypanosomiasis.

Notifiable disease- No

Ethnoveterinary treatment practices

- Refer to tables 11 and 12

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid areas with Tsetse flies.

Further Reading

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3. Simwango, M., Ngonyoka, A., Nnko, H. et al. Molecular prevalence of trypanosome infections in cattle and tsetse flies in the Maasai Steppe, northern Tanzania. Parasites Vectors 10, 507 (2017). https://doi.org/10.1186/s13071-017-2411-2

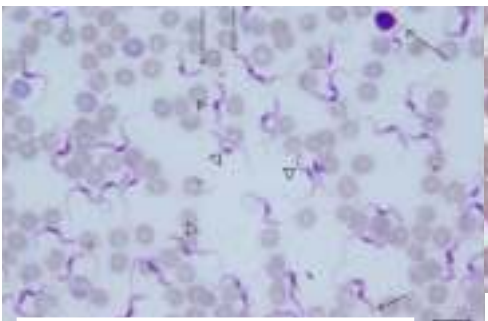


Figure 53
Trypanosome evansi in cattle blood

Pictures sourced from: https://www.hindawi.com/journals/bmri/2013/194176/

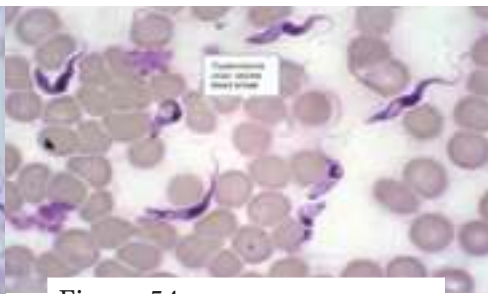


Figure 54
Trypanosome vivax in bovine blood

Pictures sourced from: https://www.msdvetmanual.com/circulatory-system/blood-parasites/trypanosomiasis#v3258185

4.14 Gastrointestinal (GIT) Parasites- Helminthiasis – NG'IRITAN / NG'IPELEI

Case definition

- Helminth parasites are found in cattle, sheep and goats in all countries and regions of the world. Many of these parasites are commonly associated with poor production and unthriftiness and can produce acute disease and even death.
- Their presence in an animal, does not mean that they are necessarily the cause of any overt disease in that animal. It is important to assess the type and level of parasitism in a herd or flock in order to be able to determine the significance of helminth infections and to recommend the most cost-beneficial control measures.

Epidemiology

- Global distribution

Aetiology

- GIT parasites in cattle, sheep and goats can be classified into four broad classes; Nematodes, Cestodes, Trematodes and Protozoa. In this discussion we have included Coccidia protozoa parasite as it often an important co-infection of helminths in livestock.
- Examples of common parasites in the groups include;
- Nematodes- Haemonchus, Ostertagia, Trichostrongylus, Cooperia, Bunostomum, Strongyloides, Nematodirus, Toxocara, Oesophagostomum, Chabertia and Trichuris.
- Cestodes- Moniezia, Cysticercus
- Trematodes- Fasciola
- Protozoa- Eimeria species

Clinical signs and Transmission

- Common stomach worms of cattle are Haemonchus placei (barber's pole worm, large stomach worm, wire worm), Ostertagia ostertagi (medium or brown stomach worm), and Trichostrongylus axei. Young animals are more affected, but adults not previously exposed to infection frequently show signs and succumb. Ostertagia and Trichostrongylus infections are characterized by profuse and persistent watery diarrhoea. In Haemonchosis there is no diarrhoea, there could be intermittent periods of constipation. Anaemia is the most significant sign. The blood loss due to Haemonchus infection causes a hyperproteinaemia and oedema particularly under the lower jaw (bottle jaw) and sometimes along the ventral abdomen. Heavy infections can result in death before clinical signs appear. Other variable signs include progressive weight loss, weakness, rough coat, and anorexia. PM lesions- Worms can be seen in the abomasum, and small petechiae may be visible where the worms have been feeding. Characteristic lesions of Ostertagia infection are small, umbilicated nodules seen as discrete, but in heavy infections they coalesce and give rise to a "cobblestone" or "morocco leather" appearance. Nodules are most marked in the fundic region but may cover the entire abomasal mucosa.
- Cooperia punctata and C pectinata are found in the small intestine and may be difficult to observe grossly.They cause profuse

diarrhoea, anorexia, and emaciation, but no anaemia; the upper small intestine shows marked congestion of the mucosa with small haemorrhages.

- Bunostomum phlebotomum (hookworm) are found in the small intestine. Infection can either be through ingestion of eggs or skin penetration by larvae. Larval penetration of the lower limbs may cause uneasiness and stamping. Adult worms cause anaemia and rapid weight loss. Diarrhoea and constipation may alternate. Hyperproteinaemia oedema may be present, but bottle jaw is rarely as seen in severe Haemonchosis. PM lesions- Worms are readily seen in the first few feet of the small intestine, and the contents are often blood-stained. As few as 2,000 worms may cause death in calves. Local oedema and scab formation of skin where larvae penetrated may be seen.
- Strongyloides papillosus (intestinal threadworm)- host is infected by penetration of the skin or by ingestion; infective larvae can be transmitted in colostrum. Infections are most common in young calves, but does not cause severe signs. Signs include intermittent diarrhoea, loss of appetite and weight, and sometimes blood and mucus in the faeces. Post mortem lesions- Large numbers of worms in the intestine produce catarrhal enteritis with petechiae and ecchymosis, especially in the duodenum and jejunum.
- Nematodirus helvetianus- Signs include diarrhoea and anorexia. Immunity to reinfection develops rapidly. PM Lesions- Thickened, oedematous mucosa.
- Toxocara vitulorum- (Roundworm) stout, whitish worm found in the small intestine of calves <6 mo old; older calves are resistant. Larvae hatching from ingested eggs pass to the tissues and, in pregnant cows, are mobilized late in pregnancy and passed via the milk to calves. Migrating larvae can be found in numerous organs: lungs, trachea, bronchi, liver, kidneys or mammary glands. Infection are not serious adult cattle. However, in calves migrating larvae can seriously damage numerous organs in adult cattle, particularly the lungs, where they cause infections with secondary bacteria and subsequent pneumonia. In calves, the adult worms in the small intestine compete for nutrients with the host, and can cause diarrhoea (often putrid), colic, enteritis, loss of appetite and weight loss. Due to the large size of the worm, a massive infection can obstruct the gut and may perforate it.
- Oesophagostomum radiatum (nodular worm)- Young animals suffer from the effects of adult worms, whereas in older animals, the effect of nodules enclosing larval worms is more important. Infection causes anorexia; severe, constant, dark, persistent, fetid diarrhoea; weight loss; and death. In older, resistant animals, the nodules surrounding the larvae become caseated and calcified, thus decreasing the motility of the intestine. Stenosis or intussusception occasionally occurs. Nodules can be palpated per rectum, and the worms and nodules can be seen readily at necropsy.
- Tapeworm Moniezia expansa - found in young cattle. Lack rostellum and hooks, and the segments are wider and long unlike for cyticercus. The eggs are ingested by the intermediate host, free-living oribatid mites, which live in the soil and grass. After 6–16 wk, infective cysticercoids are present in the mites. Infection occurs after ingestion of infected mites; Moniezia are commonly considered non-pathogenic in calves.
- Fascioliasis symptoms in cattle is determined by the number of metacercariae ingested. Acute disease occurs 2 to 6 weeks after the ingestion of large numbers of metacercariae (usually >2,000) in a short period. Acute disease manifests as distended, painful abdomen; anaemia; and sudden death. In subacute disease, cattle ingest moderate numbers (500 to 1,500) metacercariae over a long period of time. The cattle can survive for 7 to 10 weeks even in cases of significant hepatic damage, but deaths occur due to haemorrhage and anaemia. Chronic fascioliasis occurs as a result of ingestion of low numbers (200 to 500) metacercariae over a long period of time. Signs include progressive emaciation,

anaemia, unthriftiness, submandibular oedema (bottle jaw), and reduced milk production. In cattle, a partial acquired resistance develops after 5 to 6 months following infection. Post mortem lesions- Severity depends on the number of metacercariae ingested, the phase of development in the liver, and the species of host involved. During the first phase, immature, wandering flukes destroy liver tissue and cause haemorrhage. The main lesions seen are enlarged liver that is friable with fibrinous deposits on the capsule. Migratory tracts can be seen, and the liver surface has an uneven appearance. In the second phase when flukes enter the bile ducts, lesions are consistent with chronic liver damage such as cirrhosis where the damaged bile ducts become enlarged, or even cystic, and have thickened, fibrosed walls. In cattle but not sheep, the duct walls become greatly thickened and often calcified. Aberrant migrations occur more commonly in cattle, and encapsulated flukes may be found in the lungs. Mixed infections with Fasciola gigantica can also be seen in cattle. Tissue destruction by wandering flukes may create a microenvironment favourable for activation of Clostridial spores and animal can succumb to Clostridial infection.

- Coccidia are protozoan parasites; most species infecting cattle, sheep and goats belong to the genus Eimeria. All Eimeria species parasitize the intestinal epithelium of infected animals. Older animals Usually become immune to infection but often remain carriers of coccidia and continue to pass oocysts in the faeces. Young animals become infected by ingesting sporulated oocysts in contaminated food and water. The multiplication of the parasite in the intestine causes damage to the mucous membrane. The severity of this damage depends on the number of oocysts ingested. Clinical signs seen only in young animals. A prominent sign of clinical coccidiosis is diarrhoea, which is sometimes bloody. Affected animals have poor growth rates and severely affected animals may die.

Diagnosis

Field based Diagnosis

- Composite samples for faecal egg counts and oocyst identification. Composite samples are collected from 10 animals and a single (composite) dung sample is submitted to the laboratory for oocyst and faecal egg count and microscopic identification. Larval culture can also be done.

Laboratory diagnosis

- Confirmatory Diagnosis can be aided through ELISA analysis (commercially available in Europe) that enables early detection (2 to 3 weeks) of antibodies in serum or milk after infection.

Treatment

Active ingredient	Indication	Dosage* and Route**
10% Albendazole	Anthelmintic and Flukicide Effective for mature (more than 90 days old) flukes only	10 mg/kg BWT that is (1 mL per 10 kg BWT PO) Repeat after 2 to 3 weeks to target the immature flukes that have become adults
10% Triclabendazole	Anthelmintic and Flukicide Effective against all stages all stages of Fasciola hepatica from 2 day old immature to adult flukes	3 mL/25 kg BWT PO (2 mg /kg BWT) One dose
Levamisole HCl 30 mg Oxyclozanide 60 mg	Anthelmintic and Flukicide Oxyclozanide only active against the adult liver fluke	10 mL per 40 kg BWT PO Repeat after 2 to 3 weeks to target the immature flukes that have become adults
34% Nitroxyl	Anthelmintic and Flukicide Effective against immature and mature liver flukes	1.5 mL per 50 kg BWT SC only (10mg/kg)
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

Prevention/control

The principle of a parasite control strategy is to keep the challenge to young livestock by the pathogenic trichostrongyle parasites at a minimum rate. This is achieved in the following ways.

(a) Controlling the density of livestock (stocking rate). Overstocking forces, the animals to graze closer to faecal material and closer to the ground, and may result in the consumption of a higher number of infective larvae.

(b) Periodic and strategic deworming when conditions are most favourable for larval development on the pasture.

(d) Separating age groups.

(e) Reducing the effects of gastro-intestinal parasites by providing an adequate plane of nutrition.

(f) Using grazing management to minimize the uptake of infective larvae and to create safe pastures like delay animals going to pasture in the morning during rainy season to allow dew to dry off grass blades and thus allow larvae to return to the soil level.

Zoonotic potential

- Yes, important parasitic zoonosis for Fasciola, hook worm and round worm as human can serve as accidental host for the migrating forms.
- WHO estimated in 1995 that approximately half of the 2.39 million infected individuals globally were living in three Latin American countries (Bolivia, Ecuador and Peru). The Andean highlands represent the most endemic areas of the world, with high levels of prevalence of Fasciola infection among indigenous communities.

Notifiable disease – No

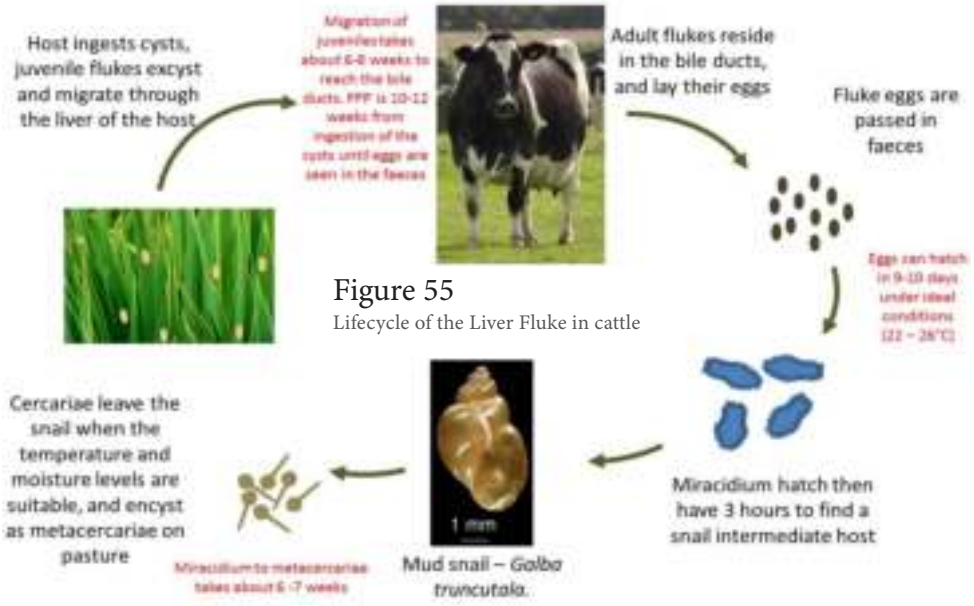


Figure 55
Lifecycle of the Liver Fluke in cattle

Pictures sourced from: https://www.msdvetmanual.com/circulatory-system/blood-parasites/trypanosomiasis#v3258185

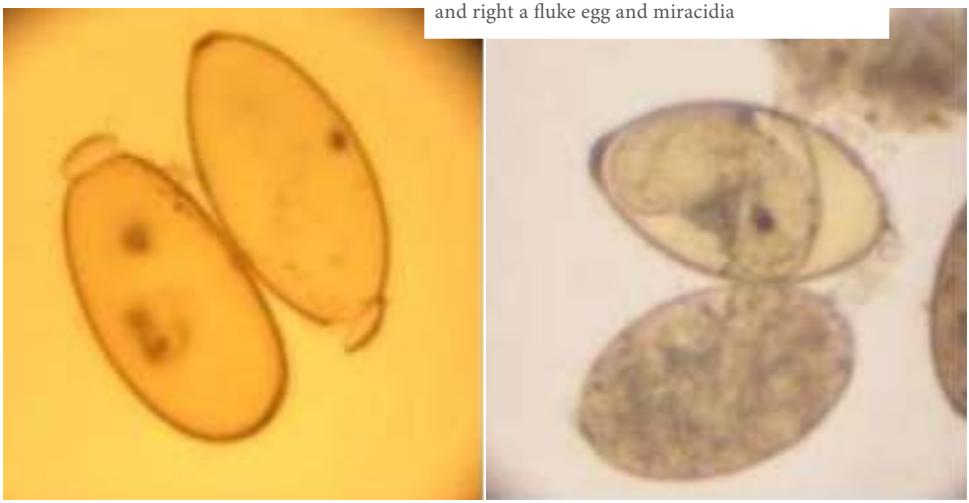
Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/

Figure 56
Liver flukes lesions seen as thickening and calcification of bile ducts of cattle liver (Pipe Stem Liver)

Figure 57

Fluke eggs under the microscope (Left hatched) and right a fluke egg and miracidia



Fluke eggs are thin-shelled, oval, operculated and have a brown-colour.

Pictures sourced from: https://projectblue.blob.core.windows.net/media/Default/Beef%20&%20Lamb/CHAWG/From%202013/Liver-fluke-in-cattle-costs-and-control.pdf

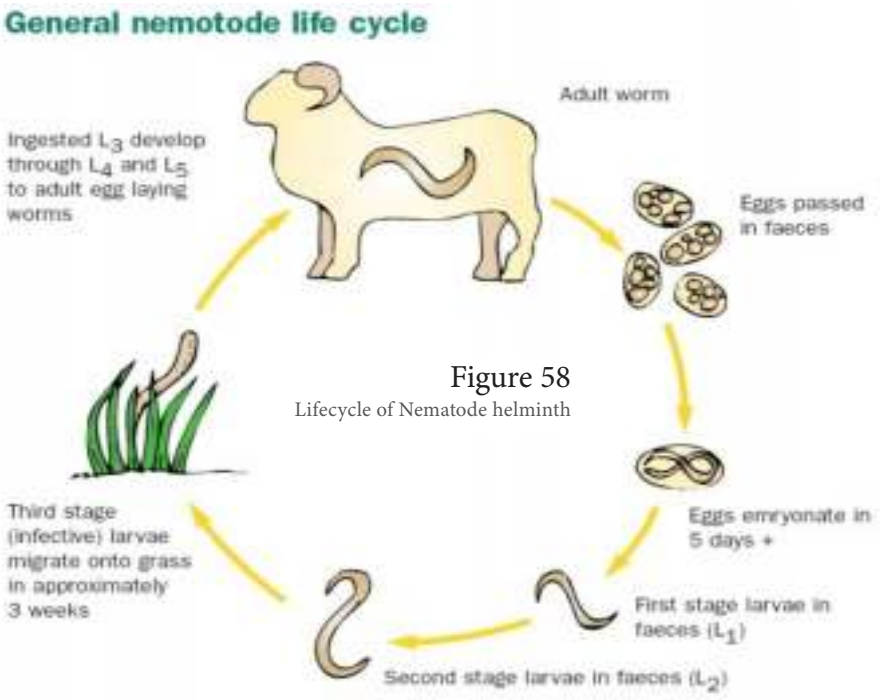


Figure 58
Lifecycle of Nematode helminth

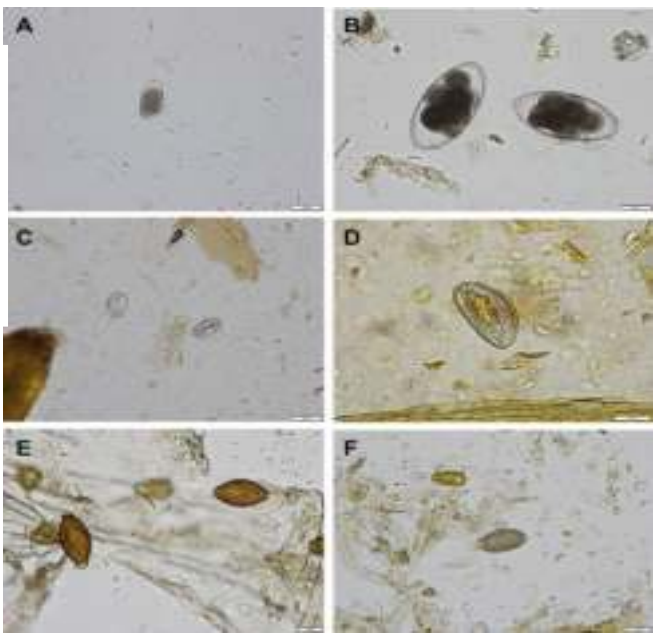


Figure 59
Eggs of gastrointestinal nematodes of livestock that are commonly seen in fecal samples. (A) Strongyle-type egg. (B) Nematodirus sp eggs. (C) Strongyloides sp eggs. (D) Skrjabinema sp egg. (E) Trichuris sp eggs. (F) Capillarid-type egg.

Pictures sourced from: Verocai, Guilherme & Chaudhry, Umer. (2020). Diagnostic Methods for Detecting Internal Parasites of Livestock. https://www.researchgate.net/publication/339055281_Diagnostic_Methods_for_Detecting_Internal_Parasites_of_Livestock

4.15 Mange in Cattle – LOTOME / EKOIKOI / LOIR

Other names: Cutaneous Acariasis, Mite infestation

Epidemiology

- Cattle are affected by 5 types of mite species.
- Clinical signs depend on the type of mite biology and ecology, for example, whether the mite burrows into the skin or whether it inhabits the hair follicles or sebaceous glands.
- Sarcoptic mange, or scabies is caused by Sarcoptes scabiei var. bovis, a skin-burrowing mite. The mite is highly contagious and zoonotic. It causes intense pruritus and papules. If left untreated the skin thickens, forming large folds, and the entire outer body surface can be affected in a few weeks.
- Psoroptes ovis is a non-burrowing mite causing psoroptic mange. The mite pierces the skin to imbibe the fluids emanating from the wound. Infection is characterised by the formation of thick crust, exudative dermatitis, alopecia, and intense pruritus.
- Chorioptes bovis, the chorioptic mange mite, typically inhabits the skin surface of the tail and lower legs and therefore the condition may be called tail, foot, or leg mange. Although C bovis can survive off the host for up to 3 weeks, chorioptic mange is relatively less pathogenic to cattle than sarcoptic or psoroptic mange.
- Demodex bovis is the most common of 3 Demodex species that can infest the hair follicles and sebaceous glands of cattle where they feed on sebum, oozing plasma, and epidermal debris. Skin lesions are susceptible to secondary bacterial infection. Young cattle appear to be more susceptible to heavy infestations, which can also result in hide damage.
- The cattle itch mite, Psorobia bos, can cause psorergatic mange of limited pathogenicity. Cases involve slight skin thickening and some scaling where low-grade pruritus can occur.
- Cattle are also susceptible to infestation by the ear mites Raillietia auris and Raillietia flechtmanni. In addition to the subclinical otitis, ear mite infestation also results in accumulation of wax.

Diagnosis

Field based diagnosis

- Skin scraping as described in chapter one
- Clinical signs can be used to make a field based presumptive diagnosis.

Treatment and control

- Treatment of mange is achieved efficiently with macrocyclic lactones (Ivermectin at a dose rate of 1 mL per 50 kg bodyweight repeated 2 weeks apart) and use of acaricides like Amitraz or synthetic pyrethroids for topical application for chorioptic and sarcoptic mange.
- Control is achieved by isolating infested animals and treating them immediately the skin lesions are seen.

Zoonotic potential

- Yes, for Sarcoptic mange

Notifiable disease

- Yes, for Sarcoptic and Psoroptic mange as they are WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Figure 60
Bovine sarcoptic mange (scabies)

Figure 61
Bovine sarcoptic mange (scabies)

Pictures sourced from: Dr. Raffaele Roncalli in MSD veterinary manual

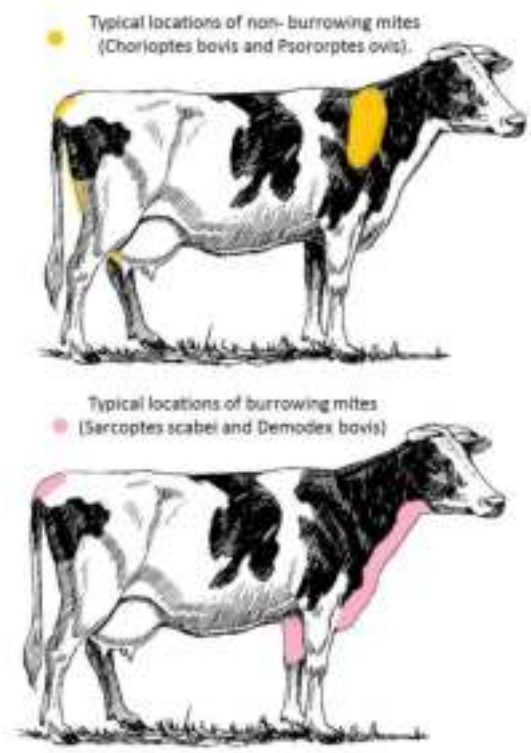


Figure 62
Skin lesion location for mites in cattle

Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/
2. WOAH (2020). Technical Disease Card https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header

Chapter 5:

Camel Diseases

Table 14: Priority camel diseases ranked in order of importance by livestock keepers in Turkana county

Conventional name	Turkana name
Trypanosomiasis	EKWAKOIT / LOTOROBUO /LOTEGERIN
Haemorrhagic Septicaemia (HS)	LOOKOT / CHOLERA
Mange	EMITINA / EKOIKOI / LOTOME
Camel cough	LOWALA / LOTURDAI
Camel pox	ETUNE ANG'IKAALA
Caseous Lymphadenitis (CLA)	ABUS/ ABUTH
Contagious Skin Necrosis (CSN)	ANG'AJUL NG'AJEMEI
Contagious ecthyma (Orf)	NG'IBUORUOK
Helminthiasis	NG'IPELEI / NG'IRITAN
Brucellosis (see Annex 2: Zoonotic diseases)	EDEKE AKIRING KA NG'AKILE that manifests as Arthritis -LOKUDO
Mastitis	LOEKETA
NASAL BOTS - Camel botfly larvae - Cephalopina titillator	EKURUT AKOU EKAAL

Table 15: Medicinal plants and traditional practices used by livestock keepers to manage priority camel diseases in Turkana County

Ethnoveterinary Treatment Strategies	Turkana name	Target Disease
Calatropis procera-roots pounded, mixed with water given as an oral drench	ETSURO	Tick infestation
Euphorbia kalisina or uhligiana- the spines/thorns are removed and succulent leaves pounded soaked in cold water and given as an oral drench	EMUS	HS, Camel cough
Gardenia ternifolia- the seeds are ground into powder and mixed with water and given as an oral drench For nasal bots livestock keepers force the animal to inhale the ground seed powder	EKORE	HS, Camel cough, Nasal bots
Aloe turkanensis –sap from succulent leaves mixed with drinking water	ECHUCHUKA	HS, Camel cough
Sarcostemma viminale – the milk sap applied topically	ELIGOI LEAVES AND TWIGS ORAL DRENCH	Contagious skin necrosis (CSN) Tick infestation Helminthiasis
Dikdik (type of Antelope) faeces – diluted in water and given as an oral drench or pounded into fine powder and animal forced to inhale	NG'ACHIN ESURO	Trypanosomiasis, Nasal bots
Hot iron or stone cauterization	AKIMAD EMACHAR KORI AMORU	Camel cough, Haemorrhagic septicaemia
Jugular bleeding	AKIGUM NG'AKOT	Trypanosomiasis, HS

NB: Text in red highlights practices that go against animal welfare principles

Table 16: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat priority camel diseases in Turkana county

Novidium® (Homidium chloride)	EDAWA LOARENG'AN	Haemorrhagic septicaemia Camel cough
Ivermectin	EDAWA ALOTOME / EDAWA LOKIRION/ EDAWA LOMOO	Mange Camel pox Nasal bots
Oxytetracycline	EDAWA LOMUNG' EDAWA ALOUKOI	Haemorrhagic septicaemia Camel cough
Penstrep - (Procaine Penicillin G and Dihydrostreptomycin)	EDAWA LOKAKUON LO AKWAAN	Haemorrhagic septicaemia Camel cough Camel pox
Acaricide	ADIP NA NG'IGURAI ADIP	Tick infestation
Tylosin	EDAWA LA NG'IUKOI	Haemorrhagic septicaemia Camel cough
Triquin	EDAWA LAKWAAN LA ANG'IKAALA	Trypanosomiasis Haemorrhagic septicaemia
Dawatrim® bolus- Trimethoprim and Sulfadiazine	EDAWA LOEREMONU	Haemorrhagic septicaemia

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

Camel diseases:

Protozoal

5.1 Trypanosomiasis – EKWAKOIT / LOTOROBOU / LOTEGERIN

Other names: Surra

Case definition

- Camel trypanosomosis (Surra) is caused by a single celled (unicellular) protozoan parasite that is transmitted by biting flies.
- Trypanosomiasis is the most important disease in camel productions systems in the world.

Distribution (Epidemiology)

- Most camels live outside Africa's tsetse belt meaning that Tsetse flies (Glossina species) are not involved in transmission of the disease. Other haematophagus flies like Tabanus, Stomoxys, Haematopota, Chrysops and Lyperosia transmit the disease through mechanical transmissions.
- Due to the wide geographic range of the haematophagus flies the disease is endemic in Africa, Asia, South and central America.

Causative agent/ Aetiology

- Trypanosome evansi, types A and B,
- T. evansi is morphologically related to T. equiperdum—a derivative of T. brucei.
- During T. evansi evolution, it lost the mitochondrial (kinetoplast) DNA making it unable to infect invertebrate vector. This means it cannot establish life-stages in the Glossina fly (Tsetse fly) like other Trypanosome species.
- T. evansi has a wide host range. Surra affects mainly camels and horses but buffaloes and cattle are also affected. Other species that develop severe disease include donkeys, mules, deer, llamas, dogs, cats, cattle and buffalo. Sheep, goats, pigs and elephants may occasional develop mild or chronic disease.
- In Pakistan, it has been found to be the most prevalent trypanosome species in donkeys.

Mode of transmission and Pathophysiology

- Trypanosoma evansi is transmitted mechanically by haematophagus biting flies.
- No developmental stage in a vector has been demonstrated which differentiates the parasite from T. brucei.
- Tabanids/Tabanus (horsefly) play the major role in transmission. The flies establish a new infection through trypanosome-contaminated mouthparts. Trypanosomes remains infective on the proboscis for a short period only.
- Cattle, sheep, goats and antelopes often carry the parasite subclinically, acting as asymptomatic reservoirs.
- Transmission can also be transplacental resulting in disease of the foetus.
- Vampire bats in South and Central America are hosts, reservoirs and vectors of T. evansi; they transmit T. evansi mechanically in their saliva, and may develop high parasitaemia which may kill the bat. Recovered bats serve as carriers.
- Carnivores may become infected after ingesting infected meat.
- Transmission in milk and during coitus has been documented.
- Numerous environmental and host factors influence the course of the disease, such as presence of other infections, nutritional status, age, pregnancy, previous exposure or immunosuppression and stress.
- The presence of the parasite in circulation

results in chemical and morphological changes in the surface of RBC resulting in increased clearance of RBC by the body that exceeds the replenishment of RBC by the bone marrow hence the severe anaemia clinical sign observed.
- Infected animals' mucous membranes are pale and the packed cell volume (PCV) drops to below 25% (v/v) and can be as low as 10% (v/v).
- The herders may notice a characteristic odour of the camel's urine and identify infected animals by this sign alone. The odour of the urine may be due to ketone bodies, which are often elevated.
- in trypanosome-infected camels, abortion in all stages of pregnancy is common. If the foetus is full term it may be born alive but weak with parasitaemia and will die within two weeks of birth.
- In endemic areas, camel herds eventually reach an endemic stable state where some animals carry the trypanosome parasite for years while others never do. Within a herd there are all forms and stages of surra from new infections to subclinical and chronic conditions. The course of the disease varies widely. In Kenya, a small percentage of animals die within 2 to 5 months of contracting the disease. Some live for up to four years with subclinical infections and some eventually self-cure.

Affected group
- Camels of any age can become infected. However, high incidence of infections is seen in young camels shortly after weaning.

Clinical signs
- In the initial phase of the disease there is fever, lacrimation, shivering, reduced appetite and mild diarrhoea.
- The animal then develops progressive anaemia and has fluctuating body temperature with initial peaks of fever of up to 41 °C and after a few weeks as the animal enters chronic phase the temperature returns to normal.
- The animal's appetite is minimally affected but the animal begins to waste despite feeding well. Wasting manifests as a drooping hump.
- Animal is unable to walk long distances and has a rough hair coat.
- Most of the clinical signs are variable but typically include weight loss, decreased stamina, oedema (base of the neck, ventrum and eyelids), anaemia, intermittent fever, poor milk production, and late term abortion or calving of premature calves.

Post mortem lesions
- Carcass is very thin (emaciated) and tissues are often pale (anaemic).
- Subcutaneous oedema of the feet, brisket, underbelly and eyelids has been reported.
- There is enlargement of the lymph nodes and spleen and pericardial effusion.

Morbidity and Mortality Rates
- Morbidity rates in camel rearing areas varies from 20–70%. Case fatality rate (CFR) in untreated camels can be as high as 100%. Surra in cattle and buffalo tends to be chronic with a much lower CFR.
- Animals subjected to stress, like malnutrition, pregnancy, and physical labour, are more susceptible to disease causing mortality of 3%.
- In Turkana county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25%.

Seasonality of occurrence
- In some countries incidence of surra increases significantly during the rainy season when biting fly populations is high
- Studies have reported varying seasonality of occurrence with some reporting outbreaks during the end of the rainy season while others reporting higher incidences during the dry season.
- In Turkana county, Camel Trypanosomiasis is endemic and occurs throughout the year with most cases reported during the long dry season AKAMU.

Diagnosis

Field based diagnosis

- Direct examination of the blood by light microscopy is a rapid diagnostic test that can be easily applied in the field. The basic technique, involves examination of fresh or stained buffy coat blood smears. Buffy coat smears are made after centrifugation of unclotted blood in a haematocrit tube-haematocrit centrifuge technique (HCT).
- The direct methods of trypanosome detection, utilise a wet blood film, Giemsa stained thick drop of blood and thin blood smears. The wet blood film is used to detect live parasite through observing parasite movement while the stained smears identify the parasite morphology and allows for parasitaemia calculation. The drawback of this method is that the animal should be in the acute phase of the disease when there is fever due to parasitaemia.
- Concentration techniques HCT is used to determine PCV and the buffy coat is Giemsa stained and is examined for the parasite. This can also be done in the field with battery-operated mini centrifuge.
- Card agglutination and Latex agglutination test are useful antibody testing that can be used in the field.

Laboratory diagnosis

- Confirmatory diagnosis is through ELISA for chronic cases that detects antibodies and is useful for large-scale surveys. Polymerase chain reaction (PCR) has also proven useful in confirming trypanosomes in infected blood or tissues.

Differential Diagnosis

- Other tsetse-transmitted trypanosomosis,
- Chronic parasitism- helminthiasis

Treatment

- Melarsomine dihydrochloride (Cymelarsan) is the latest Trypanocidal drug to be developed. It was first available for commercial use in 1992. It is used to control surra in camels via deep intramuscular injection at a dose rate of 0.25mg/kg body weight.
- The most effective drug used globally to treat Surra in camels is Quinapyramine sulphate and quinapyramine chloride (Triquin®). Triquin® is used as a curative as well as a preventive drug against T. evansi in camels. It is administered by subcutaneous injection at a dose of 8mg per kilogram body weight. Local tolerance at the injection site is low. However, the drug is quite efficient and the chemo prophylactic effect can last up to 4months. Triquin® is available in Kenya.
- Most of the drugs used to treat cattle trypanosomiasis are either not curative (homidium bromide, or Homidium Chloride=Novidium®) or are toxic at high doses for camels (diminazene aceturate = Berenil®).
- Supportive treatment with multivitamin and improving nutrition will give the animals a better chance of recovery and survival.

Prevention/control

- No vaccines are available and none are likely in the future as the trypanosome parasite is able to rapidly change its surface glycoproteins to avoid the host's immune response.
- Control measures include detection and treatment of infected animals, prophylactic treatment of susceptible animals, and protection of animals from biting flies through use of insect repellents like pour on especially during the rainy season when the biting flies' population is high.

Zoonotic potential

- None for T. evansi. Despite having a wide range of host the protozoa can only infect non-human hosts as it is susceptible to cytolysis by the trypanolytic factor that is found in normal human serum.

Notifiable disease

- No, but it is recognised by WOAH as a camel disease of economic importance.

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid grazing animals in known areas with biting flies that transmit the parasite.

Further Reading

1. D. Röttcher, D. schillinger and E. zweygarth (1987). Trypanosomiasis in the camel (Camelus dromedarius). Rev. sci. tech. Off. int. Epiz., 1987, 6 (2), 463-470. https://www.WOAH.int/doc/ged/D8495.PDF
2. World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
3. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

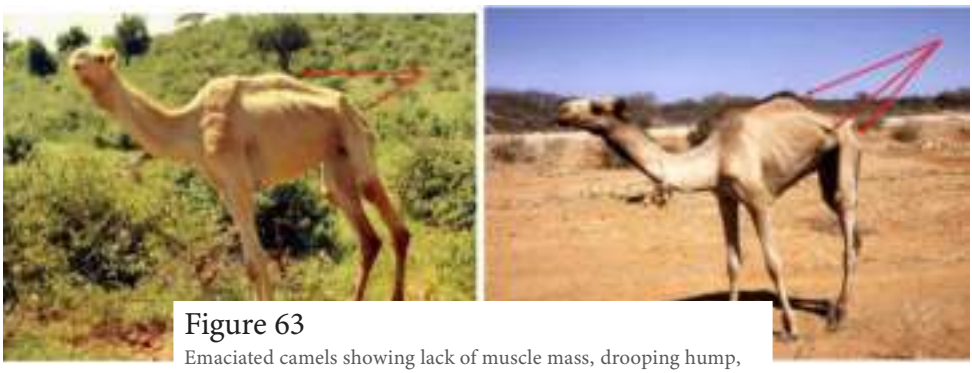


Figure 63
Emaciated camels showing lack of muscle mass, drooping hump, prominent and visible ribs, spine and hip bones (red arrows)

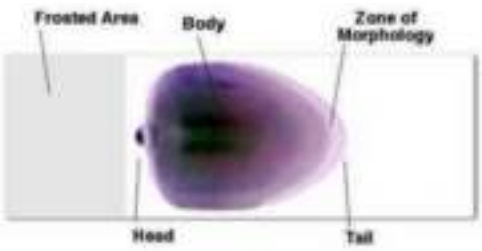


Figure 64
A good blood smear has a head, body and tail

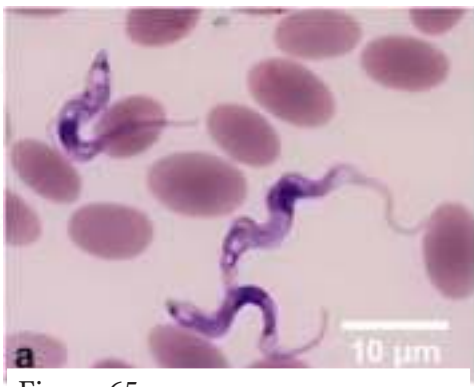


Figure 65
Giemsa-stained blood smears from camel showing Trypanosoma evansi

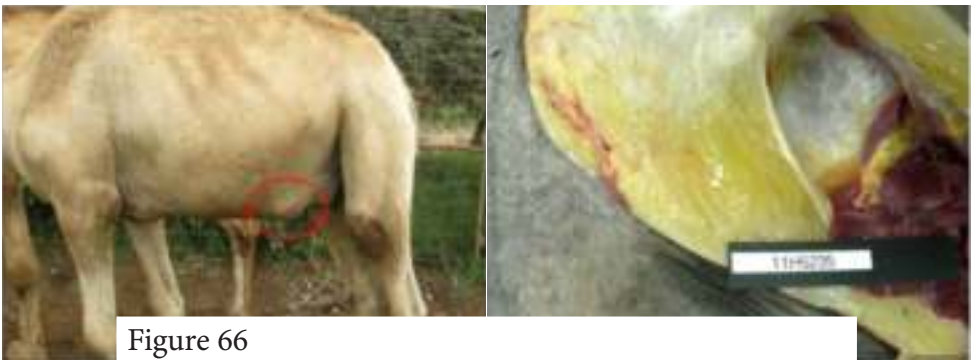


Figure 66
Ventral oedema in a camel due to Trypanosomiasis (Surra) infection and
Subcutaneous oedema (expansion of the subcutis by clear gelatinous fluid.

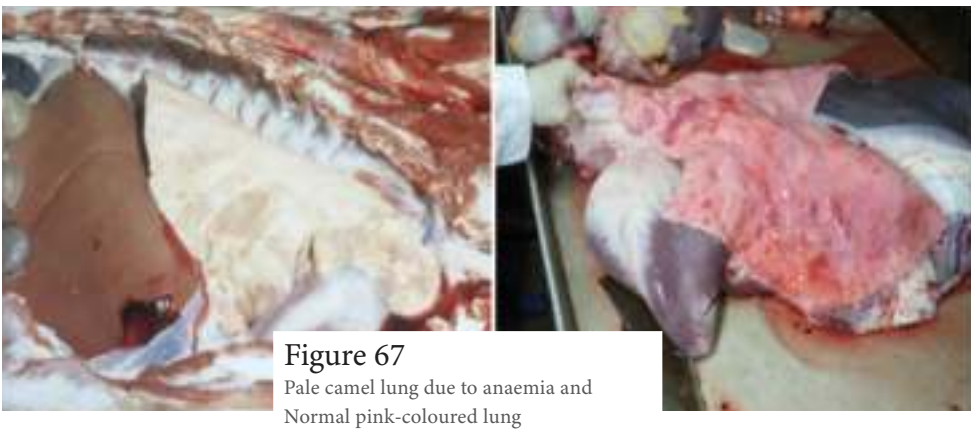


Figure 67
Pale camel lung due to anaemia and
Normal pink-coloured lung

Further Reading

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

Camel diseases:

Viral

5.2 Camel Contagious Ecthyma (CCE) -Orf – NG'IBUORUOK

Other name: Scabby mouth, Sore mouth, Contagious Pustular dermatitis or Auzdik disease

Case definition:

- Highly contagious viral dermatitis of camels characterised by pustule lesions on the lips, especially in young animals.

Aetiology and Epidemiology:

- CCE is caused by Parapoxvirus (PPV) which is a large DNA virus of the family Poxviridae. Other members of the genus PPV are Orf virus (ORFV) that affects sheep and goats, Pseudo cow pox virus (PCPV), Bovine papular stomatitis virus (BPSV) and PPV of red deer in New Zealand (PVNZ).
- The disease has a worldwide distribution
- The disease is endemic in most camel keeping countries with variations in intensity of infection, morbidity and mortality rates.
- Orf outbreaks are most common during the rainy season and affects mostly young animals. The age group at risk are those less than one year of age. Cases have been reported in one month-old camel calves.
- The major factors associated with increased likelihood of CCE occurrence are season of the year (rains), camel age (calves more susceptible), camel movements and their association with thorny trees.
- In most cases, the disease causes no mortality, but when camel calves are severely affected the pox-lesions interfere with the calves' ability to suckle or graze. Lesions can extend to eyelids leading to blindness and death.

Zoonotic potential

- Yes, transmission is through direct contact with infected animals or indirect through fomites.
- Lesions in man appear as solitary or several skin lesions that appear commonly on the fingers, hands, or forearms. The lesions start as small, firm, papules, that proliferate and progress to a severe ulcerative, pustular dermatitis. The clinical manifestation of the disease and a personal history of contact with an infected animal are sufficient to diagnose orf virus in man.

Morbidity and Mortality rate

- Morbidity rate is about 60-100% while the mortality rate is 5-20%.
- The camel calves die due to an inability to graze and suckle their dam.
- In Turkana county livestock keepers in the year 2022 estimated the annual apparent morbidity rate of 6% with no mortality or case fatality rates.

Seasonal Occurrence

- In Turkana county, CCE is endemic and occurs throughout the year, outbreaks are common during the long rainy season (AKIPORO).

Clinical signs

- Lesions first appear on the lips of affected animals as small papules that progressively develops into scabs on the lips, muzzle, nares and eyelids that later coalesce into fissured crusts on the lips. Swelling of the head and sometimes the neck is also reported.
- Superficial lymph nodes of the head region such as parotid, submaxillary, and cervical lymph nodes in severe disease can become enlarged.

Post mortem lesions

- The papular lesions and scabs are found around mouth, nostrils and on eyelids.
- Intense pruritus results in haemorrhages and extensive skin excoriation.
- There is enlargement of superficial parotid, submaxillary and cervical lymph nodes.

Diagnosis

- Clinical signs of papular lesions present around the mouth and nostrils in young camels.
- The typical parapoxvirus particles are diagnosed in scabs of camel skin using electron microscopy.
- Different serological tests are available with variable results.

Differential diagnosis

- Camel pox
- Mange
- Fungal infections

Treatment

- No treatment since it is viral.
- Local application of antiseptic solution such as povidone iodine or iodine ointment on the affected areas is recommended.
- Use of Gentian violet spray with oxytetracycline antibiotic also assists in drying lesions and prevents local secondary bacterial infection
- Antibiotic therapy is useful if the lesions become generalised and secondary infection has set in. Long acting oxytetracycline (20 to 30%) at dosage rate 1mL per 10 kilograms can be administered.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.

Prevention and control:

- Affected animals must be segregated to stop the spread of the disease in the herd.
- Any sheep or goats kept with camels should be vaccinated against ORF. A vaccine is available for sheep and goats and can be used safely in camels.
- A recombinant vaccine is under research as a bivalent vaccine for both contagious ecthyma and camel pox.

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid grazing animals in known areas with biting flies that transmit the parasite.
- Isolate sick animals by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

Further reading

Khalafalla, A.I., El-Sabagh, I.M., Al-Busada, K.A. et al. Phylogenetic analysis of eight sudanese camel contagious ecthyma viruses based on B2L gene sequence. Virol J 12, 124 (2015). https://doi.org/10.1186/s12985-015-0348-7

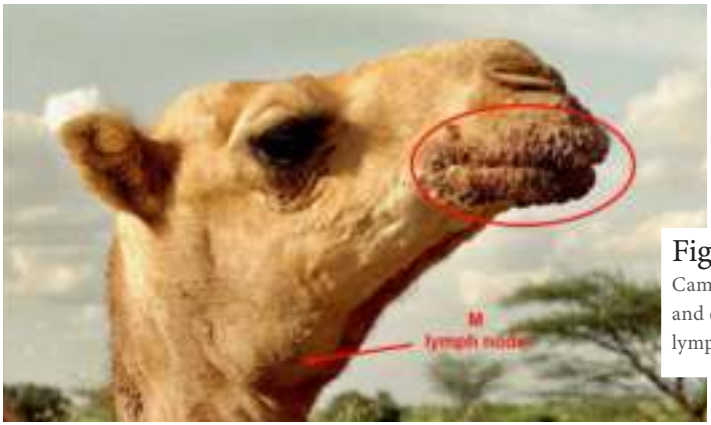


Figure 68

Camel orf oral lesions (red oval) and enlarged sub-mandibular lymph node (red arrow)

Picture source: Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21.
https://lib.dr.iastate.edu/vdpam_reports/21

Figure 69

Camel orf lesions that have coalesced into scabs, photos courtesy of Morris Ogom and Diba Denge Animal Health Staff, Marsabit County

5.3 Camel Pox- ETUNE ANG'IKAALA

Other name: Scabby mouth, Sore mouth, Contagious Pustular dermatitis or Auzdik disease

Case definition:

- Camel pox is a highly contagious viral skin disease of camels characterised by generalised pustule formation. Camel pox is an WOAH notifiable skin disease.

Causative agent

- Camel pox virus (CMLV)- CMLV genus Orthopoxvirus (OPV), is genetically similar to variola virus (VARV), the causative agent of smallpox in humans.
- Orthopox viruses are exclusively specie specific, CMLV infects only camels and variola only humans.

Zoonotic potential

- Yes, CMLV infection has been documented in camel handlers.

Epidemiology

- Disease occurs frequently during the rainy season due to increased population of suspected arthropod vectors - biting flies.
- Disease occurrence is restricted to camels and is enzootic in almost every region, where camels are reared except in Australia.
- Mortality in adult animal's ranges from 10 to 28 % and in young animals (< 4 years of age) from 25–100 %. Mortality is also influenced by presence of other immune suppressing diseases like trypanosomiasis and helminthiasis.
- In Turkana County, livestock keepers in the year 2022 estimate the apparent morbidity rate to be 2%, with no mortality or case fatality rates. The disease occurred in Turkana throughout the year with no seasonal variation.

Transmission

- CMLV is transmitted by either direct or indirect contact. Direct transmission is either by inhalation of infected nasal or oral secretion and through skin abrasion or mechanical transmission by arthropods like the camel tick Hyalomma dromedarii.
- Indirect transmission is through contact with contaminated fomites. Affected camels contaminate the environment with shed scab materials and secretions saliva, ocular and nasal discharges.
- Predisposing factors- Presence of chronically infected animals in close proximity with immune naïve animals is an important predisposing factor.
- Dried scabs shed from pox lesions contain live virus for at least 4 months. Poor nutrition and concurrent parasitic and bacterial infections often worsen the clinical disease. Animal congregating in markets, watering and grazing areas are important sources of infections.

Clinical signs

- Camel pox commonly occurs in naïve populations (herds that have not experienced an outbreak). The disease is more severe in younger animals (< 4 years).
- Fever and enlarged lymph nodes.
- Skin lesions that include vesicles or pustules with crusts that start at the head region before spreading to the neck, limbs, genitalia,

mammary glands and perineum.
- Excessive lacrimation, muco-purulent nasal discharge and abortion in pregnant camels.
- Animals may also have diarrhoea.
- Skin lesions may take up to 4–6 weeks to heal. In the generalised systemic form of the disease, pox lesions can be found in the mucous membranes of the mouth and serosal surfaces of internal organs.

Differential Diagnosis
- Contagious ecthyma (ORF)- Camel parapox virus (CPPV), Papillomatosis (wart) - Camelus dromedary papilloma virus (CdPV) and Urticaria-skin reaction to insect bites.
- Difference between camel orf and camel pox is the type distribution of lesions, in orf lesions coalesce and form crusts and are mainly located on the lips and nostrils and often cause oedema and enlargement of regional lymph nodes due to secondary bacterial infection. Camel pox lesions are mainly discrete pustular lesions that don't coalesce and occur all over the body of the animal, they are generalised. Orf is also more common in calves while camel pox occurs in all age groups.
- Warts are small, cauliflower-like lesions also occurring on the lips but can be generalised in some animals. The wart lesions are not covered by surface crusts, do not coalesce and are not associated with oedema or regional lymph node enlargement.

Post mortem lesions
- In the generalised form, the animal's external skin and internal organs have pox lesions.

Diagnosis
- Clinical and post-mortem lesions of pox like lesions are used to make a tentative field based diagnosis.

Sample collection and Confirmatory Laboratory diagnosis
- During the viraemic stage of the disease (within the first week of the occurrence of clinical signs) camel pox virus can be isolated in cell culture from blood samples.
- PCR for viral DNA and EDTA for antibodies or antigen identification.

Treatment and Control
- Viral disease there is no specific treatment
- Animals that develop secondary infection characterised by a fever or unthriftiness after 1 week should be treated with one dose of long acting oxytetracycline at dose rate of 10 mg/kg body weight IM. Multivitamin therapy can also boost the animal's immunity.
- Quarantine herds.
- Life-long immunity follows after natural infection hence there may be no need to vaccinate affected herds.
- There is commercial cell attenuated camel pox vaccine using isolates from UAE (strain CaPV298-2). The vaccine protects animal for up to 6 years. However, a booster vaccination is recommended in young animals vaccinated below 6 months as there will be maternal antibody interference.

Ethnoveterinary treatment practices
- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Avoid grazing animals in known areas with biting flies that transmit the parasite.
- Isolate sick herds by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

Further Reading

1. Vinayagamurthy Balamurugan, Gnanavel Venkatesan, Veerakyathappa Bhanuprakash and Raj Kumar Singh (2013). Camel pox, an emerging orthopox viral disease. Indian J. Virol. 24(3):295–305. https://www.ncbi.nlm.nih.gov/pubmed/24426291
2. World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
3. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

Figure 70
Generalised camel pox virus lesions (Black arrows)

Picture source: Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

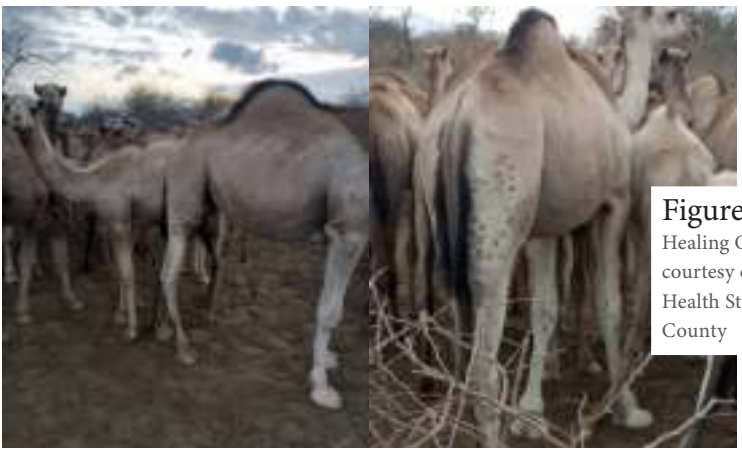
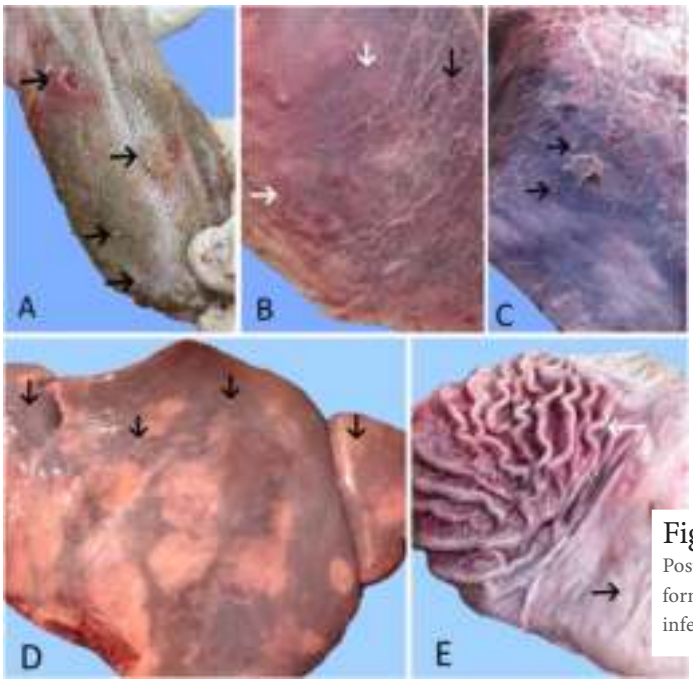


Figure 71
Healing Camel pox lesions, photo courtesy of Diba Denge, Animal Health Staff Saku, Marsabit County



A. Multiple erosive, ulcerative and vesicular lesions (arrow) in the mucosa of tongue.
B. Multiple pox lesions with haemorrhagic (white arrow) and ulcerated (black arrow) areas on lung surface
C. Multiple raised pox lesions (arrow) on lung surface.
D. Liver showing minute pox lesions (arrow) and pale areas of necrosis.
E. Abomasum showing thickened congested mucosa (white arrow) with ulcerations and pox lesions (black arrow).

Figure 72
Post mortem lesions of systemic form of camel pox in naturally infected animal

Picture source: https://www.sciencedirect.com/science/article/pii/S2405844021002917

Figure 73
Skin lesions in human due to zoonotic transmission of camel pox virus.

Photo courtesy of Thomas Lenyakopiro, CDR, Samburu County

5.4 Camel cough- LOWALA / LOTURDAI

Other names: Acute Respiratory Disease Syndrome (ARDS), Camel parainfluenza, Camel Cough, Camel Flu

Case definition

- Described as a severe respiratory infection of camels characterised by fever, cough, initial serous nasal discharge that becomes mucopurulent, laboured breathing and terminal progression to sternal recumbency with extension of the neck before death.
- The disease affects all ages of camels but weaner calves between the ages of 1 to 2 years are the most severely affected.

Distribution (Epidemiology)

- The disease has been described in all camel keeping countries globally.
- In Kenya, the most recent outbreak occurred from June 2020 and was reported in most camel keeping counties that includes Mandera, Wajir, Marsabit, Isiolo, Samburu, Turkana and Garissa.
- During the recent 2020 outbreak, camel owners reported that they had experienced a similar disease in the past which they called 'Camel flu' that presents with milder symptoms. However, the current outbreak was presenting with more severe symptoms and had a longer course duration compared to the seasonal camel flu known to many of them.
- ARDS begun during the long rainy season (April -June) with only a few cases reported. However, the long dry season (July – September) resulted in explosive outbreaks with daily reports of camel deaths in all Northern pastoralist counties of Kenya.
- There were no reports of animals having been moved to new area prior to the outbreak.

Causative agent/ Aetiology

- The definitive causative agent for acute respiratory diseases has not been conclusively determined. It is hypothesised that the disease starts as a parainfluenza 3 infection that becomes complicated by secondary bacterial infection.
- A variety of viral, fungal, bacterial and parasitic microorganisms have been associated with outbreaks of respiratory disease among camels.
- The causative agents identified are a normal flora in the upper respiratory tract of camels. The disease requires predisposing circumstances that cause the normal flora to invade respiratory tissues and cause serious disease.
- A recent 2020 metagenomic sequencing analysis of nasopharyngeal swabs sampled from 108 MERS CoV-positive dromedary camels in UAE identified other co-infecting viruses from 10 viral families including Coronaviridae, Nairoviridae, Paramyxoviridae, Parvoviridae, Polyomaviridae, Papillomaviridae, Astroviridae, Picornaviridae, Poxviridae, and Genomoviridae.
- A similar study in Sudan that analysed lung tissue identified parainfluenza virus 3 (PIV3), influenza viruses A, B and D, adenovirus, respiratory syncytial virus (RSV), and infectious bovine rhinotracheitis (IBR).
- The most common pathogenic bacteria isolated during outbreaks include; Staphylococcus aureus, Corynebacterium pyogenes, Streptococcus pyogenes, Escherichia coli, Klebsiella pneumonia, Pseudomonas

aeruginosa, Trueperella pyogenes (formerly Arcanobacterium pyogenes), Mannheimia haemolytica and Pasteurella multocida.

Mode of transmission and pathophysiology and affected group

- Direct transmission through inhalation of respiratory droplets especially due to the cough and indirect transmission through contaminated fomites.
- Studies have shown that the most commonly reported risk factors for viral or bacterial pneumonia are age and season. The highest incidence of pneumonia is reported during the cold season in all camel ages. Proliferative bronchopneumonia and pleuropneumonia are more frequent in older camels (about 10 years of age) while interstitial pneumonia and lung abscesses are more frequently seen in younger camels (6 months to 4 years of age).

Clinical signs

- The first clinical symptom observed is fever which is characterised by the animal isolating itself from the rest of the herd.
- After 2 days the animal develops a cough and serous nasal discharge that lasts for a week after which the nasal discharge becomes mucopurulent. In the second week following onset of clinical symptoms the affected animals develop a more severe cough, difficulty in breathing that forces them to extend the neck and become recumbent. If there is no treatment in the first 2 to 3 days after onset of symptoms the animals dies within 7 days after onset of symptoms especially in camels that are under 2 years.
- Recovery for those that are older than 3 years of age occurs with or without treatment and it takes 2-3 weeks from onset of clinical symptoms to recovery.
- The Kenya 2020 outbreaks estimated an apparent morbidity rate of 35%, mortality rate of 1% and case fatality of 2%.
- In Turkana county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 11%, mortality rate of 6% and a case fatality of 55%.
- The disease in Turkana county occurs throughout the year but epizootic outbreaks occur during the short dry season (AIT/NAIT).

Post mortem lesions

- The gross lesions observed during the Kenyan 2020 outbreak included hydro-peritoneum with about 60 mls of fluid. Hydrothorax with about 30mls of thoracic fluid.
- Fibrinous adhesion of the lung to the thorax. The right lung was severely congested with fibrinous bronchopneumonia appearing as red and grey hepatisation. The lung was marbled in appearance and the cut section oozed froth and purulent exudates with the lower section showing varying atelectasis.
- Thick mucoid exudates were present in the trachea and bronchial lumen. There were no gross lesions on the liver, kidney and spleen.

Diagnosis

- Samples that yield bacterial isolates are nasal, nasopharyngeal, tracheal swabs and lung tissues.
- Samples that yielded viral agents are nasal swabs and lung tissues.
- Bacterial isolation using routine culture methods with different media such as nutrient agar, blood agar, brain heart infusion, mannitol salt agar, MacConkey agar and brilliant green agar followed by identification using morphological and biochemical characteristics of the isolated strains such as colony morphology, Gram staining, spore forming ability and acid-fast staining. ELISA or PCR can be used for virus identification.
- During the 2020 outbreak, 99 nasal swabs from camels in Marsabit county submitted for bacterial culture and sensitivity yielded eight (8) genera of bacterial species that were identified on the basis of their phenotypic and biochemical properties. The isolated bacteria included Pseudomonas aeruginosa 20.2% (20/99), Klebsiella pneumoniae 19.2% (19/99); Staphylococcus aureus 15.2% (15/99); Bacillus

and Escherichia coli each with a 13.1% (13/99); Streptococcus 8% (8/99); Proteus mirabilis 6.1% (6/99) and Serratia 5.1% (5/99).

Treatment

- Antimicrobial susceptibility testing (AST) is necessary for
- All antibiotic drugs use in camels is extra-label meaning that the drug manufacturer has not indicated for camel use.
- Antibiotic treatment needs to be given in the early phase of the disease for them to be effective. Ideally before the laboured breathing sets in. Treatment is futile if given when animal has become recumbent.
- In severe cases dexamethasone can be used to reduce inflammatory accumulation of fluid in the respiratory tract. Unlike in cattle where pregnancy is supported by placenta hence can result in abortion when dexamethasone is used. Camels pregnancy is supported by the ovarian located Corpus luteum (CL). CL is not affected by dexamethasone administration.
- The 2020 Marsabit outbreak investigation revealed that bacterial isolates were sensitive to most antibiotics available in the market such as oxytetracycline, Penicillin and Streptomycin (Penstrep®), Sulphamethoxazole and Trimethoprim (Intertrim®), and Amoxycillin (Betamox®).

The dosage regimes are given in the table below.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting In severe cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM for 3 to 5 days.
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM neck muscles q48 hours PRD
Amoxycillin 150 mg Long acting	Broad-spectrum antibiotic with bactericidal action	1 ml per 10 kg BWT deep IM neck muscles q 48 hours.
Sulfamethoxazole 200 mg Trimethoprim40 mg	Broad-spectrum antibiotic with bactericidal action	Twice daily 1 ml per 10 - 20 kg BWT for 3 - 5 days. IM neck muscles
Enrofloxacin 100 mg	Broad-spectrum antibiotic with bactericidal action	Single-Dose by SC only 7.5-12.5 mg/ kg of BWT (10 to 20 ML) SC
Multivitamin	Boost immunity	Adult Camel: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

** Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q and mL- Milliliter, body weight-BWT , kg- kilograms, mg- milligram. **By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC). NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage*

Prevention/control

- There are no vaccines available in Kenya to prevent camel respiratory conditions.
- Vaccination against Pasteurellosis is routinely practiced in the Middle East. There are 3 vaccine preparations in the market; Dense bacterins combined with either alum adjuvant or oil adjuvant, and Formalin-Inactivated bacterins. The oil adjuvant bacterin provides protection for up to one year and the alum bacterin for 4–6 months.
- Maternal antibodies interfere with vaccine efficacy in calves, calves should therefore be vaccinated after 6 months.
- Ensure animals have a good plane of nutrition and internal and external parasites have been controlled. Camels in good body condition and are generally healthy are more resistant to pneumonia incidences due to their strong immunity.

Zoonotic potential - No

Notifiable disease- No

Ethnoveterinary treatment practices
Refer to tables 15 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect name)

- Isolate sick herds by ensuring they do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

Figure 74
Sternal Recumbency and nasal discharge in a camel affected by ARDS in Samburu County, April 2020.

Picture source:
Emmanuel Lesiantam,
Animal Health Assistant,
South Horr, Samburu County

Figure 75
Open mouth breathing (Left)and frothy nasal discharge with swollen submandibular lymph nodes (Right)

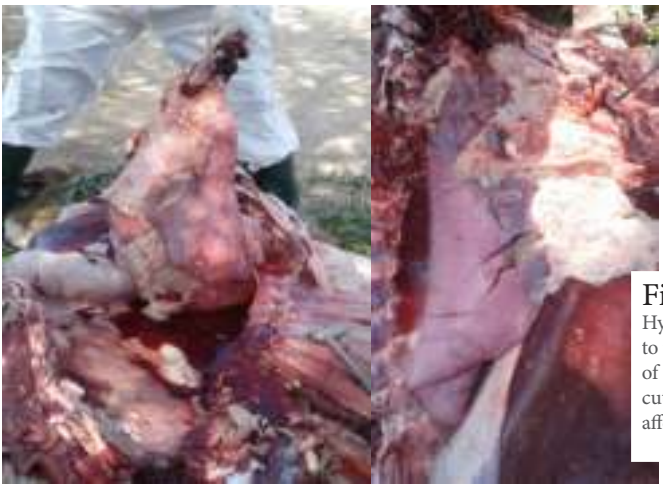


Figure 76
Hydrothorax and lung adhesions to the rib cage (Left) and exudate of mucopurulent discharge from cut lung surface (Right) in a camel affected by ARDS.

Picture source:
Emmanuel Lesiantam, Animal Health Assistant, South Horr, Samburu County

Camel diseases:

Bacterial

5.5 Haemorrhagic Septicaemia (HS)- LOOKOT / CHOLERA

Case definition

- Pasteurellosis / Haemorrhagic septicaemia is an acute fatal respiratory bacterial disease of camels caused by Pasteurella multocida and Mannheimia haemolytica.
- Disease is characterised by fever, oedema of the throat region, dyspnoea, and sudden death.
- HS has a complex pathogenesis linked to predisposing factors that causes the two bacteria that are normal flora of the upper respiratory tract to become pathogenic organisms.

Epidemiology

- Pasteurellosis occurs in camels throughout Sub-Saharan Africa, Egypt, Saudi Arabia, India, Iran, Iraq, and Russia.
- Outbreaks of HS like disease that caused large mortalities in camels (Camelus dromedarius) in Asia and in Africa have been reported since 1890.

Causative agent/ Aetiology

- HS is caused by two gram-negative coccobacillus bacteria namely Pasteurella multocida and Mannheimia haemolytica. The two bacteria have several serotypes that cause disease in camels
- HS has a complex aetiology and recent PCR studies have revealed the serotypes involved are:

- P. multocida type A, B and E (capsular and non-capsular strains)
- Mannheimia haemolytica
-M. granulomatis (new species identified)

Mode of transmission and Pathophysiology

- The disease occurs when the resistance of the body is lowered by environmental influences such as trekking or transportation over long distances, deficiencies of dietary vitamins and minerals, heavy parasitic infestation (Trypanosomiasis) and sudden changes in weather.
- The role of Parainfluenza-3 virus is not clear, however, coinfection of HS and PI-3 virus has been reported in camels.
- P. multocida is transmitted by direct contact with infected animals and indirectly through contact with contaminated fomites
- Incubation period varies from 2 to 5 days depending upon the virulence of the organism and the degree of immunosuppression of the camel. The organism enters into the blood stream through tonsils and proliferates rapidly. It causes a septicaemia in animals within 10-24 hours, leading to high temperature, swelling of the throat region, pulmonary oedema, fibrinous pneumonia, diarrhoea, and prostration. Affected camels rarely recover, and death usually occurs within 2-3 days.
- Biting arthropods are not significant vectors for transmission.

Affected group
- All age groups are equally affected

Clinical signs
- Clinical signs include increased rectal temperature (40°C), pulse, and respiration rate, dyspnoea, dullness, depression, and abdominal pain associated with haemorrhagic enteritis. There is subcutaneous swelling of the neck and submandibular space.
- Mandibular and cervical lymph nodes are enlarged and painful.
- Severe dyspnoea is characterised by camels showing dilated nostrils and open-mouthed breathing.
- Some cases, have tar coloured faeces (melena), abdominal pain, and coffee coloured urine.
- Prognosis is guarded and death occurs within 2-3 days. Both recovered and sick animals discharge the organism through excretions and secretions of the body.

Post mortem lesions
- Camels that succumb to infections have gross lesions indicating of septicaemia
- All organs are congested and serosa surfaces have petechial haemorrhages throughout the body.
- There is excess serosanguineous fluid present in the thoracic (hydrothorax) and abdominal (hydroperitoneum) cavities
- The trachea is filled with froth and there is occlusion of nasal passage with thick sticky mucous
- HS bacteria, causes bronchopneumonia and the lung cut surface oozes exudate. The lung tissue does not collapse when cavity is open

Morbidity and Mortality Rates
- Morbidity is low, but mortality can be as high as 80%. Morbidity depends on immunity and environmental conditions, including both weather and husbandry practices; morbidity is higher when animals are herded closely, are in poor condition, or exposed to wet conditions.
- Mortality is nearly 100% unless the animal is treated very early on in course of the disease
- In Turkana county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 44%, mortality rate of 40% and case fatality rate is 90%.

Seasonality of occurrence
- HS is most common during the rainy season or long dry season when animals are in nutritional stress
- In Turkana county, livestock keepers indicated that HS occurred during AKICHERES season of the year when there are light rain showers.

Diagnosis
- Tentative field based diagnosis using clinical signs and post-mortem lesions.
- Confirmatory diagnosis is through demonstrating the organisms-in live animals the organism may be difficult to culture and identify from venous puncture blood samples in addition, nasal swabs of sick animals also yield inconsistent culture results.
- Samples for isolation should be collected immediately after death (less than 4 hours) and include; blood from the heart, nasal and tracheal swabs and tissue impression smears of the spleen.
- In animals that died a long time ago which is often the case in pastoralist setting. The femur and ulna long bones can be collected for culture of the bone marrow.
- Diagnosis is through bacterial culture and identification of the organism through biochemical sugar tests.
- ELISA and PCR molecular tests can also be used on blood, nasal swabs and tissue samples.

Differential Diagnosis (DDx)

- Other Bacterial and Viral Pneumonias
- Anthrax or Clostridial infections in peracute cases that causes sudden death

Treatment

- Antimicrobial susceptibility testing (AST) is necessary for P. multocida for which resistance to commonly used antimicrobial agents has occurred.
- All antibiotic drugs use in camels is extra-label meaning that the drug manufacturer has not indicated for camel use.
- Antibiotic treatment needs to be given in the early phase of the disease for them to be effective. Before laboured breathing and recumbency sets in.
- In severe cases dexamethasone can be used to reduce inflammatory accumulation of fluid in the respiratory tract. Unlike in cattle where pregnancy is supported by placenta hence can result in abortion when dexamethasone is used. Camels pregnancy is supported by the ovarian located Corpus luteum (CL). CL is not affected by dexamethasone administration.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting In severe cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM for 3 to 5 days.
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Amoxycillin 150 mg Long acting	Broad-spectrum antibiotic with bactericidal action	1 ml per 10 kg BWT deep IM neck muscles q 48 hours.
Sulfamethoxazole 200 mg Trimethoprim40 mg	Broad-spectrum antibiotic with bactericidal action	Twice daily 1 ml per 10 - 20 kg BWT for 3 - 5 days. IM neck muscles
Enrofloxacin 100 mg	Broad-spectrum antibiotic with bactericidal action	Single-Dose by SC only 7.5-12.5 mg/kg of BWT (10 to 20 ML) SC
Multivitamin	Boost immunity	Adult Camel: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

** Once daily- SID, twice daily- BID, three times daily- TID, four times daily- QID, as needed – PRD Repeat- q and mL- Millilitre, body weight- BWT, kg- kilograms, mg- milligram. **By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC). NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage*

Prevention/control

- During outbreaks mass medication of affected herd.
- HS vaccine is commercially available but it is not marketed in Kenya. Vaccination should be done in camels using the alum precipitated or oil adjuvant Pasteurella multocida type B vaccine, before rainy season or during early phase of an outbreak. Vaccines provide protection for 6–12 months.
- There is also, a vaccine against Mannheimia haemolytica that offers protection against pneumonic Pasteurellosis.

Zoonotic potential – Yes, Pasteurella multocida can infect humans through skin wounds and immunocompromised individuals through aerosol droplets.

Notifiable disease- No

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds showing signs do not share same grazing land and watering points.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff.
- Avoid taking animals to areas with HS outbreaks.

Further Reading

1. Gluecks, I.V., Bethe, A., Younan, M. et al. Molecular study on Pasteurella multocida and Mannheimia granulomatis from Kenyan Camels (Camelus dromedarius). BMC Vet Res 13, 265 (2017). https://doi.org/10.1186/s12917-017-1189-y

2. Yaeger, Michael; Coatney, John W.; Dioli, M.; and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

Figure 77

Camel affected with HS showing fibrinous nasal discharge and open mouth breathing (Left) and spontaneous haemorrhages and swelling of the head and submandibular regions (Right)

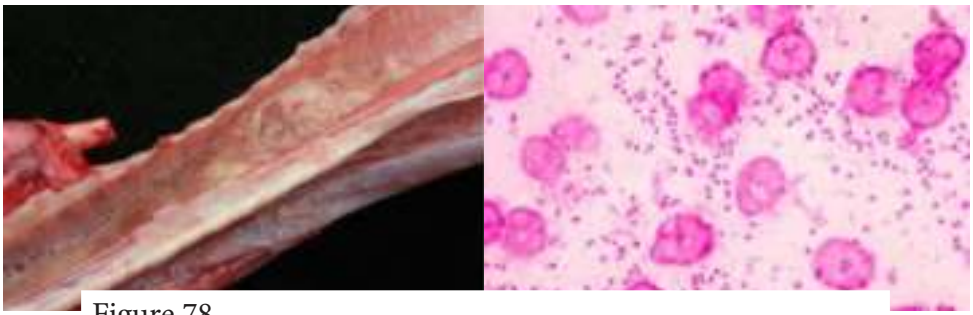

Figure 78

Frothy Thick Tracheal Fluid (Right) and Smear of HS inflammatory fluid from thorax stained with Gram stain showing Gram-negative, bipolar-staining short cocco bacilli (Black arrow)

Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

Figure 79

Pulmonary oedema demonstrated by failure of lung tissue to collapse and lung tissue hepatisation photo courtesy of Dr. Bernard Chege, Marsabit County

5.6 Caseous Lymphadenitis (CLA)- ABUS / ABUTH

Other name: Pseudotuberculosis

Case definition

- Caseous lymphadenitis (CLA) is a chronic bacterial disease of camelids, wild and domestic small ruminants, pigs and man.
- CLA lesions usually appear clinically as abscesses in the superficial lymph nodes.
- Economic losses are associated with progressive weight loss, carcass trimmings and skin condemnation at abattoirs.

Distribution

- The infection is spread by inhalation, ingestion or directly through wounds.
- CLA has been reported in all camelids that include the old world camels: Camelus dromedarius and Camelus bactrianus, including the Australian feral dromedary population and the new world camels: Llamas and Alpacas
- CLA has a high morbidity rate of 90% and mortality rate of 28%.
- Both, young and adult camels are affected by the disease.

Causative agent and pathogenesis

- Corynebacterium pseudotuberculosis is a short, irregular ovoid Gram-positive rod almost resembling a coccus. Smears from abscesses show marked pleomorphism.
- Cultured colonies are small, white and dry and can be surrounded by a narrow zone of haemolysis when sheep or cattle blood is used. The plates should be incubated at 37°C for at least 48 hours.
- Corynebacterium pseudotuberculosis is a Gram-positive rod-shaped, intracellular, facultative aerobic bacterium with worldwide distribution. The facultative, intracellular bacterium multiplies in infected phagocytes and gets disseminated via lymph or blood to secondary sites where it causes abscesses of different size.
- Bacteria has two biotypes known: ovine/caprine and equine/bovine. Recently it has been shown, that strains isolated from dromedaries in the United Arab Emirates and Kenya belong to both serotypes, serotype I (biotype ovis) and serotype II (biotype bovis) using the nitrate reduction test.
- CLA is spread primarily through direct contact of bacteria excreted from the abscesses caused by Corynebacterium pseudotuberculosis and indirectly through contaminated fomites.
- The bacterium can survive several months within the environment and the soil, making the containment of the disease difficult to control.

Clinical signs

- Development of superficial abscesses localized mainly in the cephalic, prescapular and prefemoral lymphnodes is pathognomonic of the disease in camels. The lymphnodes swellings are cold, closed and painless. They swollen external lymphnodes are the size of a lemon or orange.
- In camels, the characteristic laminated (onion ring) pattern seen in goats has not been described.
- In camelids three different types of pyogranulomatous lesions have been observed: Single abscess, when ripe and opened, the abscess extrudes thick, white-cream like pus; Large central abscess with multiple small abscesses in the peripheral connective tissue capsule and Multiple abscesses and no central abscess.
- A few cases have been seen in dromedaries

whereby the abscess breaks through the ribs and the organisms enter the lung producing severe bronchopneumonia with pulmonary caverns

Post-mortem lesions

- Camels rarely die of the disease and in most cases CLA is an incidental finding during slaughter. Most common lesions are pyogranulomatous abscess of the internal organs and lymphnodes.

Diagnosis

- Filed based diagnosis can be made through Gram stained impression smears from abscesses. CLA agent appears as a short, ovoid Gram-positive rods almost resembling a coccus.
- Bacterial culture of lymph node swabs or lymphnodes tissues reveals mucoid, opaque, greyish-white colonies the bacteria cultured can be confirmed using API biochemical tests or molecular PCR tests. bacilli arranged as Chinese letters or V shape.

Treatment and Control

- Antimicrobial treatment is often not rewarding and other treatment methods should be considered. Since erythromycin is able to penetrate tissues. It is proposed a combination of penicillin and erythromycin to treat pseudotuberculosis in camels.
- In cases with multiple abscesses, surgical and antibiotic treatments are recommended Subcutaneous ripe abscesses can be lanced and cleaned out on a daily basis with iodine solution. It is of paramount importance to provide strict aseptic methods,
- Globally, there are no commercial vaccines for camels. However, commercial ones exist for goats and sheep.
- Cull from the herd infected animals.
- Purchase of animals should only be allowed from herds with no history of abscessation

Zoonotic potential

- Yes, can infect man skin wound, always wear gloves when treating abscesses.

Notifiable disease – No

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices - None

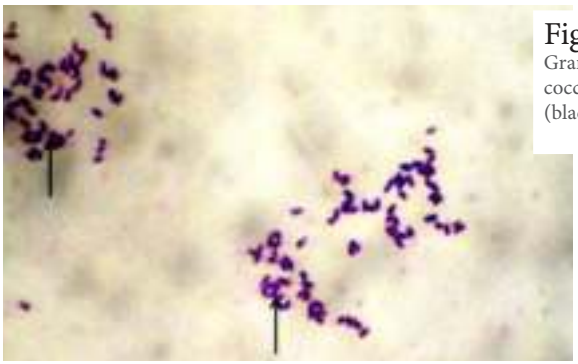

Figure 80
Grams stain of culture showing Gram positive cocco-bacilli arranged in a Chinese pattern (black arrows)

Picture source:
Singh, R. (2018). Pathology of Caseous Lymphadenitis in Slaughtered Goats Associated Infection with Corynebacterium Pseudotuberculosis. Journal of Animal Research, 8.

Further Reading

1. U Wernery and J Kinne. Caseous Lymphadenitis (Pseudotuberculosis) in Camelids: A Review. Austin J Vet Sci & Anim Husb. 2016; 3(1): 1022. ISSN: 2472-3371 https://austinpublishinggroup.com/veterinary-science-research/fulltext/avsah-v3-id1022.php

Figure 81

Cold, closed painless abscesses up to the size of a lemon or orange in a dromedary camel with bilateral inferior cervical lymph node abscesses

Picture source: https://www.researchgate.net/publication/313196136_Serological_Surveillance_of_Caseous_Lymphadenitis_in_Sudanese_and_Somali_Camels_Slaughtered_at_Al-warraq_Abattoir_Giza_Egypt/figures?lo=1

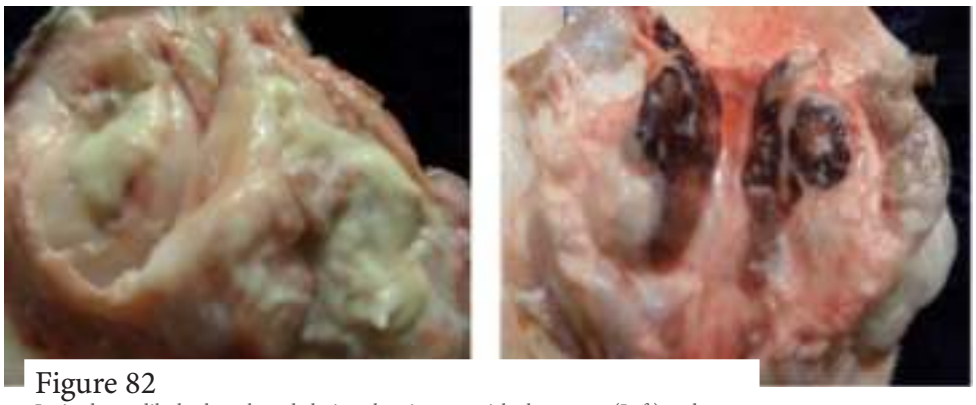

Figure 82

Incised mandibular lymph node lesion showing greenish cheesy pus (Left) and Incised inferior cervical lymph node lesion showing enlargement and congestion without abscess formation (Right)

Picture source:
https://www.bibliomed.org/mnsfulltext/31/31-1509535551.pdf?1630646409

5.7 Contagious Skin Necrosis (CSN)- ANG'AJUL NG'AJEMEI

Aetiology/ causative agent

- Contagious skin necrosis (CSN) is a chronic inflammation of the skin primarily caused by the bacteria Staphylococcus aureus.
- CSN lesions are characterized by necrosis, suppuration, and sinus formation in the skin, and enlargement of regional lymph nodes.
- Lesions predominantly occur on the skin of the back, hump, shoulders, base of the neck, and inguinal region, but may also occur on any other part of the body.
- CSN primarily affects young camels of both sexes and is more prevalent in the hot months of the year.
- Different microbial agents have been isolated from CSN lesions; the most predominant being Staphylococcus aureus.
- Other organisms isolated include; Streptococcus species, Corynebacterium pyogenes, Pseudomonas aeruginosa, Escherichia coli, Bacillus subtilis, Erysipalothrix species, Actinomyces species, Lactobacilli as well as a variety of fungal species.
- Diagnosis of CSN is usually based on clinical signs and distribution of the lesions while the causative agent (s) can be determined using standard microbiological method

Transmission and Predisposing factors

- Presence of arthropod biting flies is associated with spread of disease in a herd.
- Disease is prevalent during hot months of the year.
- Prevalence of CSN is common in young animals (<5 years). One study in Egypt found that disease prevalence increased as the age of animal increased till 5.5 years and thereafter decreased gradually by further increase in the age.

Occurrence

- CSN occurs in all regions of the world where camels are raised.

Main clinical signs

- Both male and female animals are affected equally
- The lesions start as circular sores that a few centimetres in diameter the sore area then begin to discharge fluid. A thick layer of dead skin/scab covers the lesion and after 7 to 14 days the thick scab falls off, leaving a circular ulcer that may extend deep into the skin dermal layer and does not heal
- The lesions cause irritation to the animals, camel try to rub and bite the affected skin area.
- The draining lymph nodes may become enlarged
- Rarely, does the camel develop septicaemia (blood poisoning)
- When the sore heals, it leaves a star shaped scar

Differential Diagnosis

- Abscess due to other bacteria like Corynebacterium that causes Caseous lymphadenitis
- Infected saddle wounds

Diagnosis

- Clinical diagnosis of painful skin sores, on which the hair falls out. The sores are usually found on the neck, shoulders, withers, sides and quarters, and rarely on the lower legs.
- Collect sterile swab smear and gram stain if there is a laboratory for bacterial culture and antibiotic sensitivity. Sterile swabs should be collected from the centre of the ulcer lesion once the necrotic scab is removed.
- The swab should be put in transport media like Stuart's transport medium. Where possible use swabs already pre-packed in media.

Post mortem lesions

- Animals rarely die of CSN, if they do signs will be similar to those due to septicaemia with haemorrhages seen in mucosa and serosa surfaces.

Treatment and Control

- Local treatment of sores involves washing with warm water, removal of the dead tissue, then clean with diluted lugol's iodine. Spray the sore with antibiotic spray that has gentian violet, oxytetracycline or gentamycin antibiotics. The gentian violet prevents flies from laying eggs resulting in myiasis.
- Inject a broad-spectrum antibiotic such as long-acting oxytetracycline or penicillin-streptomycin at a dosage rate of 1 mL for each 10-kilogram body weight by intramuscular route. It should be noted that organism has been shown to have high levels of antibiotic resistance and practitioners are encouraged to take bacterial swabs for culture and sensitivity especially in herds reporting recurrent occurrence.

Zoonotic potential

- No

Notifiable disease

- No

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals from herd
- Hand pick ticks
- Regular acaricide use especially pour on that work well as fly repellents.

Further Reading

1. World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header

2. A field manual of camel diseases: Traditional and modern health care for the dromedary. Compiled by Ilse Köhler-Rollefson, Paul Mundy and Evelyn Mathias. http://vetbooks.ir/a-field-manual-of-camel-diseases-traditional-and-modern-healthcare-for-the-dromedary/

3. Studies on contagious skin necrosis and trypanosomosis in camels. Maha I. Hamed, Mahmoud R. Abd Ellah, Infectious Diseases, Clinical Laboratory Diagnosis, Department of Animal Medicine, Faculty of Veterinary Medicine, Assiut University 71526, Egypt. https://pdfs.semanticscholar.org/79fa/a0bc28058cf4778b783f3ce20a2a5a37fcf9.pdf

Figure 83
Contagious skin necrosis (CSN) on the hind leg of a one-year-old camel calf. Photo: Abdul Raziq

Picture source:
https://doi.org/10.1186/1746-4269-6-16

Figure 84
CSN ulcer lesion once crust is removed, ulcer usually are filled with white pus material

Picture source:
https://pdfs.semanticscholar.org/79fa/a0bc28058cf4778b783f3ce20a2a5a37fcf9.pdf

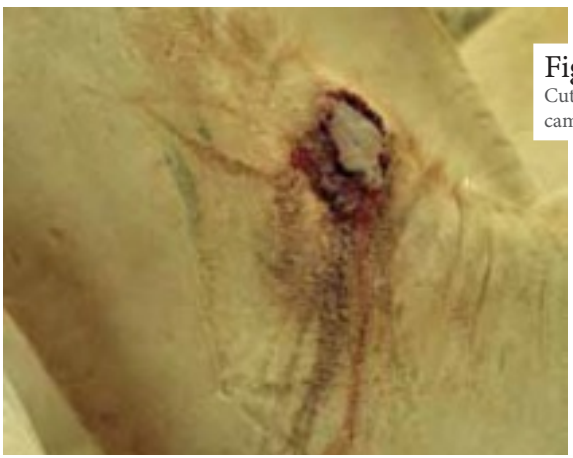

Figure 85
Cutaneous Skin Necrosis lesion on the neck of a camel, source: Maurizio Dioli

5.8 Camel Mastitis- LOEKETA

Case definition

- Mastitis is the inflammation of the mammary gland that is characterised by physical, chemical and bacteriological changes in the milk as well as pathological changes in the glandular tissue.
- The most important changes in the milk include change in the colour, presence of milk clots and presence of a large number of leucocytes.
- Clinical mastitis cases are easy to detect by manual palpation of udder for heat, pain and induration and visual examination of the milk using a strip cup.
- However, most mastitis cases are readily detectable and are referred to as subclinical mastitis and require indirect tests that depend on determining the somatic cell count (white blood cells/ leucocyte content) of the milk.
- Mastitis is not common in camels when compared to cattle. However, incidence of mastitis increase in camels that are frequently hand milked and those that have teat malformation.
- Acute mastitis has been reported to occur during the first few days following parturition or after dystocia.
- The cultural practice of tying the teats of the camels to prevent suckling of young also predisposes camels to mastitis.

Aetiology and Transmission

- Many infective agents have been implicated as causes of mastitis in camels, the commonest being bacterial infection.
- The documented common causes of bacterial mastitis in camels are Streptococcus, Staphylococcus, Micrococcus, Aerobacter and Escherichia coli.
- Streptococcus agalactiae and Staphylococcus aureus have been documented as the two most important mastitis pathogens in camels.
- Transmission mechanisms depend on the bulk of the infection in the environment, including: infected quarters; hygiene of milking personnel; susceptibility of the camel, which is related to the stage of lactation, age of the camel (older animals more susceptible) and level of inherited resistance (related to teat shape and anatomy of the teat canal); lesions on the teat skin especially the orifice; and the immunological status of each mammary gland.

Clinical signs and diagnosis

- Clinical mastitis detected easily due to changes in the secreted milk (colour, consistency, and presence of floccules) with udder changes (red, swollen). Camel may have other generalized signs such as fever and anorexia and deteriorating body condition.
- Subclinical mastitis, is difficult to diagnose and depends on various test procedures aimed at detecting the cause or products of inflammation in milk. A camel with subclinical mastitis produces less milk, but does not have a swollen udder or abnormal milk. Infection is present but can only be detected with the help of indirect methods such as the California mastitis test (CMT). See more details on how to conduct laboratory analysis in Chapter 4 section 4.5- Bovine mastitis.

Treatment

- Although some authors have suggested daily intra mammary infusion with antibiotic preparations as used in cattle, there is opposition to this practice because of the particular anatomy of the camelidae udder and

because of the difficulty in administering such treatment.49 Therapeutic approach in treating acute mastitis is via systemic antibiotics (e.g. trimethoprim–sulfamethoxazole or penicillin/ Aminoglycoside) and anti-inflammatory drugs (flunixin meglumine), with regular stripping of the mammary glands. Hydrotherapy is beneficial in reducing local oedema. The teat of the camel udder contains sometimes three separate teat canals that open independently into the teat sphincter. The separate canals drain separate gland complexes.50 This implies that for intra mammary treatment of mastitis, not only must each quarter but also each gland complex be treated separately, that is, one intra mammary tube per gland complex. Great caution is necessary when applying intra mammary treatment to camels. The teat canal openings in camel are smaller than those of the cow and thus require smaller cannula. Unhygienic and traumatic application of intra mammary treatment is very likely to do more harm than good. Treatment of chronic mastitis is very difficult and the condition often results in the loss of the affected quarter.

- For subclinical mastitis The best treatment period is during the non-lactation (dry) phase which proved it cures about 70% of environmental infection caused by Streptococci

Prevention and Control

- The main principles of mastitis control include the elimination of existing infection, prevention of new infection and monitoring udder health status. Elimination of existing infections can be achieved by appropriate therapy of infected cases. Prevention of new infections through monitoring udder health status, providing supplementation of the required nutrients and maintaining hygienic measures.

Further Reading

Jilo K, Galgalo W, Mata W. Camel mastitis: a review. MOJ Eco Environ Sci. 2017;2(5):194-202. https://doi.org/10.15406/mojes.2017.02.00034 :

Article is also the source of pictures below

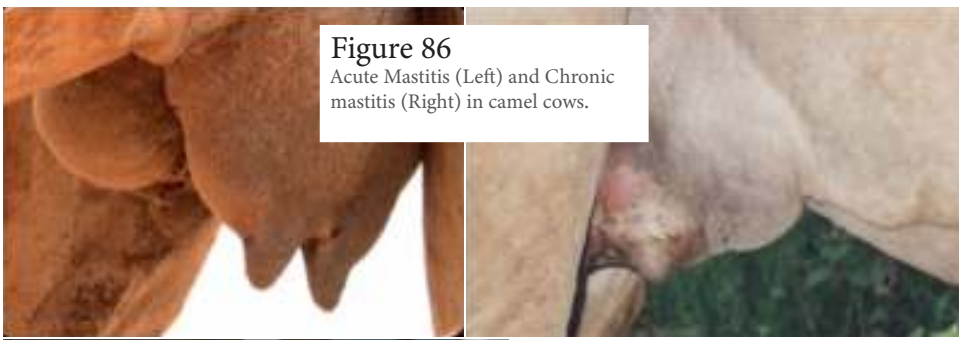

Figure 86
Acute Mastitis (Left) and Chronic mastitis (Right) in camel cows.

Figure 87
CMT paddle used to test for mastitis

Camel diseases:

Parasitic

5.9 Camel Mange- EMITINA / EKOIKOI /LOTOME

Aetiology: Mange is caused by the mite (Sarcoptes scabiei var cameli).

- **Clinical signs:** The mite burrows through the skin producing irritation and intense pruritus, causing the camel to scratch against objects. Affected areas may appear reddened (inflamed) and the hair will have been rubbed off. Infection leads to loss of body condition, due to decreased grazing time.
- **Transmission and age group affected:** The disease is highly contagious and is more common in the young. It can spread directly from animal to animal, particularly when it is cold and animals huddle together. It can also be spread by fomites (saddles, harnesses) or in areas of common use (bedding, tree trunks, dust baths). Young calves that are in poor body condition are more susceptible.
- **Morbidity and mortality rate:** In young animals' morbidity rates can be as high as 100% while mortality rate is lower at 30%.
- **Seasonal occurrence:** Occurs all year round.
- **Diagnosis**- Mange is easy to diagnose clinically from the pruritus, alopecia and encrusted plaques. Clinically affected animals are recognised by large areas devoid of hair, with thickened skin and folds affecting the hind limbs and neck. Chapter 1 describes the process of collecting a skin scrapping for field based confirmation of the parasite.
- **Differential diagnosis**- other skin diseases that cause thickening of the skin include tick infestation, mineral deficiencies, recovered pox lesions and ringworm infections.
- **Zoonotic potential** – Yes man can be an accidental host. Close contact with infected camels may result in the spread of disease to caretakers.
- **Notifiable disease**- No.
- **Treatment**- Mange is treated effectively with acaricides like organochlorine (Amitraz®, Tix fix®), organophosphates like Malathion. These are applied as a spray, although hand application with a cloth is used for areas that are least accessible.
- Treatment should be repeated after an interval of 8-15 days.
- Ivermectin is a macrocyclic lactone is effective against mange two subcutaneous injections, 2 weeks apart combined with acaricide wash results in clinical resolution of signs.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.
- **Prevention**- From the prophylactic standpoint, it would be desirable to treat the entire herd but this may be impracticable. Priority should be given to young camels and breeding males.

Ethnoveterinary treatment practices

- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals in the herd and create a separate night kraal/boma.
- Migrate out of the area or Relocate the camel boma
- Regular acaricide use or hand pick ticks.

Further reading

1. Richard D. (1987). Camel mange. Rev. sci. tech. Off. int. Epiz., 1987, 6 (2), 475-477.
https://www.WOAH.int/doc/ged/D8497.PDF

2. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

Figure 88

Mange skin lesions in a camel calf red arrows show alopecia, encrusted plaques and hyperkeratosis of the skin

Picture source: Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21.
https://lib.dr.iastate.edu/vdpam_reports/21

5.10 Dermatophytosis

Other name: Ring worm

Definition and Epidemiology

- Camel dermatophytosis also known as ringworm is a highly contagious fungal dermatitis that characterised by circumscribed crusty hairless lesion, that are 1 to 2 cm in diameter that are distributed over the head, neck, shoulder, limbs and flanks. The fungal lesions are described as having a 'moth-eaten' appearance of the camel calf's wool.
- The disease is mostly found in young calves less than 3 years of age
- Trichophyton verrucosum is the most common fungi affecting young camels while older animals are affected by T. mentagrophytes.
- The morbidity rate in young calves is estimated to be 43.5% while the overall herd incidence rate of all age groups is 11.2%.
- The disease is more prevalent during the rainy and cold seasons of the year when calves hurdle together due to the wet conditions. The persistent wet woolly skin of calves weakens the natural skin immunity barrier providing ideal conditions for infection.

Aetiology and Transmission

- The disease is caused by a specialised group of zoonotic fungi called dermatophytes. Dermatophytes are filamentous fungi which invade keratinized tissues of humans and animals, causing mild to severe, localized and/or diffuse infections.
- Transmission of dermatophytes is usually by direct contact with infected host (animals or humans) or asymptomatic carriers and/or indirect contact with infected exfoliated skin or hairs in the environment.

Clinical signs:

- The incubation period of the disease is 8-30 days. Skin lesions are extremely variable in distribution from multi focal to generalized lesions. The typical skin lesions are circular alopecic areas with peripheral expansion and that have the centre covered by grey powdery crust.
- The periphery spreading lesions may become confluent and produce a moth- eaten appearance of the wool.
- Lesions are found in the head, neck, shoulders with extension to the flanks and legs.
- Generalised skin infections lead to emaciation.
- Mixed infection of dermatophytosis and Sarcoptic scabiei (Mange), other fungal infections like Microsporum gypseum and bacterial infection with Dermatophilus congolensis especially in rainy season have been reported.

Diagnosis

- Field based diagnosis can be done through Direct examination of skin scrapings digested with 20% potassium hydroxide (KOH) and examined under low and high power light microscope. Procedure for skin scraping is

described in detail in chapter 1.
- Routine 'gold standard' culture of samples takes 5 weeks to grow on Sabouraud's dextrose agar (SDA). Molecular diagnostic tests can shorten the confirmatory diagnostic period.

Differential diagnosis
- Mange
- Other Dermatomycosis species
- Dermatophilosis due to Dermatophilus congolensis bacteria.

Zoonotic potential – Yes,
Notifiable disease- No
Treatment
- Optimal therapy of dermatophytosis requires a combination of topical antifungal therapy,
- concurrent systemic antifungal therapy and environmental decontamination.
- In pastoralists setting systemic therapy and environmental decontamination may be impractical. The following treatment regime has been used; Wear gloves, Shave the affected woolly areas and remove scabs, Topical application of 2% solution of tincture iodine or 10% iodine solution once daily for three weeks (duration is difficult to adhere to in pastoralists settings). Systemic treatment can be done with Griseofulvin10 mg/kg body weight for 7 days in mild infections; in severe cases 2 to 3 weeks (impractical in pastoralists settings)
- Dermatophytes can remain viable in infected soil for many years. Environmental decontamination can be done with 10% hypochlorite solution (Jik™).

Prevention
- Isolate infected animals from herd by ensuring they sleep in a different boma/kraal.
- Treat infected animals immediately with iodine based solutions.
- Regular ectoparasite control.
- Ensure good nutrition of calves.
- There is a vaccine commercially available. However, it is not marketed in Kenya. The vaccine is a live attenuated vaccine against T. verrucosum and T. mentagrophytes and is administered every five years.

Ethnoveterinary treatment practices
- Refer to tables 14 and 15

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Isolate sick animals in the herd and create a separate night kraal/boma.
- Migrate out of the area or Relocate the camel boma
- Regular acaricide use or hand pick ticks.

Further reading

1. Abdulaziz M. Almuzaini, Salama A. Osman & Elhassan M.A. Saeed (2016) An
outbreak of dermatophytosis in camels (Camelus dromedarius) at Qassim Region, Central of Saudi Arabia, Journal of Applied Animal Research, 44:1, 126-129, DOI: 10.1080/09712119.2015.1021806: http://dx.doi.org/10.1080/09712119.2015.1021806

Figure 89
(a) Cutaneous lesions, (b) Colonies on SDA and (c) Distorted hyphae with chlamydospores in chains confirming T. verrucosum.

Picture source:
Abdulaziz M. Almuzaini, Salama A. Osman & Elhassan M.A. Saeed (2016) An outbreak of dermatophytosis in camels (Camelus dromedarius) at Qassim Region, Central of Saudi Arabia, Journal of Applied Animal Research, 44:1, 126-129. http://dx.doi.org/10.1080/09712119.2015.1021806

5.11 Helminthiasis – NG'IPELEI / NG'IRITAN

Aetiology

- There is a scanty information on camel helminths with most literature available being country specific prevalence coprological (faecal) studies based on flotation and sedimentation techniques.
- Although the camel is not a true ruminant as it has only 3 stomachs (C1, C2 and C3), the fact that it shares the same habitat with cattle and small ruminants means that most helminth or gastrointestinal parasites are the same. Refer to Chapter 4 Cattle diseases section 4.14 Helminths for more information on the classification of helminths of ruminants.
- Based on studies in Ethiopia, Iran and Saudi Arabia the most common helminths genera are Nematodes mainly -Trichostrongylus, Haemonchus, Strongyles, Strongyloides, Nemaotdirus and Trichuris. Trematodes like Paramphistomum and Fasciola and cestodes like Moniezia as well as protozoa such as Eimeria have been identified.
- Prevalence rate of GIT helminths was higher in young animals as adult camels develop resistance as seen in adult cattle.
- There is need for more research to identify the species of helminth genera involved and their pathophysiology in camels.

Clinical signs and Treatment

- Light infections in camels is associated with decreased productivity manifested by retarded growth, poor weight gain and decreased milk production.
- Heavy infection is characterised by apathy, anorexia, nutrient deficiency, progressive wasting manifested by atrophy of the humps and decreased abdominal volume (unfilled flanks even after watering), diarrhoea, constipation, intestinal obstruction, anaemia, colic, prostration, and even death after a few weeks or a few months.
- Refer to Chapter 4: Cattle diseases section 4.14 for detailed clinical signs linked to each genera of helminth.
- Ivermectin at a dose of 0.2 mg/kg and albendazole at a dose of 10 mg/kg has been shown to substantially reduce nematode egg counts. Although of slightly lower efficacy, levamisole (at a dose of 10 mg/kg) has been shown to be able to reduce the parasite burden.

Further Reading

1. Hadush, Angesom. (2019). Major helminth parasites of Camelus dromedarius in afar pastoral area of Ethiopia. Iraqi Journal of Veterinary Sciences. 33. 117-122. 0.33899/ijvs.2019.125524.1046.

2. ShibabawBejano, Zelalem Worku, Nega Mokennen, Tolesa Ibsa, Befikadu Zewde, Alemneh Kassa. Gastrointestinal Tract Parasites of Camel in and Around Dire Dawa. Biomed J Sci & Tech Res 42(2)-2022. BJSTR. MS.ID.006714.

5.12 Camel nasal bots- EKURUT AKOU EKAAL

- The oestrid fly, Cephalopina titillator (Clark), causes nasopharyngeal myiasis in camels, the fly is widely distributed in many camel breeding areas triggering health hazards and severe economic losses in camels. Heavy infestation has a poor prognosis even with treatment
- Most infested camels developed clinical signs of nasal discharge, restlessness, loss of appetite, difficulty in breathing, frequent sneezing, and snoring. Post-mortem examination reveals blockage of the nasopharynx by larvae and/or mucofibrinous secretions. The most effective early treatment is Ivermectin at a dose rate of 0.2 mg/kg.

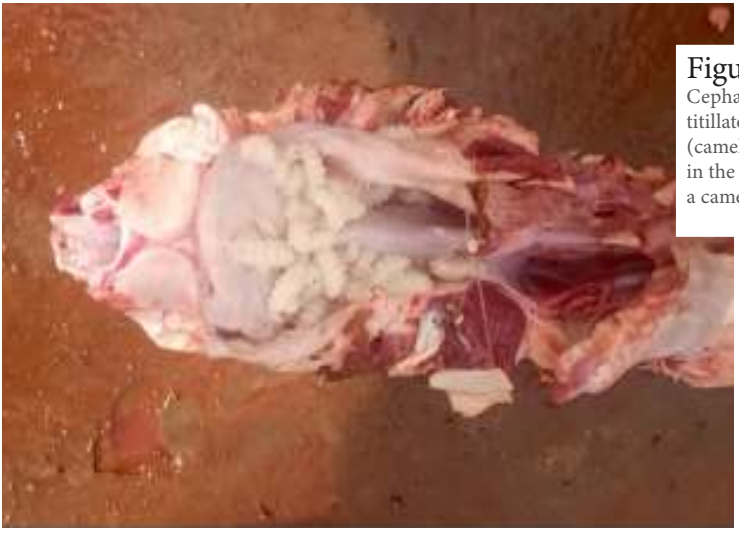

Figure 90
Cephalopina titillator larvae (camel nasal bot fly) in the nasal cavity of a camel

Photo credit, Diba Denge, Animal Health Technician, Marsabit County

Chapter 6:

Sheep Diseases

Table 17: Priority sheep diseases ranked in order of importance by livestock keepers in Turkana county

Conventional name	Turkana name
PPR	LOMOO
Sheep and Goat Pox	ETUNE
Helminthiasis	LOPEELEI / NG'IRITAN / LOBOLIBOLIO
Mange	LOTOME / EKOIKOI / LOGIRIGIRA / LOIR
Brucellosis (see Brucellosis Annex 2)	EDEKE AKIRING KA NG'AKILE / AKIYEC
Contagious ecthyma (Orf)	NG'IBUORUOK
Foot rot	EKICHODONU
Coccidiosis	EREMONU ANG'AKOOT
Enterotoxaemia	LORENG MALITENY
Infectious keratoconjunctivitis (Pink eye)	EDEKE ANG'AKONYEN
Pneumonia	LOUKOI
Babesiosis	LONYANG / LORENG'EKUL
Coenurosis – larval stage of dog tapeworm Taenia multiceps	LOKOU / LOIIRA

Table 18: Ethnoveterinary treatment strategies used by livestock keepers to manage priority sheep diseases in Turkana county

Medicinal plants botanical name	Turkana name	Target Disease
Tobacco (chewing tobacco) (Nicotiana tabacum)- soaked and given as an oral drench or chewed and spat into the eye of sick animal	ETABA	Pneumonia, HS, Babesiosis,
Cissus quadrangularis- a tree whose leaves and branches are pounded soaked in cold water and given as an oral drench	EGIS	Pneumonia, HS, Babesiosis, PPR,
Euphorbia kalisina or uhligiana- succulent leaves boiled or pounded and mixed with cold water and given as a drench or applied topically	EMUS	Pneumonia, HS, Babesiosis, PPR, Mange, Sheep pox
Aloe turkanensis- succulent leaves pounded mixed with water and given as an oral drench or applied topically	ECHUCHUKA	Pneumonia, Babesiosis, PPR, Mange, Sheep pox
Cadaba farinose- tree whose bark is dried, pounded and mixed with water to be given as an oral drench	ERENG	Pneumonia, Babesiosis, PPR,
Dalechampia scandens- shrub whose roots are pounded mixed with water and given as an oral drench	EKWANG'A	Pneumonia, Babesiosis, PPR,
Balanites pedicellaris- tree whose leaves are pounded and mixed with water and given as an oral drench	ELAMACH	Pneumonia, Babesiosis, PPR,
Balanites rotundifolia - tree whose seeds are crushed and mixed with water to be given as an oral drench	EBEI	Pneumonia, Babesiosis, PPR,
Boscia coriacea- ree whose seeds are crushed and mixed with water to be given as an oral drench	EDUNG	Pneumonia, Babesiosis, PPR,

Cont. Table 18: Ethnoveterinary treatment strategies used by livestock keepers to manage priority sheep diseases in Turkana county

Traditional Practice	Turkana name	Target Disease
Prosopis julliflora - leaves dried and topically applied	ETIRAE	Pneumonia, Babesiosis, PPR,
Neem tree (Azadirachta indica)- leaves boiled with water given as an oral drench	MWARUBAINI	Pneumonia, Babesiosis, PPR,
Hot iron or stone cauterization/ application on the body system affected	AKIMAD EMACHAR KORI AMORU	Pneumonia, Babesiosis, Sheep pox, PPR
Hoof trimming using knives	AKIKUL ANG'IMAGER	Foot rot, Laminitis
Milk fat given as an oral drench	AKIDEDIET	Helminthiasis
Ash mixed with water and given as an oral drench	EKURON	Pneumonia
Carbonated drink /soda coca cola	COCA COLA	Bloat, Enterotoxaemia
Sewing needle	EKIPIRET	Removal of foreign material from the hoof
Mesenteric fat	ASEPAE	Topical application on wounds
Sugar	ESUGARI	Topical application in the sick eye (Pink eye infection)
Magadi soda- mixed with water and given as an oral drench or applied on skin lesion	AMAKAT	Pneumonia, Babesiosis, HS, Sheep pox, PPR, Helminthiasis

NB: Text in red highlights practices that go against animal welfare principles

Table 19: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat priority cattle diseases in Turkana county

Type of (VMP)	Active ingredient	Turkana name	Target disease
Antibiotic	Oxytetracycline injectable Adamycin®	EDAWA LOMUNG' / EDAWA ALOUKOI	Pneumonia, Babesiosis, Sheep pox, PPR,
	Tylosin	EDAWA LA NG'IKOUI / EDAWA LOUKOI	Pneumonia
	Procaine Penicillin G and Dihydrostreptomycin (Penstrep®)	EDAWA LOKAKOUN LO AKWAAN	Pneumonia, Sheep pox
	Trimethoprim and Sulfadiazine Dawatrim® bolus	EDAWA LOEREMONU	Helminthiasis, PPR, Enterotoxaemia. Babesiosis
Antiprotozoal	Oxytetracycline spray Alamycin®	EDAWA LOPUS	Mange
	Homidium Chloride (Novidium®)	EDAWA LOARENG'AN	Babesiosis
Antiparasitic / Anthelmintic	Ivermectin	EDAWA ALOTOME/ EDAWA LOMOO/ EDAWA LOKIRION	Mange, PPR, Sheep pox
	Albendazole	NAOSIN / EDAWA LOA ANG'IPEELEI	Helminthiasis, Enterotoxaemia
Acaricide	Amitraz, Pyrethroids (Ectopor®) organophosphate	ADIP NA NG'IGURAI / ADIP	Mange

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

Sheep Diseases:

Bacterial

6.1 Enterotoxaemia – LORENG MALITENY

Other names; Over eating or Pulpy Kidney disease

Case Definition:

- An acute disease of sheep and goats that is characterised by sudden and high death rates in lambs and kids and at post mortem haemorrhagic enteritis where sections of the small and large intestinal walls have a deep blue-purple appearance and intestinal contents are watery and bloody. The kidneys on palpation have abnormally soft consistency described as being pulpy.
- Enterotoxaemia as the name suggests is a blood poisoning due to a toxin (toxaemia) produced by an intestinal dwelling bacteria.

Aetiology and Pathophysiology

- Enterotoxaemia is caused by a gram-positive, rod-shaped bacteria called Clostridium perfringens type D.
- The bacteria are normally found in the soil and as part of the normal microflora of the gastrointestinal tract of healthy sheep and goats.
- However, under specific predisposing conditions, the bacteria rapidly reproduce in the animal's intestine, producing large quantities of the epsilon toxin.
- Young animals are more susceptible to the toxin when compared to adult animals that acquire a toxin tolerance with age following low dose exposure over time.

Predisposing factors and Epidemiology

- Overgrowth of Clostridium perfringens type D bacteria occur in the following conditions:
- Excessive consumption of milk, consumption of lush green pasture at the beginning of the rainy season and feeding on high grain concentrates.
- Reduced Immunity due to disease or heavy gastrointestinal parasitism such as nematodes, cestodes (tapeworms) and coccidia. Heavy tape worm infestation causes a reduction in the motility of the intestinal tract.
- Disease is distributed worldwide with an estimated morbidity rate of 2%, mortality rate of 3% and case fatality rate of 30%. Disease incidence reports of unvaccinated sheep and goats in India have found a prevalence rate 56% in lambs, 46% in kids and 4% in adult sheep.
- In Turkana county, livestock keepers in the year 2022 estimated an apparent morbidity and mortality rate of 3% and case fatality rate of 100%. The livestock keepers indicated the disease was common during the long rainy season AKIPORO.

Clinical signs

- Sudden death is the main manifestation of the per acute form of the disease in young lambs although some of the animals may be dull, depressed and anorexic.
- In acute cases, there is frothy salivation, green or pasty diarrhoea, staggering, recumbency, opisthotonus, colonic convulsions coma and death.
- Colic and bloat may occur. Affected adult

sheep often lag behind the rest of the flock and show nervous signs. Champing of the jaws, blindness, salivation, rapid and shallow respiration, atonic rumen and pasty faces may also be evident.

Post-mortem lesions

- Haemorrhagic enteritis with ulceration of the mucosa.
- Fluid-filled pericardial sac in young lambs and in older animals, haemorrhagic areas on the myocardium.
- Petechiae and ecchymosis haemorrhages of the abdominal muscles and serosa of the intestine.
- Bilateral pulmonary oedema and congestion is also common.
- Rumen and abomasum contains feed. There is also undigested feed in the Ileum.
- Rapid post-mortem autolysis of the kidneys in adult sheep has led to the popular term pulpy kidney disease. Pulpy kidney is not a common feature in young lambs and is rarely found in goats and cattle. In goats the disease can also cause haemorrhagic or necrotic enterocolitis.

Diagnosis	Treatment	Control
Field based diagnosis can be made through making Giemsa stained smears of intestinal contents that can be examined for large numbers of gram-positive, rod-shaped bacteria Confirmatory diagnosis made through ELISA and PCR analysis of faecal and intestinal contents **Differential Diagnosis** • Plant poisoning • Bloat • Thiamine deficiency	Treatment of the animals is usually ineffective, due to the rapid progression of the disease in the animal. Attempts can be made to treat animals that are not too sick with; • Clostridium perfringens C & D antitoxin (5 mL subcutaneously) • Antibiotics, especially penicillin • Anti-bloating medication • Intramuscular thiamine (vitamin B1) to prevent or treat the encephalomalacia **Ethnoveterinary practice** • Refer to tables 17 and 18	The most effective control method is annual vaccination. KEVEVAPI does not have the vaccine source from private sources. **Ethnoveterinary practice** • Restrict lambs from suckling all the time by ensuring the lambs are separated from ewes most of the day. • Restrict access to large quantities of lush pasture by delaying the time the animals are released in the morning or feeding the animals on hay before being released to graze on lush pastures.

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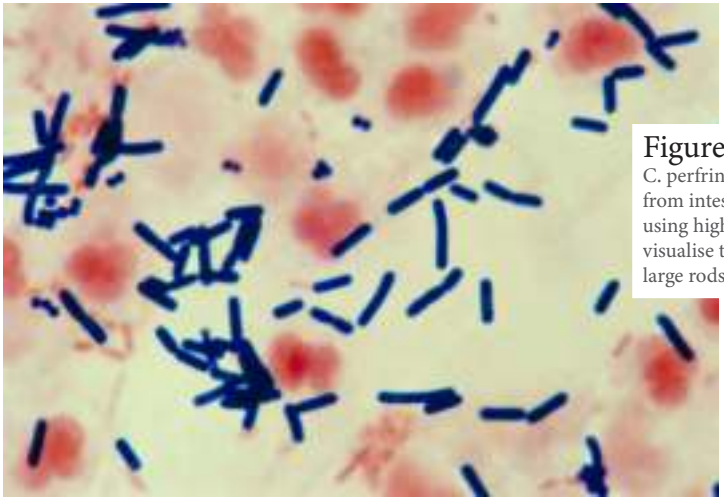
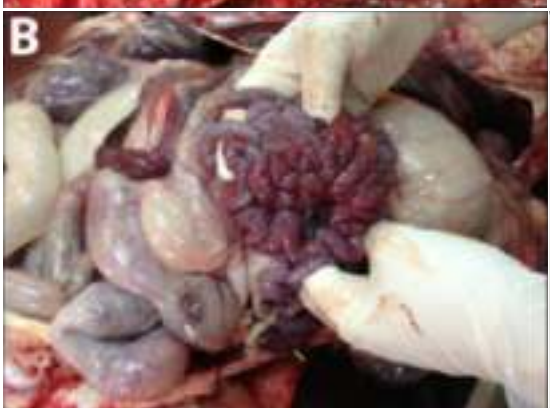


Figure 91

C. perfringens, Gram-stained smear from intestinal epithelial surface using high power objective to visualise the intensely gram-positive large rods that lack spores.



The large and small intestines show severe congestion and oedema with distension of intestine with gas (A, B). Congestion and enlargement of the kidney (C).

Figure 92

Post mortem findings of sheep that died of enterotoxaemia

Photo credit: http://www.sciencepub.net/nature/ns1108/003_19105ns1108_15_21.pdf

6.2 Foot rot- EKICHODONU

Case definition and Aetiology

- Foot rot is a contagious bacterial hoof infection that causes severe lameness in sheep and goats.
- The bacteria are normally found in the soil and are easily carried onto a farm via infected animals or the soles of the shoes. Two types of soil borne bacteria cause the disease - Dichelobacter nodosus (formerly Bacteroides nodosus, a Gram-negative, obligate anaerobe) and Fusobacterium necrophorum).
- Both thrive in moist soil conditions and are difficult to control or eliminate once they contaminate the soil in farms where sheep and goats are kept.
- In Turkana county, livestock keepers in the year 2022 estimated an apparent morbidity rate of 3% with no mortality rate.
- The disease is most common during the long rainy season (AKIPORO).

Clinical signs and Field based diagnosis

- Foot rot is an inflammation between the toes that usually affects one foot.
- Foot rot can occur in one or more feet, causing severe lameness.

Typically, animals are seen grazing on their knees. It occurs when both bacteria cause a dual infection of the tissues of the foot.

- The interdigital space of the foot has dark exudate and the discharge is foul smelling.
- If not treated early, the bacterial toxins break down the hoof wall and sole of the foot, resulting in the hoof wall loosening and detaching from the foot.
- Predisposing factors include; overgrown, cracked or damaged hooves; poor diets deficient in certain minerals also predispose animals to poor hoof health.

Treatment

- Systemic treatment with antibiotics with or without trimming of the hoof is effective. One injection of 20% long acting oxytetracycline at 1ml per 10kgs deep intramuscular is effective. One can also administer an analgesic like phenylbutazone.
- Trimming of the claws is recommended to remove excess tissue that provides a place for the bacteria to thrive.
- After feet have been trimmed, affected animals should stand for at least 5 minutes with all feet in a medicated foot bath (10% copper or zinc sulphate) and feet should be dry before going back to pasture.
- Foot dipping should be repeated once a week for four weeks and non-responding animals should be culled out of the herd.

Ethno veterinary treatment and control

- Wash affected leg interdigital space with salty water
- Ensure animals night boma area drains well especially during the rainy season.

Control

- Do not purchase lame animals. Thoroughly inspect feet before purchase. Observe herd of origin for lameness of other animals.
- Trim hooves regularly.

- Provide good drainage to all areas in pastures and paddocks where water tends to pool, or fence these areas off. This is where the bacteria often collect.
- Keep goat houses or bomas dry and clean.
- Ensure regular hoof care management and always check feet of limping goats.

Differential diagnosis

- Foot and mouth disease
- Tick infestation of the lower limb (dew claw area or interdigital space)
- Foot scald
- Foot trauma- thorns pricking interdigital space
- Foot abscess with other bacteria

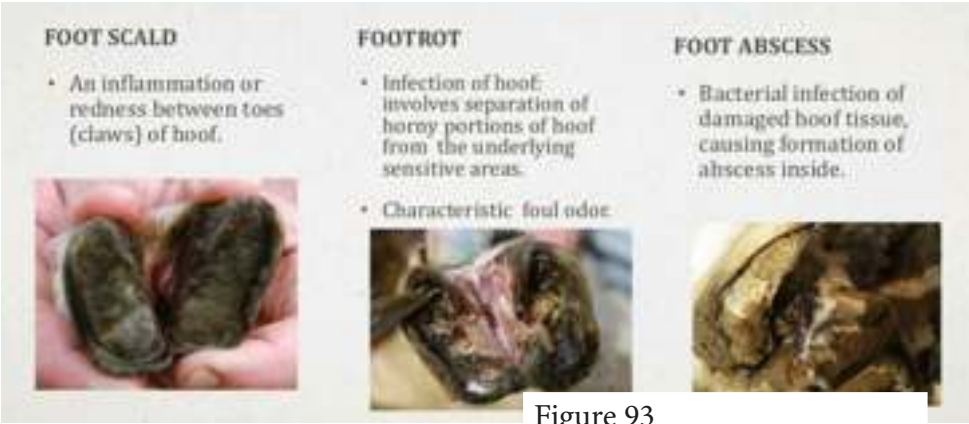


Figure 93
Common hoof conditions in goats and sheep

Figure 94
Diagram depicting differences of foot abscess, foot scald and benign or virulent foot rot

Picture source: https://www.canr.msu.edu/news/tactics-to-win-the-battle-against-foot-rot

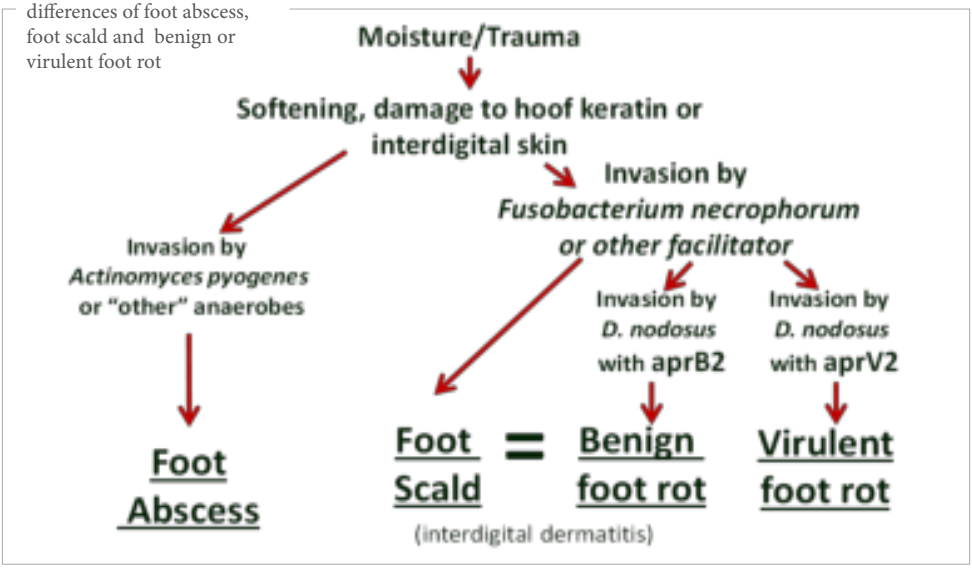


Figure 95

Comparing hoof that is overdue for trimming and one that is properly trimmed

The picture shows an overdue hoof that has the hoof walls curling inwards and is long at the toe. The in curling hoof wall traps mud, manure thus predisposing animal to foot rot. A properly trimmed hoof is flat at the bottom and has a box shaped hoof shape where both toes are the same length.

Figure 96

Hoof trimming procedure in sheep and goats

6.3 Pneumoniae- LOUKOI

Other name- Haemorrhagic septicaemia- LOOKOT

Aetiology

- The most common cause of pneumonia in sheep are due to two bacteria Mannheimia haemolytica and Pasteurella multocida. The two bacteria are natural inhabitants of sheep's respiratory system and only cause problems when the animal is stressed and the immunity is compromised.
- In Turkana county, pneumonia cases are common during the short dry season locally known AIT/NAIT. The apparent morbidity rate in the county is 16% with 8% mortality rate and 50% case fatality rate.

Predisposing factors

- The risk factors of the disease include stressful environmental conditions such as cold weather and wet weather, overcrowding in pens, excessive dust, poor housing.

Pathophysiology

- The virulence of Pasteurella bacteria is mediated by the action of several factors, including endotoxin, leucotoxin, and capsular polysaccharide. The leucotoxin is mainly important, as it is specifically toxic to ruminant leukocytes, resulting in fibrin deposition in lungs and on pleural surfaces. The lipopolysaccharide endotoxin causes adverse reactions in the lungs and also systemic circulatory failure and shock. The capsular polysaccharide inhibits phagocytosis of the bacteria and assists in attachment to the alveolar epithelial surface.
- Survival of the acute phase of pneumonic Pasteurellosis depends on the extent of lung involvement and damage in the lower respiratory tract. Sheep and goats that recover may have chronic respiratory problems, including reduced lung capacity and lack of weight gain if ≥20% of the lung is damaged.

Clinical signs

- Animals may be found dead without prior signs- especially during a sudden down pour.
- Fever of above 40.6oC and as high as 42oC.
- Respiratory distress and purulent discharge from the nostrils.

Diagnosis	Treatment	Control
Nasal swabs are of no significance and diagnosis can only be confirmed at necropsy: The most common pathological observation in acute cases is heavy cyanotic lungs with purplish-red solid areas which exude frothy haemorrhagic fluid when incised.	Treatment using penicillins is effective against Pasteurella pneumonia in sheep. Parenteral administration of Amoxiclav of Penstrep is recommended as the first line of treatment. Other effective treatments are floroquilones (enrofloxacin) and Oxytetracycline.	The ultimate control of Pasteurella pneumonia in sheep is by vaccination –KEVEVAPI has a vaccine (Pastrevax™). Other control measures include isolation of sick animals and quarantine of new additions to the flock, proper housing (good ventilation and drainage), avoiding mixing of age groups and avoiding exposure to extreme weather conditions.

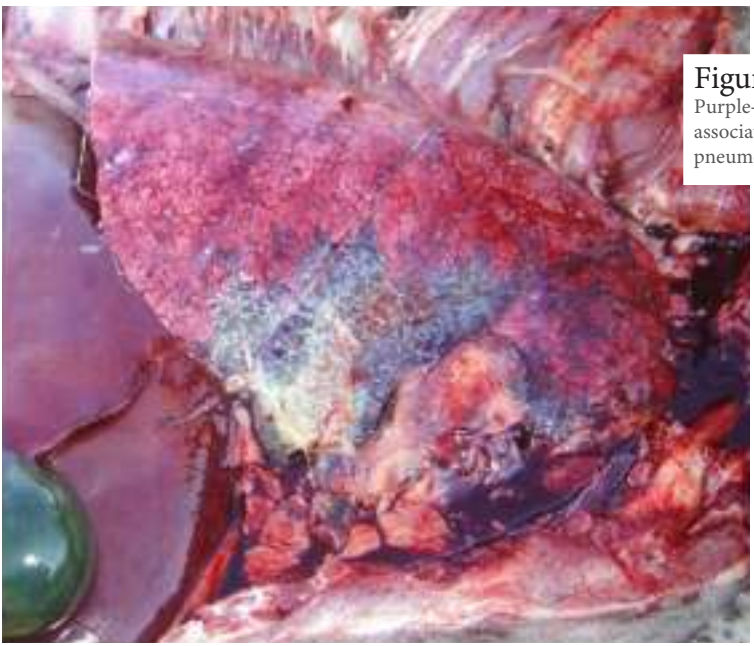


Figure 97
Purple-red lesions on lung associated with Pasteurella pneumonia in sheep.

Photo Credit:
Bruce Watt

6.4 Infectious keratoconjunctivitis (Pink eye)- EDEKE ANG'AKONYEN

Aetiology

- In sheep and goats, pinkeye is primarily caused by the microorganisms Mycoplasma conjunctivae and Chlamydia. These are not the same microorganisms that cause pinkeye in cattle (Moraxella bovis), so the vaccine used to prevent pinkeye in cattle is not effective in goats.
- In Turkana county pink eye has an apparent morbidity rate of 4% with no case fatality rate. The disease occurs throughout the year.

Clinical Signs

- Pinkeye progresses rapidly once a sheep or goat is infected. The first signs of pinkeye are squinting with excessive blinking (blepharospasm).
- Watery eye discharge that later become yellow or green due to pus.
- The eye is red and swollen shut in some cases.
- There is formation of new blood vessels and increased inflammatory cells in the cornea that causes if to be cloudy.
- In severe cases, wound-like ulcers may appear in the centre of the eyes
- Pinkeye can cause temporary blindness in goats, or permanent blindness in severe and untreated cases.

Diagnosis

- Presumptive diagnosis of infectious Keratoconjunctivitis is based on ocular signs.
- Microbial culture may help to confirm the causative organisms and effective antibiotic to use.

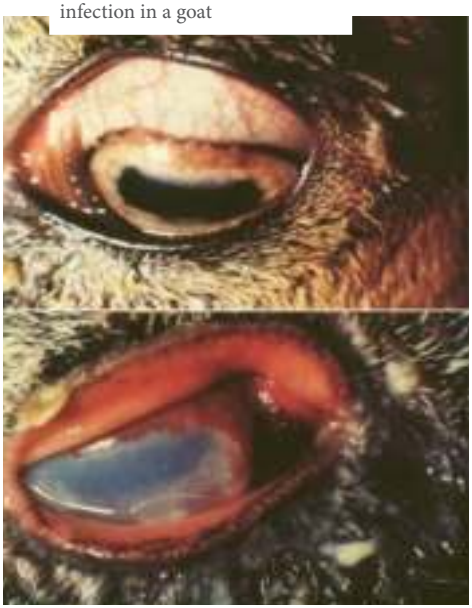
Treatment

- Topical tetracycline eye ointment or cloxacillin eye ointment (opticlox®) works well. (Please do not use the popular eye powder preparation. It worsens the condition as it increase the irritation in the eye).
- 20% Oxytetracycline long acting at a dose rate of 1 ml per 10 kgs body weight is effective for severe cases given intramuscular or subcutaneous. Tylosin at 1 ml per 20 kgs body weight given intramuscular is also effective. If the infection is caused by Chlamydia, any antibiotic including the eye ointment with Cloxacillin is effective.

Control

- Always purchase animals from disease free herds.
- Prevent stress when managing the herd because stress increases the hormone cortisol, which lowers immunity.
- Quarantine newly purchased animals to avoid the possibility of introducing sick animals into the herd.
- Improved management practices help prevent infectious Keratoconjunctivitis.

Figure 98
Different stages of Pink eye infection in a goat



Top picture:
Eye of goat with keratoconjunctivitis. Epiphora, injection of the palpebral and bulbar conjunctiva, transparent conjunctival follicles at the dorsal limbus, and corneal neovascularization are present. The anterior segment otherwise appears normal.

Bottom picture:
Eye of goat with severe keratitis is characterized by mucopurulent ocular discharge, corneal oedema, and corneal neovascularization.

Picture Source: https://www.researchgate.net/publication/21066303_Keratoconjunctivitis_Associated_with_Neisseria_Ovis_Infection_in_a_Herd_of_Goats/figures?lo=1

Sheep Diseases:

Viral

6.5 Peste de Petits Ruminants (PPR) - LOMOO

Other name: Sheep and goat plague

Case definition

- PPR is a highly contagious disease affecting domestic and wild small ruminants. It is characterised by multisystemic clinical signs that include bilateral nasal and ocular discharge, pneumoniae, necrotic stomatitis and diarrhoea.

Aetiology and Pathophysiology

- PPR is caused by a virus of the family Paramyxoviridae, genus Morbillivirus.
- In 2016 the virus name was changed from Peste des Petits Ruminants virus (PPRV) to Small ruminant morbillivirus (SRM). However, PPRV is still commonly used by people working in the field.
- The virus through nucleic acid sequencing is differentiated into a single serotype that has four lineages (1–4).
- The virus is antigenically similar to rinderpest virus, measles virus and canine distemper virus.
- Virus is susceptible and is inactivated by alcohol, ether and common detergents and disinfectants like phenol and 2% Sodium hydroxide (Jik). The virus can survive for long periods in chilled and frozen tissues.
- PPR virus enters the body through the respiratory tract and spreads throughout the body via both the lymphatic and vascular systems. As the PPR virus is both lympho- and epithelio-tropic, infection usually results in conjunctivitis, rhinotracheitis, ulcerative stomatitis, gastro-enteritis and pneumonia.

Epidemiology and Transmission

- PPR was first described in 1942 in Côte d'Ivoire, West Africa. Since then the disease has spread to the rest of Africa, the Middle East, Asia and Europe.
- Based on the World organisation for Animal Health (WOAH) reports, the disease is found in more than 70 countries globally which are home to 1.7 billion heads or 80 % of the global population of sheep and goats.
- WOAH estimates that PPR when introduced into a new area can cause a high morbidity mortality rate of 90 and 70% respectively. In endemic areas, morbidity rates are lower at 30% while mortality rates can be as high as 50% in young lambs and kids that are under 1-year-old.
- Sheep and goats that recover from PPR develop an active immunity and have antibodies that last for more than 4 years. Studies indicate that PPR immunity may be life-long.
- In Turkana county livestock keepers in the year 2022 estimated that PPR had an apparent annual morbidity rate of 20%, mortality rate of 14% and case fatality rate of 65%.
- PPR virus is found in tears, nasal discharge, coughed secretions and milk. Transmission is mainly through direct contact with aerosol droplets between animals living in close proximity and indirect transmission through contaminated fomites such as feed, pasture and water troughs.
- There is no disease carrier state.
- PPR outbreaks are common during the rainy season or the dry cold season that coincides

with increased local livestock movement or trade.
- PPR is endemic in Turkana county with cases reported all year round. However, explosive outbreaks occur during the short dry season (AIT/NAIT).

Clinical signs
- Incubation period is between 3 and 6 days. WOAH has set the period for trade purposes to be 21 days.
- PPR clinical severity depends on PPRV lineage, age (young have a more severe disease), host species (severe in goats) and breed (indigenous breeds less susceptible) and the health status (nutrition level and parasitism).
- Infected animals present clinical signs similar to those that were once seen with rinderpest (RP) in cattle. However, in PPR there are two differing signs, development of crusting scabs along the lips and pneumonia in later stages of the disease.
- PPR has various clinical forms described in literature. Peracute form that is frequent in goats from immuno-naïve herd. Animals develop a high fever (40-41°C) are depressed and die in 1 or 2 days. This form has high mortality.
- Acute form is characterised by fever (40–41°C) for 3–5 days, depression, anorexia, serous ocular and nasal discharge that becomes mucopurulent and profuse. The discharges crusts over and occludes the nostrils and eyes causing respiratory distress. The mucopurulent discharge may persist for up to 14 days. Within 4 days of onset of fever, the gums become hyperaemic, and erosive lesions develop in the oral cavity with excessive salivation. Severe, watery, blood-stained diarrhoea is common in later stages that also occurs with bronchopneumonia that is evidenced by coughing and abdominal breathing. Abortions may also occur. Dehydration, emaciation, hypothermia and death may occur within 5–10 days. Survivors undergo long convalescence.
- Sub-acute form- common in endemic areas animals develop fever and serous ocular and nasal discharge, diarrhoea and if severe the animal may develop dehydration and prostration. Mortality is low.

Post-mortem lesions
- Erosive stomatitis of lower lips, gum, free portion of the tongue, hard palate, pharynx and upper third of the oesophagus in severe cases. In Turkana county, livestock keepers described the oral lesions as those seen when one consumes a hot liquid like tea.
- The rumen, reticulum and omasum rarely have lesions most lesions are found in the small and large intestine.
- There are streaks of haemorrhages and sometimes erosions: in the first portion of the duodenum and the terminal ileum. Severe cases have necrotic or haemorrhagic enteritis with ulceration of Peyer's patches.
- Linear haemorrhages of the lining of the cecum and colon give a stripe appearance called "zebra striping" of the large intestine.

PPR eradication
- WOAH and FAO, in their joint strategy for control and eradication of PPR, have set the goal of eradicating the disease by 2030. The strategy has three components, (i) the technical step-wise approach (Stage 1 to Stage 4) to control and eradicate the disease (ii) the strengthening of Veterinary Services in order to be able to carry out the technical component and (iii) the control of other priority small ruminant diseases like CCPP and SGP together with PPR in view of increasing the impact of the control efforts.

Diagnosis
- Field based diagnosis- PPR rapid diagnostic test using ocular conjunctival or nasal swabs.

Confirmatory diagnosis
- Specimens to be collected include whole blood and tissue samples such as lymph node (mesenteric and bronchial), spleen, and lung for antigen isolation and identification using PCR and ELISA for antibody analysis. ELISA does not differentiate antibodies due to vaccination or disease.

Differential diagnosis
- Contagious caprine pleuropneumonia
- Coccidiosis
- Pasteurellosis (may occur as secondary infection to PPR)
- Contagious ecthyma
- Foot and mouth disease

Treatment
- Viral diseases have no treatment. Treatment is mainly to prevent adverse effects of secondary bacterial infection with the main bacteria involved being Pasteurella. Broad spectrum antibiotics like long acting Oxytetracycline, Penicillin streptomycin and Sulphonamides may be used at the recommended dosage given by the manufacturer.

Control
- Vaccination is the most effective method of controlling PPR. Isolation, quarantine and movement control are used to limit its spread during an outbreak.
- KEVEVAPI has a vaccine called PESTEVAX™

Ethnoveterinary treatment practices
- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Isolate sick animals
- Migrate out of outbreak area or avoid areas with active PPR outbreak
- Avoid sharing of pasture of watering points with sick herds
- Report outbreak to CDR, ward administrator or government vet officer
- Present animals for vaccination

Zoonotic potential – None

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Figure 99

Buccal lesions, including vesicular to erosive lesions in an 8 month lamb (Left) and mouth lesions showing areas of dead cells in a goat (Right)

Lesions commonly seen during the early stages of the diseases

Picture source:
Mohebbi et al (2018). Report of an Outbreak of Peste Des Petits Ruminants in Iran, Clinical, Epidemiological and Pathological Study. Recognizing PPR: A field manual http://www.fao.org/3/x1703e/x1703e00.htm

Figure 100

Nodular lesions around the mouth are a common finding in goats in the later stages of PPR infection

Picture source:
Recognizing PPR: A field manual http://www.fao.org/3/x1703e/x1703e00.htm

Figure 101

"Zebra striping" in the large intestine due to linear haemorrhage along the tips of the folds of the lining of the caecum and colon.

Picture source:
Recognizing PPR: A field manual http://www.fao.org/3/x1703e/x1703e00.htm

NB: This is not an endorsement of the product

Figure 102
PPR rapid test kit/ pen side kit

SPECIES:	Sheep and goat (with additional field evidence for camel and antelope)
RAPID:	Results within 20 minutes
EASY:	Requires no laboratory equipment
SIMPLE:	Only requires swab sample from eyes or nose of animal
DETECTS:	All four known genetic lineages of PPRV
VALIDATED:	With experimental and field samples
SENSITIVE:	84% (relative to PCR) and can detect before clinical signs
SPECIFIC:	95% (100% with ocular sample)

6.6 Sheep and Goat pox (SGP) - ETUNE

Case definition

- Sheep pox and goat pox are viral diseases of sheep and goats characterised by fever and sudden appearance of generalised skin nodules that extend into internal organs (particularly the lungs) and death.

Aetiology, Epidemiology and Transmission

- Sheep pox virus (SPPV) and goat pox virus (GTPV) are the causative agents of SGP and they belong together with lumpy skin disease virus to the genus Capripox virus from the family Poxviridae.
- SGP is endemic in Africa, the Middle East, Asia and Southern Europe countries of Bulgaria, Kazakhstan, Mongolia, Greece and Russia.
- Based on WOAH reports, SGP in endemic areas has a morbidity rate of morbidity rate: of 70 to 90% and mortality rate of 5 to 10%. In immune naïve herds with exotic breeds, mortality can reach 100%.
- All breeds of domestic and wild sheep and goats are susceptible to infection.
- Direct transmission occurs through the aerosol route while indirect transmission occurs through contact with contaminated fomites or through mechanical routes via bites of insects or iatrogenic through hypodermic injection.
- Infection results in solid and enduring immunity. In Turkana county, livestock keepers estimated an apparent annual morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25%.
- SGP is endemic in the county with most outbreaks occurring during the long rains (AKIPORO).

Clinical signs

- Incubation period is between 8 and 13 days
- Fever, dullness and Inappetance rarely anorexia
- There is development of rhinitis and conjunctivitis that manifests as increased mucopurulent nasal and ocular discharge.
- There is enlargement of all the superficial lymph nodes, in particular the prescapular lymph nodes.
- Breathing becomes laboured and noisy due to pressure on the upper respiratory tract from the swollen retropharyngeal lymph nodes and developing lung lesions.
- Development of widespread nodular skin lesions that are readily seen on the muzzle, ears and areas free of wool or long hair. Palpation of animal detects skin lesions not readily seen.
- If affected animal survives the acute phase of the disease, the skin lesions become necrotic and form scabs that persist for up to 6 weeks, leaving small scars. The skin lesions are susceptible to fly strike.

Post mortem lesions

- Skin, oral and respiratory mucous membranes and organs have the characteristic nodules. Nodules are seen on the surface of the lungs, kidney, liver and testicles.
- All superficial and internal lymph nodes are enlarged.

Diagnosis

- Field based diagnosis- clinical signs can be used as tentative diagnosis
- Confirmatory- biopsy skin lesions and whole blood in live animals and lymphnodes and affected tissue samples like lungs for dead animals. ELISA for antibody detection should be done within the first week after clinical signs begin. After that PCR diagnosis to detect viral genome.

Treatment

- Viral diseases have no treatment. Treatment is mainly to prevent adverse effects of secondary bacterial infection with the main bacteria involved being Pasteurella. Broad spectrum antibiotics like long acting Oxytetracycline, Penicillin streptomycin and Sulphonamides may be used at the recommended dosage given by the manufacturer.

Control

- Quarantine and movement control during outbreaks
- Isolate sick animals
- Vaccination is the most effective control strategy. Live, attenuated virus vaccines induce longer immunity than inactivated virus vaccines. KEVEVAPI has a SGP vaccine called S&G Vax™.

Ethnoveterinary treatment practices

- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals
- Present animals for vaccination
- Report outbreak to authorities

Figure 103
Lung lesions in sheep and goat pox

Picture source:
Flock&herd at flockandherd.net.au

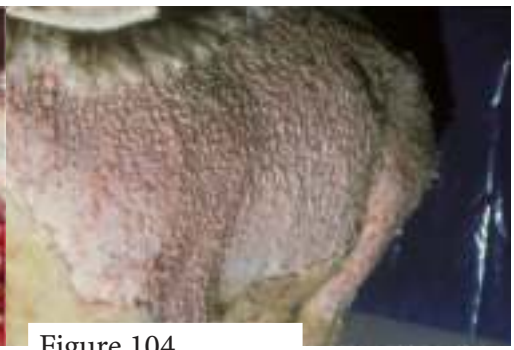

Figure 104
sheep pox skin lesions

Picture source:
Hurisa et al., 2018

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Figure 105
SGP lesions in a goat kid and adult in Marsabit county

Picture source:
Diba Denge

Figure 106
Early SGP skin lesions in a goat in Marsabit county

Picture source:
Elema Bule, AHA ILLeret Ward, Marsabit County

6.7 Foot and mouth disease (FMD)

Refer to chapter 4: Cattle diseases, section 4.1 Foot and Mouth Disease (FMD)

Ethnoveterinary treatment practices

- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals and avoid areas with outbreaks
- Present animals for vaccination
- Do not buy animals from the markets during an outbreak (especially goats as they do not show clinical signs but are carriers of disease). Disease in cattle is more severe when compared to the disease in sheep. Goats are more resistant to FMDV as they show no or less severe clinical signs.

6.8 Contagious ecthyma (Orf) – NG'IBUORUOK

Other names- scabby mouth, contagious pustular dermatitis, sore mouth

Case definition

- Contagious viral skin and mucosa disease which causes scabby lesions usually around the area of the mouth, lips and nose. Orf virus infects mainly sheep and goats (goats have a more severe disease).
- Orf also occurs in camelids, deer, reindeer, dogs, cats and squirrels.
- Orf is very contagious, it is spread through direct animal-to-animal contact entering through damaged skin. It is often self-limiting but is zoonotic, although is considered more of an occupation hazard for people working with livestock.

Aetiology

- Caused by a virus from the parapoxvirus genus in the Poxvirus family.
- Humans infected with orf virus develop ulcerative lesions or nodules on their hands.

Clinical signs

- Mouth lesions are typically found on the lips, muzzle, and in the mouth.
- Early in the infection, lesions appear as blisters that develop into crusty scabs.
- Sheep and goats may get sores on their lower legs and teats, especially when ewes or does are nursing infected lambs or kids. Young animals may have difficulty nursing and may require bottle or tube feeding. Nursing ewes or does with lesions on their udders may abandon their lambs, and older animals with oral lesions may also require nutritional support.
- Except in rare cases, animals recover completely from sore mouth infections within a month. Particular breeds, especially Boer goats, may be especially susceptible and may have severe infections.
- Animals can become infected more than once in their lifetime but repeat infections usually occur after a year's time and are generally less severe.

Diagnosis

- Field based diagnosis using clinical signs
- Mouth lesions and whole blood can be submitted for ELISA and PCR analysis

Differential diagnosis

- PPR- in goats especially due to nodular/ scab like lesions around the lip commissures (junction). However, in PPR the animals also have bilateral nasal and ocular mucopurulent discharge and diarrhoea and are very sick.
- Dermatophilosis- especially in sheep, lesions appear as discrete "bottle-brush" / "paint brush or lumpy skin lesions on muzzle and margins of the ears.
- Warts- discrete growth that have a narrow base attachment (neck) to the skin and are not contagious. Only 1 or 2 animals gets warts in a flock.

Treatment

- Treatment of individually infected animals is not necessary unless lesions are severe. In which case topical disinfecting with iodine based products or application of milking salve on mouth and udder lesions can be used.

Oxytetracycline Gentian Violet spray has also been used to prevent fly struck, control secondary bacterial infection and dry scabs.

- Severely affected kids require good nursing care to ensure that they are eating and drinking and can be an antibiotic combination of 200mg/kg BWT procaine penicillin and 250mg/kg BWT dihydrostreptomycin sulphate once a day for 3 days. A petroleum based soft emollient cream can also be applied once a day until lesions resolve. Does and ewes may require one treatment of long acting antibiotic either oxytetracycline or amoxicillin based if they develop mastitis. Oxytetracylcine is not indicated for young animals as the molecule is deposited in bones and teeth (binds to calcium) causing discolouration of teeth and delays bone growth.

Prevention

- Isolate newly purchased animals.
- Ensure young kids drink colostrum.
- KEVEVAPI has a vaccine called ORFVAX. The vaccine is applied to scarified skin on the inside of the thigh. This is done by making 3 to 4 short superficial scratches about ½" apart with a sterile hypodermic needle. These scratches should break the skin but should not cause bleeding. The vaccine is applied using a small piece of cotton wool moistened with the reconstituted vaccine. A mild local reaction occurs within one week and immunity develops within 14 days. Annual revaccination is recommended.

Zoonosis potential- Yes, wear gloves when treating sick animals
Notifiable disease – No

Ethnoveterinary treatment practices

- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Prompt treatment of cases using traditional or conventional methods so as to avoid the disease spreading to other animals
- Prevent animals from accessing thorny browse during the rainy season.

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2. MSD Veterinary Manual https://www.msdvetmanual.com/

Figure 107
Ten weeks old lamb with severe orf before and after 2 week Penstrep treatment

Figure 108
Contagious ecthyma mouth lesions in goats

Figure 101 shows orf infection in goats. Clinical picture of orf on the oral commissures of an adult goat (A), which recovered completely after 4-5 weeks (B) after first appearance of the clinical lesions. Proliferative Orf lesions on the gums and tongue in a 1-week-old kid in a small goat flock owned by a farmer in Bidar, India (C). Lesions have covered the entire anterior and lateral portion of the tongue (T), while lesions on the lower jaw (J), particularly on the gum (G) portion, have covered the entire lower incisors. Two kids with oral lesions died 3-4 days after showing clinical signs.

Picture source:

Hosamani, Madhusudan & Scagliarini, Alessandra & Veerakyathappa, Bhanuprakash & Mcinnes, Colin J & Singh, Raj. (2009). Orf: An update on current research and future perspectives. http://dx.doi.org/10.1586/eri.09.64

6.9 Progressive Interstitial Pneumoniae (PIP)/Chronic Interstitial Pneumonia (CIP)

Other names: Maedi, Maedi-Visna, Zwoegersiekte, La bouhite, Graaff-Reinet disease

Case definition

- A chronic viral disease in sheep that clinically manifests as progressive interstitial pneumonia characterised by dyspnoea (Maedi) and wasting (Visna). Visna is the neurologic form of the disease in sheep, resulting initially in unilateral pelvic paresis that progresses to hind limb paralysis. In goats, a similar chronic disease called Caprine Arthritis Encephalitis (CAE) affects the nervous system and joints resulting in progressive arthritis and neurological signs.
- In both CIP and CAE clinical disease is not treatable and animals have to be culled for lack of production or animal welfare reasons.

Aetiology and Epidemiology

- Caprine arthritis-encephalitis (CAE) and Maedi-Visna (MV) are persistent lentivirus infections of goats and sheep. The viruses are collectively called the Small Ruminant Lentiviruses (SRLVs).
- Reported seroprevalence for (CIP/PIP) in sheep varies widely from 1 to 50%. In the Western parts of North America, a seroprevalence of 49% has been reported, in north Atlantic region a prevalence of 9% has been documented and in Laikipia Kenya a prevalence of 29% is reported.
- CAE has a worldwide distribution with most studies reporting a high prevalence level of above 70%.
- The causal lentivirus, persists in lymphocytes, monocytes, and macrophages of infected sheep and goats in the presence of a humoral and cell-mediated immune responses.
- Seropositive sheep and goats must be considered infected and capable of transmitting the virus. Transmission occurs via the oral route, usually by ingestion of colostrum or milk that contains virus particles, or by inhalation of infected aerosol droplets. Intrauterine infection may also occur.
- Direct contact with blood or other body secretions, which might contain free viral particles and infected macrophages or epithelial cells may also occur

Clinical signs

- CIP/PIP clinical signs rarely occur in sheep under 2-years-old and are most common in sheep over 4 years old. The disease progresses slowly, with wasting and increasing respiratory distress as the main signs. Coughing, bronchial exudate, depression, and fever are seldom evident unless secondary bacterial infection occurs. A non- inflammatory, indurative mastitis (udder becomes hard and firm) may occur. The encephalitic form of visna causes head tilt and circling, whereas the spinal form causes unilateral pelvic limb proprioceptive deficits progressing to paresis and eventually to complete paralysis.
- Caprine arthritis encephalitis (CAE) is a multisystemic contagious viral disease of goats. The disease is typically spread from mother to kid through the ingestion of colostrum or milk. CAE virus may also be spread among adult goats through contact with body secretions including blood and faeces of infected goats.
- There are 5 major forms of CAE in goats: arthritis, encephalitis (inflammation of the brain), pneumonia, mastitis, and chronic wasting. The arthritic form of the disease

is most common in adult goats, while the encephalitic form is common in kids. The chronic wasting form of the disease can occur either separately or in addition to any other form of CAE.

- Arthritic CAE signs in adults include- Lameness (may be sudden), stiffness, reluctance to walk, abnormal posture, reluctance to rise, weight loss, swollen joints and walking on knees
- Encephalitic CAE seen mostly in kids 1 to 6 months old signs include- Incoordination, inappropriate placement of limbs, progressive paralysis, depression, blindness, head tilt, seizures and death.
- Pneumonic CAE is common in adults, signs include- deep, chronic cough, difficulty breathing and weight loss.
- Mastitic CAE signs include- hard, swollen udder(s), decreased milk production
- In addition, CAE virus may also cause a chronic wasting disease in which goats continue to lose weight although appetite is unaffected.

Post-mortem lesions

- Macroscopic lesions of CIP/PIP are confined to the lungs and associated lymph nodes. The lungs do not collapse when the thorax (with obvious rib indentations) is opened and are abnormally firm and heavy (~2 kg; 2–4 times normal weight). Early lung changes may be difficult to detect, but later in the disease, lungs are mottled by gray and brown areas of consolidation. The mediastinal and tracheobronchial lymph nodes are greatly enlarged and oedematous. All lesions are progressive and result from the cellular immune response of the host, and not directly from viral damage.
- CAE gross lesions include swelling of carpal joints due to increased synovial fluid, and enlarged and hard joint heads, there is also articular cartilage ulceration on the joint cavity, and severely swollen mesenteric lymph nodes. In the neurological form there is asymmetric, brownish pink, swollen areas, most commonly located in the cervical and lumbosacral spinal cord segments.

Diagnosis:

- Field based diagnosis using clinical signs and post mortem lesions.
- Confirmatory diagnosis is collection of whole blood in live animal for ELISA and PCR testing or post mortem collection of lung, lymph node and joint tissues for virus genetic identification through PCR.
- Differential diagnoses of progressive pneumonia include pulmonary adenocarcinoma, pleural abscesses, and pulmonary caseous lymphadenitis. Listeriosis, scrapie, rabies cerebrospinal nematodiasis, and space-occupying lesions should be considered when the neurologic form (visna) of the disease is seen.
- Differential diagnosis for progressive paresis and paralysis exhibited by young kids should include enzootic ataxia, spinal cord abscess, cerebrospinal nematodiasis, spinal cord trauma, and congenital anomalies of the spinal cord and vertebral column. If a neurologic examination indicates brain involvement, polioencephalomalacia, listeriosis, and rabies should be considered. The pulmonary form of caseous lymphadenitis may have a similar clinical presentation to the pulmonary form of CAEV in adult goats.

Control

- Currently, there is no practical, effective treatment, and no vaccines are available. Therefore, the only means for control and prevention is serologic testing and culling of positive animals. This is not practical in Turkana county and animal service providers

should advise livestock keepers to cull animals with CIP or CAE as they will continuously shed the virus in the environment and pass it to off springs.
- Because of the long incubation period and time to seroconversion once signs appear it is advisable to cull animals.
- Pregnant does and ewes showing signs once they deliver young. The livestock keeper should immediately separate neonates from dam to prevent suckling of colostrum.
- Isolated dams should be fostered to other health dams or fed on heat-treated colostrum, and later raised on milk replacer.

Zoonotic potential- None identified

Notifiable disease- Yes, WOAH listed disease, report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Cull sick animals as there is no treatment and the longer the sick animal stays in the flock the more it infects other.

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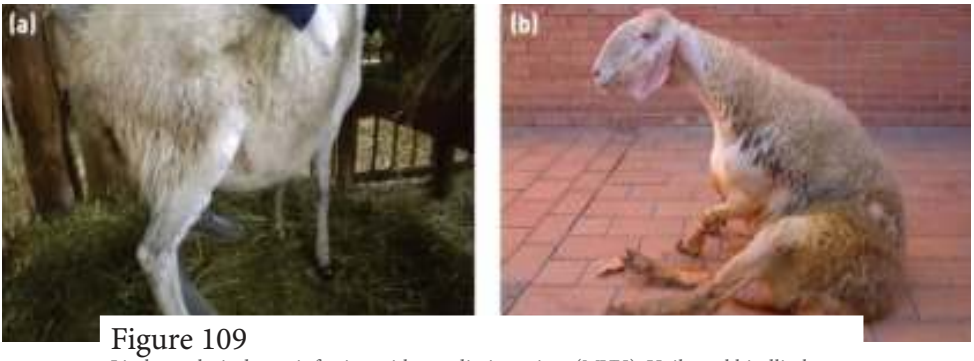

Figure 109

Limb paralysis due to infection with maedi-visna virus (MVV). Unilateral hindlimb paresis (a) early stages of the disease and later ataxia and hindlimb paralysis (b).

Picture source:

Diagnosing limb paresis and paralysis in sheep (2015) http://dx.doi.org/10.1136/inp.h5547

Figure 110

Appearance of a goat infected with CAE (advanced stage) in which the animal is hitting its head against the pen wall (left picture), has depressive behaviour (middle picture), and has joint inflammation (right picture).

Picture source:

Short communication: Genetic parameter estimates for caprine arthritis encephalitis in dairy goats (2020) https://doi.org/10.3168/jds.2019-17740

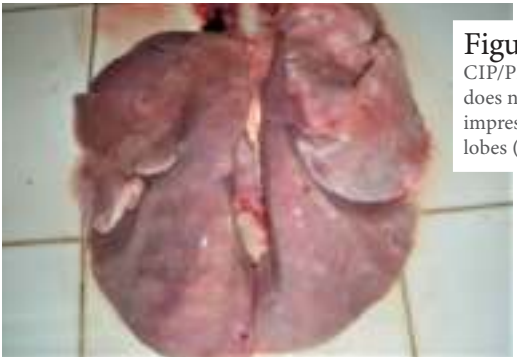

Figure 111

CIP/PIP affected lung is enlarged and heavy and has does not collapse when thoracic cavity is open. Note rib impressions on the costal surfaces of the diaphragmatic lobes (black arrow)

Picture source:

Ovine Progressive Pneumonia: Diagnosis and Seroprevalence in the South of Sonora, Mexico (2021) https://doi.org/10.1155/2021/6623888

Sheep diseases:
Protozoal

6.10 Babesiosis – LONYANG / LORENG'EKUL

Refer to chapter 4: Cattle diseases, section 4.12 Babesiosis

Ethnoveterinary treatment practices
- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Clear bush and infested pasture through burning
- Migrate out of heavily tick infested areas.

6.11 Coccidiosis- EREMONU ANG'AKOOT

Case definition

- Coccidia are single-celled, microscopic parasites that live and reproduce in animal cells. Coccidia of sheep and goats are host specific and almost all kids and lambs will become infected.
- Significant damage to the cells lining the small intestine, caecum and large intestine results in diarrhoea which may contain mucus or blood. This is often accompanied by straining, pain, weight loss and sometimes death of the lamb and kids.
- Recovered animals have poor growth rates due to damage of epithelial lining of the intestine and reduced ability to absorb nutrients from ingested food.
- Most of the different Eimeria species found in sheep are harmless and may even provide beneficial effects. Distinguishing between an infection with coccidia and the disease coccidiosis is very important in lambs.

Aetiology, Epidemiology and Pathogenesis

- Numerous species of Eimeria species cause disease in goats in Kenya. A study estimated the species prevalence in goats to be E. arloingi (71%) followed by E. ninakohlyakimovae (65%) and E. hirci (59% percent).
- The disease is severe and often fatal in kids.
- In sheep, only two harmful (or pathogenic) species cause clinical disease (Eimeria ovinoidalis and Eimeria crandallis) when there is either a heavy infestation in lambs or they have lowered immunity. Disease is mainly seen in lambs 4 to 8 weeks old and rarely seen in lambs over 3 months old.
- Lambs and kids take in pathogenic coccidia oocysts (eggs) by mouth. Inside the intestine, the oocysts hatch, invade the mucosal intestinal cells and multiply dramatically. The coccidia emerge by bursting the cells open - damaging the inside lining of the intestine. A dramatic number of oocysts are shed in faeces and this can be many million times higher than the number that were ingested. It takes two to three weeks from infection via the mouth to the passing of oocysts from the rectum.

Clinical Signs

- Coccidia are host-specific and each animal species is susceptible to infection with various coccidia species. In goats and sheep, Eimeria spp. are most common.
- The protozoan parasite goes through its life cycle in the small intestine, destroying the epithelial cells.
- Transmission of oocytes is via the faecal-oral route. This process involves oocysts being passed when an animal defecates, and another animal ingests the contaminated substrate.
- Clinical cases vary from loss of appetite and decrease in weight gain to severe cases involving chronic diarrhoea, fluid faeces containing mucus and blood, straining in attempt to pass faeces, loss of weight, and dehydration and death.

Post-mortem lesions

- Lesions are confined to the small intestine, which may appear congested, haemorrhagic, or ulcerated, and have scattered pale, yellow to white macroscopic plaques in the mucosa.

Diagnosis

- Clinical signs of bloody diarrhoea and post mortem lesions
- Microscopic analysis of faeces to observes oocysts.
- In lambs, clinical signs of severe, bloody diarrhoea and straining can be highly suggestive of coccidiosis but the disease should be confirmed. Faecal samples can be taken to identify a significant oocyst count of pathogenic species of coccidia. Samples with a high faecal coccidia count alone may not be significant and they should be speciated to confirm the oocysts are the harmful type at an accredited laboratory. Many lambs have a very high count of harmless oocysts.

Treatment

- Oral solution or bolus of Sulfadiazine400 mg Trimethoprim80 mg at 1 g per 10 kg body weight for 4 - 7 days.
- Extra label use of the poultry product Bayticol® that contains Toltrazuril is effective as a single dose at 1 ml/2.5 kg. It is reported to be very effective at reducing oocyte shedding as the drug is effective at all intracellular developmental stages of the parasite. Poultry products containing Amprolium have also been used effectively in goats.

Control

- Good hygiene is essential in livestock bomas especially where lambing and kidding occurs.
- Feeding troughs should be elevated or located outside the sleeping areas
- Ensure new born kids feed on colostrum as it provides immunity to coccidiosis for the first several weeks of the neonate's life.
- If possible, separate young animals from older animals
- Stress contributes to outbreaks of coccidiosis. Sheep and goats should be handled minimally and handled calmly and gently. It is particularly important to minimize stress at weaning.

Figure 112
Eimeria species unsporulated oocysts in goat kid faecal sample

Picture source:
https://www.boergoatprofitsguide.com/goat-care-for-beginners/

Figure 113
Small intestine of a goat with multiple raised nodules consistent with coccidiosis.

Picture source:
https://tvmdl.tamu.edu/2020/06/15/pathologic-diagnosis-of-coccidiosis-in-goats/

Sheep Diseases:
Parasitic

6.12 Helminthiasis – LOPEELEI / NG'IRITAN / LOBOLIBOLIO

Epidemiology

- Helminths affecting sheep and goats can be divided into nematodes (round worms), trematodes (flukes) and cestodes (tape worms).
- Nematodes include: Haemonchus contortus, Nematodirus species, Bunostomum species, Ostertagia circumcinta, Trichostrongylus species
- Cestodes include: Monenzia expanza is the most common cestode of sheep
- Trematodes include: Fasciola hepatica (liver fluke) is the most common trematode of sheep
- Important helminths of sheep and goats are Haemonchus contortus and liver flukes- Fasciola hepatica.

Haemonchosis- Barber's pole worm
Transmission and Pathophysiology

- H. contortus passes through six stages of life which include egg, four larval stages, and adult. Female adults lay numerous eggs with an average of more than 1200 eggs a day. The eggs are passed through faeces to pastures. The infective larval stage is L3.
- The L3 move out of the faeces, onto the soil, then randomly migrate horizontally and vertically up the grass sward independent of free water. The larvae do not feed, relying on stored energy for survival. In tropical climates like in Turkana county, the survival of L3 is less than five weeks, as the larvae are more active and consume energy stores. This provides an opportunity for spelling of pastures (that is periods without grazing) that can be used as a control method.
- The L3 larvae infective stage of H. contortus are consumed when the animals graze the pasture sward. Once ingested, a range of factors derived from the host sheep will influence survival and establishment of the L3 larvae within the abomasum. These include age, breed, health, and nutritional status of the animal as they impact on an individual's animal ability to mount an immune reaction to the H. contortus larvae.
- In the abomasum, the larvae burrow into the mucosa into the gastric pits where they use their lancet to cut the abomasal tissue and induce haemorrhages and ingest the leaking blood. The larvae then mature into adults. Female worms start producing eggs 12 to 15 days after ingestion and produce. The infected animals may lose up to 30 µL of blood every day due to one parasite.
- Previous research had indicated that hypobiosis, arrested development of ingested larvae occurred in the host during dry or drought conditions. However, a recent study in West Africa that found a high incidence rate of 77.8 to 100%) with no definite seasonal distinction. There was however, a clear seasonal trend in the worm burden of animals; higher burdens were evident during the rainy rather than the dry season. The study found no significant hypobiosis during the unfavourable dry season (November to March).

Clinical signs

- Clinical signs are associated with anaemia and hypoalbuminemia. The magnitude

and consequences of the anaemia and hypoalbuminemia depend on parasite burden, their stage of development, the age of animal and immune response.

- Haemonchosis clinical syndrome is categorised into three stages; hyper acute, acute, and chronic
- Hyper acute haemonchosis is rare and occurs in animals exposed to heavy burdens of
- H. contortus (up to 30,000 larvae per animal) over a short period of time. This form is common in young animals. Death is sudden with or no previous clinical signs of anaemia. Bloody diarrhoea may be the only sign.
- Acute haemonchosis is the most common form and is characterized by lower burdens of H. contortus (approximately 2000–20,000 larvae per animal), resulting in a less severe loss of blood and anaemia that develops over a longer period of time. Animals show clinical signs associated with anaemia (conjunctiva colour that varies from the normal, red-pink colour to extreme white) and hyperproteinaemia (specifically hypoalbuminemia). The main clinical signs are lethargy, weakness, increased respiratory and heart rates, pale mucous membranes and bottle jaw. Hyperproteinaemia occurs due to blood loss, leakage of protein into the abomasal lumen through disrupted cell junctions and increased permeability and decreased protein absorption due to abomasal epithelial cell loss. There is also increased protein use to repair damaged tissues and mount an immune response. Hypoalbuminemia results in decreased intravascular oncotic osmotic pressure and oedema that may be generalized or localized in the intermandibular space (referred to as "bottle jaw" or cervical region. Diarrhoea is not a key feature of Haemonchus infection unless there is concurrent infection with other helminth species or coccidiosis.
- In chronic or subclinical haemonchosis, sheep and goats ingest few numbers of H. contortus as the environment is not suitable for the development of the infective larvae. Infection manifest as a reduction in productivity, feed conversion and growth rate.

Diagnosis and Post mortem lesions

- Field based diagnosis – clinical signs of anaemia and bottle jaw that is supported with the use of FAMACHA Score card. The score card allows rapid anaemia classification in the field. The FAMACHA system was developed in South Africa and is based on conjunctival mucous membrane colour in small ruminants on a scale of 1–5 with various stages of anaemia related to haemonchosis with their measured Packed Cell Volume (PCV). A value of 1 (red) is considered non-anaemic and a PCV of 35%, 2 a PCV of 25%, 3 a PCV of 20%, 4 a PCV of 15%, through to a value of 5 (white) and a PCV of 5%, being severely anaemic.
- Post-Mortem diagnosis- Examination of dead small ruminants is another rapid, accurate method of confirming a diagnosis, as it allows the visualization of the H. contortus parasites in the abomasum of affected animals. The parasites have a characteristic red and white, twisted "barber's pole" appearance related to the blood-filled intestine spiralling around the white reproductive tract of the female nematodes.
- Confirmatory diagnosis- Confirming the presence of parasites through faecal egg counts and larval culture and haematology testing of EDTA blood for anaemia indicators: that include Complete Blood Cell Count (CBC), PCV and evaluation of Mean Corpuscular Volume (MCV) and Mean Corpuscular Haemoglobin Concentration (MCHC) and reticulocyte (immature RBC) counts. Molecular methods as a confirmatory test have a high sensitivity but have limitations due to the inhibition effects of faeces on the polymerase chain reaction (PCR) and their accessibility to veterinarians in the field. Development of suitable PCR tests is on-going, with droplet digital PCR showing promise. using the McMaster method for faecal egg count has a specificity but a lower sensitivity when compared to molecular methods.

Treatment and control

- Widespread anthelmintic resistance limits the effectiveness of both treatment and prevention of haemonchosis. Successful management of haemonchosis relies on the early recognition of infection through periodic monitoring of H. contortus burdens (FAMACHA score and or faecal egg count), strategic use of anthelmintics and grazing management.
- Regular deworming with broad-spectrum anthelmintics 2 weeks after the onset and at the end of the rainy seasons. Proper dosing based on manufacturers instruction that is guided by the weight of animal is important. Use of a weigh band can help minimise under dosing or overdosing incidences.
- Broad-spectrum anthelmintics currently available belong to five different chemical groups:

1) Benzimidazoles (white or blue drenches) like fenbendazole, oxfendazole and albendazole,
2) Imidazothiazoles (yellow drenches) like levamisole
3) Macrocyclic lactones (clear or watery drenches or injectable like Ivermectin and doramectin.
4) Amino-acetonitrile derivatives-is not readily available as it a new anthelmintic compound (2010). Monepantel effectively controls adult and L4 stages of ruminant nematodes, including multidrug resistant helminths.
5) Spiroindoles like Derquantel has been approved as a combination anthelmintic product with abamectin. This is also a new anthelmintic that is not readily available. The combination is effective against adult and L4 stages of ruminant nematodes that have shown multi-drug resistance.

- In Turkana county, strategic use of anthelmintic can be used due to the high cost of treating the large small ruminant herds. Animals with FAMACHA score of 3 and above should be the only ones treated at the correct dosage. In addition, the timing of treatment should be at the beginning of the rainy season and at the end. Pasture management such as pasture rotation to allow desiccation of larvae to high environmental temperatures and delaying animals from accessing pasture when there is morning dew are good strategies used by livestock keepers.
- Barbervax®, a vaccine containing Haemonchus contortus gut membrane glycoprotein antigens have been developed and efficacy trials conducted. Results indicated that naturally infected vaccinated sheep had significant egg count reductions compared with controls. Vaccination also prevented the periparturient rise in egg shedding of ewes, as well as egg shedding in lambs.

Ethnoveterinary practices

- See tables 17 and 18

Ethnoveterinary control practices- Delay animals grazing early morning during the rainy season, avoid contaminated pastures.

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4. Teixeira, M., Matos, A.F.I.M., Albuquerque, F.H.M.A. et al. Strategic vaccination of hair sheep against Haemonchus contortus. Parasitol Res 118, 2383–2388 (2019). https://doi.org/10.1007/s00436-019-06367-x

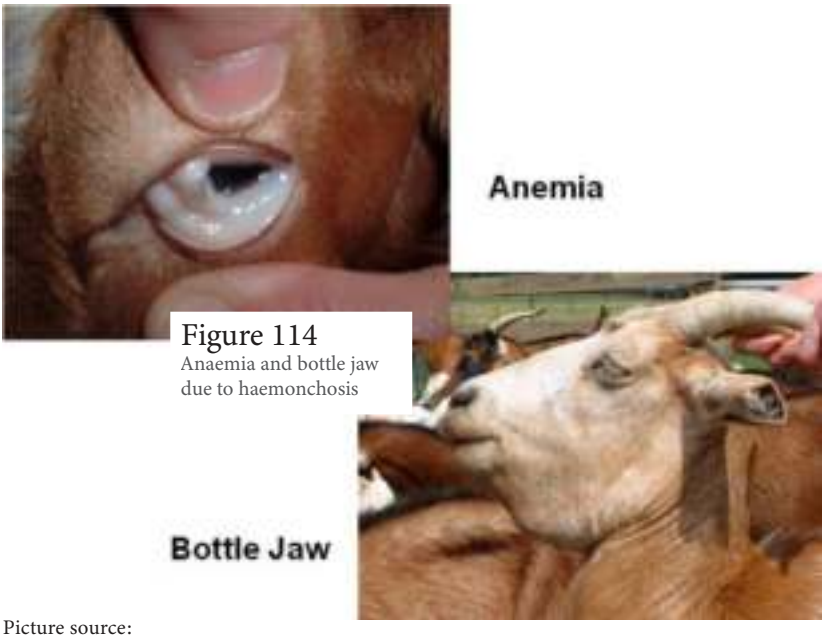


Figure 114
Anaemia and bottle jaw due to haemonchosis

Picture source:
Anaemia and bottle jaw due to haemonchosis
Photo credit- MSD

Figure 115
Haemonchus contortus parasites in the abomasum of an affected lamb, demonstrating the characteristic red and white twisted "barber's pole" appearance.

Picture source:
Professor K G Thompson, Massey University, New Zealand

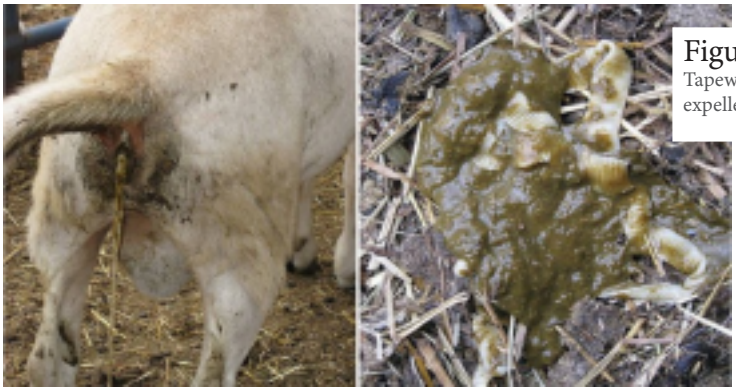


Figure 116
Tapeworm segments being expelled with diarrhoea in a goat

Picture source:
Dr. Ann Zajac Virginia-Maryland College of Veterinary Medicine

Oestrus ovis larvae cause cavitary myiasis in small ruminants. The larvae are an obligate parasite of the nasal and sinus cavities of sheep and goats. In endemic areas, the O. ovis larvae have been found in humans' eyes and nasopharyngeal airway. The female fly (adult female fly (Oestrus ovis) commonly known as the sheep nasal bot fly. Flies around the head of its host to deposits larvae at a few centimetres from sheep nostrils. In case of the fly presence, the animals shake their heads and hide the muzzle on the soil or in the others sheep's wool. Once deposited the larvae enter the nostrils where they use the hooks and spines to provoke inflammation and mucous nasal discharge which the larvae feed on. Sheep infested with larvae are often seeing sneezing and have muco purulent nasal discharge. Animals may also lose weight. Treatment involves injecting Ivermectin at a dose rate of 0.2mg/kg BWT given SC once or repeated after 2 weeks if the clinical signs persist. In heavy infestation diagnosed through post mortem of the nasal cavity a combination of Ivermectin and closantel (10mg/ kg orally) can be used.

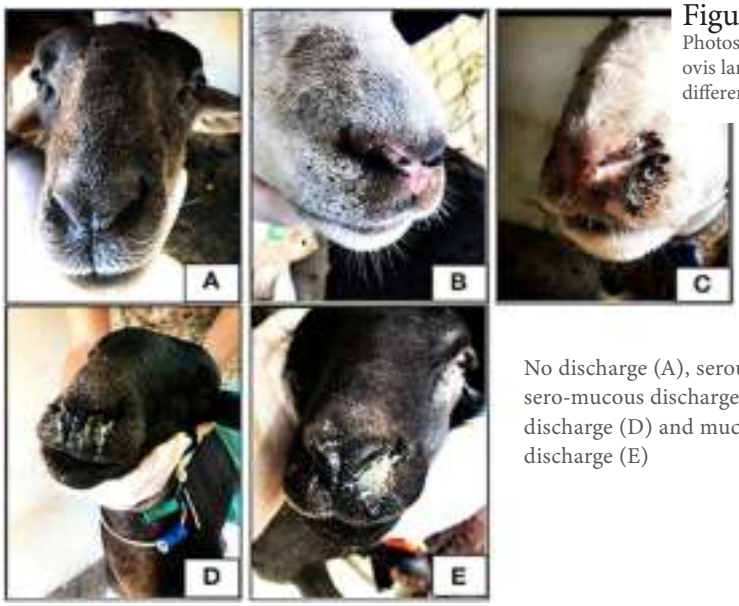


Figure 117
Photos of lambs with oestrus ovis larva infestation showing different nasal discharge scores.

No discharge (A), serous discharge (B), sero-mucous discharge (C), thick mucous discharge (D) and mucopurulent thick discharge (E)

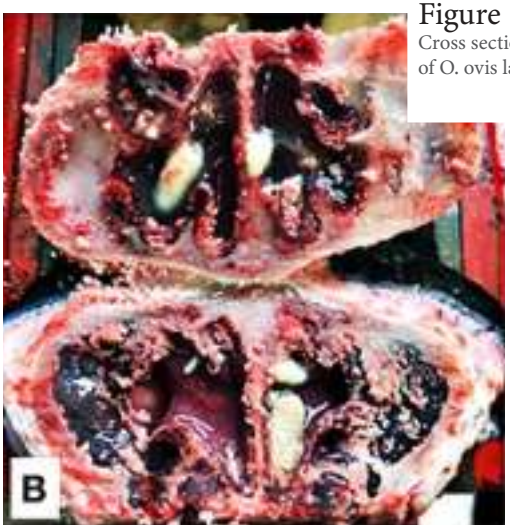


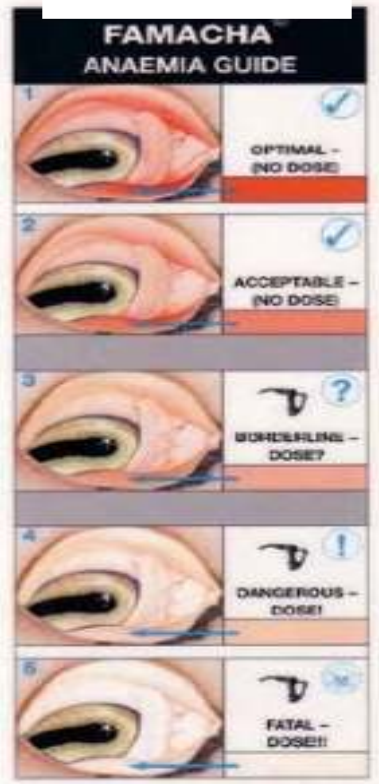
Figure 118
Cross section of the nasal sinuses for the recovery of O. ovis larvae from naturally infested lambs (B)

Picture source:
Prophylactic Effects of Ivermectin and Closantel Treatment in the Control of Oestrus ovis Infestation in Sheep (2022) https://doi.org/10.3389/fvets.2021.798942https://doi.org/10.3389/fvets.2021.798942

Figure 119
Microscopic appearance of helminth eggs in ruminants

Picture source:
https://link.springer.com/10.1007/978-3-642-27769-6_4307-1

Figure 120
FAMACH score card showing when to administer anthelmintics



Picture source:
https://u.osu.edu/sheep/2010/06/16/use-famacha-correctly/

6.13 Coenurosis – LOKOU / LOIRA

Case definition

- Fatal parasitic disease affecting sheep and goats. It is caused by the larval stage of Taenia multiceps, the adult tapeworm is found in the intestines of dogs. Sheep and goats are intermediate hosts and do not support the larvae that hatches from the eggs to mature to adult forms. Instead the aberrant larvae migrate to the nervous tissue and forms cysts in the brain.
- The larval stage, Coenurus cerebralis, is typically found in the central nervous system (CNS) of wide range of livestock. Cysts in sheep are mainly in the brain and spinal cord. However, in goats, in addition to the central nervous system (CNS) they can have cysts in musculoskeletal system, subcutaneous, connective tissue, peritoneal and pelvic cavities, the liver and the lungs.
- Zoonotic parasite as evidenced by multiple cases of coenurosis in humans caused by T. multiceps.

Aetiology and Epidemiology

- The causative agent is Coenurus cerebralis, the larval stage of Taenia multiceps, a tapeworm that infests the small intestine of carnivores.
- Contamination of pastures grazed by sheep by dog faeces can result in larval invasion of the central nervous system and clinical disease. The life cycle is completed when the carnivorous definitive host ingests infested sheep's brain.
- In Turkana county, livestock keepers in the year 2022 estimated an apparent morbidity and mortality rates 1% and a 100% case fatality rate.
- In Turkana county the disease can occur at any time of the year, but livestock keepers indicate the signs manifest more during the long dry season especially when animals are watered after a period of water depravation.

Pathophysiology

- The adult tapeworm, Taenia multiceps, inhabits the dog guts where it produces eggs that are defecated onto pasture. The eggs can survive on pasture for several weeks.
- Sheep and other intermediate hosts become infected by consuming pasture contaminated with Taenia multiceps eggs.
- The eggs hatch into larvae within the gut of the intermediate host. The larvae migrate into the brain of the intermediate host, a process that may last several months, where they form cysts. The symptoms associated with Coenurosis vary with the location and size of the cysts as well as with the pressure the cysts on the cerebrum.
- Molecular evaluation of cerebral and non-cerebral coenuri from sheep and goats established that the larval stage of Taenia multiceps in the brain and other aberrant sites are different. Larvae found away from CNS in goats are now called Taenia gaigeri and Taenia skrjabini in sheep.

Clinical signs

- Circling in one direction, Tilting, lifting or lowering the head
- Constant loud bleating and excitement,
- Weight loss
- Cysts found on post mortem on brain or spinal cord and in goats in other tissue.

Diagnosis	Treatment	Control
Diagnosis is confirmed on necropsy by the presence of Coenurus cerebralis cysts in the brain of sheep. Clinical signs alone cannot be used as a diagnostic method. The best diagnostic method that is not feasible in Turkana is clinical signs combined with accurate localization of the cyst using diagnostic imaging.	Treatment with medication is not usually successful, farmers resort into culling affected animals	The most effective means of controlling coenurosis, is through the regular anthelmintic treatment of farm dogs with an effective taeniacide (i.e. praziquantel at 5 mg/kg body weight) at 6- to 8-week intervals and the proper disposal of sheep carcasses to prevent scavenging by herding and stray dog and wild carnivores Most of the naturally occurring cases of clinical coenurosis in sheep have been observed in young animals. Suggesting the existence of an age-related resistance to T. multiceps infection in sheep, although the mechanism by which this may occur is unknown. There is a vaccine under development.

Differential diagnosis

- Listeriosis, nasal bots syndrome, louping ill, scrapie, sarcocystosis, polioencephalomalacia and cerebral echinococcosis.
- The age of an infected animal may aid diagnosis, since the symptoms of cerebral coenurosis typically manifest in young animals; however, immunocompromised animals as well as animals raised in isolation may develop coenurosis when adults.

Zoonotic potential- Yes for only coenurosis

Notifiable disease- No

Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Cull affected animals

References

1. Varcasia, A., Tamponi, C., Ahmed, F. et al. Taenia multiceps coenurosis: a review. Parasites Vectors 15, 84 (2022). https://doi.org/10.1186/s13071-022-05210-0

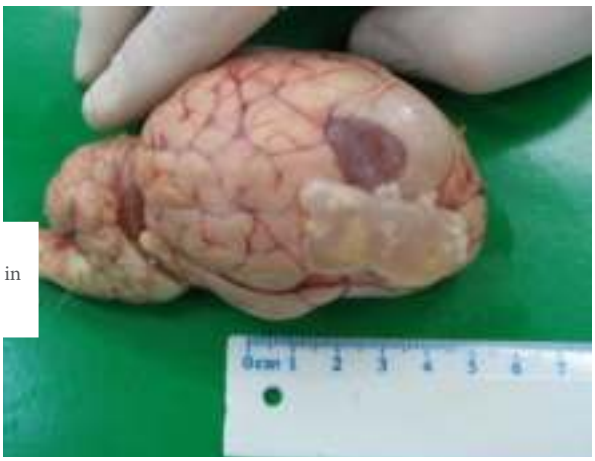


Figure 121
Coenurus cerebralis larval cyst in sheep brain.

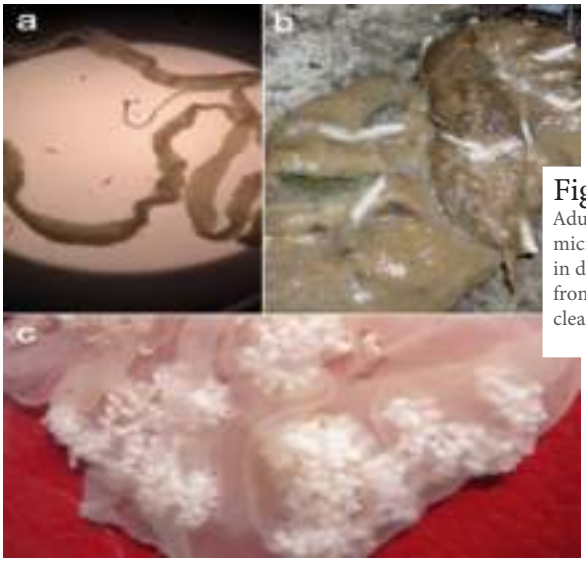


Figure 122

Adult Taenia multiceps under light microscopy. b Proglottids of T. multiceps in dog faeces. c Mature coenurus isolated from sheep; clusters of protoscoleces are clearly visible without magnification

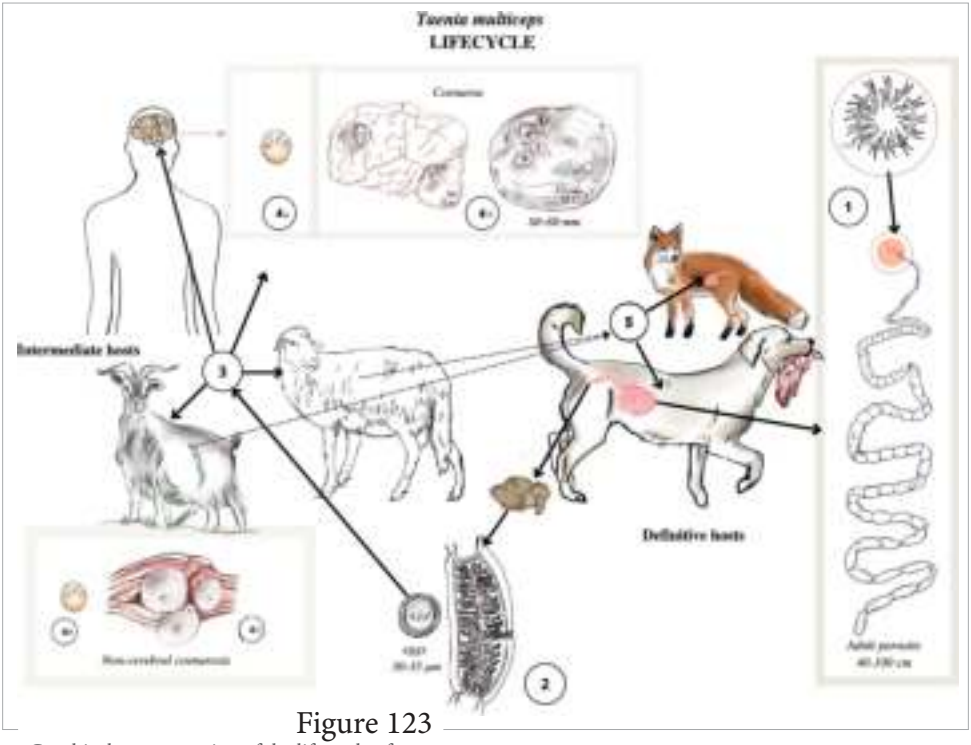


Figure 123

Graphical representation of the life cycle of Taenia multiceps

Picture source:
Varcasia, A., Tamponi, C., Ahmed, F. et al. Taenia multiceps coenurosis: a review. Parasites Vectors 15, 84 (2022). https://doi.org/10.1186/s13071-022-05210-0

6.14 Mange- LOTOME / EKOIKOI / LOGIRIGIRA/ LOIR

Case definition

- Mite infestations cause skin irritation leading to oozing (exudation) and skin thickening. The intense irritation leads to disruption of feeding patterns and weight loss.
- Mange occurs worldwide and in all seasons of the year in the tropics.

Aetiology

The most important parasitic mite species of sheep are:

- Psoroptes (P) ovis that causes psoroptic mange, also called sheep scab occurs worldwide.
- Sarcoptes (S) scabiei var. ovis that causes sarcoptic mange, also called scabies occurs worldwide.
- Chorioptes (C) ovis that causes chorioptic mange, also called leg mite, foot scab occurs worldwide.
- Demodex (D) ovis, responsible for sheep demodectic mange

The most important parasitic mite species of goats are:

- S scabiei var caprae
- C. bovis
- Demodex caprae infests goats.
- In Turkana county, livestock keepers in the year 2022 estimated the apparent morbidity rate of 3% with no mortality. Mange cases occurred through the year but more cases are seen when animals are in nutritional stress during the long dry season (AKAMU).

Clinical signs goats

- This mite infests the head and face. Lesions manifest with formation of crusts and intense pruritus.
- Affected animals have decreased reproduction, meat gain, and milk yield.
- In goats, S scabiei var caprae is responsible for a generalized skin condition characterized by marked hyperkeratosis. Lesions start usually on the head and neck and can extend to the inner thighs, hocks, brisket, ventral abdomen, and axillary region.
- S scabiei var caprae has zoonotic. Consistent with other animal variants of Sarcoptes, zoonoses are initiated from direct contact with infected animals but are self-limiting infestations.
- C bovis in goats is similar to that in cattle, with papules and crusts seen on the feet and legs.

Treatment and Control

- Hot lime sulphur spray or dip is labelled for use against sarcoptic, psoroptic, and chorioptic mites in sheep. Treatment should be repeated every 12 days if needed.
- Certain formulations of synthetic Pyrethroids or organochlorines (Amitraz) are labelled for use in sheep and goats. As with cattle, they are not considered the compound of choice but if used, the animals should be thoroughly wetted with the product and re-treated in 10–14 days.
- Macrocyclic lactones (Ivermectin) is the drug of choice and given as two doses given 7 days apart at a dose rate of 0.2mg/kg BWT given SC.

Pathophysiology and Clinical signs in sheep Psoroptes ovis (sheep scab)

- Adult Psoroptes female mite lays 1 to 3 eggs a day, a total of about 50 to 100 eggs in their lifetime. Adult life lasts for about 50 days. The shortest life-cycle duration from eggs to eggs of the next generation is about 10 to 14 days.
- Psoroptes mites do not dig tunnels in the skin, but the mite faeces cause an allergic reaction of the host's skin, which reacts producing

exudations and skin thickening and hardening (lichenification) with formation of papules, scales and crusts (excoriations), mostly with wool loss.

- The mites feed on the exudates and secretions produced by the affected skin. Large scabs may develop that spread to cover the entire body in 2 to 3 months if left untreated. Mites concentrate at the edge of the growing scabs. Mites are one host parasites.
- Transmission within a herd is mostly by physical contact. Mites do not actively jump or crawl from one host to another one, but are passively transmitted when animals come in close contact. Psoroptic mites and eggs can survive 2 to 3 weeks off the host (in clumps of fallen wool or on inanimate objects like fence posts).
- Sheep scab lesions affect the back, the flanks and the shoulders. Infestations remains unnoticed until wool loss becomes evident, which mostly means that the whole flock is already infested. Affected animals suffer from intense itching (pruritus) and react vigorously scratching, biting and rubbing against objects, which causes injuries that can be infected with secondary bacteria. All this leads to weight loss and wool loss, reduced milk production, and general weakness that makes the affected animals more susceptible to other diseases. Left untreated it is often fatal, especially for lambs. Hides of affected animals are downgraded or rejected at slaughter.
- Diagnosis based on clinical symptoms, confirmed by examining skin scrapings of affected parts under the microscope for visualization of the mites. Psoroptic mites are not infectious to humans.

Sarcoptes scabiei var. ovis

- Sarcoptic mites of sheep are a species-specific strain of Sarcoptes scabiei, a mite species that infests also cattle, pigs, other livestock and also humans. (zoonosis) causes scabies.
- Sarcoptic mites spend their whole life on the same host. Mites do not actively jump or crawl from one host to another one, but are passively transmitted when animals come in close physical contact. However, sheep can pick mites from the immediate environment or fomites. There are no external vectors that transmit the mites.
- The mites dig tunnels beneath the skin. Their saliva has potent digestive enzymes that dissolve the skin tissues. They feed on the resulting liquids. They do not suck blood.
- Adult females deposit their eggs in tunnels, which hatch in 3 to 5 days. The whole development through several larval and nymphal stages can be completed in less than 2 weeks. Adults live for 2 to 3 weeks. Off the host the mites survive only a few days.
- Disease not as severe as that of Psoroptes mites. Mite digging causes skin irritation, which is enhanced by allergic reactions to the saliva. The affected skin develops pimples and papules that become crusty, and shows hardening, thickening, and folding.
- Skin lesions mainly seen in non-wooly skin and frequently start on the head, then later spread to sides of the neck and fore legs.

Chorioptes ovis

- Chorioptic sheep mites (also called "leg mites", or "foot scab") are less harmful when compared to psoroptic or sarcoptic mites. They are not transmitted to humans.
- They have chewing mouthparts and neither suck blood, nor dig tunnels as sarcoptic mites, but bite the outer skin layers and feed on skin debris, fat, lymph or exudates. The whole development through several larval and nymphal stages can be completed in about 32 weeks. Off the host the mites survive only a few days.
- Preferential sites or chorioptic mites are the

hoofs and lower part of the legs.
- Can affect the scrotum, the face and the lips. Affected parts show formation of scales and crusts. Rams are often more affected and spread the disease in the flock, especially if they are permanently confined. Severely infested rams may suffer from partial paralysis and low reproductive performance.
- Itching is not as severe and the scratching and biting reactions of affected animals are less vigorous.

Treatment and Prevention
- Two injections with a macrocyclic lactone (Ivermectin) with 7 to 10 days' interval.
- Topical acaricide and pour-ons are not reliable for controlling psoroptic mites.
- Infected premises/ boma should be kept vacant for at least 2 weeks. This allows surviving mites or eggs to die.
- There are no vaccines that will protect sheep by making them immune to the mites.
- There are no repellents, natural or synthetic that will keep mites away from sheep.

Ethnoveterinary treatment practices
- Refer to tables 17 and 18

Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Isolate sick animals and if possible build a separate night boma for them
- Migrate from boma and leave after you have burned the infested livestock boma

Figure 124
Sarcoptic Mange in a goat showing alopecic areas of the skin

Picture source:
https://www.researchgate.net/publication/319454346_THERAPEUTIC_MANAGEMENT_OF_SARCOPTIC_MANGE_IN_GOAT_A_COMPARATIVE_STUDY_ALLOPATHIC_AND_HERBAL_PRODUCT/figures

Figure 125
Location of ectoparasite in goats indicates if it is a mite or flea infestation

Picture source:
https://www.farmhealthonline.com/US/disease-management/goat-diseases/mange-in-goats/

Figure 126
Sarcoptic mange in a sheep flock

Picture source:
https://link.springer.com/article/10.1007/s12639-012-0157-5

Further Reading
Merck Veterinary Manual. https://www.merckvetmanual.com

Chapter 7:

Goat diseases

Table 20: Priority Goat diseases ranked in order of importance by livestock keepers in Turkana county

Conventional name	Turkana name
Contagious Caprine Pleuropneumonia (CCPP)	LOUKOI
Sheep and Goat pox	ETUNE
Mange	LOTOME / EMITINA
Helminthiasis	LOPELEI / NG'IRITAN
PPR	LOMOO
Anaplasmosis	LONYANG' / LOPID
Foot rot	EKICHODONU
Brucellosis (see Annex 2: zoonotic diseases)	AKIYEC / EDEKE AKIRING KA NG'AKILE
Heart water	AMIL
Contagious ecthyma (Orf)	NG'IBUORUOK
Caseous lymphadenitis	ABUS/ ABUTH / LONG'ARURWEI
Infectious keratoconjunctivitis (Pink eye)	EDEKE ANG'AKONYEN
Coenurosis – larval stage of dog tapeworm Taenia multiceps	LOKOU / LOIIRA

Table 21: Medicinal plants and traditional practices used by livestock keepers to manage priority Goat diseases in Turkana County

Botanical and common name	Turkana name	Target Goat Disease
Aloe Turkanensis- succulent leave sap given as an oral drench	ECHUCHUKA	CCPP Anaplasmosis Heart water
Euphorbia kalisina or uhligiana- the spines/ thorns are removed and the succulent leaves pounded and soaked in cold water and given as an oral drench	EMUS	PPR Heart water Anaplasmosis
Azadirachta indica- Neem tree leaves are boiled or soaked in water and given as an oral drench when cool	MWARUBAINI	PPR Heart water Goat pox
Keinia squarrosa- succulent leaves pounded and soaked in cold water and given as a drench	ELILA	CCPP Heart water
Sugar- dissolved in water and given as an oral drench	ETHUGARI	Anaplasmosis
Cissus quadrangularis succulent part of a plant are pounded and soaked in cold water and given as an oral drench or applied topically	EGIS	CCPP Heart water PPR Goat pox Mange
Traditional practice	Turkana name	Target Disease
Hot iron or stone cauterization of affected body part. ECHAPAT- on the throat and NAMARAN - on the ribs	AKIMAD EMACHAR KORI AMORU	CCPP

NB: Text in red highlights practices that go against animal welfare principles

Table 22: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat priority Goat diseases in Turkana county

Type of VMP	Active ingredient	Turkana name	Target Goat disease
Antibiotic	Oxytetracycline	EDAWA LOMUNG' / EDAWA ALOUKOI	PPR , CCPP, Heart water, Goat Pox, Anaplasmosis
	Tylosin	EDAWA LA NG'IUKOI	PPR , CCPP, Heart water, Goat Pox, Anaplasmosis
	Procaine Penicillin G and Dihydrostreptomycin (Penstrep®)	EDAWA LOKAKUON LO AKWAAN	PPR, Goat Pox, Heart water
Immune boster	Multivitamin	EDAWA LO APITAMIN	PPR , CCPP, Heart water, Goat Pox, Anaplasmosis
Antiparasitic / Anthelmintic	Ivermectin	EDAWA ALOTOME / EDAWA LOKIRION / EDAWA LOMOO	Goat pox Mange
	Albendazole	NAOSIN / EDAWA LO ANG'IPEELEI	PPR , Mange
Acaricide	Amitraz Synthetic Pyrethroids (Ectopor®) Organophosphate	ADIP NA NG'IGURAI / ADIP	Mange Anaplasmosis

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

Goat Diseases:
Bacterial

7.1 Heart water- AMIL
Other name: Cowdriosis

Aetiology
- An infectious, noncontagious tick-borne disease of ruminants caused by bacteria Ehrlichia
- Ruminantium (formerly Cowdria ruminantium) and transmitted by the ticks, Amblyomma variegatum.
- In Turkana county the apparent morbidity rate is 6%, mortality rate 4% and case fatality rate of 67%. The highest incidence of the disease is during the short dry season AIT/NAIT.
- It is estimated by South African based research studies that mortalities due to Heart water are under diagnosed and in most cases they are more than double those due to Babesiosis and Anaplasmosis.

Physiopathology
- Once in the host, the organisms replicate first within the regional lymph nodes with subsequent dissemination via the bloodstream to invade endothelial cells of blood vessels elsewhere in the body. In domestic ruminants, there is a predilection for endothelial cells of the brain. Organisms can often be found in colonies (commonly but mistakenly referred to as morulae) within the cytoplasm of endothelial cells.

Clinical signs
In peracute cases:
- Sudden death may occur within a few hours of developing a fever, sometimes without any apparent clinical signs; Others display an exaggerated respiratory distress and/or paroxysmal convulsions.

In the acute form;
- Anorexia and depression along with congested and friable mucous membranes.
- Respiratory distress slowly develops along with nervous signs such as a hyperaesthesia, a high-stepping stiff gait, exaggerated blinking, and chewing movements.
- Terminally, prostration with bouts of opisthotonus; "peddling," "thrashing," or stiffening of the limbs; and convulsions are seen.

In subacute cases;
- The signs are less marked and CNS involvement is inconsistent.

Treatment
- Oxytetracycline at 10 mg/kg/day, IM is effective as a cure if administered early in the course of heartwater infection.
- A higher dosage of oxytetracycline (20 mg/kg) is usually required if treatment begins late during the febrile reaction or when clinical signs are evident. In such cases, the first treatment should preferably be given slowly IV. A minimum of three daily doses should be given regardless of temperature; if fever persists, oxytetracycline treatment should continue for a fourth and fifth day.
- If the fever still does not abate, a potentiated sulphonamide at 15 mg/kg/day, IM, has been successful. The withdrawal times for milk and meat after treatment with short- or long-acting oxytetracycline, and sulphonamides must be observed

- Corticosteroids have been used as supportive therapy (1 mg/kg, IM).
- Diazepam may be required to control convulsions.
- Affected animals must be kept quiet in a cool area with soft bedding and be totally undisturbed; any stimulation can pre-empt a convulsive episode and subsequent death.

Control

- Control of tick infestation is a useful preventive measure but endemic stability needs to be considered.

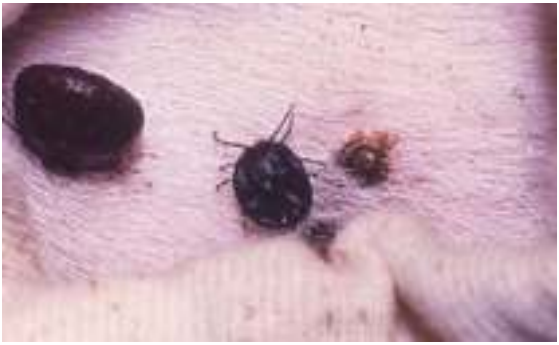


Figure 127
Amblyomma tick feeding on an animal

Picture sources: https://www.cfsph.iastate.edu/diseaseinfo/disease-images/?disease=heartwater

Figure 128
Sheep, lung. Interlobular septa are distended with oedema fluid (left) and the right picture the Sheep, lung. is not collapsed and hyperaemic, and the bronchi contain frothy fluid (pulmonary oedema)

Picture sources: https://www.cfsph.iastate.edu/diseaseinfo/disease-images/?disease=heartwater

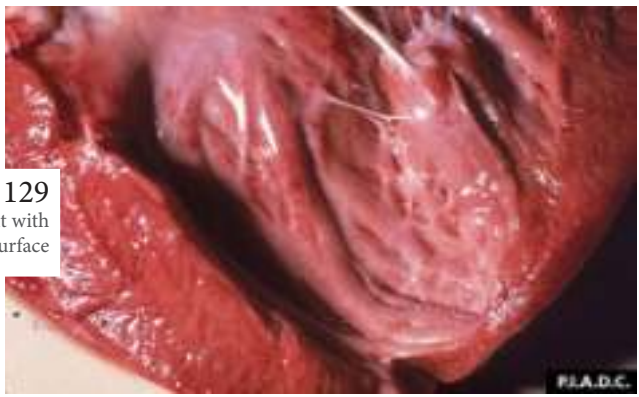


Figure 129
Heart of a small ruminant with haemorrhages on the endocardial surface

Figure 130
Sheep, kidney with multiple petechiae on the cortical surface (left picture) and right cut surface numerous haemorrhages

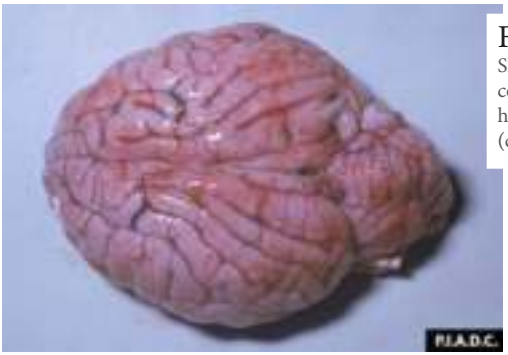


Figure 131
Sheep, brain. The leptomeninges are congested and contain many small haemorrhages. Gyri are flattened (cerebral oedema)

Figure 132
Small ruminant, abomasum (right) and small intestine (left) the mucosa contains disseminated petechiae and coalescing ecchymotic haemorrhages

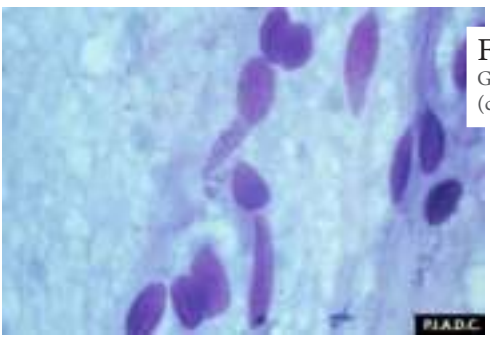


Figure 133
Goat, brain smear. An endothelial cell with a morula (cluster) of Ehrlichia ruminantium

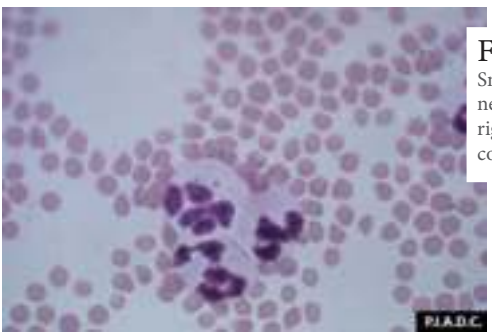


Figure 134
Small ruminant peripheral blood smear (left) the neutrophil contains a few Ehrlichia ruminantium and right picture is a buffy coat smear. Several neutrophils contain E. ruminantium morulae

Picture sources: https://www.cfsph.iastate.edu/diseaseinfo/disease-images/?disease=heartwater

7.2 Contagious Caprine Pleuropneumonia (CCPP) - LOUKOI

**Notifiable disease and should be reported to national and county DVS through KABS.

Case definition and Epidemiology

- CCPP is a severe highly contagious fatal disease that affects the respiratory tract.
- In immune naive herds the morbidity rate may reach 100% and the mortality rate can be as high as 80%. CCPP causes major economic losses in East Africa and the Middle East, where it is endemic
- During the only confirmed outbreak in wild ruminants, the morbidity rate was 100% in wild goats and 83% in Nubian ibex. The mortality rates in these two species were 82% and 58%, respectively.
- Goats are the primary hosts but Sheep may be affected, during CCPP outbreaks affecting mixed goat and sheep herds the bacteria has been isolated from healthy sheep, and their role as a possible reservoir must be considered.
- CCPP was first reported in Europe in Thrace, Turkey, in 2002, but it did not spread to neighbouring countries of Greece and Bulgaria.

Causative agent

- CCPP is caused by bacterium Mycoplasma capricolum subspecies capripneumoniae (Mccp). Formerly known as Mycoplasma sp. type F-38.
- Genetic studies have grouped Mccp isolates into two major clusters (2 or 3) depending on the geographic region.
- Mccp is closely related to M. capricolum subsp. capricolum and more distantly related to other members of the "Mycoplasma mycoides cluster" such as M. mycoides subsp. capri or M. leachii. Mccp lesions are only confined to the thoracic cavity. However, mycoplasmas of the mycoides cluster have thoracic as well as prominent lesions in other organs and/or parts of the body.
- Mccp is inactivated by most of the routinely used disinfectants for example phenol (1%) inactivates it in 3 minutes. The bacteria cannot survive for long in the external environment. on average it only survives outside the host for up to 3 days in tropical areas.

Morbidity and Mortality rates

- In Turkana county, livestock keeper in the year 2022, estimated an apparent annual morbidity rate of 16% with a 7% mortality and 44% case fatality rate.
- The disease is endemic in Turkana and occurs throughout the year.

Transmission

- Transmitted during close contact and by inhalation of respiratory droplets.
- Chronic carriers may exist, but this remains unproven. Some outbreaks have occurred in endemic areas when apparently healthy goats were introduced into flocks.
- Outbreaks of the disease often occur after heavy rains (e.g. after the monsoons in India), after cold spells or after transportation over long distances. This may be because recovered carrier animals shed the infectious agent after the stress of sudden climatic or environmental changes.

Clinical signs

- The incubation period is 6 to 10 days. Peracutely affected goats can die within one to three days with minimal clinical signs. Acute symptoms include fever, lethargy, violent coughing, extended necks, laboured breathing, loss of appetite and abortions.

Control

- Treatment with Tylosin at 10 mg/kg/day, IM, for 3 days, has been effective, as has oxytetracycline (15 mg/kg). Tylosin dosage is 0.3 – 0.4 mL per 10 kg bodyweight for 3 to 5 days. On the first day two injections can be given 12 hours apart.
- Vaccines used to control disease in Kenya, KVEVAPI CAPRIVAX that is administered to animals over 3 months of age via subcutaneous injection at the rate of 1ml per animal. Revaccination should be carried out every 6 months

Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/
2. OIE (2017). Technical Disease Card. https://www.woah.org/app/uploads/2021/03/contagious-caprine-pleuro.pdf

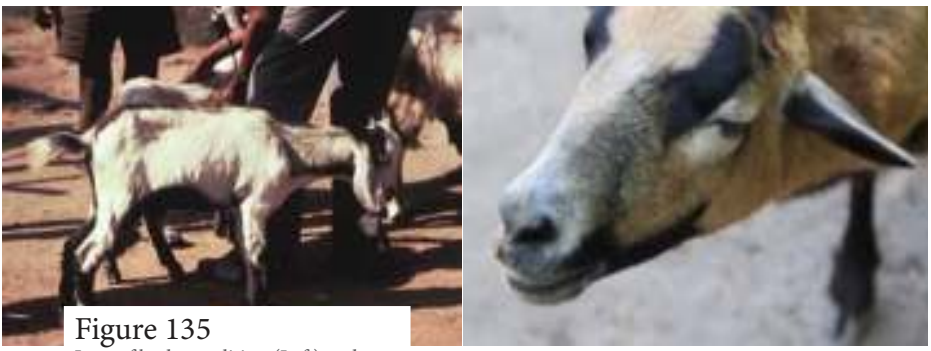


Figure 135
Loss of body condition (Left) and open mouth breathing in goats affected with CCPP

Picture source:
https://www.semanticscholar.org/paper/Contagious-caprine-pleuropneumonia-in-Beetal-goats-Hussain-Auon/a2ffc6fb72ef9c23e40087760efade8e82d7ec85/figure/1

Figure 136
CCPP lesions showing abundant fibrin on lung lobes (Left) and large quantities of pleural fluid and "port wine"–coloured hepatized lung lobes in a goat

Picture source:
https://www.msdvetmanual.com/respiratory-system/respiratory-diseases-of-sheep-and-goats/contagious-caprine-pleuropneumonia

7.3 Mastitis in Goats- LOEKETA

Case definition, Aetiology and Epidemiology

- Mastitis in goats is an acute to chronic inflammation of the udder mainly caused by bacterial infection. Trauma may be a predisposing factor to bacterial mastitis.
- The bacterial organisms that infect the udder of does are similar to those in cows. Coagulase-negative staphylococci are generally the most prevalent and can cause persistent infections that result in increased cell counts and low-grade mastitis with some recurring clinical episodes. The level of infection and incidence of mastitis due to Staphylococcus aureus tends to be low (<5%) but can result in persistent infections that do not generally respond to therapy. Streptococcal intramammary infections can occur in both subclinical and clinical cases but are less frequent than in cattle. Streptococcus agalactiae is not a common mastitis pathogen in does.
- Mycoplasma infections, primarily M mycoides (large colony type) and M putrefaciens, sometimes cause serious outbreaks of mastitis in goats (Contagious Agalactia). The latter also causes septicaemia, polyarthritis, pneumonia, and encephalitis, together with serious disease and mortality in suckling kids. M capricolum has also been reported to cause severe mastitis in goats and infection in kids. Does usually recover after 4 weeks.
- Gram-negative organisms cause intermittent infections that may be severe but are usually self-limiting. Trueperella (formerly Arcanobacterium) pyogenes sometimes produces multiple, nodular abscesses in the udder and loss in the functionality of the udder in does.
- Does can also signs of mastitis from caprine arthritis and encephalitis and ovine progressive pneumonia secondary to systemic infection. Agalactia is common due to the hardening of the udder from fibrosis.
- Does that have aborted and have uterine infection should be treated immediately as the same microorganisms that cause abortion in does also cause mastitis.

Diagnosis

- Diagnosis, control, and treatment of bacterial mastitis in does are similar to those in cows. However, monitoring subclinical mastitis with somatic cell counts (SCCs) in does is difficult because of poor discrimination between infected and non-infected animals, especially in the later stages of lactation. This is partially because a of a higher proportion of epithelial cells in caprine milk than in bovine milk. As lactation progresses, shedding of epithelial cells into milk increases; thus, SCCs >1,000,000 cells/mL are common in healthy does in late lactation.

Treatment

- The treatment should be based on the results of the microbiologic culture obtained from milk samples. This is not practical in Turkana setting.
- Before treatment the affected udder half should be dried off by stopping the kid from suckling the half or stop milking and infusing commercially Intramammary infusion of 2%

chlorhexidine solution into affected half twice a day for 5 to 10 days. This many not also be practical in Turkana setting.

- Intramammary infusions for dairy cows also work in goats but one should observe for developing irritation as the drug concentration may be strong for the doe's udder tissue.
- Most practitioners prefer systemic antibiotics combined with anti-inflammatory drugs like glucocorticoids. Correct dosage and duration as well as withdrawal period should be observed. Antibiotics like cloxacillin, amoxicillin plus clavulanic acid, erythromycin, and tetracycline have been recommended to treat mastitis in goats. However, cure rates vary from animal to animal and according to the severity of the case.
- Glucocorticoids, can be administered early in the course of disease. Administration of dexamethasone in the mammary gland has been reported to reduce swelling.
- Treatment during the dry-off period is an efficient method for the cure of subclinical mastitis

Control

- Proper milking procedures and good environmental sanitation are needed to reduce the prevalence and spread of infection.
- Chronically infected does should be culled, including does with M mycoides infections and those that do not recover from M putrefaciens or M capricolum infections.
- Kidding pens must be disinfected and bedding removed daily.
- When purchasing animals palpate mammary glands to note if the consistency is normal.

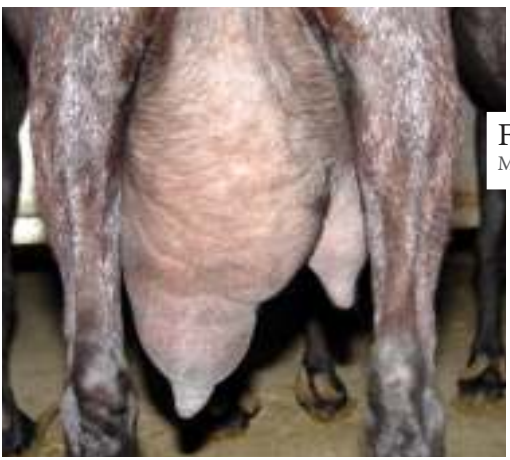


Figure 137
Mastitis in a goat

Picture source:
https://www.premier1supplies.com/w/mastitis-control-program/

7.4 Anaplasmosis – LONYANG / LOPID

Refer to Chapter 4: Cattle diseases section 4.9: Anaplasmosis

7.5 Foot rot- EKICHODONU

Refer to Chapter 6: Sheep diseases section 6.2 Foot rot

7.6 Infectious Keratoconjunctivitis (Pink eye)- EDEKE ANG'AKONYEN

Refer to chapter 6: Sheep diseases section 6.4: Infectious keratoconjunctivitis (Pink eye)

7.7 Caseous Lymphadenitis (CL)- ABUS / ABUTH / LONG'ARURWEI

Case definition and Epidemiology

- Caseous lymphadenitis (CL) is a chronic, contagious bacterial disease that manifests clinically as abscesses of external/ peripheral and/or internal lymph nodes and organs.
- Peripheral lymphnodes when rupture produce very thick pus that has no foul smell (non-odorous).
- The peripheral form is characterised by abscesses of single or multiple peripheral palpable lymph nodes while the internal CL form manifests as chronic weight loss and ill thrift.
- Field based diagnosis is through the clinical signs while confirmatory is culture of lesions discharge to isolate Corynebacterium pseudotuberculosis.
- The ideal control strategy is to cull affected small ruminants from the herd as they serve as a constant source of infection. This is not possible in extensive production systems as practiced in Turkana county. The best cause of prevention and control is therefore, treatment that consists of sustained antimicrobial therapy to reduce the numbers of active draining lesions and isolation from other herd mates until lesions are dry and/or resolved.
- Occurs worldwide and the most common site of entry is the skin after an injury that may result from shearing, tagging, tail docking, castration, or other environmental hazards resulting in skin trauma. Contact with purulent material draining from open, active lesions most commonly serves as the source of bacteria.

Aetiology and Pathogenesis

- C pseudotuberculosis is a gram-positive, intracellular coccobacillus.
- Two biotypes have been identified- nitrate-negative group that infects sheep and goats, and a nitrate-positive group that infects horses. Isolates from cattle are a heterogeneous group.
- All strains produce an exotoxin called phospholipase D that enhances dissemination of the bacteria by damaging endothelial cells and increasing vascular permeability. The second virulence factor is an external lipid coat that provides protection from hydrolytic enzymes of the host phagocytes.
- Once the bacteria have entered the body, they move to the lymph nodes via the regional draining lymphatic system. Internally, the bacteria establish infection not only in the lymph nodes but also in the viscera. Replication of bacteria occurs in the phagocytes, which then rupture and release bacteria. The ongoing process of bacterial replication, followed by attraction and subsequent death of inflammatory cells, forms the characteristic abscesses associated with CL.
- The incubation period varies from 1 to 3 months, culminating in development of encapsulated abscesses. C pseudotuberculosis is hardy in the environment and can survive on fomites such as bedding and wood for 2 months and in soil for 8 months.

Clinical signs

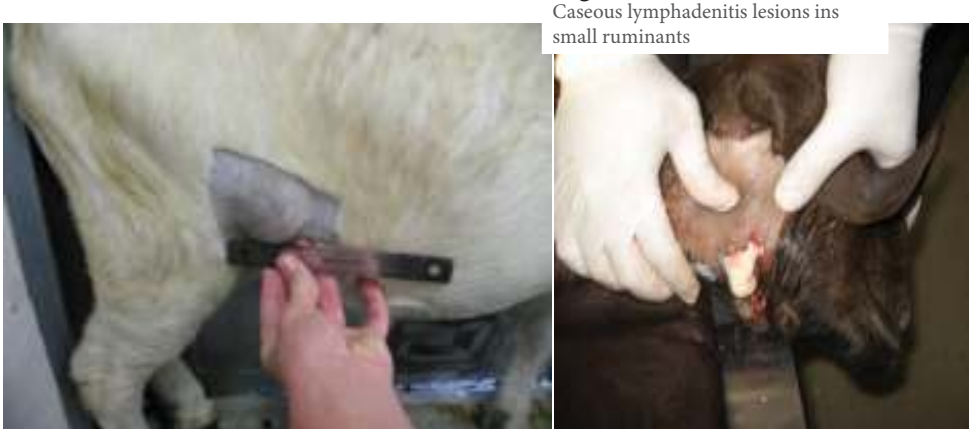
- External caseous lymphadenitis is characterised by development of abscesses in the peripheral lymph nodes. Common sites include the submandibular, parotid, prescapular, and prefemoral nodes. Less commonly, abscessation of supramammary or inguinal lymph nodes occurs, If left untreated, these lesions eventually mature into open draining abscesses. The purulent material from these lesions has no odour and varies in consistency from soft and pasty in goats to thick and caseous in sheep. Once natural draining occurs, the skin lesion heals with scarring. Recurrence is common even with systemic antibiotic and local treatment.

- The internal form of CL presents as chronic weight loss and failure to thrive. Other clinical signs depend on the organs of involvement, which may include any of the major organ systems. Lung abscessation is a common site of visceral involvement in internal CL; therefore, signs of chronic ill thrift with cough, purulent nasal discharge, fever, and tachypnoea with increased lung sounds are common lung lesions signs. The internal CL form is more common in sheep and has been termed the "thin ewe syndrome."
- The incidence of abscesses and development of clinical disease with either the external or internal form increases with age.
- In sheep, abscesses often have the classically described laminated "onion-ring" appearance in cross section, with concentric fibrous layers separated by caseous exudate. In goats, the abscesses are less organized, and the exudate may be soft and paste-like.

Treatment

- Antimicrobial treatment (intra-lesion (1 to 2% Povidone iodine irrigation) and/or systemic antibiotic like Tylosin combined with Penicillin.
- The practice of injecting abscesses with formalin should be strongly discouraged, as this is an extra-label use of a potent carcinogen in food-producing animals.
- Penicillin alone, although effective in vitro, is unlikely to penetrate the capsule of developed abscesses, as are many, if not most, of the water-soluble or moderately lipid-soluble antimicrobials.
- Recent studies have shown that administration of one dose of tulathromycin a macrolide like Tylosin or erythromycin at 2.5 mg/kg, either SC directly into the abscess cavity, or two doses at 2.5 mg/kg, administered at the same time, one SC and one intralesionally, can resolve the lesions without lancing the abscess. Further, effective concentrations of tulathromycin can be achieved within walled-off abscesses caused by C pseudotuberculosis after a single dose at 2.5 mg/kg, SC. The highly lipid-soluble property of tulathromycin may be particularly helpful in cases of internal CL, when abscesses are not accessible for other forms of treatment. Despite the efficacy of intralesional and parenteral administration of tulathromycin in many cases, recurrence remains a problem. Therefore, use of these drugs cannot be considered curative but rather an acceptable alternative to manage cases of CL when culling from the herd or flock is not an acceptable option for the owner

Figure 138
Caseous lymphadenitis lesions ins small ruminants



Further reading

MSD Vet Manual https://www.msdvetmanual.com/circulatory-system/lymphadenitis-and-lymphangitis/caseous-lymphadenitis-of-sheep-and-goats

Goat Diseases:
Viral

7.8 Sheep and Goat Pox- ETUNE
Refer to Chapter 6: Sheep diseases under section 6.6: Sheep and Goat pox

7.9 Peste des Petits Ruminants (PPR)- LOMOO
Refer to Chapter 6: Sheep diseases under section 6.5 Pest des Petits Ruminants (PPR)

7.10 Contagious Ecthyma (Orf)- NG'IBUORUOK
Refer to Chapter 6: Sheep diseases under section 6.8 Contagious ecthyma (orf)

Goat Diseases:

Parasitic

7.11 Helminthiasis- LOPELEI / NG'IRITAN

Refer to Chapter 6: Sheep diseases section 6.12 Helminthiasis

7.12 Coenurosis- LOKOU / LOIRA

Refer to Chapter 6: Sheep diseases section 6.13 Coenurosis

7.13 Mange- LOTOME / EMITINA

Refer to Chapter 6: Sheep diseases section 6.14 Mange

Chapter 8:

Chicken Diseases

Table 23: Priority Chicken diseases ranked in order of importance by livestock keepers in Turkana County

Conventional name	Turkana name
Newcastle disease (ND)	EREMONU LOPUS-Greenish diarrhoea LONG'EDA ANG'IKIENY –Sudden death
Infectious Bronchitis (IB)	LOWALA ANG'IKIENY / LOUKOI
Fowl pox	ETUNE ANG'IKIENY / LOMERIMERI
Coccidiosis	EROMONU ANG'AKOT ANG'IKIENY- Bloody diarrhoea AREMOR ANG'IKUKUI- Diarrhoea
Fowl Typhoid	EREMONU LONYNAG- Yellowish diarrhoea (Most likely does not occur in county as diarrhoea in fowl typhoid is whitish in colour)
Helminthiasis	NG'IPEELEI / NG'IRITAN
Ectoparasite infestation	ASASA - Mites NG'IKADESDES - Fleas EMADANG' - Ticks ELACHIT - Lice
Infectious Coryza	LOKIO ANG'AKONYEN
Crop impaction	EDEKE ETOLE

NB: As there is no post mortem conducted in birds it is difficult to verify if the above mentioned diseases actually occur in Turkana county. There is need for chicken necropsy training for animal health service providers in the county so that they can confirm diseases in poultry.

Table 24: Ethnoveterinary practises used by livestock keepers to manage priority chicken diseases in Turkana County

Medicinal plant botanical name and Traditional Practice	Turkana name	Target Chicken Disease
Ash – topically applied	EKURON	Ectoparasite infestation and Fowl pox
Kitchen soot- mixed with drinking water	NG'MUDUI	ND, Coccidiosis, Infectious Bronchitis, Helminthiasis and Infectious coryza
Used engine oil- topical application	AOILOLO	Ectoparasite infestation and Fowl pox
Oil paraffin- topical application	AKIMIET ATAA	Ectoparasite infestation and Fowl pox
Cooking oil- topical application	AKIMIET NAKIPOET	Ectoparasite infestation and Fowl pox
Aloe turkanensis –sap from succulent leaves mixed with drinking water	ECHUCHUKA	ND, Coccidiosis, Infectious Bronchitis, Helminthiasis and Infectious coryza
Euphorbia kalisina or uhligiana- the spines/ thorns are removed and the succulent leaves pounded and soaked in cold water and given as an oral drench	EMUS	ND, Coccidiosis, Infectious Bronchitis, Helminthiasis and Infectious coryza
Pepper powder- put in drinking water	EPILIPILI	ND, Coccidiosis, Infectious Bronchitis, Helminthiasis and Infectious coryza
Azadirachta indica- Neem tree leaves are soaked in water and given as an oral drink	MWARUBAINI	ND, Coccidiosis, Infectious Bronchitis, Helminthiasis and Infectious coryza
Salvadora persica (tooth brush tree)- bark crushed and mixed with water and given orally to bird	ESEKON	Crop impaction
Magadi soda- dissolved in water and given as a drinking water	AMAKAT	ND, Crop impaction
Local brew –put in the drinking water	CHANGAA	Crop impaction, ND, Coccidiosis, Infectious bronchitis
Doxycycline capsules intended for human use- powder put in the drinking water	EDAWA LOPUS	Coccidiosis , Infectious bronchitis , Infectious coryza
Amoxycillin capsules intended for human use- powder put in the drinking water	EDAWA LOLING'ANYANG'	Coccidiosis , Infectious bronchitis, Infectious coryza
Paracetamol (Panadol®) tablet intended for human use crushed and put in drinking water	EDAWA LOAKWAN	ND, infectious bronchitis, Infectious coryza, fowl pox

Table 24: Ethnoveterinary practises used by livestock keepers to manage priority chicken diseases in Turkana County

Medicinal plant botanical name and Traditional Practice	Turkana name	Target Chicken Disease
Artemether/lumefantrine Coartem® -intended for human use - Malaria treatment tablet crushed and put in drinking water	EDAWA LOEMALERIA	Infectious bronchitis, Infectious coryza, ND, Coccidiosis
Sevin® powder (Carbaryl)- topical application	KUKU DUST	Ectoparasite infestation
10% Oxytetracycline injection- put a few drops in drinking water	EDAWA LOMUNG' EDAWA ALOUKOI	Infectious bronchitis, Infectious coryza, ND, Coccidiosis

NB: Text in red highlights practices that go against animal welfare principles and will contribute to AMR. Use of human drugs in poultry should be discouraged and should form part of extension messages.

Chicken diseases:

Viral

8.1 New Castle Disease (ND)- EREMONU LOPUS/ LONG'EDA ANGIKIENY

Other names- Avian pneumoencephalitis, Ranikhet disease, Fowl pest, Fowl plague

Local dialect name: EREMONU LOPUS- Greenish diarrhoea and LONG'EDA ANGIKIENY- sudden death

Case definition

- ND is a highly contagious and fatal viral disease affecting over 250 bird species with chicken being the most susceptible.
- ND is a multisystemic disease involving the respiratory, neurological, reproductive and gastrointestinal body systems.

Distribution (Epidemiology)

- Endemic globally, sero-surveillance studies in Kenya have shown a Morbidity rate of 5 to 17.8% and mortality rate of 50-100%.
- Based on Turkana county livestock keepers' perception (2022) ND's apparent annual Morbidity rate is 23% with a Mortality rate of 22% and Case fatality rate of 95%.
- ND in Turkana county occurs throughout the year with most outbreaks reported during the long rainy season (AKIPORO).
- ND, is the most significant disease of poultry in Kenya.

Age susceptibility

- All ages, but young birds have the highest mortality rates.

Aetiology

- Virulent strains of avian orthoavulavirus 1 (AOaV-1), causative agent formerly known as avian paramyxovirus1 (APMV 1).
- It is a single-stranded RNA virus from Paramyxoviridae family.
- AOaV-1 has 11 serotypes
- Original classification was based on virulence; Lentogenic (low virulence viruses); Mesogenic (moderate virulence), and Velogenic (highly virulent) and tissue tropism (predilection). The virus exhibits tissue tropism where some strains only attack the nervous system, others the respiratory, and others the digestive systems.
- In the new classification - Velogens and mesogens strains are now called virulent NDV (vNDV) they cause Newcastle disease and are WOAH reportable infections.
- Infections with lentogens are now called low virulence NDV (loNDV) and are widely used as live vaccines and are not reportable.

Mode of transmission

- Main risk factors for ND outbreaks are introduction of new birds into the flock and purchase of birds from markets.
- AOaV-1 is stable in nature and remains infectious for weeks at low temperatures, and if protected by bird's feathers and within eggs survives for over 250 days.
- Direct transmission is via inhalation or

ingestion (faecal/oral route). Birds shed virus in faeces and respiratory secretions. Reservoir and carrier hosts are a source of direct infection as they shed the virus,
- Indirect transmission is via fomites. Virus survival is prolonged by the presence of faeces, for example on eggshells.

Clinical signs and Pathophysiology
- Incubation period is 2 to 12 days.
- There is sudden death and high mortality within 24-48 hours
- Birds that survive are drowsy or extremely depressed with most birds are seen sitting on their back hock joints.
- Birds have ruffled feathers are anorexic and have a sudden drop in egg production.
- The most common gastro intestinal sign is greenish to yellowish diarrhoea.
- The main respiratory signs are dyspnoea (gasping) with rales (gurgling or rattling sounds), sneezing, coughing, nasal and ocular watery discharge. Swelling of head and neck tissues.
- Nervous signs include muscular tremors (twitching) of head and neck, partial paralysis of wings and legs, circling, spasms and torticollis (twisted neck) and opisthotonus (star gazing).
- Recovered birds beyond may have permanent nervous (tremor) or permanent reproductive impairment.
- Clinical signs may be seen in birds vaccinated with the live vaccine where they have a marked drop in egg production, some birds may produce eggs that are misshapen, abnormally pale, rough, or thin-shelled with watery albumin.

Post-mortem Gross lesions
- Swelling of periorbital area or entire head.
- Oedema of the interstitial or peritracheal tissue of the neck especially at the thoracic inlet.
- Diphtheritic membranes in the oropharynx, trachea and oesophagus.
- Pulmonary congestion and oedema.
- Pinpoint haemorrhages of the proventriculus gland.
- Multifocal, necrotic haemorrhagic areas on the mucosal surface of intestines especially at lymphoid foci such as the gut-associated lymphoid tissues (GALT) of cecal, tonsils and Peyer's patches.
- Though not pathognomonic, ulceration/ necrosis of Peyer's patches is suggestive of Newcastle disease.

Diagnosis
- Tentative/Presumptive- clinical signs and PM gross lesions but are not specific to ND.
- Laboratory investigation is needed.
- Samples collection should be from recently dead birds (less than 8 hours) or moribund birds that have been killed humanely.
- Samples- Tracheal, oropharyngeal and cloacal swabs or faeces obtained from live birds or pooled organ samples taken from dead birds. Contact laboratory to get advice on what transport media to use.
- Confirmatory diagnosis- antibody or viral antigen detection using ELISA, or other serological assays like Haemagglutination (HA) and Haemagglutination inhibition (HI) or molecular virus identification using RT-PCR for the detection and typing of virus.

Public health risk
- Zoonotic disease - Causes conjunctivitis in humans, that is mild and self-limiting.

Differential Diagnosis (DDx)
- Other viral diseases - highly pathogenic avian influenza, laryngotracheitis, fowl pox (diphtheritic form) and infectious bronchitis.
- Bacterial diseases: fowl cholera, psittacosis (Chlamydiosis), Mycoplasmosis, salmonellosis
- Fungal diseases like Aspergillosis
- Management errors such as deprivation of water poor feed and air quality.

Treatment
- Recovered birds may be immune but remain carriers that shed virus when stressed.
- No treatment as it is a viral disease
- Infected birds should be slaughtered.

Prevention/Control

- Humane destruction of all infected birds.
- Thorough cleaning and disinfection of houses/ burning house
- Proper carcass disposal
- Endo and Ecto parasite control
- Allow 21 days without poultry before restocking
- Isolate new birds for 1-week
- Regular vaccination

Vaccination Schedule

- Locally available live attenuated vaccine (AVIVAX-L™ and AVIVAX-F™) is used for vaccinations against AOaV-1 in Kenya.
- Thermostable I-2 vaccine (derived from lentogenic LaSota/F strains) is administered in three doses (as eye drops): first dose 4 days to 3 weeks old, second dose at 4–8 weeks and the last dose at the age of 8–18 weeks.
- To overcome the problem of continuous ND outbreaks even in vaccinated flocks, some commercial farmers combine live and/or inactivated vaccines, which are based on an older AOav-1 genotypes that has been reported to be inefficient in reducing viral replication and shedding.
- Kenchic has introduced a hatchery vaccination in all day-old chicks against Gumboro, Newcastle and Infectious Bronchitis diseases. The TRANSMUNE vaccination against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases.

Reporting criteria

- Notifiable disease- WOAH listed
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS)

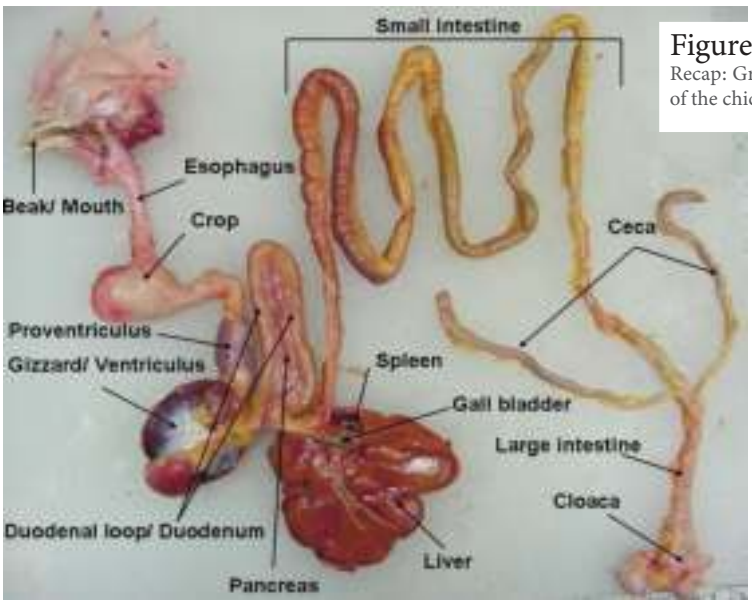


Figure 139
Recap: Gross anatomy of the chicken

Picture source:
https://www.premier1supplies.com/w/mastitis-control-program/

Figure 140
Clinical signs of Newcastle disease (clockwise order): 1) chicken showing severe depression,
2) torticollis
3) greenish watery diarrhoea and
4) death

Figure 141
Chicken, neck with marked subcutaneous and peritracheal oedema (Left) and Marked pulmonary congestion and oedema (Right)

Figure 142
Chicken proventriculus and ventriculus. Severe, acute multifocal mucosal haemorrhages

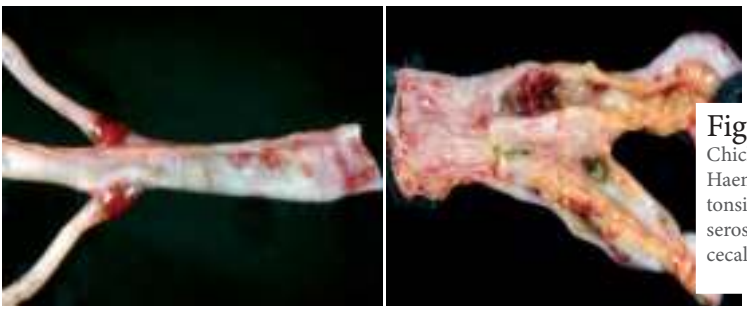


Figure 143
Chicken, cecal tonsils. Haemorrhage of the cecal tonsils visible through the serosa (Left) and necrosis of cecal tonsil (Right)

8.2 Infectious Bronchitis (IB)- LOWALA ANG'IKIENY / LOUKOI

Case definition:

- acute, highly contagious upper respiratory tract disease. characterised by coughing, sneezing and tracheal rales.
- In laying hens, causes decreased egg production and quality.
- Some virus strains cause nephritis (Kidney inflammation) and cystic oviduct syndrome (false layer syndrome).
- Causes clinical disease in chicken only and no other bird species.
- Second most important disease of chicken after New Castle Disease (ND). ND causes high mortality while Infectious Bronchitis (IB) causes low mortality but high production losses.

Distribution (Epidemiology)

- Worldwide
- IB virus (IBV) has many antigenic types, some IBV types are widespread, others are regional.
- Severity of disease and body systems involved influenced by:

o strain of the virus, age, immune status, and diet of the chicken and exposure to cold stress.

- Co-infection with Mycoplasma (M) gallisepticum, M synoviae, Escherichia coli, and/or Avibacterium paragallinarum worsen infections.
- Morbidity rate in flocks with no immunity is 100% but morbidity is low 5% but can be as high as 60% due to bacterial co-infections.
- Based on Turkana county livestock keepers' perception (2022). IB apparent annual Morbidity rate is 6% with a Mortality rate of 4% and Case fatality rate of 67%.
- IB in Turkana county occurs throughout the year with most outbreaks reported during the long rainy season (AKIPORO).

Age susceptibility

- All ages of birds.
- Young chicks more susceptible.

Causative agent (Aetiology)

- IBV is an avian coronavirus that is an enveloped and single-stranded RNA virus with characteristic spike-like projections on the surface of its envelope.
- Variation of the viral spike protein gives rise to multiple strains of the virus, which may vary regionally.
- IBV quickly mutates and requires constant surveillance to identify IBV types circulating in a specific region.
- Different antigenic virus strains do not cross-protect.
- Recent discoveries of IBV and IBV-like strains in other bird species such as geese, ducks, and pigeons. May play a role in the spread of IBV to chickens.

Mode of transmission

- Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks.
- Virus is shed via aerosol droplets and faeces. IBV can persist in the bird's intestinal tract for several weeks or months
- Frequency of airborne transmission is not known but studies have shown of transmission

occurring in commercial farms over 1 kilometre apart.
- Incubation period is 24 to 48 hours, peak virus excretion from the respiratory tract occurs for 3 to 5 days after infection.
- Direct and indirect transmission via aerosol droplets, ingestion of contaminated feed and water, and contact with contaminated equipment and clothing (fomites).

Clinical signs and Pathophysiology
- Symptoms manifest 18 to 24 hours' post-exposure and may last for 2-3 weeks.
- Conjunctivitis, coughing, sneezing, tracheal rales and facial swelling.
- Respiratory distress is due to increased mucous in the trachea.
- In addition to above signs, adult birds, have a sharp decrease in egg production.
- Damage to the reproductive tract causes production of misshapen, wrinkled, thin soft eggs of poor internal quality for several weeks.
- Infection in birds that have not started laying (pullets) can cause false layer syndrome as the oviduct is damaged but ovaries are intact. The ovaries continue to produce and the yolks accumulate in the abdomen of the animal. With time, the non-functional oviduct fills with clear fluid and distends (cystic).
- In addition, accumulation of yolks, causes abdominal distention and forces the birds to stand upright (penguin-like posture).

Post-mortem Gross lesions
- Trachea, sinuses, and nasal passages contain serous, catarrhal, or caseous exudates,
- Air sacs are cloudy and thickened. If complicated by infection with E coli, there may be caseous airsacculitis, perihepatitis, and pericarditis.
- Female birds infected when young have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and ovaries with regressed follicles.
- Infection with nephropathogenic IBV strains cause swollen, pale kidneys, with the tubules and ureters distended with urates. Kidneys may be atrophied.

Diagnosis
- Tentative/Presumptive- clinical signs and clinical history
- Laboratory investigation needed due to large number of diseases that show similar symptoms.
- Samples collection should be from recently dead birds (less than 8 hours), moribund birds that have been killed humanely or live birds.
- Samples for acute respiratory disease include swabs from the upper respiratory tract of live birds or tracheal and lung tissues from dead birds.
- Samples from nephritis or egg production problems should be tissue samples from the kidneys or oviduct, respectively in addition to respiratory specimens.
- Confirmatory diagnosis- antibody or viral antigen detection.
- Laboratory will inform if they can Conduct rising antibody analysis using ELISA or Molecular virus identification using RT-PCR.
- There is need for sustained molecular surveillance so as to ensure early detection of novel strains of IBV.

Public health risk
- No data indicating zoonotic potential.

Differential Diagnosis (DDx)
- Viral diseases: Newcastle Disease, Infectious laryngotracheitis, infectious coryza.
- Bacterial diseases: Mycoplasma gallisepticum (Mycoplasmosis)
- Management errors such as deprivation of water, poor feed and air quality.

Treatment

- None as it is viral.
- Antibiotics can be used if there is evidence of secondary bacterial infection that manifests as increasing mortality rates with cloudy air sacs, peri hepatitis and pericarditis.

Prevention/Control

- Attenuated live and killed vaccines are available, but different antigenic types of the avian coronavirus causing the disease do not cross-protect, complicating control efforts.
- No medication alters the course of IBV infection, as it is a viral disease. Antimicrobial therapy may reduce mortalities caused by complicating bacterial infections.
- In cold weather, increasing the ambient temperature may reduce mortalities, and reducing the protein concentrations in feed and providing electrolytes in drinking water may assist in outbreaks caused by nephropathogenic strains.
- The live-attenuated vaccines used for immunization may produce mild respiratory signs. These vaccines are initially given to 1- to 14-day-old chicks by spray, drinking water, or eye drop, and birds are commonly revaccinated approximately 2 weeks after the initial vaccination.
- Revaccination with a different serotype can induce broader protection. Attenuated or adjuvant inactivated vaccines can be used in breeders and layers to prevent egg production losses as well as to pass protective maternal antibodies to progeny.
- There are many distinct types of IBV, and new or variant types, which are not fully controlled by existing vaccines, are identified relatively frequently.
- Variant viruses historically arise from mutations accumulating over time as the virus replicates (genetic drift). However, recombination can occur in coronaviruses and may result in unique viruses that may or may not cause disease.
- Selection of vaccines should be based on knowledge of the most prevalent virus type(s) in the area. The correlation between IBV type and protection is imperfect, and selection of the most appropriate vaccine, or combination of vaccines, may require experimental assessment in vivo.

Vaccination Schedule

- Kenchic has introduced hatchery vaccination in all day-old broilers against Gumboro, Newcastle and Infectious Bronchitis diseases.
- TRANSMUNE vaccination against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases in day-old chicks at the hatchery. The farmer will only do one single Newcastle disease vaccination in the Broiler farm at day 14 instead of 2 vaccinations as done previously

Reporting criteria

- Notifiable disease- WOAH listed
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS)

References

1. Al-Rasheed, M., Ball, C. & Ganapathy, K. (2021). Route of infectious bronchitis virus vaccination determines the type and magnitude of immune responses in table egg laying hens. Vet Res 52, 139 (2021). https://doi.org/10.1186/s13567-021-01008-7
2. Najimudeen, Shahnas, Mohamed S. H. Hassan, Susan C. Cork, and Mohamed F. Abdul-Careem. (2020). Infectious Bronchitis Coronavirus Infection in Chickens: Multiple System Disease with Immune Suppression. Pathogens 9, no. 10: 779. https://doi.org/10.3390/pathogens9100779

Figure 144

Changes in Laid eggs due to IBV infection N: Normal Egg; D: Deformed Egg; SL: Shell less Egg

Picture source: https://www.researchgate.net/profile/Tofazzal-Md-Rakib

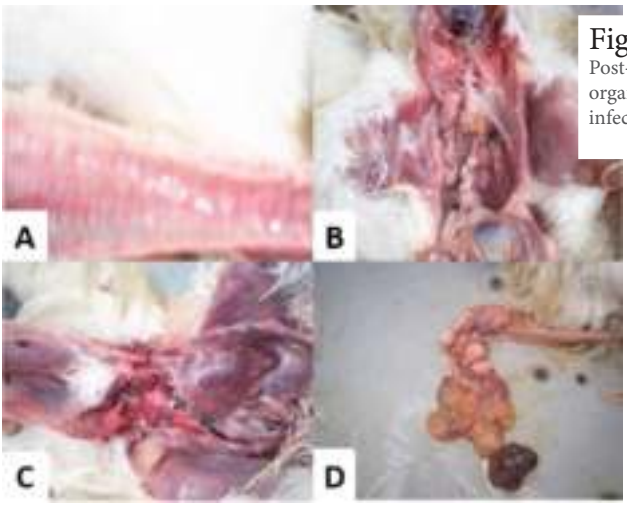


Figure 145

Post-mortem lesion in different organs of chicken caused by infectious bronchitis virus

A) Congested trachea;
B) Swollen, pale or marbled kidneys with urate deposits in the tubules;
C) Visceral gout in chicken with wide spread deposits of uric acid on serosal surface of the body;
D) Misshappen ova and oviduct

Picture source: https://www.researchgate.net/profile/Tofazzal-Md-Rakib

8.3 Fowl pox- ETUNE ANG'IKIENY / LOMERIMERI

Other name: Avian pox. There are three strains of virus that cause avian pox: Fowl pox, pigeon pox and canary pox.

Causative agent: An avipoxvirus. The virus is resistant and can survive in the environment for long periods in dried scabs.

Transmission:

- Direct contact through abrasions of the skin
- Aerosol infection from recovering birds that have skin lesions (scabs) and sharing same houses
- Indirectly through mechanical vectors like mosquitoes, other biting insects or individuals handling one bird after the other for immunization or other reasons.
- In Turkana county the estimated apparent morbidity rate is 1% with no mortality rate.
- The disease in the county is most common during the short dry season (AIT/NAIT).

Main clinical signs:

There are two forms of the disease:

1. Skin or cutaneous form (dry pox): this is characterized by nodular wart-like lesions on various parts of the un-feathered skin of chicken's head, neck, comb, wattles, eyelids, legs, and feet. In some cases, lesions are limited to the feet and legs. The lesion is initially a raised, blanched, nodular area that enlarges, becomes yellowish, and progresses to a thick, dark scab. Localization of lesions around the nostrils may cause nasal discharge. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes. Only a few birds develop cutaneous lesions at one time. Multiple lesions usually develop and often coalesce. Lesions in various stages of development may be found on the same bird.
2. Diphtheritic form (wet form): This is characterized by small white nodules in upper respiratory and digestive tracts. These nodules merge together to form raised-yellow white cheesy patches. These lesions cause difficulty in breathing. It has been found that most cases of diphtheritic fowl pox are characterized by the formation of massive yellow cheesy necrotic masses in the larynx and adjacent trachea The bird in such cases die suddenly from asphyxiation (lack of oxygen).

Post mortem Findings:

- Wart-like lesions of the head particularly of the comb and around the eyes, or yellow cheesy lesions in the larynx and adjacent trachea on post-mortem, are diagnostic of fowl pox.

Differential Diagnosis:

- Infectious laryngotracheitis, necrotic dermatitis, leg mites and thrush (Candidiasis) and canker (Trichomonas gallinae).

Diagnosis:

- Tentative diagnosis is based on flock history and presence of lesions.

Prevention and control:

- Vaccination effectively prevents the disease and may limit spread within actively infected flocks. In high-risk areas, vaccination with an attenuated vaccine of cell-culture origin in the first few weeks of life and revaccination at 12–16 weeks is often sufficient. Vaccinated

birds should be examined 1 week later for swelling and scab formation ("take") at the site of vaccination. Absence of "take" means the birds are not protected and should be vaccinated again.

Zoonotic potential- None reported

Notifiable disease- No

Figure 146
Cutaneous form of fowl pox showing (white arrows) scab-like lesions on the combs of chicken

Figure 147
Diphtheric form of fowl pox infection showing yellow cheesy oral lesions

8.4 Infectious Bursal Disease (IBD)- Gumboro) – EREMONU EKWAN

- There are no reports of occurrence in Turkana county
- Acute and highly contagious viral infection in young chickens that attacks the bursa of Fabricius that is located inside the vent area of the bird.
- IBD is found throughout the world (except New Zealand) and is endemic
- Caused by a virus - infectious bursal disease virus (IBDV). IBDV is the only species of virus under the Avibirnavirus genus in the Birnaviridae family of viruses.
- Virus is extremely hardy and can survive in a wide range of environmental conditions, making it difficult to eliminate or decontaminate.
- The bursa of Fabricius plays an important part in the disease. The bursa is the "assembly plant" for the immune system. The bursa produces B cells which, in turn, produce antibodies to help the immune system fight off disease challenges or response to vaccination. The B-cell system develops during embryogenesis (formation and development of an embryo after fertilization) and the first few weeks after hatch.
- Disease manifest one of two forms, depending on the age of the chicken when it becomes infected. The subclinical form occurs in chickens less than 3 weeks of age, and there are usually no clinical signs of disease at this time. However, affected chickens are left with permanent and severe immunosuppression. Most infections that occur in the field are of the subclinical variety, and this is the more economically important form of the disease.
- Clinical form affects chicks 3- to 6-weeks old. There is rapid increase in mortality rate. Clinical symptoms may include trembling, ruffled feathers, poor appetite, dehydration, huddling, vent pecking, and depression. Chickens are more susceptible at 3-6 weeks of age when Bursa is at its maximum rate of development and filled with B cells. The affected birds discharge whitish diarrhoea similar to fowl typhoid. On necropsy, lesions are found in the bursa of Fabricius. The bursa is swollen and/or inflamed; may be oedematous (excessive fluid present) and hyperaemic (excessive blood present); and may have a gelatinous, yellowish fluid inside. In severe cases, haemorrhages or necrosis (death of tissue) may be present. A few days after the initial infection, the bursa will begin to atrophy (waste away) and shrink rapidly in size. Often, the kidneys may appear swollen in birds that are necropsied. However, this is likely related to severe dehydration and not directly related to the IBD virus itself.
- Transmission is primarily through the faecal-oral route. Chickens infected with IBD virus shed the virus in the faeces. Feed, water, litter/bedding material, and equipment can become contaminated with the virus. Other chickens become infected by ingesting the virus. Litter beetles have been shown to carry the virus. There is no transovarial transmission.
- Diagnosis is via serology– ELISA or PCR as the clinical form can be confused with Mareck's disease that may also affect the bursa of Fabricius.
- There is no known treatment as it is a virus

and no known zoonotic transmission.

- Control is through vaccination of breeders to produce maternal immunity that is passed on to the chick. Kenchic in Kenya has introduced TRANSMUNE vaccination against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases in day-old chicks at the hatchery.

Zoonotic potential- None reported

Notifiable disease- No – livestock keepers should ensure they purchase chicks from hatcheries or breeder farms that vaccinate against IBD.

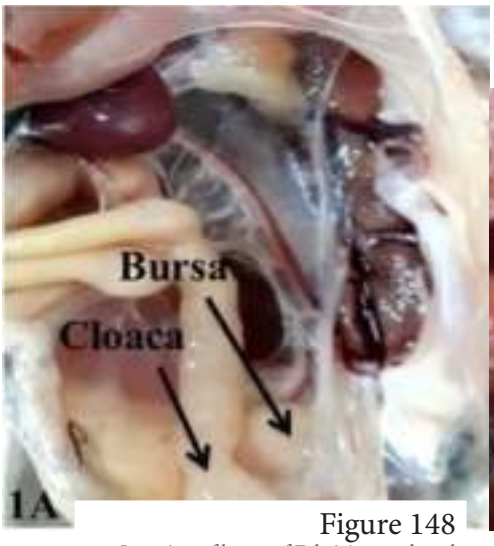


Figure 148
Location of bursa of Fabricius on dorsal aspect of cloaca in a healthy chicken

Picture source: Ayman, Ummay & Alam, Md & Das, Shonkor. (2020). Age-related development and histomorphological observations of bursa of Fabricius in sonali chicken. Journal of Advanced Biotechnology and Experimental Therapeutics. 3. 20-28. 10.5455/jabet.2020.d103.

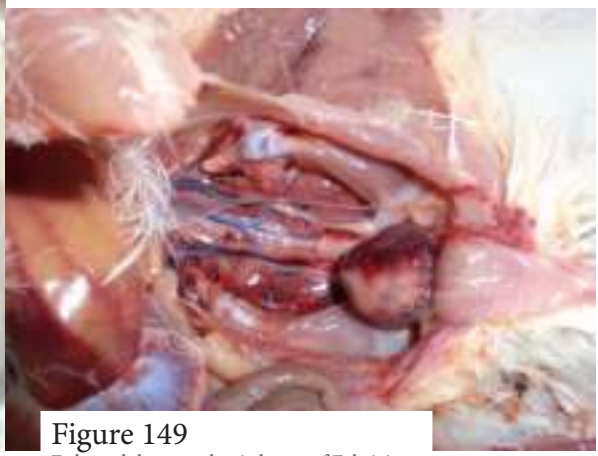


Figure 149
Enlarged, haemorrhagic bursa of Fabricius in a chicken infected with virulent IBDV

The IBDV can also cause haemorrhages in skeletal muscles.

Photo source: MSD veterrinary manual

8.5 Marek's Disease (MD)

- There are no reports of occurrence in Turkana county
- Other name- fowl paralysis
- Viral disease caused by a herpes virus. The disease causes changes in many of the nerves and may cause tumours in major internal organs.
- Chickens are the main species affected, although the disease occurs rarely in some other types of birds.
- Young birds are most susceptible and most deaths occur between 8 and 20 weeks of age, although in some cases the disease may be seen in birds as young as 3-4 weeks of age or as old as one year of age.
- The virus is highly infectious and once introduced into a flock it spreads rapidly to unvaccinated birds, most chickens in an unvaccinated flock become infected. Infected chickens carry the virus for life whether they develop the disease or not, and continue to shed the virus for long periods. The virus is shed from the feather follicles and spreads readily in fluff and dust, gaining entry when the bird breathes infected dust particles. This material can also be carried by people and equipment.
- The virus can survive in the environment for as long as several months at room temperature. It is not spread from the hen to the chicken through the egg.
- Disease manifests in two forms the nervous and visceral.
- In the nervous form- there is progressive paralysis of one or more of the limbs or, less often, the neck or wings. The sciatic nerve (the main nerve supply to the leg) is commonly affected The birds are unable to stand, become paralysed, appear uncoordinated and slowly waste away from lack of food and water. In most cases the paralysis comes on quickly. In some cases, the eyes may be affected, resulting in blindness.
- In the visceral form- disease occurs as tumours in internal organs, including the ovaries, liver, spleen, kidney and heart. Sometimes the liver and spleen are swollen without distinct tumours being present. Birds may show signs of depression, paralysis, loss of appetite, loss of weight, anaemia (pale combs), dehydration (shrunken combs), and sometimes diarrhoea. Some birds die without any clinical signs being noticed.
- Most birds that develop Marek's disease die.
- Diagnosis in the field is based on clinical signs, history of age and post-mortem lesions.
  - Enlargement of nerves such as the sciatic nerve are commonly seen at post-mortem.
- Differential diagnosis from the viral disease known as lymphoid leucosis that causes tumours in organs, but does not cause paralysis. It is usually seen in birds over 16 weeks of age, whereas Marek's disease is commonly seen in younger chickens.
- No treatment as it is viral- diseased birds should be promptly removed from the flock and humanely destroyed. Other birds in the flock are likely to be infected at this stage and should be closely monitored for signs and culled.
- Vaccination alone will not prevent Marek's disease. It is important to maintain good

biosecurity measures to ensure that vaccinated chicks will develop immunity before they are exposed to a severe challenge of virus.
- Chicks need to be reared separately so that they are free from the infected fluff and dust of older birds.
- Sourcing birds that have been bred for genetic resistance to the disease.

Zoonotic potential- None reported

Notifiable disease- No – livestock keepers should ensure they purchase chicks from hatcheries or breeder farms that vaccinate against IBD.

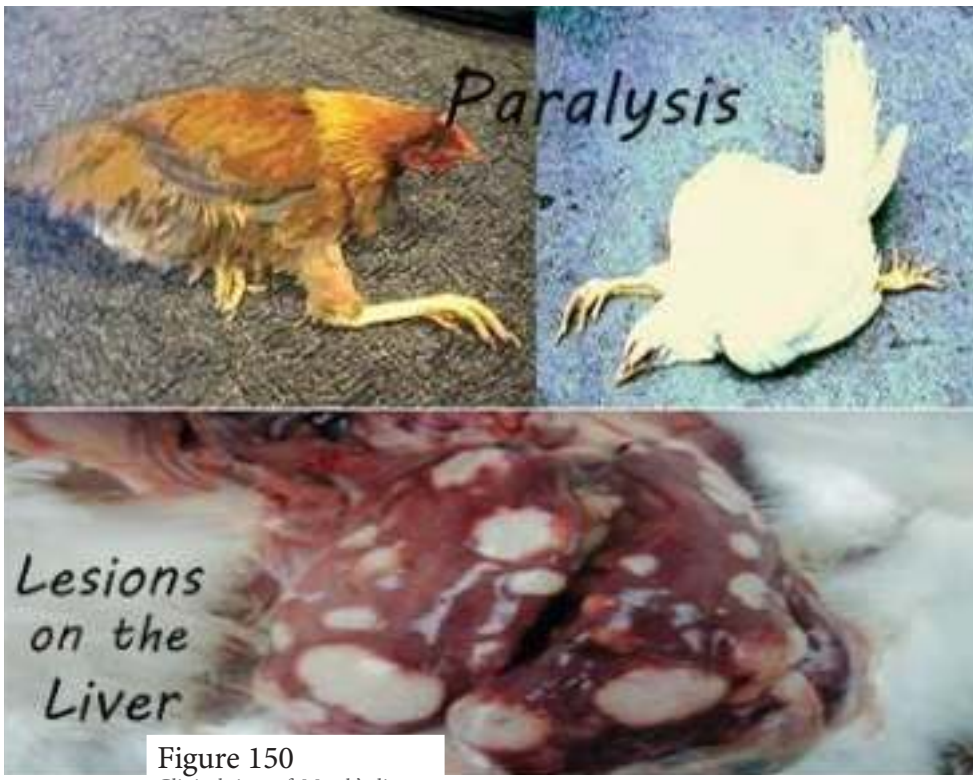


Figure 150
Clinical signs of Marek's disease

Picture source: https://learningcenter.kukuchic.co.ke/articles/37

Chicken Diseases:

Bacterial

8.6 Infectious coryza- LOKIO ANG'AKONYEN

Other name: Fowl Coryza

** Though mentioned by livestock keepers, the clinical signs indicate that the disease may not occur in Turkana county.

Causative agent: A bacteria known as Avibacterium paragallinarum (previously known as Haemophilus paragallinarum and Haemophilus gallinarum)

Transmission:

- Through direct contact, airborne droplets and contamination of drinking water
- Infected flocks are a constant threat to uninfected flocks.
- Chronically ill or healthy carrier birds are the reservoir of infection for A. paragallinarum.
- In Turkana county infectious coryza has the apparent morbidity rate of 2% with no mortalities. The disease occurs throughout the year but incidence peaks are seen during the short dry season locally known as AIT or NAIT.

Clinical Signs:

- All ages are affected though disease is usually less severe in juvenile birds.
- Causes acute inflammation of the upper respiratory tract.
- Serous to mucoid nasal discharge, coughing, sneezing, facial oedema and conjunctivitis hence the typical facial oedema (swollen head-like syndrome).
- Males may have swollen wattles
- Respiratory rales may be heard in birds with lower respiratory tract infection
- Birds may have diarrhoea, decreased feed and water consumption
- Mortality is usually lower than new castle disease but severity of disease can be complicated by other factors like poor housing, parasitism, inadequate nutrition or occurrence of concurrent diseases such as fowl pox, infectious bronchitis, infectious laryngotracheitis, Mycoplasma gallisepticum infection and Pasteurellosis, resulting in increased mortality.
- Prompt disposal of dead birds through burning.

Post-mortem findings:

- In acute cases, only the infraorbital sinuses may be involved and contain copious, grayish, semifluid exudate evident on gross inspection. In chronic cases this exudate may become consolidated
- Acute catarrhal inflammation of mucous membranes of nasal passages and sinuses
- Catarrhal conjunctivitis and subcutaneous oedema of face and wattles
- Other lesions may include: air sacculitis, conjunctivitis, pericarditis, perihepatitis, and sinusitis

Diagnosis:

- Laboratory diagnosis of the bacteria through bacterial culture. However, polymerase chain reaction testing of live flock assay has been reported to provide more accurate results.
- Isolation of a gram-negative, catalase-negative organism from chickens in a flock with a

history of a rapidly spreading disease is diagnostic for infectious coryza.
- A catalase specific test is essential as non-pathogenic haemophilic organisms, which are catalase-positive, are present in both healthy and diseased chickens.

Treatment:
- Early antimicrobial treatment with supportive care of infected birds to aid recovery is recommended.
- Antibiotics such as erythromycin and oxytetracycline are commonly used. Sulphonamides, including trimethoprim-sulfamethoxazole combinations in drinking water can also be used. New generation antimicrobials like, fluoroquinolones, macrolides are also considered effective.
- Manufacturer's instructions on drug use and withdrawal periods should always be followed.

Prevention and control:
- Once there is infection, the farm will likely have carrier birds and therefore flock clearance and disinfection are recommended.
- An "all-in/all-out" management is important as part of sound farm management and biosecurity practices.
- Ensure disinfection of facilities before entry of new flock.
- Vaccines/bacterins are not readily accessible in Kenya

Figure 151
Chicken with swollen facial tissue due to infectious corvza

8.7 Fowl Typhoid- Pullorum disease- EREMONU EKWANG / EREMONU LONYANG

****Though mentioned by livestock keepers in which they described signs of yellow diarrhoea the disease may not occur in Turkana county.

- EREMONU EKWANG means white diarrhoea while EREMONU LONYANG'-means yellow diarrhoea
- The historical name for fowl typhoid is bacillary white diarrhoea.
- Pullorum disease is caused by Salmonella enterica. Disease is characterized by very high mortality in young chickens and turkeys up to 3 to 4 weeks of age.
- Affected birds huddle near the heat source, are anorectic, weak, depressed, and have white faecal material pasted to the vent area. In addition, the birds may have respiratory disease, blindness, or swollen joints.
- **Transmission** can be vertical (transovarian) but also occurs via direct or indirect contact with infected birds (respiratory or faecal) or contaminated feed, water, or litter.
- **Infection transmitted** via egg or hatchery contamination usually results in death during the first few days of life up to 2–3 weeks of age.
- Survivors are small in size and frequently become asymptomatic carriers with localized infection of the ovary. Some of the eggs laid by such hens hatch and produce infected progeny.
- Recent evidence of vector borne transmission of Salmonella enterica enterica serovar Gallinarum and fowl typhoid disease mediated by the poultry red mite, Dermanyssus gallinae (De Geer, 1778).
- **Post mortem lesions**- Lesions in young birds usually include unabsorbed yolk sacs and classic gray nodules in the liver, spleen, lungs, heart, gizzard, and intestine. Firm, cheesy material in the ceca (cecal cores) and raised plaques in the mucosa of the lower intestine are sometimes seen. Occasionally, synovitis is prominent. Adult carriers usually have no gross lesions but may have nodular pericarditis, fibrinous peritonitis, or haemorrhagic, atrophic, regressing ovarian follicles with caseous contents.

- **Field based diagnosis** is from observation of necropsy lesions and should be confirmed by isolation, identification, and serotyping of S enterica Gallinarum.

- **Treatment and control**- Freedom from infection and elimination of positive birds and flocks is key to control. Treatment will not eliminate the carrier state and is never recommended. Control is based on routine serologic testing of breeding stock to assure freedom from infection. In addition, management and biosecurity measures should be taken to reduce the introduction of S enterica Pullorum from feed, water, wild birds, rodents, insects, or people. Birds should be purchased from sources free of S enterica Pullorum.

- In Kenya the vaccine is available form KEVEVAPI- Fowlvax™ that is an Inactivated Salmonella gallinarum (Fowl typhoid) Vaccine with multi-strain inactivated Fowl typhoid vaccine prepared from Salmonella gallinarum. The strains included are CN 175, 176, 177, 178, and 179.

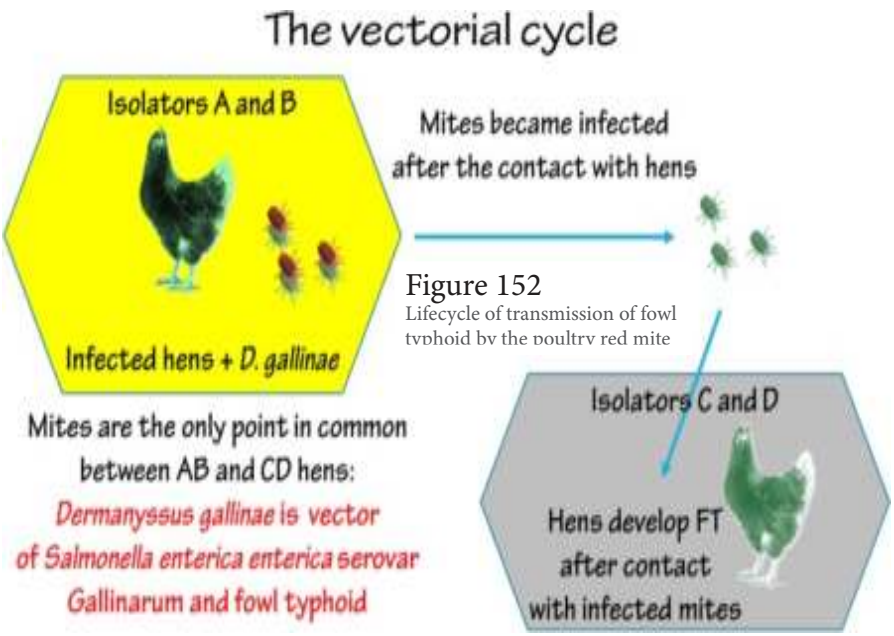


Figure 152
Lifecycle of transmission of fowl typhoid by the poultry red mite

Pictures credit source: Cocciolo, G., Circella, E., Pugliese, N. et al. Evidence of vector borne transmission of Salmonella enterica enterica serovar Gallinarum and fowl typhoid disease mediated by the poultry red mite, Dermanyssus gallinae (De Geer, 1778). Parasites Vectors 13, 513 (2020). https://doi.org/10.1186/s13071-020-04393-8

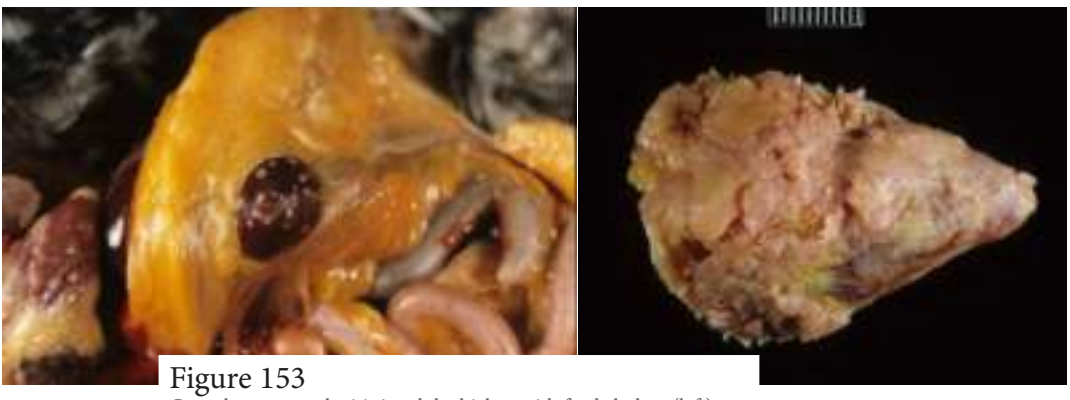


Figure 153
Granulomatous splenitis in adult chicken with fowl cholera (left) and Granulomatous pericarditis due to fowl typhoid

Photo Courtesy of Dr. David E. Swayne. MSD veterinary manual

8.8 Fowl Cholera

Other names: Avian cholera, Avian Pasteurellosis, or Avian Haemorrhagic Septicaemia

****** Not reported in Turkana county**

Causative agent:

- Bacteria - Pasteurella multocida, has different serotypes which vary in pathogenicity. The bacteria produce endotoxins. Invasion and multiplication of a strain in necessary to produce sufficient quantities of endotoxins that contribute to the virulence.
- Contagious bacterial disease that occurs as a septicaemia of sudden onset with high morbidity and mortality
- Common in birds more than 6 weeks old. Those that recover remain carriers for life
- Bacteria can survive for one month in faecal droppings and for 2-3 months in decaying carcass and soil. However, it is susceptible to sunlight, drying and disinfectants.

Transmission:

- Spread of P. multocida within a flock is mainly by excretions from mouth, nose, and conjunctiva of diseased birds. These excretions contaminate the environment, particularly feed and water.
- Chicken become infected by drinking contaminated feed and water.

Main Clinical Signs:

Acute cases:

- Signs occur for a few hours then lead to death, therefore death may be the only sign
- Other signs may be fever, anorexia, ruffled feathers, mucous discharge from the mouth, diarrhoea and increased respiratory rate
- Cyanosis mostly occurs prior to death, most evident in un-feathered areas of the head (comb and wattles)
- Diarrhoea is initially watery and whitish in colour but later becomes greenish and contains mucous
- Birds that survive acute septicaemic stage, later succumb to debilitating effects of emaciation and dehydration or become chronically infected or recover
- High flock mortality

Chronic cases - signs are related with localized infections such as:

- Swelling of the wattles, sinuses, legs or wing joints, foot pads and or sternal bursae
- Exudative conjunctival and pharyngeal lesions
- Torticollis
- Tracheal rales and dyspnoea may result from respiratory tract infections
- Lameness not paralysis
- Chronically infected birds may succumb, remain infected for long periods, or recover
- Chicken can also be asymptomatic

Post-mortem Findings:

- Marked congestion of the carcass, pinpoint haemorrhages throughout the internal organs and multiple necrotic areas (areas of dead tissue) in the liver.
- The liver may be enlarged and also show very small haemorrhages on the surface.
- In the laying hen's free yolk may be present in the abdominal cavity.
- In the less severe disease, oedema of the lungs (accumulation of fluid) and pneumonia

(inflammation of lungs) are seen.
- In chronic cases, arthritis (inflammation) of the hock and foot joints, and swelling of one or both wattles.

Diagnosis:
- Flock history, clinical signs and post mortem findings are indicative.
- Confirmed through laboratory examination.
- Isolate and identify P. multocida from specimens of heart blood, liver, and spleen.
- In acute cases, characteristic bipolar organisms may be observed in Giemsa-stained smears of heart blood.
- PCR can be used to identify carrier animals within flocks.
- Avibacterium gallinarum and Gallibacterium anatis biovar haemolytica are 2 closely related bacteria that may be isolated from diseased poultry and incorrectly identified as Pasturella multocida.

Treatment:
- Antibiotics reduce mortality but won't eliminate bacteria from the flock. Eradication of infection requires depopulation, followed by thorough cleaning and disinfection of houses.
- Antibacterial chemotherapy informed by sensitivity testing is recommended as strains of P. multocida vary in susceptibility to chemotherapeutic agents and resistance to treatment develops quickly. Use of broad spectrum antibiotics like oxytetracycline and sulphonamides fortified with multivitamins as oral treatment in water can be attempted if sensitivity testing is not accessible.

Prevention and Control:
- Good management practices, including a high level of biosecurity, are essential to prevention.
- Controlled entry into poultry houses must be observed as rodents, wild birds, pets, and other animals may be carriers.
- Disinfect poultry houses and use disinfectants in footbaths at points of entry/exit. Remember, the bacteria are susceptible to ordinary disinfectants, sunlight, drying, and heat.
- Vaccination of healthy flock with live attenuated vaccines to induce immunity. Vaccines are available for administration by wing-web inoculation.
- Prompt disposal of dead birds through burning.

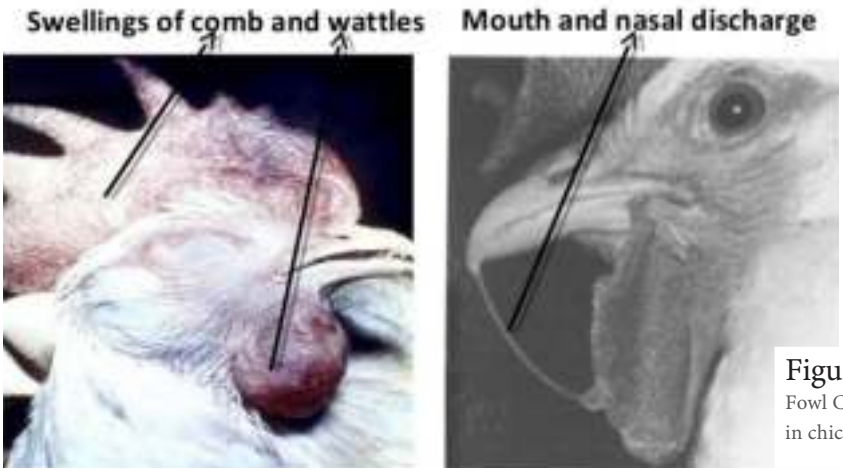


Figure 154
Fowl Cholera signs in chicken

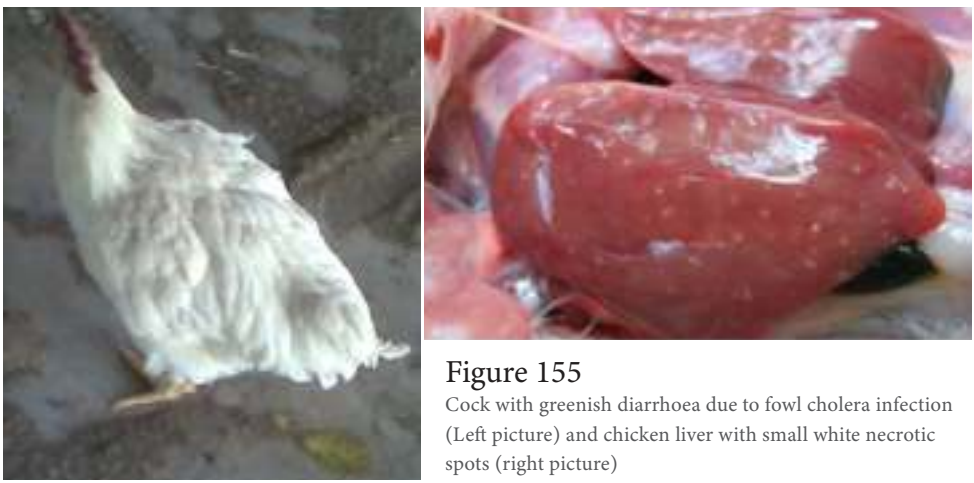


Figure 155
Cock with greenish diarrhoea due to fowl cholera infection (Left picture) and chicken liver with small white necrotic spots (right picture)

Picture source:
https://www.veterinariadigital.com/en/articulos/main-avian-diseases-found-in-india/

8.9 Chronic Respiratory Disease (CRD)

*** Disease does not occur in Turkana county

Aetiology:
- Bacteria of the genus Mycoplasma (M gallisepticum).
- Affected animals develop respiratory symptoms such as sneezing, nasal discharge or dyspnoea.
- Disease more severe in Turkeys than chicken, in chickens, infection may be inapparent or result in varying degrees of respiratory distress, with slight to marked rales, difficulty breathing, coughing, and/or sneezing.
- Morbidity is high and mortality low in uncomplicated cases but can reach 30% in cases complicated with other bacteria or viruses.

Transmission:
- Mycoplasma gallisepticum spreads via eggs, airborne transmission and indirect or mechanical routes such as introducing infected birds to an existing flock, or via bird transport containers.
- M. Gallisepticum can reside in a flock with few indications of its presence until the flock or individuals is stressed sufficiently to show signs of respiratory disease.

Clinical Signs
- Sniffling, sneezing, coughing and other signs of respiratory distress like rales.
- When suspected, observe the birds from a distance to get a good idea of the symptoms as birds often do not display signs of disease once handled.
- Sometimes CRD slowly spreads through the flock causing poor production, retarded growth and wet noses. In these cases, there are usually few mortalities unless the underlying stressors increase.

Post-mortem lesions
- Lesions that appear are very variable, depending on whether there are other concomitant infections
- Sinusitis, tracheitis, air sacculitis, thickening and turbidity of the alveoli, exudative accumulations, fibrinopurulent pericarditis and perihepatitis could be observed.

Differential diagnosis
- Infectious Coryza, Infectious bronchitis, Infectious laryngotracheitis and Fowl cholera.
- Treatment
- Antibiotics can help to control the disease but reducing stress is an essential component of managing the disease once it is in the flock.
- Most strains of M gallisepticum are sensitive to a number of broad-spectrum antibiotics, including Tylosin, Tetracyclines, and others but not to penicillins or those that act on the cell wall.
- Tylosin or tetracyclines have been commonly used to reduce egg transmission or as prophylactic treatment to prevent respiratory disease in broilers and turkeys.
- Antibiotics may alleviate the clinical signs and lesions but do not eliminate infection. Regulations on the use of antibiotics in food animals are rapidly evolving and should be consulted before use.

Prevention

- Obtain chicks or poults from M gallisepticum-free breeder flocks that have been vaccinated.
- Commercial vaccine is available but not easily accessible in Kenya for small scale farmers.
- Management issues must be addressed before the birds arrive. Ensure birds are free of M. Gallisepticum on introduction. An all-in, all-out system with thorough cleaning down between batches and acquiring vaccinated birds helps.
- Ensure the nutrition and flock dynamics (size, age distribution, presence of roosters) are also optimal.
- Housing that is difficult to clean and thus accumulates manure, dust and vermin leads to many common ailments in poultry.

Figure 156
Post-mortem lesions of a bird with CRD with abdominal sac thickened and cloudy with blood vessels visible

There are many causes of airsacculitis in chickens and turkeys, but M gallisepticum should always be considered as the primary causative agent.

Photo credit:
MSD veterinary manual

Chicken Diseases:

Protozoal

8.10 Coccidiosis – EROMONU ANG'AKOT ANG'IKIENY

EROMONU ANG'AKOT- bloody diarrhoea

Causative agent: Protozoan parasite of the Eimeria species that infect various sites in the intestines where it multiplies causing damage. There are about nine species that infect chicken and about seven that infect turkeys. Eimeria species are considered host specific meaning those that infect chicken do not infect turkeys and vice versa. Eimeria life cycle takes 7 to 9 days.

Transmission:

- The sporulated oocyst is the infective stage of the parasite and is shed by both Infected and recovered birds.
- Chicken get infected when they ingest the infective form of the oocysts (sporulated oocysts)
- Oocysts can be spread mechanically by animals, wild birds, insects, clothing and human footwear, contaminated equipment, or in some cases, by wind/dust while spreading poultry-house dust and litter over short distances.

Main Clinical Signs:

Coccidiosis occurs in two forms:

Large intestine/ Caecal coccidiosis is caused by Eimeria tenella. Disease is severe and signs include:

- Bloody diarrhoea
- Reduced weight gain
- High mortality
- Unthriftiness and emaciation

Small Intestinal coccidiosis is caused by Eimeria necatrix, E. acervulina, E. brunetti, E. maxima, E. mitis, E. mivati, E. necatrix and E. praecox.

- Severe weight loss
- Droppings contain blood
- High morbidity.
- Decreased egg production
- Depressed feed and water consumption

Post mortem Findings:

The main lesions are bloody intestinal content, enteritis and mottled intestinal wall. The lesions are different depending on the species as follows:

- E. acervuline: lesions include numerous whitish, oval or transverse patches in the upper half of the small intestine, which may be easily distinguished on gross examination.
- E. mivati: lesions develop in the anterior part of the small intestine. In severe infections, E. mivati may cause reddening of the duodenum that may be visible through the serosa of the distal duodenum and proximal jejunum.
- E. necatrix: major lesions are found in the anterior and middle portions of the small intestines. Small white spots, usually intermingled with rounded, bright- or dull-red spots of various sizes, can be seen on the serosal surface. This appearance is sometimes described as "salt and pepper." The white spots are diagnostic for E. necatrix if clumps of large schizonts can be demonstrated microscopically.
- E. maxima: develops in the small intestine, where it causes dilatation and thickening of the wall; petechial haemorrhage; and a reddish, orange, or pink viscous mucous exudate and fluid. The exterior of the midgut often has numerous whitish pinpoint foci, and the area

may appear engorged.
- E. tenella: haemorrhagic typhlitis (inflammation of the cecum). Recognized by accumulation of blood in the ceca and by bloody droppings.
- There may be accumulations of clotted blood, tissue debris, and oocysts in ceca of birds surviving the acute stage.

Diagnosis:
- Tentative diagnosis is based on flock history, clinical signs and lesions.
- Post mortem examination within one hour of death before the post mortem changes take effect in the intestinal mucosa.
- A definite diagnosis is made by observation of Eimeria Oocysts in the laboratory.
- Note that the finding of a few oocysts by microscopic examination of smears from the intestine indicates the presence of infection, but not a definite diagnosis of clinical coccidiosis.
- Coccidia and mild lesions are present in the intestines of birds 3–6 weeks old in most flocks. Diagnosis should be based on finding lesions that confirm the microscopic findings.

Prevention and Control:
- Prevention is mainly with anti-coccidia drugs, good hygiene, disinfection and sanitation of premises and providing clean feed and water.
- Early treatment during outbreaks will prevent large scale bird losses.
- Use of antibiotics combined by supplemental provision of vitamins A and K are incorporated in the feed for treatment. Medication used include: amprolium, sulphonamides and toltrazuril.
- A species-specific immunity develops after natural infection, the degree of which largely depends on the extent of infection and the number of reinfections.

Figure 157
Damage to the intestinal wall caused by E. necatrix

8.11 Helminthiasis – NGI'PEELEI / NG'IRITAN

Causative agents:

- Mainly nematodes (roundworms) and cestodes (tapeworms) Ascarids are the main round worms that infect chicken. Gapeworms are found in the trachea, capillaria in the intesines, heterakis in the ceca, trichostrongyles and tapeworms in the intestines while gizzard worms in the gizzard.

Transmission:

- Direct transmission from bird to bird by ingestion of infective eggs or larvae.
- Indirectly through an intermediate host (insect, snail or slug).
- Helminthiasis cases are reported throughout the year with no seasonal prevalence.

Main Clinical Signs:

- General unthriftiness and inactivity
- Depressed appetite
- Suppressed growth
- In severe cases, may result in death
- Gapeworms cause chickens to gasp with neck extended

Diagnosis:

- Detection of worm eggs by faecal flotation allows for the reliable confirmation of the presence of worms. Note that absence of eggs in a sample does not necessarily mean worms are absent.

Treatment:

- Fenbendazole is effective against nematodes when given in drinking water at 125 mg/litre for 5 days. Do not administer during moult as it interferes with feather re-growth
- Albendazole can be administered as a single oral suspension at a dose of 5 mg/kg bird weight. This is reported to be effective against A galli, H gallinarum, and C obsignata. It has also has been reported effective against cestodes if administered at 20 mg/kg
- Levamisole is reported to be effective against A dissimilis, H gallinarum, and C obsignata if given in drinking water at 0.03%–0.06%.
- Other medications such as piperazine citrate are available in the market and should be used according to manufacturer's instructions.

To reduce the potential spread of resistance, treatment should be limited to birds with severe infection that show clinical signs of disease.

Prevention and Control:

- Improvement of management and sanitation is key. In free range birds, move the chicken, although the benefit that may result will be of short duration.

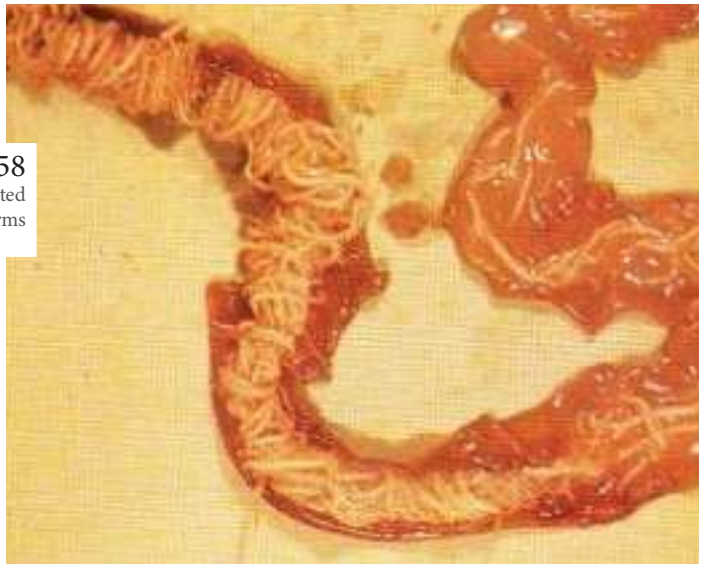


Figure 158
Chicken intestinal tract impacted by Ascarid worms

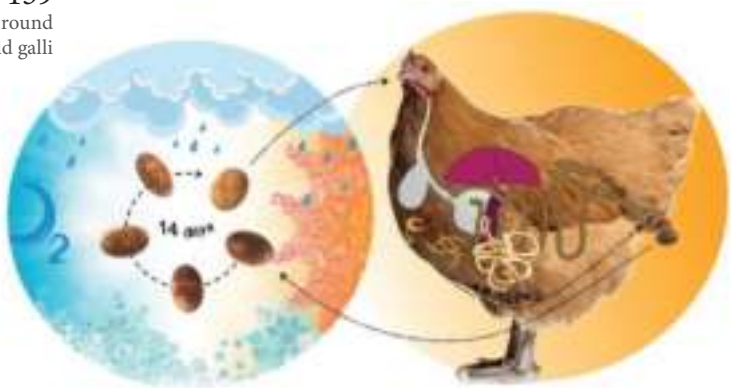


Figure 159
Life cycle of the large round worm of chicken Ascarid galli

Picture source:
https://core.ac.uk/download/pdf/211564736.pdf

The table below summarises important helminth parasites pathogenicity in chicken

Parasite	Intermediate host or lifecycle	Organ infected	Pathogenicity
Nematodes:			
Ascaridia galli	Direct	Small intestine	Moderate
Capillaria annulata (also known as Eucoleus annulata)	Earthworm	Esophagus, crop	Moderate to severe
Capillaria caudinflata (also known as Aonchotheca caudinflata)	Earthworms	Small intestine	Moderate to severe
Capillaria contorta (also known as Eucoleus contorta)	None or earthworms	Mouth, esophagus, crop	Severe
Capillaria obsignata (also known as Baruscapillaria obsignata)	Direct	Small intestine, ceca	Severe
Cheilospirura hamulosa	Grasshoppers, beetles	Gizzard	Moderate
Dispharynx nasuta	Sowbugs	Proventriculus	Moderate to severe
Gongylonema ingluvicola	Beetles, cockroaches	Crop, esophagus, proventriculus	Mild
Heterakis gallinarum	Direct	Ceca	Mild, but transmits agent of histomoniasis
Oxyspirura mansoni	Cockroaches	Eye	Moderate
Strongyloides avium	Direct	Ceca	Moderate
Subulura brumpti	Earwigs, grasshoppers, beetles, cockroaches	Ceca	Mild
Syngamus trachea	None or earthworm	Trachea	Severe
Tetrameres americana	Grasshoppers, cockroaches	Proventriculus	Moderate to severe
Trichostrongylus tenuis	Direct	Ceca	Severe
Cestodes:			
Choanotaenia infundibulum	House flies	Upper intestine	Moderate
Davainea proglottina	Slugs, snails	Duodenum	Severe
Raillietina cesticillus	Beetles	Duodenum, jejunum	Mild
Raillietina echinobothrida	Ants	Lower intestine	Severe, nodules
Raillietina tetragona	Ants	Lower intestine	Severe

8.12 Ectoparasites

There are a number of ecto-parasites that infest chicken as shown in the table:

English name	Causative agent
Fleas – NG'IKADESDES	The main flea pest is the sticktight flea; Echidnophaga gallinacean. Attach on the skin of the head
Lice - ELACHIT	Chewing lice; Order Mallophaga. They feed on feathers, feather debris, skin scales and other dermal scurf.
Ticks - EMADANG	Argasid Ticks (soft-shelled ticks) are most common. Argasid ticks are nocturnal feeders and favor the soft un-feathered skin beneath the wings

- External parasitism in chicken flocks i occurs throughout the year with no seasonal outbreaks reported.

Main clinical signs:

Ectoparasites affect chicken by:

- Transmitting diseases to chicken- ticks transmit spirochetosis.
- Sucking blood causing birds to be weak, less productive and more vulnerable to disease. If too many, cause anaemia and death in young birds.
- Birds spend more time scratching leading to reduced growth and egg production.
- Lice cause irritation and damage to feathers that results in depluming.

Diagnosis:

- Heavy mite infestation is characterized by anaemia and the appearance of black mite dirt that is the exoskeleton casts and excreta of the mites.
- Mites also cause dermatitis of the vent area.
- Birds parasitized with soft-shelled ticks (Argas species) show multiple hematomas associated with biting sites.
- Chronic infestation of the legs of free-roaming chickens with scaly leg mites, Knemidocoptes mutans, results in proliferation of scales overlying the shanks and feet. The gross appearance of the lesion is pathognomonic of Scaly Leg Mites. The diagnosis may be confirmed by microscopic examination of scales to detect the mite.
- Evidence of mature adult lice on examination of chicken which appear as brown tiny parasites attached to the skin of the head is diagnostic for lice infestation.
- Under the microscope lice egg clusters ("nits") are observed as spherical white structures adherent to the shafts of feathers.

Treatment:

- Ectoparasites may be treated with a carbamate insecticide such as Carbaryl 7.5% insecticide powder – external use (Sevin Poultry dust) applied as a powder to birds at two-week intervals. Cages and housing can be treated with carbamate dust topically. Application should ensure it is up to the base of the feathers.
- Only approved insecticides should be applied to poultry or used in the vicinity of housing to avoid contamination of the food chain. Insecticides should be used in accordance with manufacturers' label instructions.

Prevention and Control:

- Ectoparasites are also found in the poultry premises like wall cracks, perch joints, rough floor, in litter and in nest boxes. These surfaces should also be dusted with the acaricide powder.
- Isolation of all new birds and application of acaricide powder before allowing them to mix with the flock should be done
- Regular spraying of the chicken house with acaricide applications recommended for use in chicken such as Ectomin 100 EC (cypermethrin) at a ratio of 1:1.
- Birds can also be dipped to allow better penetration of the chemical. Immerse the chicken one by one into the Ectomin solution. Always follow manufacturer's instructions on use to ensure birds are not poisoned.

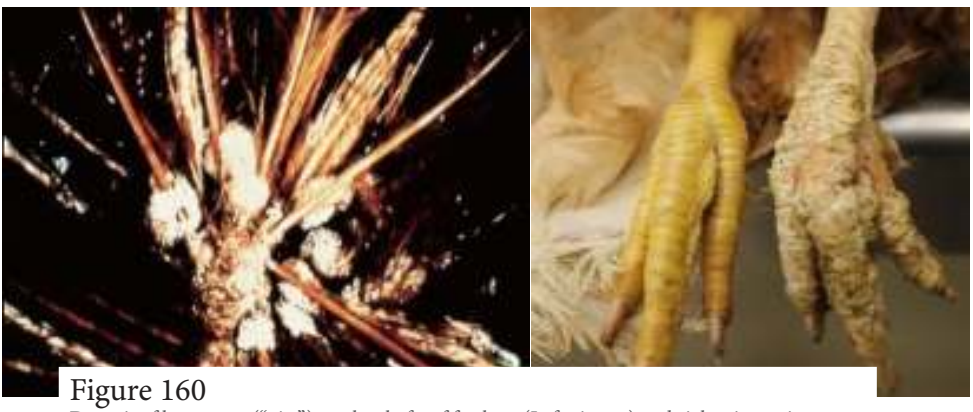


Figure 160

Deposit of louse eggs ("nits") on the shafts of feathers (Left picture) and right picture is a comparison of the normal leg compared to scaly leg of a chicken infested with scaly leg mite

Table 25: Vaccination schedule for indigenous birds

Day/Week	Vaccination	Mode	Remarks
Day 1	Mareks	Subcutaneous injection	Done at the hatchery
Day 10	Gumboro 1st dose	Drinking water	If done at Hatchery do not give. Use boiled water
Day 18	Gumboro 2nd dose	Drinking water	If done at hatchery do not give. Use boiled water
3 weeks	New castle 1st doze	Eye drop or drinking water	If done at hatchery do not give Use boiled water
3 weeks (hot areas) 6 weeks (other areas)	Fowl pox	Wing slab	Use skilled personnel
8 weeks	Newcastle 2nd dose Fowl Typhoid	Eye drop or drinking water Intra muscular injection	If done at hatchery do not give. Use boiled water Use skilled personnel
18 weeks	Newcastle (3rd dose	Eye drop or drinking water	If done at hatchery give 2nd dose now and repeat every 3 months
19 weeks	Deworm using levamisole, albendazole, piperazine, pyrantal pamoate	Drinking water	Repeat every 3 months

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Picture Source: Picture book of infectious poultry diseases (2010). Produced by FAO ECTAD Southern Africa. https://www.itpnews.com/uploads/2017/03/color%20atlas%20of%20poultry%20disease%20-%2001.pdf?direct_access_media=1

8.13 Crop impaction- EDEKE ETOLE

- The crop is an enlarged part of the oesophagus located to the right of the trachea at the thoracic inlet. The walls of the crop are thin, and the organ is attached to the skin and clavicle by loose connective tissue.
- There are 2 muscles that support the crop and aid in the emptying process. The main function of the crop is to store feed while the proventriculus and gizzard are full of feed. Shortly after feed is ingested, it is normal for the crop to be distended and full of feed. As the feed in the gizzard is moved into the intestine, the crop begins to contract, emptying its content into the oesophagus and supplying more feed to the proventriculus and gizzard.
- Crop impaction occurs when the stored material cannot be propelled forward into the oesophagus. Crop impaction normally develops when a group of fibers get tangled together into a mass that is too big to pass from the crop to the oesophagus.
- Crop impaction is more common in free-range birds as they have access to foreign objects (long pieces of plastic or thread). Sick birds, especially those with intestinal problems, are more likely to consume large amounts of roughage or grit.
- Worms can over-populate the intestinal tract to the point of blocking off the entire passage, causing the crop to become impacted.
- Under normal circumstances, the crop will usually empty in 2-4 hours. In birds with crop impaction, the crop will not empty. On palpation, the contents of the crop will feel firm. Birds will show reduced appetite, weight loss, and depression.
- Diagnosis is through palpation of the crop early in the morning before the birds has fed. If there is any content in the crop in the form of a mass with variable consistency, then the crop is impacted.
- Treatment - with early detection, a gentle massage of the crop may be all that is needed. Adding warm water into the crop before the massage can help to hydrate the content and make it softer. To inject water into the crop, a special needle is used: 1.5-2 inches long with a rounded blunt end that is approximately 3 mm in diameter. The rounded end is designed to prevent trauma in the oral cavity. If that needle is not available, any soft tubing connected to a syringe will work but it may take longer) to direct water into the crop. Use ~3-10 mL of lukewarm water, depending on the size of the bird. Cooking Oil can also be added to the water as a lubricant. Massage the crop gently for 3 minutes. The process may have to be repeated several times for a couple of days. If the previous treatment is unsuccessful, surgery may be needed (impractical in field conditions).

Figure 161
Crop impaction in a chicken

Reference- https://extension.psu.edu/crop-disorders-of-chickens-i-crop-impaction

Chapter 9

Donkey Diseases

Table 26: Priority donkey diseases as ranked by livestock keepers in Turkana County

Conventional name	Turkana name
Faecal impaction	KORIANG'
Tetanus	LOTERU / ETEREGEGE / ESIDENG'DENG'IT
Anthrax (See Annex 2: zoonosis diseases	ENOMOKERE
Helminthiasis	NG'IPEELEI / NG'IRITAN / NG'ILOMUN
Mange	LOTOME / LOGIRGIRA
Saddle wounds	NG'AJEMEI
Rabies (see Annex 2: zoonotic diseases)	LONG'OKUO NG'IKEREP
Sarcoid	AMODING
Laminitis	EKAMUCHURUT
Strangles	LONYANG' SURUMA
Babesiosis / Equine piroplasmosis (There is no record of Anaplasmosis in donkeys)	LONYANG / LOPID

Table 27: Medicinal plants and traditional practices used to treat donkey diseases by livestock keepers in Turkana County

Botanical and common name	Turkana name	Target disease
Aloe Turkanensis- succulent leave sap given as an oral drench	ECHUCHUKA	Strangles Pneumonia Anaplasmosis
Cissus quadrangularis succulent part of the plant is pounded and soaked in cold water and given as an oral drench	EGIS	Faecal Impaction
Azadirachta indica- Neem tree leaves are boiled or soaked in water and given as an oral drench when cool.	MWARUBAINI	Anaplasmosis
Cyperus species- a sedge plant pounded and soaked in cold water and given as an oral drench	EKERIAU	Strangles
Nicotiana tabacum- chewing tobacco ground and given as an oral drench	ASIJI	Strangles
Maerua crassifolia – fruits/seeds pounded with a little water and paste topically applied.	ERENG	Mange
Boscia coriacea- fruits/seeds pounded with a little water and paste topically applied.	EDUNG	Mange
Traditional practice	Turkana name	Target Disease
Manual extraction of impacted faeces using a curved small ruminant rib or a hand	AKUTAN AMARAN	Faecal Impaction
Oral drench of fat from the tail of a sheep	AKURING EMESEK	Faecal Impaction
Oral drench Magadi soda salt	AMAKAT	Faecal Impaction
Bleeding from jugular vein or ear vein (drain approximately 500 mls)	AKIGUM NG'AKOT	Strangles
Hot iron or stone cauterization of affected body part	AKIMAD EMACHAR KORI AMORU	Haemorrhagic septicaemia Pneumonia Laminitis
Topical application of fat from the tail of a sheep	AKIWOS AKIMIET ASONYOK KORI AKIDEDIET	Mange
Topical application of used engine oil	AKIWOS AOILLOLO	Mange
Scrub skin with sand then wash off with clean water	AKIRIGARE ASINYEN KA NG'AKIPI	Mange
NB: Text in red highlights practices that go against animal welfare principles		

Table 28: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority donkey diseases

Type of Veterinary Medicinal Product VMP	Active ingredient	Turkana name	Target Donkey disease
Antibiotic	Oxytetracycline	EDAWA LOMUNG' EDAWA ALOUKOI	Strangles Anaplasmosis Pneumonia
	Tylosin	EDAWA LA NG'IUKOI	Pneumonia
	Procaine Penicillin G and Dihydrostreptomycin (Penstrep®)	EDAWA LOKAKUON LO AKWAAN	Pneumonia Strangles
Anti-protozoal	Homidium Chloride (Novidium®)	EDAWA LOARENG'AN (RED TABLET) / LA ARENGAN	Strangles Anaplasmosis Pneumonia
Antiparasitic	Ivermectin – oral drench of injectable drug	EDAWA ALOTOME / EDAWA LOMOO/ EDAWA LOKIRION	Mange Anaplasmosis Helminthiasis Feacal impaction
Acaricide	Amitraz Synthetic Pyrethroids (Ectopor®) Organophosphate	ADIP NA NG'IGURAI ADIP	Mange

Text in red indicate extra-label use of drugs. The practice will encourage AMR and may cause drug reaction

Table 29: List of notifiable Equine (Donkey) diseases that should be reported to DVS and WOAH*

African horse sickness
Anthrax
Contagious equine metritis
Dourine (T. equiperdum)
Equine encephalomyelitis (Eastern and Western)
Equine infectious anaemia
Equine influenza
Equine piroplasmosis
Equine rhinopneumonitis
Equine viral arteritis
Glanders (B. mallei)
Japanese encephalitis
Rabies
Vesicular stomatitis
Venezuelan equine encephalomyelitis
West Nile fever

Donkey Diseases:

Bacterial

9.1 Strangles - LONYANG' SURUMA

Other names- Horse distemper

Case definition

- Strangles is a highly contagious Upper Respiratory tract (URT)disease of equines characterised by abrupt onset of fever, mucopurulent nasal discharges, coughing and abscesses of the submandibular and retropharyngeal lymph nodes.
- The disease is caused by the bacteria Streptococcus equi subsp. equi (S. equi) and affects horses and donkeys of all ages.
- The bacteria can persistently and asymptomatically colonize the upper respiratory tract of animals for years and this serves as a source of new infection. Carrier animals are important for maintenance of the bacteria between epizootics and initiation of outbreaks on premises previously free of disease

Epidemiology

- Occurs in sporadic and epidemic outbreaks.
- Strangles occurs in all equine rearing parts of the world.
- Affects horses, donkeys and mules of all ages.
- In donkeys, it is common in donkeys that are less than two years old (except those under four months of age, which are protected by colostrum-derived passive immunity).
- Previous infection confers immunity; some animals may contract the disease for a second or third time. Especially if they have underlying chronic diseases or are malnourished.

Aetiology, Pathogenesis and Transmission

- Streptococcus equi subspecies equi which is a Gram-positive, beta-haemolytic coccobacillus.
- The bacterium is an obligate parasite of the URT that relies on its host for survival and inter epizootic maintenance.
- Survival requires moisture and protection from sunlight.
- Transmission is through direct or indirect contact with oral and nasal secretions. After entry through the oral or respiratory systems the bacteria attach to the cells of the tonsillar crypts and the ventral surface of the soft palate then moves to the lymph nodes.
- Subsequent migration of neutrophils into the lymph nodes causes their swelling and abscessation
- Communal drinking points and overcrowding are important risk factors.
- S. equi survives for several weeks in water troughs, but not in soil or pasture.
- Recovered animals are a persistent source of infection as they may infection in the pharynx and guttural pouches for months to years.

Clinical signs

- Incubation period of 1 to 3 weeks.
- Suddenly development of complete anorexia and depression.
- High Fever (39.4°–41.1°C)
- Serous nasal discharge that rapidly becomes copious and mucopurulent.
- Dyspnoea and dysphagia due to enlargement of retropharyngeal lymph nodes.

- Death by asphyxiation due to swollen URT lymphnodes.

About 20% of clinical cases have a severe or complicated form that includes;

- Development of suppurative necrotic bronchopneumonia secondary to the aspiration of pus from internal ruptured abscesses or metastatic infection to the lungs.
- Guttural pouch infection with empyema due to rupture of abscesses in the retropharyngeal lymph nodes.
- Metastatic infection, also known as "bastard strangles", results in the formation of abscesses in the lungs, mesenteric lymph nodes, liver, spleen, kidneys and brain.
- Purpura haemorrhagica which is a rare complication caused by bleeding from capillaries which results in red spots on the skin and mucous membranes.

Diagnosis

- Field based diagnosis using characteristic clinical signs
- Confirmed by bacterial culture of exudate from abscesses or nasal swab samples.
- Haematology analysis reveals leucocytosis due to neutrophilia.
- Nasopharyngeal swab samples for PCR are also used widely in strangles diagnosis.

Treatment

- Topical application of warm compresses (hot fomentation) with or without dissolving epsom salt in the warm water should be applied to sites of lymphadenopathy twice a day to facilitate maturation of abscesses.
- Aseptic lancing of mature abscesses to facilitated drainage improves recovery.
- Ruptured abscesses should be flushed with dilute (3%–5%) povidone-iodine solution for several days until discharge ceases.
- Administration of non-steroid based pain killers like phenylbutazone (dose of 4.4 mg IV once) to reduce pain and fever in sever disease should be considered.

Antimicrobial therapy is controversial.

- Initiation of antibiotic therapy after abscess formation may provide temporary clinical improvement in fever and depression, but it ultimately prolongs the course of disease by delaying maturation of abscesses. Administration of penicillin during the early stage of infection (less than 24 hours after fever onset) will arrest abscess formation.
- Antibiotic therapy is only indicated in life saving situation like when the animal develops dyspnoea, dysphagia, prolonged high fever, and severe lethargy and anorexia.
- The other disadvantage of early antimicrobial treatment is that the animal fails to mount a protective immune response, rendering the donkey susceptible to another bout of infection after recovery.
- If antimicrobial therapy is indicated, the combined antibiotic Penstrep® (procaine penicillin G and dihydrostreptomycin) should be the first choice drug. For the short acting preparation give 1 ml per 20 kg body weight (BWT) for 3 days and for the long acting preparation give once at 5 mls of Penstrep 20/20 LA per 50 kg BWT.

Control and prevention

- Clinically affected animals should be separated from healthy ones and should not share watering troughs with the rest.

- Quarantine of sick animals should not be less than 14 days and where possible introduction to the herd should be after two negative nasal swabs. However, this is not possible in Turkana context. Livestock keepers should integrate animals into herd when the animal has stopped having nasal discharge and abscesses have stopped leaking pus.
- Immunity after infection is prolonged and is associated with local mucosa antibody production.
- Several vaccines have been developed but protection is only 50% for those that are not administered through the intranasal route. The intranasal vaccines were associated with side effects including development of disease in animals.
- No vaccination is done in Kenya as there is no safe vaccine.

Zoonotic potential- None reported
Notifiable disease- No

Figure 162
A donkey with typical Strangles clinical signs

Picture credit: The clinical companion of the donkey (2021). The Donkey Sanctuary. https://www.thedonkeysanctuary.org.uk/sites/uk/files/2018-03/the-clinical-companion-of-the-donkey.pdf

9.2 Tetanus- LOTERU / ETEREGEGE

Other names- Lock jaw

Case definition

- Tetanus is a toxic reaction to a specific poison (toxin) that blocks transmission of inhibitory nerve signals to muscles. This leads to severe muscle contraction and an exaggerated response to stimuli without a relaxation phase.
- The toxin is produced by the bacterium Clostridium tetani in dead tissue. Most mammals are susceptible, but horses and humans are the most sensitive of all species.

Aetiology and Epidemiology

- Bacteria Clostridium tetani Clostridium tetani is found in soil and intestinal tracts.
- It is introduced into the body through wounds, particularly deep puncture wounds. Sometimes, the point of entry cannot be found.
- The bacteria remain in the dead tissue at the original site of infection and multiply. As bacterial cells die and disintegrate, the potent nerve toxin is released. The toxin spreads and causes spasms of the voluntary muscles.

Clinical signs

- Incubation period varies from 1 to several weeks but usually takes an averages of 10 to 14 days.
- Clinical signs begin as localized stiffness, of the muscles of the jaw, neck and hind limbs, near the region of the previous deep puncture wound.
- General stiffness becomes pronounced about later spasms and painful sensitivity to touch become evident. As the disease progresses, the reflexes increase in intensity and the animal is easily excited into more violent, general spasms by sudden movement or noise. The spasms may be so severe that they cause bone fractures. Spasms of head muscles cause difficulty in grasping and chewing of food, hence the common name, lockjaw.
- Donkeys also has flared nostrils, protrusion of the third eyelid, difficulty breathing and inability to lie down or stand.
- Donkey may have extensor rigor 'saw-horse stance' with stiff rigid legs and hind limbs stretched out behind.
- Collapse and death from respiratory arrest (usually within 3 to 10 days of the onset of
- clinical signs)

Diagnosis

- Field based diagnosis should be based on wound history and clinical signs.

Treatment and Prevention

- Treatment may be successful if done early but this is rarely the case as by the time the clinical signs start they toxin effect are irreversible.
- Treatment involves thorough cleaning and disinfection of the wound, the use of antibiotics, and tetanus antitoxin (which helps protect against the effects of additional toxin being released).
- Recovering animals as part of supportive nursing care should be kept in a dark, quiet area. This is not practical in Turkana context.
- Immunization can be accomplished with tetanus toxoid and is usually recommended

for all horses and donkeys. Yearly booster injections of toxoid are advisable.

- Breeding females' animals should be vaccinated during the last 6 weeks of pregnancy and the foals vaccinated at 5 to 8 weeks of age. In high-risk areas, foals may be given tetanus antitoxin immediately after birth and every 2 to 3 weeks until they are 3 months old, at which time they can be given toxoid.

Zoonotic potential- None reported
Notifiable disease- No

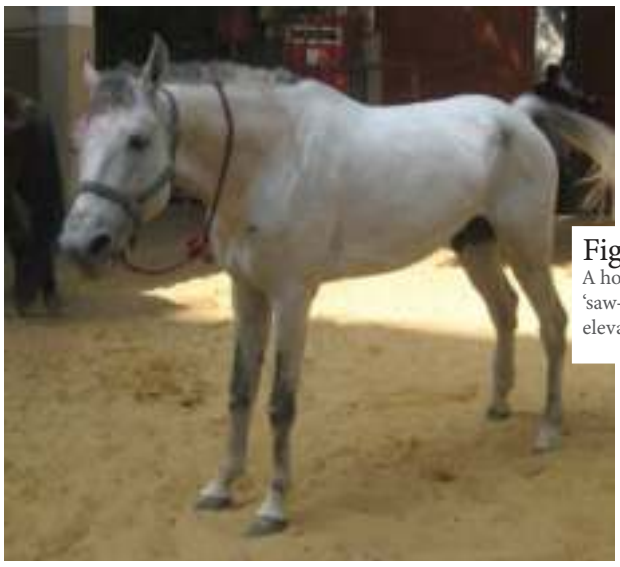


Figure 163
A horse showing classic signs of Tetanus 'saw-horse stance'. Note the erect ears, elevated tail and stiff-legged rigid stance.

Picture credit: The Working Equid Veterinary Manual (2016). A BROOK publication https://www.thebrooke.org/our-work/working-equid-veterinary-manual

9.3 Wounds - NG'AJEMEI

Aetiology

- Most wounds result from repetitive trauma or predisposing factors like poor harnessing, and carrying of inappropriate loads. In Turkana county donkeys also get wounds from predation due to hyena bites.
- Wounds and abscesses in donkeys when discovered by the owner or animal health service provider are always advanced and with possible secondarily infection. This is because the long coat of the donkey hides wounds and any grooming or examinations are done infrequently.
- When managing wounds, the cause of the wound has to be addressed in order to prevent re-occurrence. This may involve education and advice on alternative options for the owner or the community.
- The prevalence of skin wounds in Turkana county was estimated by livestock keepers to be 4% with no reported fatalities.
- Wounds that become infected are swollen, hot and painful to touch. There may also be an unpleasant smell or pus oozing from the wound.

Management of Saddle wounds

- The Golden Period is a theoretical time frame of 8 hours after which a contaminated wound is described as infected. Within approximately 8 hours any bacteria present within the wound will adhere to the tissue surface and is unlikely to be removed by wound flushing and debridement. This has implications when determining a wound management protocol (e.g. open wound or closed wound). If more than 8 hours have passed it is preferable to allow a wound to heal by second intention. Suturing an infected wound will worsen the prognosis as the wound could develop deep sited infection and cause suture breakdown.
- Livestock keepers need to be trained on how to assess wounds severity so as to be able to seek veterinary intervention early. They also need to be taught basic first aid to manage severe wounds as they wait for the animal health practioner.
- Wounds that require urgent veterinary intervention include

1. Wounds that have continuous or excessive bleeding (haemorrhage)- need immediate attention before the vet arrives. Ask the livestock keeper to keep the donkey calm and still as possible. If bleeding wound is on the leg they should take care not to get injured. They should take a clean cloth and apply pressure directly to the wound. If blood seeps through the cloth they should apply a second layer of cloth on top, without removing the first so as not to dislodge the clot forming.
2. Wounds that penetrate or puncture the entire skin thickness.
3. Skin wounds on or close to a joint especially the knee, hock and elbow joints.
4. Avulsed wounds (partial or complete tearing away of skin and the tissue beneath) as seen in predation wounds.
5. Skin Wounds of the lower leg can be serious even if they don't seem deep.

Management of severe wounds involves identification of all involved structures (synovial cavities, bones, soft tissues), control of active haemorrhage and determining if there is need for surgical intervention. Surgical intervention should be conducted by a trained veterinary surgeon, surgical intervention is indicated if there is continuous and extensive blood loss that does not respond to pressure application, tendon injury, penetration of a synovial structure or body cavity like the thoracic or abdominal and extensive avulsion of the skin and underlying tissues.

Steps of treating skin wounds;

1. Wash the wound by running cold, clean water over the wound to remove dirt and to allow visualisation of the wound depth and margins.
2. Clean the wound using a gauze swab and antiseptic solution that is diluted according to manufacturer's instructions.
3. Trim the hair around the wound to help keep the wound clean.
4. Infected wounds should be lavaged with 1 to 2% povidone Iodine. Current research indicates that hydrogen peroxide used to clean infected wounds actually works against wound healing and should not be used
5. In case of infected puncture wounds establish proper drainage by lancing open the puncture wound area.
6. Give tetanus toxoid, analgesic such as phenyl butazone and appropriate antimicrobial therapy such as Penstrep®. For the short acting preparation give 1 ml per 20 kg body weight (BWT) for 3 days and for the long acting preparation give once at 5 mls of Penstrep 20/20 LA per 50 kg BWT. As a general rule do not give corticosteroids as they delay the inflammatory process of forming granulation tissue which is one of the processes of wound healing.
7. If skin wounds are due to rabid animal, ensure you administer a rabies vaccine to animal at a post exposure dosage on Day 0, Day 3, Day 7, Day 14 and Day 28. If one is not able to give all the days, the general practice is that the animal should receive not less than 3 doses with the first dose given within 3 days of the bite.
8. Repeat wound lavage with Povidone iodine every day with 1 to 2 % povidone iodine until the pus discharge clears.

Zoonotic potential- None reported (except if bite wounds are from rabies cases)

Notifiable disease- No

Figure 164
Open wounds over the carpus joint (left) and how to shave around a wound in a horse

Pyoderma

- This is secondary bacterial infection common in all skin conditions regardless of the primary cause.
- It is caused by many bacteria which are part of the normal flora of the skin. The bacteria invade the broken skin and colonise in the damaged tissue.
- Common causal bacteria include; Staphylococcus species (predominant bacteria in pyoderma), Corynebacterium species and Dermatophilus congolensis that causes 'rain scald'.
- Predisposing factors include damp skin due to warm and wet weather, poor grooming, skin wounds, abrasion and pruritic conditions (mange) resulting in excoriation.
- Clinical signs - Staphylococcus infection – forms crusts in a circular pattern (similar to ringworm). The skin appears wet due to exudate. Encrusted papules and pustules are evident. In severe cases a deep pyoderma with ulceration develops. There are signs of pruritus and discomfort. Inflammation – causes redness, swelling and pain. (Staphylococcus lesions are usually very painful.)
- Treatment - Clip the affected area and clean with dilute antiseptic. An application of topical anti-bacterial solution may be necessary; in severe or chronic cases systemic antibiotics may be indicated (trimethoprim sulfamethoxazole).
- Zoonotic potential- Yes, Consider the human health risks of Staphylococcus infection particularly antibiotic-resistant strains known as MRSA (Methicillin Resistant Staphylococcus Aureus). Always use gloves when managing wounds.

Proud flesh (excessive granulation tissue)- AMODING

- Wounds heal via granulation from the depth and epithelialisation from the edges. Usually these two act in synchrony resulting in a fast healing process.
- Proud flesh occurs when granulation exceeds the epithelialisation process and it occurs as an excessive pink tissue forming a large protruding growth which prevents epithelialisation. Proud flesh is common on the distal limbs of horses and donkeys but can occur anywhere.

Management of excessive granulation tissue

1. Using a scalpel blade, trim the excess granulation tissue to just below the skin surface. Granulation tissue has an excellent

blood supply and one should expect profuse bleeding. Be careful not to trim the newly laid epithelium around the edges of the wound. The aim is to encourage epithelialisation so the wound heals.

2. After trimming apply corticosteroid cream to the centre of the wound only so as to inhibit further granulation, make sure you the cream does not reach the skin edges.
3. Firmly bandage the wound – the pressure will prevent further bleeding.
4. Apply the corticosteroid cream to the granulation tissue every third day for 3–5 applications after debridement. If exuberant granulation tissue reforms, repeat the process. Do not use other chemicals such as potassium permanganate or copper sulphate.
5. Prevention of excessive granulation is possible through early treatment of wounds and cleaning wounds thoroughly to prevent infection. Where possible bandaging severe distal limbs wounds and preventing excessive walking also limits the development of proud flesh.

Figure 165
Proud flesh on the face of a horse and dorsal coronary band

Sarcoids- AMODING

- Proud flesh should be differentiated from Sarcoid.
- Sarcoids are skin tumours and are difficult to treat as they recur after surgical excision.
- Studies have proposed that sarcoids could be due to as a bovine papilloma virus, transmitted by biting flies. The virus infects epithelial cells to induce uncontrolled growth resulting in sarcoids.
- Sarcoids are benign (they do not metastasise) but are locally aggressive and can become secondarily infected.
- Sarcoids have different types - Verrucose Crusty and wart-like should be differentiated from ringworm. Fibroblastic Ulcerated and very vascular and should be differentiated from squamous cell carcinoma. Occult which are small lesions with an area of alopecia and develop a crusted surface and nodular encapsulated discrete masses found within the dermis/epidermis. Mixed sarcoid types also occur.
- Sarcoids can be found on any part of the body; however, they are common around the head (particularly the verrucose type), eyes, groin, ventral midline and axilla.

Diagnosis

- Visual diagnosis is sufficient. A biopsy can trigger further growth and should be avoided when a sarcoid is suspected.

Treatment

- Leave a sarcoid alone if possible. If the lesion is small and interfering with a harness, surgical excision is possible. Lesions frequently recur following surgery, as sarcoids are locally invasive.
- Some studies indicate a protocol of injections with BCG. There has been a varied success rate, and there is a small risk of anaphylactic shock. Sarcoids of the limbs and axilla are more difficult to treat and have a high recurrence rate.

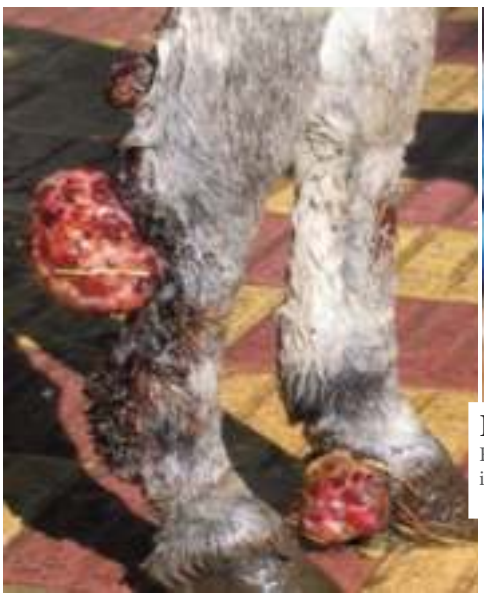


Figure 166
Fibroblastic sarcoid (left) and Nodular sarcoid in donkeys

9.4 Dermatophilosis

- **Dermatophilosis** ('rain scald') is caused by Dermatophilus congolensis (bacteria) and is spread by carrier animals. Pre-disposing factors for infection include moisture and abrasion.

- **Clinical signs**- Appearance of small crusty lumps with hair standing up (paintbrush) with underlying skin moist and inflamed. Lesions are found mainly on the dorsal surfaces of the body, caudal pastern due to hobbles or a muddy environment and the skin under harness such as the flanks as well as on the face of the donkey.

- **Diagnosis**- make Impression smear of the underside of crusts that are stained with Giemsa and look for 'railroad track' cocci (bacteria joined together in a line)

- **Treatment**- Minimise exposure to wet, muddy environments. Rugs and saddle padding should be removed to avoid contact with sweaty skin. Shave or clip the affected area. Apply dilute antiseptic and leave open to the air to dry. Remove loose scabs and crusting which harbour the bacteria. Burn the removed scabs, as bacteria within this material can infect other donkeys and horses. Treat with penicillin or trimethoprim sulfamethoxazole for 7 days. Protect lesions on the lower limb by applying a waterproof barrier cream (petroleum jelly or hydrous wool fat).

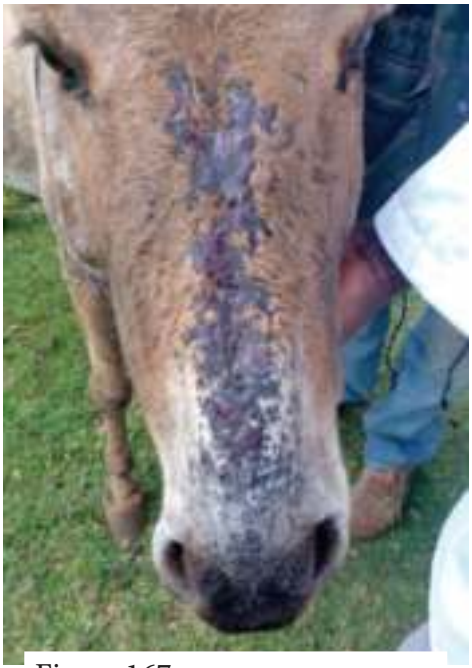


Figure 167
Dermatophilosis on the face of a donkey

Donkey diseases:
Protozoal

9.5 Equine Trypanosomiasis - LONYANG / LOPID

Case definition

- Trypanosomiasis are infectious diseases affecting both humans and animals.
- The causative organism is a protozoa haemoparasites called Trypanosoma that is transmitted by Tsetse fly (Glossina species) or mechanically by hematophagous biting flies of the species Tabanus and Stomoxys.

Aetiology and Epidemiology

- Tsetse flies are distributed in a 'belt' across 37 countries in Africa; trypanosomiasis is endemic in these areas. For T. congolense and T. brucei, development within the tsetse is an essential stage in the life cycle. All domestic animals are at risk of infection in tsetse areas. T. brucei gambiense and rhodesiense cause disease in humans resulting in a fatal condition known as sleeping sickness. Horses cannot be kept in tsetse areas without trypanocidal prophylaxis; however, donkeys have been kept in these areas. Although resilient, donkeys are not resistant to tsetse transmitted trypanosomiasis.
- Human African trypanosomiasis (HAT), also known as sleeping sickness, is an important public health disease caused by Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense. T. brucei gambiense causes the chronic form of HAT in west and central Africa while T. rhodesiense induces an acute form which is found in eastern and southern Africa. The human-infective parasites can be transmitted to livestock and wildlife; the animals also serve as reservoir host for HAT.
- During the last three decades, HAT has been controlled effectively and this has allowed the World Health Organisation (WHO) to include it in the WHO roadmap for eradication, elimination and control of neglected tropical diseases, with a target set to eliminate HAT as a public health problem by 2050. Achieving these goals requires investigation of animal reservoirs especially in equines and especially so in donkeys as they are often an ignored species when formulating control and eradication programmes.
- The disease in animals is known as African Animal Trypanosomiasis (AAT).
- Equine trypanosomiasis caused by species of the genus Trypanosoma is a complex of infectious diseases called dourine, nagana and surra. These diseases are characterized by overlapping clinical features that can be defined by their mode of transmission.
- Nagana is caused by T. vivax, T. congolense and/or T. brucei subspecies and is transmitted by tsetse flies; surra is caused by T. evansi and is mechanically transmitted by biting flies; while dourine is due to T. equiperdum and is sexually transmitted.
- Knowledge of the different transmission modes is important so as to design appropriate control measures. This can only be done if animal health service providers gain a better understanding of the epidemiology of equine trypanosomiasis by identifying trypanosomes that naturally infect horses and donkeys.
- T. equiperdum that is transmitted mechanically when mating is the only trypanosome that is not transmitted by an

invertebrate vector. The pathogenesis of dourine differs from other trypanosomes in that it is primarily a tissue parasite which rarely invades the blood. There is no known natural reservoir of the parasite other than infected equids. Dourine is confined to parts of Africa, Asia, Central and South America.

- A 2020 prevalence study in Chad that collected 286 blood samples from horses and donkeys found a 19% prevalence for Trypanosoma parasite on Rapid Diagnostic Test (RDT), a 13% prevalence on Capillary Tube Centrifugation (CTC) test and 35% prevalence on PCR revealed. Trypanosomes of the subgenus Trypanozoon (T. brucei, T. evansi and T. equiperdum) were the most prevalent (29%), followed by T. congolense forest (12%), T. congolense savannah (5%) and T. vivax (5%). Two donkeys and one horse were found with T. b. gambiense infections that causes HAT.
- In Kenya, tsetse flies are endemic in 38 out of 47 counties. Since 2009, Kenya has not reported a case of HAT apart from two cases detected in 2012 from European tourists returning from the Maasai-Mara National Reserve. The Kenya Tsetse and Trypanosomiasis Eradication Council (KENTTEC), established in 2012 has made significant progress in controlling animal trypanosomiasis (AAT). However, 'nagana' is still a major problem in Kenya. Several trypanosome species such as T. b. brucei, T. congolense, T. vivax, T. simiae, and T. suis still occur in many parts of the country partly due to the extra risk of transmission by other biting flies and the fact that most livestock are kept under the extensive production system that is characterised by highly mobile herds in search of pasture and water.

Clinical signs

Nagana- Tsetse transmitted Trypanosomiasis

- T. brucei clinical signs include anaemia, icterus (jaundice), enlarged lymph nodes and petechial haemorrhages of the mucous membranes (third eye lid and vulva mucosa are good sites to check for petechiation). The animals also have parasitaemia 'waves' of intermittent high fever (41˚C) which occurs as host immunity responds to changes in the proteins on the parasite surface. T. brucei can cause a serious and acute disease in donkeys and horses with mortality occurring within 14–90 days if left untreated.
- T. congolense and T. vivax have a low prevalence and cause milder and more chronic clinical disease when compared to T. brucei. The main clinical signs are anorexia, wasting and generalised oedema around 14 days after infection. T. vivax can also be transmitted mechanically, and therefore can spread beyond the tsetse belt.

Surra- Mechanically transmitted by biting flies Trypanosomiasis

- T. evansi clinical signs include severe weight loss, progressive weakness, anaemia, haemoglobinuria, intermittent fever, petechial haemorrhage of mucous membranes, oedema of limbs, lower abdomen and thorax and severe neurological signs. The mortality rate in untreated horses is almost 100%. The severity of the disease depends on the parasite strain and factors including stress and the health of the equid. Animals subjected to stress, such as malnutrition and physical labour, are more susceptible to the disease.
- Chronic forms persist for several months up to 2 years, providing a reservoir of infection for other animals. There is considerable variation in host species susceptibility; severe rapidly fatal disease is common in horses, whereas donkeys and mules tend to develop chronic mild or subclinical infections.
- There are rare reports of T. evansi infection in humans, these is thought to be rare anomaly

that occurred as the individuals were immuno-compromised.

Dourine- Sexually transmitted Trypanosomiasis

- T. equiperdum clinical signs include- A mucopurulent discharge from the penis or vulva, and oedema around the genital area (is the most common initial presenting sign). Generalised clinical signs include fever, oedema, anaemia and wasting. Approximately a month following inoculation, urticarial reactions erupt all over the body. Progressive paralysis is a possible sequel and these cases are frequently fatal. Abortion is also common.
- The protozoa parasite may persist for years in donkeys and mules without showing clinical signs; mortality can be high in untreated horses.

Diagnosis

- Microscopic evaluation of centrifuged blood (CTC) test.
- Serologic tests (RDT and ELISA) and trypanosome antigen detecting tests (PCR).
- T. equiperdum rarely produces haemoparasites so blood smear detection is not usually effective. Microscopy of direct smears from the fluid of infected genitalia may detect parasites; however, serology is the most reliable test for dourine.

Treatment

- Treatment is not recommended for dourine as it is never 100% effective; recovered animals can still infect others. Castration may be an option in males; however, euthanasia should be discussed with the owner. It is important to reiterate that infected animals should not be used for breeding.
- Quinapyramine salts only for treatment do not use for prevention in endemic areas- The drug is used for T. evansi at a dose rate of 3 to 5 mg/kg of a 5% solution split between three injection sites. Quinapyramine dimethylsulphate and quinapyramine chloride (Triquin) is the most commonly available drug but it is poorly tolerated (particularly in horses). Only administer it deep IM injection using a long narrow-gauge needle, as SC injection results in sloughing of skin and wounds that take months to heal.
- Homidium bromide (250 mg) dissolved in sterile water, prepared as 1–2.5% solution for SC or IM injection at 1 mg/kg. With both prophylactic and curative properties, this is most effective against T. vivax
- Do not use Diminazene aceturate- Just like in camels it causes severe side effects which can be fatal.

Control

- Attempts to develop a vaccine have been futile as the parasite overrides the immune defences by rapidly changing the surface proteins. Each time an antibody response is mounted, the coat changes and the defences become useless.
- Prophylactic treatment is not recommended. Although in the short term this may reduce the number of clinical cases, it has led to drug resistance and is no longer encouraged.
- Vector control through topical application of fly repellents (especially when biting fly population is high during the rainy season) and dung removal are important control strategies. Also avoidance of tsetse infested areas if possible is encouraged. and do not house equids close to stagnant water.
- Treat infections early so as to prevent spread to other animals.

Zoonotic potential- Yes

Notifiable disease- Yes- Dourine is a WOAH listed disease. Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

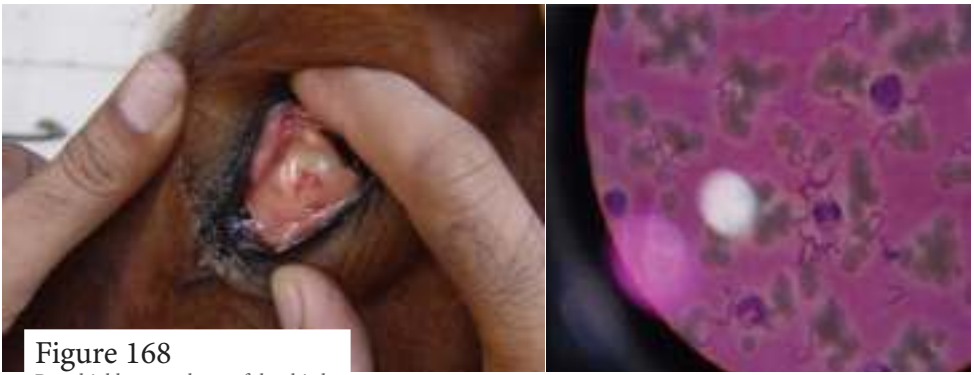


Figure 168
Petechial haemorrhage of the third eyelid in a horse and Trypanosomes in a blood smear

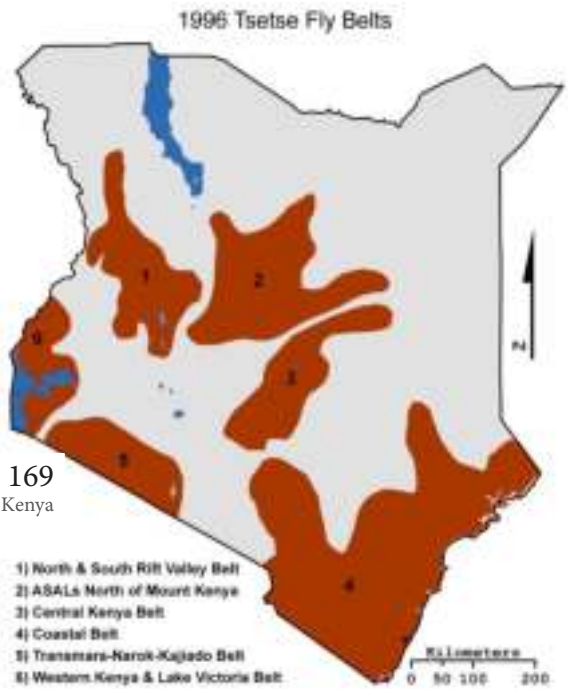


Figure 169
Map of the Tsetse belt in Kenya

9.6 Babesiosis- Equine Piroplasmosis (EP)- LONYANG / LOPID

Case definition and Epidemiology

- Tick-borne infection caused by two haemoprotozoa parasites- Babesia caballi and Theileria equi. This disease affects horses, donkeys and mules
- B. caballi and T. equi are endemic in many tropical and subtropical countries in Mediterranean Europe, Africa, the Middle East, Asia, and Central and South America. Equine piroplasmosis is a notifiable disease in many countries and is reportable to the WOAH by National director of veterinary services.
- Twelve species of ixodid ticks of the genera Dermacentor spp. (central Asia), Hyalomma spp. (Middle East and Africa), and Rhipicephalus spp. (Africa and South America) are vectors for EP.
- T. equi is also iatrogenic transmitted by contaminated needles and syringes.
- Carrier equids remain sources of infection for tick vectors for up to 4 years after infection.

Pathogenesis and Clinical Signs

- The causative agents of EP are intracellular parasites which invade red blood cells inducing haemolysis, and development of haemolytic anaemia.
- The severity of clinical signs relates to the parasite burden, and disease can manifest as acute or chronic, mild to severe. Fatalities may occur in the first 48 hours of infection but chronic disease often develops. B. caballi is clinically milder than T. equi; severe anaemia is very rare with a B. caballi infection. Donkeys tend to develop the chronic form of the disease and the signs are often unspecific.
- Clinical signs of acute EP include - Intermittent fever (> 40˚C), sudden sweating, anaemia, icterus, petechial haemorrhage of third eyelid, rapid heart rate and respiratory rate, Oedema of muzzle, limbs, ventral abdomen and thorax, hind limb weakness, reluctance to move, tremors, haemoglobinuria and dry faeces. Death within a few days (mortality 5–10% in endemic areas and up to 50% in naïve horses)
- Clinical signs of chronic EP - Inappetance, weight loss and poor performance.

Diagnosis

- The number of parasites in the blood varies throughout the course of infection. In acute cases with clinical signs the haemoparasites are readily visible on a blood film, appearing as dark dots, rings or pear-shaped marks within red blood cells. It may be difficult to detect parasites in animals in the latent or chronic stages of disease, particularly with B. caballi. Laboratory techniques include PCR and antibody ELISA can be used for chronic cases.

Treatment

- Drugs used to treat EP are toxic, so administer treatment only to animals with severe clinical signs or if parasites are present in > 50% of RBCs.
- Imidocarb dipropionate (Imizol)- efficacy of this medication is variable and toxic side effects are common. Administration can cause severe colic and diarrhoea. Administer 2.4 mg/kg as a single deep IM injection; donkeys are more susceptible to toxic side effects so use a low dose (1–2 mg/kg). A treatment protocol of 4 injections at 4 mg/kg at 72-hour intervals has been recommended for

complete elimination of T. equi in endemic areas complete parasite elimination will reduce endemic stability which is not advisable so only treat to alleviate acute severe symptoms.

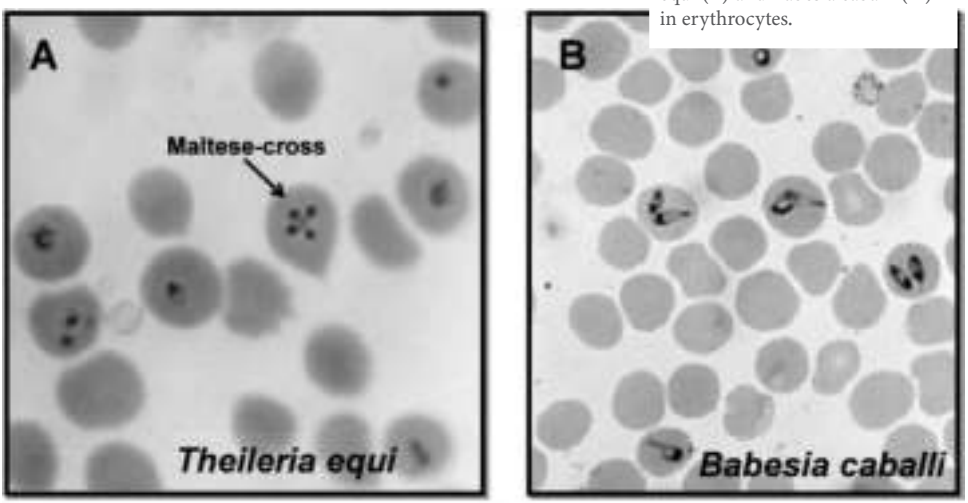
- Diminazene aceturate- avoid this drug in equids unless other drugs are unavailable, as it has a low therapeutic index and toxic side effects are common. Administration of 3.5 mg/kg IM reduces the clinical signs within 24 hours. (In areas of resistance, horses require even higher doses; 2 doses 24 hours apart at 5 mg/kg for B. caballi, 6–12 mg/kg for T. equi.) However, this drug is associated with marked side effects even at the lower dose rate. At higher doses, the risk and severity of such side effects would be increased and is not recommended. Toxicity can be treated with calcium salts.

Endemic stability- 'Infected but not affected'- this is an epidemiological state in which severe clinical disease is scarce despite high levels of infection in the population. In endemic areas where equids are faced with a low level of continuous challenge, immunity develops and reduces the severity of disease. Disruption of this low-level challenge, through control of ticks or constant use of treatment drugs might result in an increase in clinical disease incidence. Under conditions of epidemiological stability, when hosts are under frequent exposure to EP, foals are protected passively by maternal antibodies acquired via colostrum. This protection can last for up to 9 months. Infections of foals in this period will induce immunity without any overt signs of the disease.

Zoonotic potential- No

Notifiable disease- Yes- WOAH listed disease. Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

Figure 170
Blood smear showing Theileria equi (A) and Babesia caballi (B) in erythrocytes.



Picture credit: Kumar, Sanjay & Kumar, Rajender & Sugimoto, Chihiro. (2009). A Perspective on Theileria Equi Infections in Donkeys. The Japanese journal of veterinary research. 56. 171-80. https://www.researchgate.net/deref/http%3A%2F%2Fwww.ncbi.nlm.nih.gov%2Fpubmed%2F19358444

Donkey Diseases:
Parasitic

9.7 Mange- LOTOME / LOGIRGIRA

Aetiology and Clinical Signs

- Mange is caused by microscopic parasites called mites that burrow into the skin causing skin irritation that manifests as continuous licking or scratching. Mange can be contributing cause of pastern dermatitis.
- The most common mites found infesting equids are Chorioptic sp. Other genera of mange mites, such as Sarcoptes sp., Psoroptes sp., and Demodectic sp., are rarely diagnosed due to their susceptibility to macrocyclic lactone dewormers (ivermectin and moxidectin) which are mostly used in equine husbandry.
- Sarcoptes scabiei var equi is rare but is the most severe type of mange. The first sign of infestation is intense pruritus due to hypersensitivity to mite products. Early lesions appear on the head, neck, and shoulders. Regions protected by long hair and lower parts of the extremities are usually not involved. Lesions start as small papules and vesicles that later develop into crusts. The skin becomes lichenified, forming folds. If infestations are not treated, lesions may extend over the whole body, leading to emaciation, general weakness, and anorexia.
- Chorioptic mange is caused by infestation with Chorioptes bovis (formerly C. equi) and is the most common form of mange. Lesions caused by C bovis start as a pruritic dermatitis affecting the distal limbs around the foot and fetlock. Papules are seen first, followed by alopecia, crusting, and thickening of the skin. A moist dermatitis of the fetlock develops in chronic cases. Infested animals may stamp their feet or rub one foot against the opposite leg or object. Chorioptic mange is a differential diagnosis for "greasy heel" in working equids. The signs subside in hot weather but recur with the return of cold weather.
- Demodectic mange is caused by infestation with Demodex equi or D.caballi. Demodex mites infest hair follicles and sebaceous glands. D equi lives on the body, and D caballi on the eyelids and muzzle. Demodectic mange is rare but can manifest as patchy alopecia and scaling or as nodules. Pruritus is absent; therefore, secondary infections due to excoriation are rare. Therapy is rarely done, although there is limited evidence that the macrocyclic lactones may be effective. Lesions have also been reported to resolve without treatment.

Diagnosis

- Mites burrow in the skin and therefore are not visible to the naked eye.
- Skin scraping of affected area or margin of affected area should be done.
- See Chapter 1 for more details on mite diagnosis.

Treatment

- Hot lime sulphur spray or dip is labelled for use against sarcoptic, psoroptic, and chorioptic mites in equids. Treatment should be repeated every 12 days if needed, following the species-specific dilution on the label.
- Although not labelled for treatment of mange, two doses of oral ivermectin at 200 mcg/kg given 14 days apart (field studies), or a single treatment of oral moxidectin at 400 mcg/kg can be used.

Control

- Affected animals should be separated from those that appear to have no active signs of the disease.
- Insecticide application (pyrethrins, carbamates) may be of value in reducing re-infestation of recently treated animals, and the spread of the condition.
- Clipping of excess hair in affected regions, such as the pasterns, may aid in the management of mites.
- Some animals with few or no clinical signs may harbour chorioptic mites, and serve as carrier animals.

9.8 Dermatophytosis ('ringworm')

- Common in young or immune-compromised animals.
- Lesions are caused by fungal species that utilise keratin, Trichophyton and Microsporum.
- The incubation period is 1–4 weeks and the infection spreads slowly amongst a group of equids.
- Ringworm is a zoonotic skin condition and one should wear gloves when treating affected animals.
- Clinical signs - Crusting and scaling of skin that has multifocal, sharply demarcated areas of hair loss, classically a coin-shaped appearance. Initial lesions appearing as raised, swollen lumps (urticaria) with variable pruritus
- Treatment - Generally equids recover over the course of several months. Affected equids are a source of infection to other animals and humans throughout recovery. If severe, topical antifungals are indicated. Follow the instructions as these medications can be toxic. Wash daily with dilute iodine (10% solution). Systemic antifungals (griseofulvin) require a prolonged treatment period for efficacy, and compliance can be problematic. Griseofulvin should not be used in pregnant mares as the medication is teratogenic. Isolate the affected animal and monitor animals in contact for signs of similar skin lesions.

Zoonotic potential- Yes for Sarcoptic mange – always use gloves

Notifiable disease- No

Ethnoveterinary practice- Refer to table 31.

Figure 171
Mange skin lesions in donkeys

Picture source: https://twitter.com/4helpinganimals/status/1242755217952624641

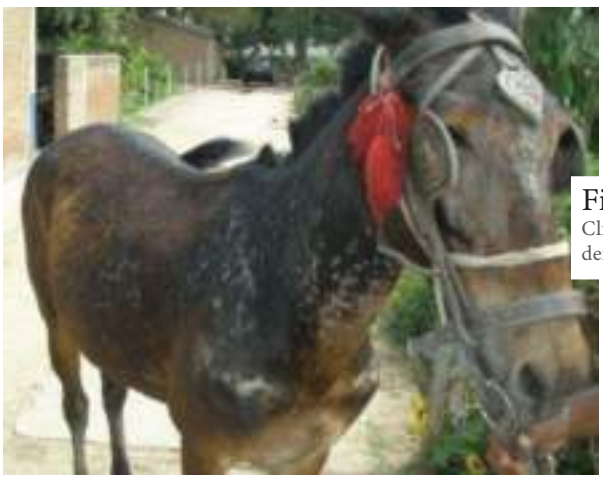


Figure 172
Clinical appearance of dermatophytosis in a horse

9.9 Helminthiasis- NG'IPEELEI / NGIRITAN / NGI'LOMUN

Type of parasite	Predilection Sites
Large strongyles (Strongylus spp.particularly vulgaris)	Adults Large Intestines Larvae Intestinal Wall
Small strongyles (cyathostomes)	Adults Small Intestine Larvae Liver And Lungs
Roundworms (parascaris equorum)	Adults Large Intestine Larvae Intestinal Wall
Pinworms (Oxyuris equi)	Adults Large Intestines Larvae Intestinal Wall
Tapeworms (Anaplocephala spp.)	Adults Large And Small Intestine Larvae Forage Mites
Thread worms (stronglylooides westeri)	Adults Small Intestine Larvae Lungs And Other Tissues
Liver fluke (rarely causes diseas in equids) Fasciola hepatica - temperate Fasciola gigantica - tropical)	Adults Liver Larvae Snail

Aetiology

- Common helminths of donkeys and predilection sites.

Clinical signs

- Clinical signs are varied, including poor coat health, anorexia, weight loss, diarrhoea, colic, rectal prolapse, lack of vigour, poor work performance, and productive loss.
- Large strongyle- S.vulgaris larval stages migrate through the cranial mesenteric arteries and adjacent branches. Migration induces thrombus formation and thickening of arterial walls. Blockage of the blood supply to the intestine results in infarction and severe colic. If the cranial mesenteric artery is completely occluded the intestines become necrotic; this condition is fatal. S. edentus. larvae causes liver damage, and large numbers of adults in the intestine result in unthriftiness, weight loss and anaemia.
- Small strongyle- Cyathostome infection are more moderate than that of large strongyles but include loss of condition, peripheral oedema, malaise, weight loss, poor appetite, lethargy and disrupted intestinal motility. Mass emergence of larvae results in a marked

pathology, 'verminous enteritis', with profuse diarrhoea, colic, weight loss, dehydration, inappetance, dullness depression and protein-losing enteropathy. Mortality rates can be as high as 40–70%.

- Roundworms - Parascaris equorum is one of the largest endoparasites in livestock. Clinical signs include - Mild coughing and nasal discharge in the migratory phase. Affected equids remain bright and alert. Light intestinal infections are tolerated well with no clinical signs. Heavy infections result in unthriftiness, a dull coat, poor growth in young stock, and lethargy. Younger animals can show signs of colic if a heavy roundworm burden causes a blockage of those parts of the digestive tract with a particularly narrow lumen. Clinical signs mainly in foals as adults generally develop resistance.
- Pinworm- Oxyuris equi are a common parasite in equids but are often non-pathogenic. Adult worms live in the lumen of the colon. After fertilisation the female migrates to the anus and deposits eggs in yellowish white streaks on the perineal skin. Pinworms rarely cause gastro intestinal signs other than Intense pruritus around the anus that can lead to self-trauma.
- Tapeworms (Anoplocephala spp.)- cause mild infestations are considered non-pathogenic; however, moderate to severe tapeworm burdens have been associated with spasmodic colic, intussusception and intestinal rupture.
- Liver fluke infestation is low in equines.
- Stomach bot - Gasterophilus spp. the larvae of the bot fly. Presence of larvae in the buccal cavity may cause inflammation, although this is rare. Attachment. to the rectal mucosa has been associated with rectal prolapse

Diagnosis

- The McMaster technique is a widely accepted protocol for the detection of nematode eggs in equine faeces. Refer to chapter 1 for more details.

Treatment

- Ivermectin - Used in treatment of all major helminth parasites at a dosage rate of 0.2 mg/kg PO
- Benzimidazoles like fenbendazole are used for all helminth but action is slow - at a dosage of 7.5 mg/kg PO
- Pyrantel emboate – Indicated for treatment of large and small strongyle, Oxyuris, Parascaris equi and Anoplocephala spp. At a dose rate of 19 mg/kg PO.

Control

- Grazing management practices can be combined with drug intervention to minimise the spread of helminths between animals. Rest heavily grazed ground, and limit density of numbers to reduce pasture contamination.

9.10 Rectal prolapse

Is defined as protrusion of one or more layers of the rectum through the anus. causes of rectal prolapse include heavy worm infestation, malnutrition, feeding of dry feed (bran) without adequate water, overloading or overworking. Treatment is aimed to reduce the prolapsed mass as well as correcting the spasmodic colic and tenesmus. To reduce intestinal motility, administer hyosine at the dose rate of 0.5 mg/kg body weight intravenously. Administer flunixine meglumine to reduce the colic at the dose rate of 1.1 mg/kg body weight intravenously. Ensure the donkey is properly restrained by administering low dose of xylazine hydrochloride at the dose rate 0.5 mg/kg intravenously for sedation and 5 ml of 2% lignocaine hydrochloride solution in the first intercoccygeal space. Confirm the effect of analgesia on the perineum by pricking the area with a sterile needle. Then proceed to manually reduce the prolapsed mass by applying sugar granules to reduce oedema and apply liberal lubrication like glycerine. Reinforce with a purse-string suture if there is a tendency for recurrence.

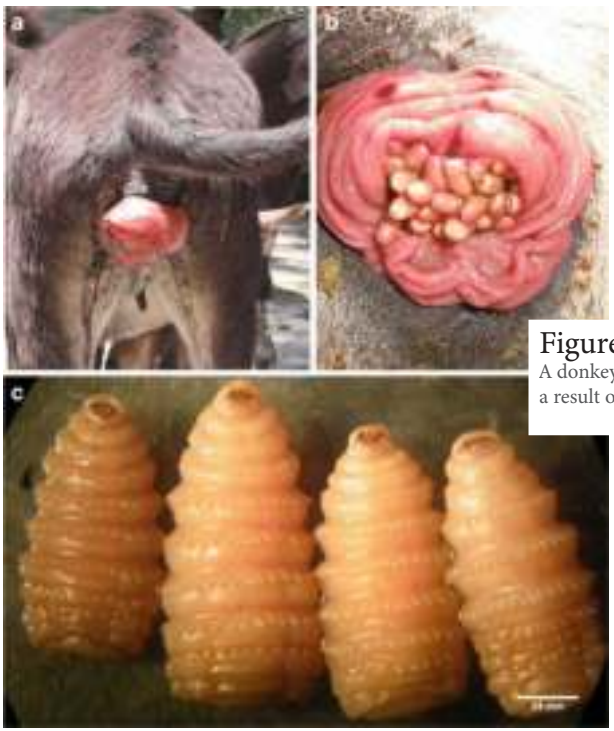


Figure 173
A donkey with rectal prolapse as a result of helminth infestation

Donkey diseases:
Viral

9.11 African Horse Sickness

Aetiology and Epidemiology
- Causative agent is an Orbivirus of which there are nine serotypes.
- Virus is spread by arthropod vectors, usually a Culicoides midge. It is important to realise that African Horse Sickness (AHS) is not contagious between individual equids but will spread in a population from the biting vectors, hence the importance of vector control.
- AHS is a seasonal disease endemic to Sub-Saharan Africa and parts of Southern Africa and mainly occurs during the warm rainy season when Culicoides midges' population is high.

Clinical signs
- All equids of all ages are affected (mortality rate 70–90%).
- The disease has several manifestations disease depending on which form is present.
- Acute lung form occurs in horses– it has a short incubation period of 3–5 days, high fever, laboured breathing, coughing and profuse frothy nasal discharge. Mortality rate is high. Up to 95% of horses die within a week, often from 'drowning' in their own pulmonary secretions.
- Cardiac form or subacute occurs in donkeys- Has an incubation period of 7–14 days, Fever followed by swelling over the head, eyelids, lips, cheeks and under the jaw, Conjunctival swelling which can be significant. Mortality rate is about 60%, death results from heart failure.
- Mixed form a combination of the above two types- The incubation period is between 5–7 days and is indicated initially by mild respiratory signs which progress to the typical swellings of the cardiac form.
- Mild or Horse Sickness Fever form- Seen in zebras and African donkeys, this form occurs with mucosal congestion and conjunctivitis. Animals usually recover.

Diagnosis
- A presumptive diagnosis can be made based on clinical signs or post-mortem lesions in areas with a known vector presence.
- Laboratory confirmation through ELISAs or PCR.

Treatment
- Viral infection- there is no treatment give supportive care
- Extreme acute form may have to be euthanised.

Prevention
- AHS is notifiable in many countries; report suspected cases to the relevant governing bodies. AHS is a WOAH listed disease.
- Live attenuated vaccines are available commercially in South Africa; these vaccines are polyvalent but may not contain the serotype causing a local outbreak. It is essential that the vaccines are administered annually.
- Vector control using repellents or other locally available substances that are not harmful to equids.

Figure 174
African Horse Sickness in a donkey in Kenya

Donkey Diseases:
Undetermined Causes

9.12 Colic

- Colic' is a general term meaning abdominal pain. There are a large number of known causes of colic in equids, so a thorough work-up is necessary. Colic is a symptom, not a diagnosis.
- Most, 80% of colic cases resolve spontaneously or with simple medical treatment, with a further 10% requiring intensive medical treatment.
- Inflammation of the stomach or intestinal walls This could be due to parasitic worm damage, sand ingestion, bacterial infection or ingested foreign bodies (e.g. plastic bags). It can lead to altered motility and diarrhoea.
- Stretching of the intestinal wall This is due to distension with gas (tympany) or food material (grain overload, or colonic impaction).
- Altered intestinal motility Peristaltic movements may be abnormally increased (spasm), decreased or absent (ileus). Changes in motility may be caused by dehydration, dietary changes, parasitic worms, toxins, or obstruction from inappropriate ingested materials. They often lead either to diarrhoea or to constipation.
- Loss of blood supply to an area of intestine (ischemia) - caused by intestinal torsion, strangulation, intussusception or total blockage. It leads to the release of toxins into the blood, inflammation and severe pain followed by necrosis and death of the affected section of intestine.
- Unlike in horses that have dramatic colic signs. Donkeys show subtler pain symptoms which may be overlooked. They must be examined thoroughly in order to assess the severity of colic. Dullness and depression are signs of colic as well as subtle changes in behaviour.

Approach to a colic case

Take a detailed history of the case by asking the following questions

1. When did the colic signs start?
2. Has the animal had colic before? If so, when was the last time?
3. When did it last pass faeces? How much? What consistency?
4. Does it have diarrhoea?
5. When was the last de-worming treatment?
6. When did the animal last eat? What does it eat? Has the diet changed recently?
7. When did it last drink and how much?
8. When did it last pass urine?
9. Is the animal pregnant?
10. History of teeth problems?

Diagnosis

- Nasogastric intubation- one must be trained
- Rectal examination- refer to the reference section of this chapter to refer to the book - The Working Equid Veterinary Manual for a detailed guide on rectal examination in equine
- Faecal sampling- to rule out helminthiasis

Management of a colic case

- Analgesics to control the pain
- If gastric reflux is not present, and there is evidence of gut sounds, give oral fluids by nasogastric tube. 3–6 litres can be given, depending on the size of the animal, and electrolytes can be added. This can be

repeated every 2–4 hours; if the impaction is large this may be necessary. Because foreign body impactions are difficult to distinguish from food impactions, laxatives such as mineral oil are often used. However, frequent administration of fluids per nasogastric tube has been shown to be as effective, if not more so. Refer to Section 6.2 on fluid therapy for information on how to make an isotonic solution suitable for nasogastric intubation.

- Food should be withheld and the treatment repeated until the animal starts to pass faeces again.
- Recent research on treatment of large colon impactions shows that oral fluid hourly is the most effective way to treat an impaction.

Figure 175
Colic signs in the donkey may appear more subtle than in the horse, including sitting down and stretching out.

Figure 176
Colic sign in a horse: Flank-watching

Faecal impaction-Impaction colic-KORIANG

- One of the most commonly reported causes is due to impacted ingesta in the large intestine ("impaction colic").
- Most impactions occur at the pelvic flexure of the colon.
- Predisposing factors – feeding on inappropriate vegetation, Turkana county the livestock keepers indicated that faecal impaction occurred when donkeys fed on Prosopis

Julliflora. The other main causative agent is dental disease- missing teeth or dental abnormality (shear, wave, step, undershot, overshot or diastema). Older donkeys are also more susceptible.

- Most donkeys that get impaction do not survive as it may require early medical or surgical intervention.

9.13 Laminitis – EKAMUCHURUT

- Laminitis is inflammation of the 'laminae', the sensitive soft tissue structure inside the hoof that joins the pedal bone to the hoof wall. As a result, the pedal bone loses its support and becomes unstable.
- The pedal bone then rotates or move downwards within the hoof, causing pain and irreversible damage.
- Laminitis is an very painful condition that is seldom reversible and can be fatal.

Figure 177
X-ray of a normal donkey foot (left) vs one with laminitis (right)

Note how the pedal bone has lost its parallel relationship with the front part of the hoof wall.

Picture source: https://www.thedonkeysanctuary.org.uk/what-we-do/knowledge-and-advice/for-owners/laminitis-indonkeys#:~:text=Laminitis%20is%20inflammation%20of%20the,causing%20pain%20and%20irreversible%20damage.

- Potential causes of laminitis are: a) An infection in any part of the body, e.g., uterus or chest infections; b) Excess weight being placed on one foot because the donkey has pain in the opposite foot; c) Access to too much grass; d) Access to cereal or other sugar rich feed; e) Hormonal imbalances/disturbances and Equine Metabolic Syndrome (similar to Type 2 Diabetes in humans).
- Donkeys do not behave in the same way as horses when they have severe foot pain. A donkey will tend to lie down more than usual or adopt subtle weight shifting as it stands.
- Evidence of bouts of laminitis is revealed by 'laminitic rings' on the hoof wall. These can be distinguished from other 'event rings' (e.g. sudden diet change) because they are not parallel with each other but diverge towards the heels.
- Main treatment is to give analgesics like phenyl butazone (avoid steroids as they worsen the laminitis). Hoof trimming may also be useful. Refer to the reference section of this chapter to refer to the book - The Working Equid Veterinary Manual for a detailed guide on rectal examination in equine for more detailed information on laminitis.
- Severe long standing cases cannot be treated and donkey should be euthanised.

Figure 178
Rings of abnormal hoof tissue which diverge towards the heels.

Picture source: https://www.littlelongears.org/wp-content/uploads/2017/04/Laminitis-In-Donkeys-from-The-Donkey-Sanctuary.pdf

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Annex:

1: Livestock Vaccination Schedule for Turkana County

Livestock Disease	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sept	Oct	Nov	Dec	Notes
Peste des Petits Ruminants (PPR)													PESTEVAX™ vaccine (KEVEVAPI) – for goats 4 months & older.
Sheep and goat pox (SGP)													S & G VAX™ -(KEVEVAPI) sheep/goats over 5 weeks of age; unless in outbreak up to 3 weeks old. Annual vaccination recommended.
Contagious Caprine Pleuropneumonia (CCPP)- Goats only													CAPRIVAX™ (KEVEVAPI) Goats over 3 months old, repeat every 6 months
Contagious Bovine Pleuropneumonia (CBPP)													CONTAVAX ™- (KEVEVAPI) Annual booster recommended
Lumpy Skin Disease (LSD)													LUMPIVAX™ (KEV-EVAPI)- Annual booster recommended
Foot and Mouth Disease (FMD)													FOTIVAX TM- (KEVEVAPI)- Twice a year or for the fortified oil base once - annual booster recom-mended
New Castle Disease (ND)													Vaccinate at day 4, repeat week 4, week 16, then every 4 months Avivax I-2™ thermotolerant (KEVEVAPI)
Rabies (Include dogs and cats)													There are many brands in the market. Once a year. Post exposure regime Day 0,3,7,14,and 28

- Cattle
- Goats
- Sheep
- Chicken
- Donkeys

Guiding notes

1. Follow manufacturer's instructions for dosage and acceptable vaccine combinations.
2. Camel vaccines are not readily available in the Kenyan Market.
3. Based on CDVS reports the county had once imported HS camel vaccine from the Middle East. The vaccine reduced HS incidences for 3 years. The county should consider sourcing for HS vaccines for Camel and Cattle.
4. Kenchic has introduced TRANSMUNE vaccine against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases in day-old chicks at the hatchery. The farmer will vaccinate only once for ND in the Broiler farm at day 14 instead of 2 vaccinations done previously.

Annex:

2: Zoonotic diseases

- A zoonosis (zoonotic disease or zoonoses -plural) is an infectious disease that is transmitted between animals to humans (or from humans to animals).
- About 60% of human infections have an animal origin and 75% of all new and emerging human infections have zoonotic origin. In addition, 80% of pathogens infecting animals are "multi-host," meaning that they move among different specie hosts including humans.
- Zoonotic diseases emergence is not only about the relationship between domestic animals or wildlife and people, but it is also about the complexity of the system as a whole and the interactions between biotic and abiotic components. Biodiversity is a key factor that supports healthy ecosystems thus reduces zoonoses risk.
- All zoonotic diseases mentioned below are notifiable and should be reported to the county and national director of veterinary services.
- Management of zoonotic diseases require a One Health approach this means there is need for a multi-disciplinary team that investigates the links between the environment (weather, climate, soil, vectors and vegetation factors), susceptible animal and human host and behaviour in at-risk populations. The one health approach improves information sharing and leverages on discipline expertise that translates to early detection, early response thus preventing large scale outbreaks.

ANTHRAX- ENOMOKERE

Other names: Splenic fever, Siberian ulcer, Charbon, Milzbrand and Wool sorters disease

Causative agent:

- Gram positive, Spore-forming bacteria called Bacillus anthracis that exists in two forms vegetative form when in the ruminant body and spore form in the environment.
- The bacteria produce extremely potent toxins, which are responsible for illness.
- Anthrax spores are resistant and can survive in the environment for decades, making the control or eradication of the disease difficult.

Transmission and Predisposing factors

- Soil-borne transmission
- Anthrax does not spread from animal to animal or from person to person.
- Bacteria produce spores on contact with oxygen. These spores are extremely resistant and survive for years in soil, or on wool or hair of infected animals.

Main route of transmission

- Ingestion or inhalation of spores or through entry of spores through broken skin especially in humans.

Occurrence

- Anthrax is mainly a disease of ruminants and is found all over the world on all continents except Antarctica. Each country has endemic areas with frequent outbreaks. Outbreaks are sporadic in response to unusual weather patterns which can cause spores that were dormant in the soil to come to the surface where they are ingested by domestic and wild ruminants that germinate and cause disease.

Main clinical signs

Clinical signs are rarely seen;

- Ruminant animals often found dead with no indication that they had been ill.
- Blood that does not clot is seen exuding from body openings.
- The carcass undergoes rapid decomposition and will not have rigor mortis (stiffen after death).
- In ruminants there may be digestive upsets, fever, depression and sometimes swellings of the body. These symptoms last four days before death.
- In carnivores, if animal feeds on infected ruminant carcass there may be an intestinal form of the disease with presents with fever and cramps from which animals sometimes recover.

Humans

More than 95% of human anthrax cases take the cutaneous form and result from handling infected carcasses or hides, hair, meat or bones.

- In humans, anthrax manifests in three distinct patterns (cutaneous, gastrointestinal and pulmonary). The cutaneous form is the most common (95%) form and is characterised by a boil-like skin lesion that eventually forms an ulcer with a black center (eschar).
- The most susceptible group that contracts anthrax are veterinarians, agricultural workers, livestock producers or butchers.
- The spores enter the body through cuts or scratches in the skin and cause a local infection

that if not controlled may spread throughout the body. The digestive form occurs when the spores are eaten. Tragically people who lose their animals may also lose their lives when they consume meat from an animal that has died of anthrax.
- The deadliest form of the diseases is the pulmonary form also known as 'wool sorters disease'. The pulmonary form is rare under natural circumstances. However, it is the form that occurs when anthrax spores are laboratory engineered to act as biological weapon.

Differential Diagnosis
- Black quarter (Clostridium chauvoei)
- Peracute bovine Babesiosis
- Chemical poisoning (heavy metals, other poisons)
- Plant poisoning
- Snake bite,
- lightning strike
- Metabolic disorders such as lactic acidosis, magnesium deficiency and bloat.

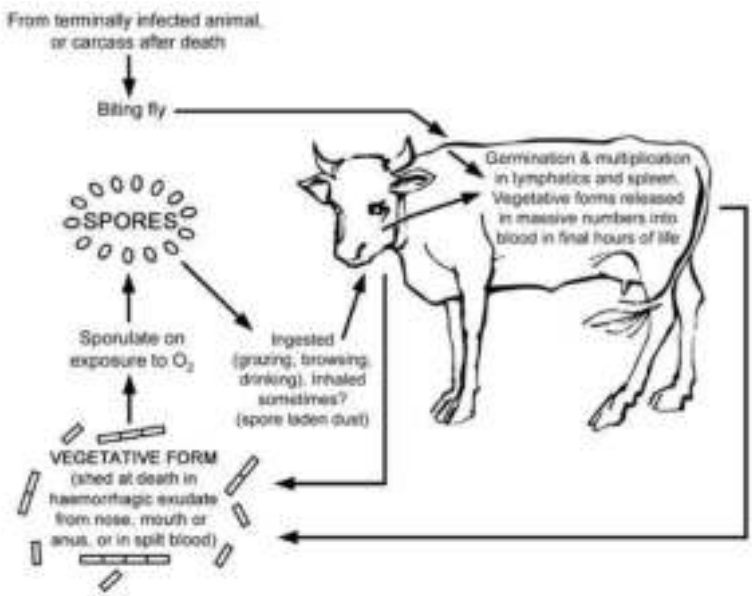
Post mortem lesions
- Do Not Open Carcass with dark blood that does not clot oozing from all orifices and with rigor mortis absent or incomplete, has marked bloating and rapid decomposition.
- If the carcass is inadvertently opened, septicaemic lesions are common and include blood that is dark and thickened and fails to clot readily. Haemorrhages of various sizes are common on the serosal surfaces of the abdomen and thorax as well as on the epicardium and endocardium. Oedematous, red-tinged effusions commonly are present under the serosa of various organs, between skeletal muscle groups, and in the subcutis.
- The most characteristic necropsy finding is a markedly enlarged black to dark red spleen

Diagnosis
- Clinical signs of sudden death with blood oozing from all orifices, rapid decomposition of carcass that lacks rigor mortis development.

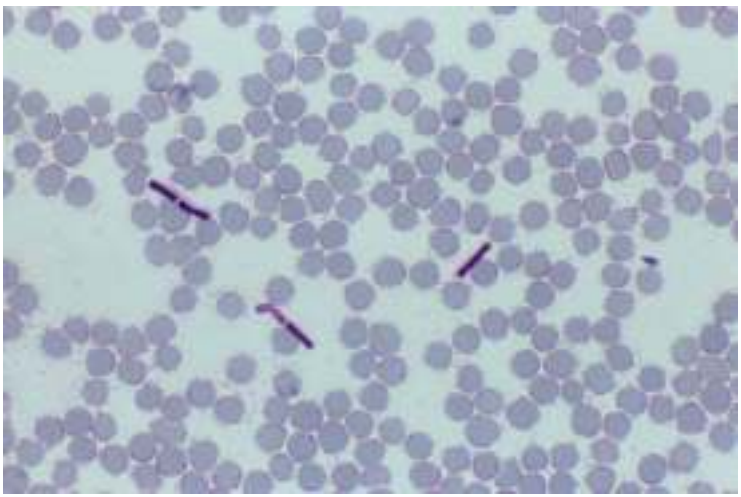
Sample Collection
- Anthrax is diagnosed by examining blood smears for the presence of the bacteria. Samples must be collected carefully to avoid contamination of the environment and to prevent human exposure to the bacteria.
- Giemsa stained blood smears made from a cut surface of the base of the ear of a fresh carcasses contains large numbers of B. anthracis vegetative forms which can be seen under a microscope.
- Cultured and isolation of bacteria is prohibited.
- Blood smear is the main confirmatory diagnostic test for laboratories.

Treatment and Control
- Anthrax is susceptible to most antibiotics especially penicillin group. Prompt treatment of animals at the earliest signs can be effective. Treatment with antibiotics counteracts the vaccine though, so antibiotics should not be given to animals that are recently vaccinated.
- Vaccination is the main control strategy and is done once a year. The vaccine Blanthrax™ that combines anthrax and black quarter is the most common in Kenya and is available from KEVEVAPI.
- Proper disposal of dead animals is critical-carcass should not be opened, since exposure to oxygen will allow the vegetative form of the bacteria to form spores, infected areas should be quarantined until all susceptible animals are vaccinated and all carcasses disposed of preferably by incineration or alternatively, by deep burying with quick lime poured over the carcass.

Anthrax Transmission cycle in cattle



Picture source: https://infonet-biovision.org/AnimalHealth/Diseases-Killing-very-fast-Killer-diseases



Blood smear stained with Giemsa showing characteristic Bacillus anthracis bacteria

Picture source: https://repository.up.ac.za/handle/2263/32656

RABIES – LONG'OKOU NG'IKEREP

Other names: Mad dog disease, Hydrophobia

Definition:

- Rabies is an acute, progressive viral encephalomyelitis that principally affects carnivores and bats, although any mammal can be affected. The disease is fatal once clinical signs appear.

Causative agent

- Virus- RNA virus of the genus Lyssavirus in the family Rhabdoviridae
- Lyssaviruses are highly neurotropic and have a long incubation period
- There are over 16 different lyssaviruses
- Rabies virus can be transmitted between mammals, whether they belong to the same or different species

Transmission and Predisposing factors

- All mammals are thought to be susceptible to the rabies virus the disease is a fatal zoonosis that causes encephalitis. It is 100% fatal.
- Following the bite of an infected animal, the virus is placed under the skin surface. Here the virus multiplies for a period of days or weeks. It then travels along the peripheral nerves and via the axons to the dorsal root ganglia. Here further multiplication of the virus occurs. The virus passes to the central nervous system and is capable of passing from cell to cell. The final path of its journey takes it back along axons to the salivary glands and many other deep organs like the heart, skeletal tissue, kidney and cornea.
- The virus is then transmissible through saliva after 3 days to up to years. Most commonly between 1 to 3 months. The incubation period is directly related to the extent of the inoculation and the closeness of the brain. Thus bites on the face and neck are particularly significant and will, in general, be associated with a short incubation period. The incubation period is both prolonged and variable. Typically, rabies virus remains at the inoculation site for a considerable time. The unusual length of the incubation period helps to explain the effective action of local infiltration of rabies immune globulin during human post exposure prophylaxis, even days after exposure. Most rabies cases in dogs develop within 21–80 days after exposure.
- There reports of infection in animals (carnivores) due to Ingestion of infected meat but this has not been reported in humans.
- Human to human spread is possible in theory but has not been reported to date. All ages are at equal risk.
- Transmission is mainly through the saliva of an infected animal. Saliva becomes infectious a few days prior to the onset of clinical signs. Infection occurs primarily via bite wounds, or infected saliva entering an open cut or wound or mucous membrane, such as those in the mouth, nasal cavity or eyes.
- Occasional, though rare, transmission by inhalation of infected aerosol has been described. This is common when humans enter caves or houses with bat faeces (Guano). There are reports of laboratory workers getting infected by brain tissue (hence virus isolation or brain tissue handling should not be attempted unless in a Biosecurity level 3 or 4 laboratories.

Occurrence

- Rabies is found throughout the world, with the exception of Antarctica
- Rabies is a zoonotic disease that can affect all mammals. Carnivores circulate different rabies virus (RABV) variants and act as a reservoir for rabies.
- Rabies infection is maintained in two epidemiological cycles, one urban and one

sylvatic. In the urban rabies cycle, dogs are the main reservoir host. This cycle predominates in areas of Africa, Asia, and Central and South America. Canine-mediated rabies is one of the most important zoonosis and is estimated to cause up to 70,000 human deaths per year mostly affecting people in rural areas and especially children.

- The sylvatic (or wildlife) cycle is the predominant cycle in the northern hemisphere.

Main clinical signs

- On set of clinical signs vary with the distance of infecting bite to the nervous tissue it may take a few days to more than a year.
- Clinical course may be divided into three phases—prodromal, furious and paralytic. However, this division is of limited practical value because of the variability of signs and the irregular lengths of the phases. During the prodromal period, which lasts approximately 1 to 3 days, animals show only vague nonspecific signs, which intensify rapidly. The disease progresses rapidly after the onset of paralysis, and death is virtually certain a few days thereafter. Some animals die rapidly without marked clinical signs.
- Furious Rabies: This is the most common form (80%).In this form animals are aggressive is pronounced. This is the classic "mad-dog syndrome," although it may be seen in all species. There is rarely evidence of paralysis during this stage. The animal becomes irritable and, with the slightest provocation, viciously and aggressively uses its teeth, claws, horns, or hooves. The posture and expression is one of alertness and anxiety, with pupils dilated. Noise may invite attack. Such animals lose caution and fear of people and other animals.
- Carnivores with the furious form of rabies frequently roam extensively, attacking other animals, including people, and any moving object. They commonly swallow foreign objects. As the disease progresses, muscular incoordination and seizures are common. Death results from progressive paralysis.
- Paralytic Rabies: manifest by ataxia and paralysis of the throat and masseter muscles, often with profuse salivation and the inability to swallow. Dropping of the lower jaw is common in dogs. Owners frequently examine the mouth of dogs and livestock searching for a foreign body or administer medication with their bare hands, thereby exposing themselves to rabies. These animals may not be vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, and coma and death follow in a few hours.
- Species Variations- Cattle with furious rabies can be dangerous, attacking and pursuing people and other animals. Lactation ceases abruptly in dairy cattle. The usual placid expression is replaced by one of alertness. The eyes and ears follow sounds and movement. A common clinical sign is a characteristic abnormal bellowing (sounds like the animal has a hoarse voice), which may continue intermittently until shortly before death. Horses and donkey's mules frequently show evidence of distress and extreme agitation. These signs, especially when accompanied by rolling, may be interpreted as evidence of colic. As in other species, horses may bite or strike viciously and, because of their size and strength, become unmanageable. Rabies should be suspected in terrestrial wildlife acting abnormally. The same is true of bats that can be seen flying in the daytime, resting on the ground, paralyzed and unable to fly, attacking people or other animals, or fighting.

Differential Diagnosis

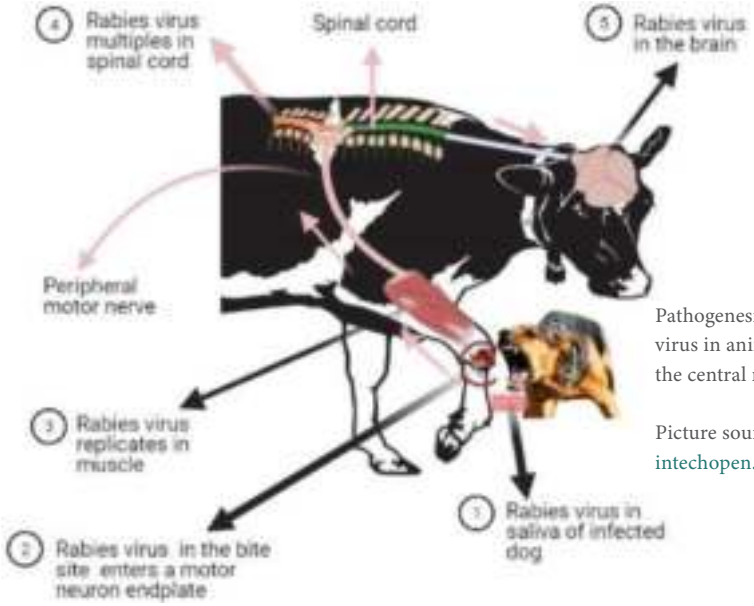
- Heart water
- Chemical toxicity
- Acute lead poisoning,
- Acute salt (sodium toxicosis) and water deprivation
- (Cerebrocortical necrosis) Polioencephalomalacia (PEM) is an important neurologic disease of ruminants that is seen worldwide in Cattle, sheep, goats, and camelids. PEM associated with two types of dietary risks: altered thiamine status and high sulphur intake.

Diagnosis

- Immunofluorescence microscopy (direct fluorescent antibody test) on fresh brain tissue is the test of choice. Brain tissues examined must include the medulla oblongata and cerebellum (and should be preserved by refrigeration with wet ice or cold packs). Immunofluorescence microscopy on fresh brain tissue allows direct visual observation of a specific antigen-antibody reaction.
- Diagnosis should only be done by a qualified laboratory, designated by the local or state health department in accordance with established standardized national protocols for such viral testing.
- Confirmatory tests include PCR and histology of brain tissue.
- A definitive pathologic diagnosis of rabies can be based on the findings of Negri bodies in the brain or the spinal cord. Negri bodies contain rabies virus antigens and can be demonstrated by immunofluorescence. Both Negri bodies and rabies antigens can usually be found in animals or humans infected with rabies, but they are rarely found in bats.

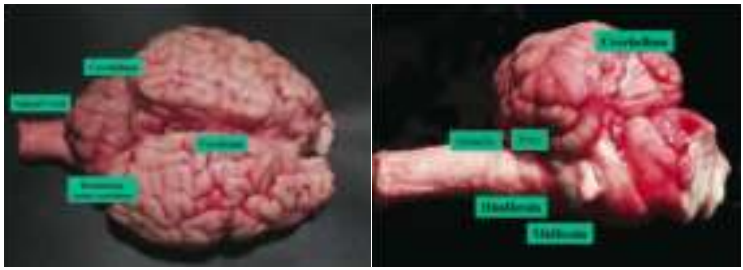
Treatment and Control

- Vaccination and registration of dogs, with control of stray populations
- Oral vaccination of wildlife can reduce rabies in reservoir populations
- Notification of suspected cases, and euthanasia of dogs with clinical signs and dogs bitten by a suspected rabid animal
- Reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine
- Mass immunization of dogs by campaigns and by continuing vaccination of young dogs
- Stray dog control and euthanasia of unvaccinated dogs
- Available animal vaccines use killed vaccines. A rabies control programme should consider: Socio-cultural framework, including public awareness, the promotion of responsible pet ownership and animal welfare. This includes annual rabies vaccination and neuter of animals.

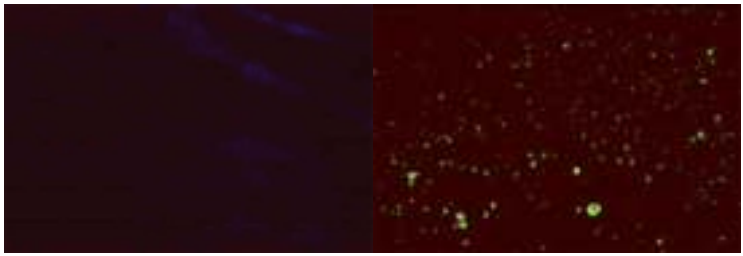


Pathogenesis and spread of rabies virus in animals from the bite site to the central nervous system.

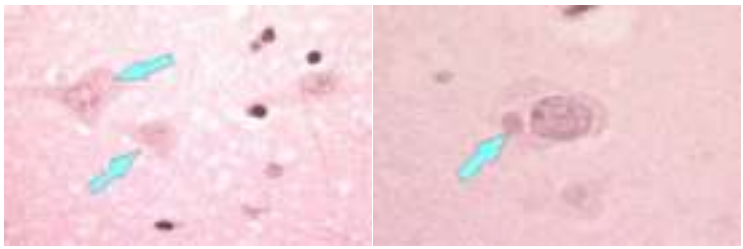
Picture source: https://www.intechopen.com/online-first/77118



The brain tissue is tested for the presence of Negri bodies at the cerebellum or brain stem (brain stem is made up of the midbrain, pons and medulla)

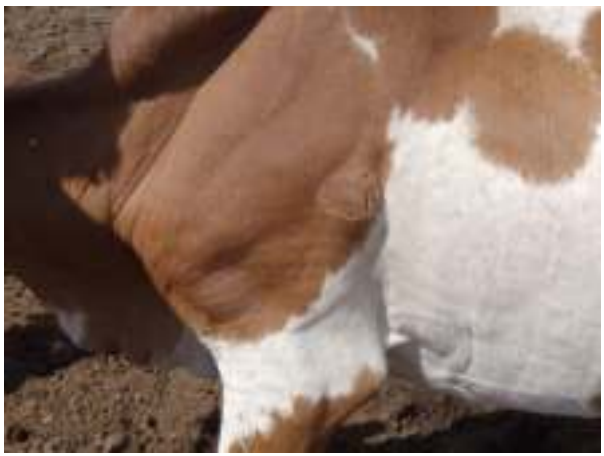


Direct fluorescent antibody test that is negative (Left) and positive (Right) for the rabies virus.



Histology brain tissue with neuron without Negri bodies (right) and with Negri bodies (left)

Picture source: http://uwrfbio353koob.blogspot.com/p/histology.html



A cow bitten by a Rabid donkey in Kajiado county, 2021

Photo credit: Dr. Pauline Gitonga

Rift Valley Fever

Definition

- Rift Valley fever (RVF) is a peracute or acute, mosquito-borne, zoonotic disease of domestic and wild ruminants in Africa, Madagascar, and the Arabian Peninsula. Large outbreaks of clinical disease are usually associated with heavy rainfall and localized flooding. During epidemics, the occurrence of abortions in livestock and deaths among young animals, particularly lambs, together with an influenza-like disease in people, is characteristic. However, infections are frequently subclinical or mild.

Causative agent:

- RVF virus (RFV)belongs to the genus Phlebovirus family Bunyavirus.
- The RNA virus has little genetic diversity, but has different pathogenicity.
- The virus is transmitted by mosquitoes and occurs in inter epizootic intervals (10 years). RVF has caused serious disease in laboratory workers and infected samples should be handled at biosecurity level 2 and 3 laboratories.

Occurrence

- Endemic in many tropical and subtropical regions of Africa, Madagascar, and the Arabian Peninsula. RVF was originally confined to the Rift Valley region of eastern and southern Africa, the virus recently expanded its range, with major outbreaks seen in Egypt since 1977, West Africa since 1987, Madagascar since 1990, and the Arabian Peninsula in 2000. Particularly large epidemics with large numbers of human cases occurred in Egypt in 1977–1978 and in Kenya in 2006–2007.
- RVF is considered a threat to regions where competent mosquito vectors are present.
- Sporadic, large epidemics have occurred at 5–10 year intervals in drier areas of eastern Africa, and less frequently in southern Africa. Outbreaks are usually associated with periods of abnormally heavy rainfall or, in some cases, with localized flooding due to dam building or flood irrigation.
- During inter-epidemic periods, the virus remains dormant in eggs of floodwater-breeding Aedes mosquitoes in the dry soil of small dambos or pans. This transovarial transmission is believed to be the most important inter-epidemic survival strategy for the virus.
- However, inapparent cycling of the virus between vectors and wild or domestic mammalian hosts has been shown to occur in many areas. RVF virus may also spread by movement of viremic animals and possibly by wind-borne mosquitoes. When emergence of infected mosquitoes, or introduction of virus to an area, coincides with abnormally wet conditions and the presence of a highly susceptible host population, a large epidemic may ensue when the virus is amplified in ruminants and spread locally by many species of mosquitoes or mechanically by other insects.
- In Africa where insect vectors are present continuously, seasonality is not seen.

Transmission and Predisposing factors

- People are readily infected through blood aerosols from infected animals during slaughter, or by exposure to infected animal tissues, aborted foetuses, mosquito bites, and laboratory procedures.
- Therefore, veterinarians, farm labourers, and abattoir workers are particularly at risk.
- People can also act as amplifying hosts and introduce the disease (via mosquitoes) to animals in uninfected areas.

- Elnino climatic conditions favours breeding of mosquito vectors (Aedes, Anopheles, Culex, Eretmapodites and Mansonia). Mosquito species act as reservoirs for RVF virus during inter-epidemic periods and increased precipitation in dry areas leads to an explosive hatching of mosquito eggs; many of which harbour RVF virus.
- During inter epidemic period immuno-naïve domestic ruminants' animal populations are present and when Infected Aedes feed on domestic ruminants they act as biological amplifier of RVF virus.
- Satellite imaging has been used to confirm historic importance of precipitation in RVF outbreaks and in forecasting high-risk areas for future outbreaks.
- There are more human deaths from communities that consume raw infected meat during home slaughters during outbreaks.
- There is possible human transmission through consumption of raw milk
- Virus regularly circulates in endemic areas between wild ruminants and mosquitoes' disease is usually inapparent.

Clinical signs

- Storm of abortion in goats, sheep (mainly), cattle and camel,
- High mortalities in young lambs
- In mature ruminants- bloody nasal discharges, haemorrhagic fetid diarrhoea, dysgalactia and jaundice during abnormally prolonged heavy rainfall.
- Mortality rate in lambs is high (90-100%), goat kids and calves (70%), adult sheep and s 10-30%, adult goats, cattle, camels (5-10%).

Humans

- Influenza-like syndrome: fever (37.8–40°C), headache, muscular pain, weakness, nausea and epigastric discomfort, photophobia and recovery occurs within 4–7 days. Complications of retinopathy, blindness, meningo-encephalitis, haemorrhagic syndrome with jaundice, skin petechial spots and death.

Post mortem lesions

- Appropriate bio-security and biosafety must be exercised as RVF is a potent zoonotic pathogen.
- Focal or generalised hepatic necrosis (white necrotic foci of about 1 mm in diameter).
- Congestion, enlargement, and discoloration of liver with sub capsular haemorrhages.
- Brown-yellowish colour of liver in aborted foetuses.
- Widespread cutaneous haemorrhages, petechial to ecchymotic haemorrhages on parietal and visceral serosa membranes.
- Haemorrhagic enteritis and Icterus

Differential Diagnosis

- Bluetongue
- Enterotoxaemia of sheep
- Ephemeral fever
- Brucellosis
- Leptospirosis
- Q-fever
- Plant poisoning
- Bacterial septicaemias
- Anthrax

Diagnosis

Field based diagnosis

- Clinical signs- storm of abortion in goats, sheep (mainly), cattle and camel, high mortalities in young animals (lambs and kids), bloody nasal discharges in mature animals, haemorrhagic diarrhoea and jaundice during abnormally prolonged heavy rainfall.

Sample Collection

- Donning of full personal protective clothing is a must
- Most laboratories will require serum or blood to conduct competitive ELISA (antibody testing) or PCR tests (antigen identification). IgM antibodies signify a recent infection while IgG a past infection.

Treatment and Control

- There is no treatment of sick animals, supportive therapy to animals with clinical

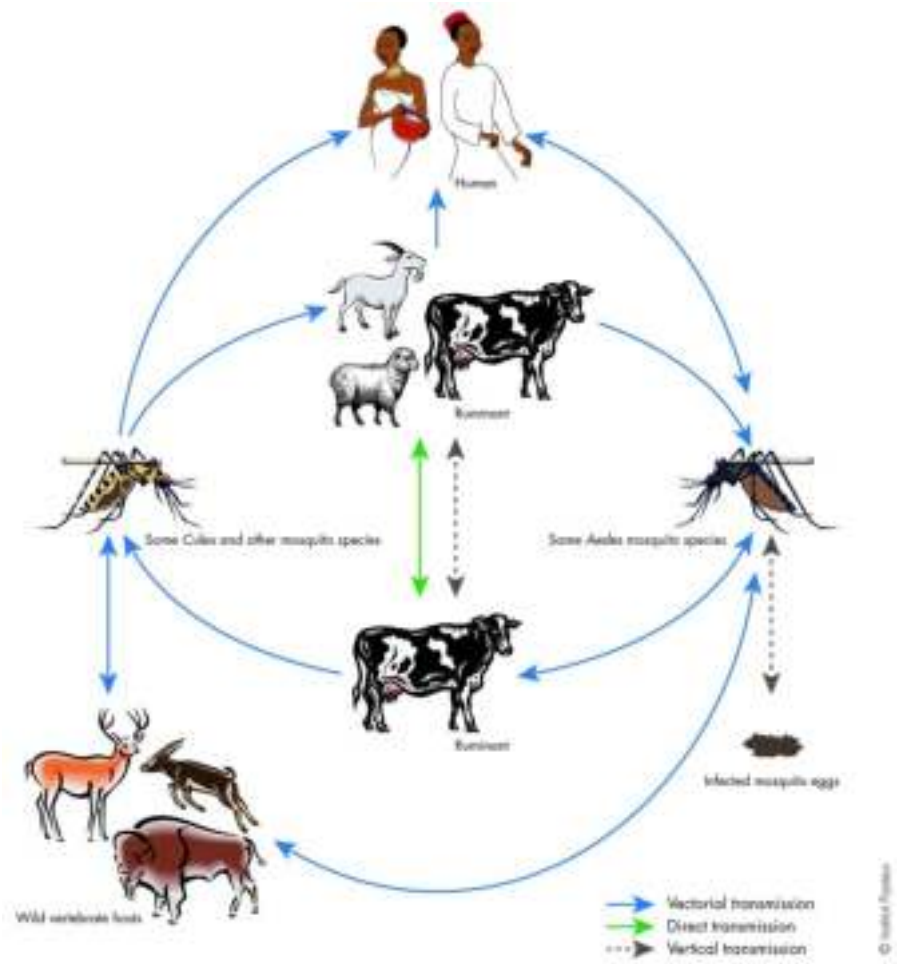
signs. Care should be taken not to spread infection through re-use of hypodermic needles or self-infection during treatment of the sick animals.

- Conduct Participatory disease surveillance (PDS) Surveillance during inter-epizootic period. Targets the period prior to and during the early warning of heavy rain by international and national meteorological agencies. It will be crucial at this point to assess the capacity in critical areas such as coordination, sero-surveillance, laboratory diagnosis and response.
- Develop platforms for crowd sourcing of information from the community. Creation of public awareness on the risk factors and need to report the occurrence of risk factors such as heavy rains, flooding, abortions in animals or mosquito build up should be encouraged.

NB: Vaccination should not be done in areas with suspected clinical disease. This is because vaccination team have a high likelihood of spreading infection through equipment and have a high chance of self-infection as most animals having no clinical sign but are amplifying the virus.

- Quarantine and close of slaughter slabs and houses, livestock markets and movement restriction
- Scale up public health messages to ensure people stop home slaughter or touching aborted foetuses without protection
- During the inter-epizootic phase without predisposing factors, the following control measures may be applied:

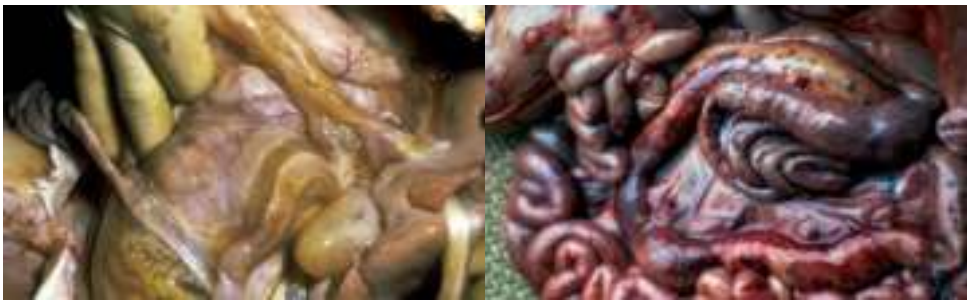
- Vaccination in identified high risk areas- RIFTVAX TM®- is a freeze-dried live attenuated vaccine prepared from Smithburn's attenuated strain of RVF virus.
- Public education should inform livestock keepers that vaccination is associated sometimes with early embryonic death, congenital CNS anomalies and arthrogryposis, or abortion or stillbirths.



Rift Valley Fever (RVF) Transmission cycle

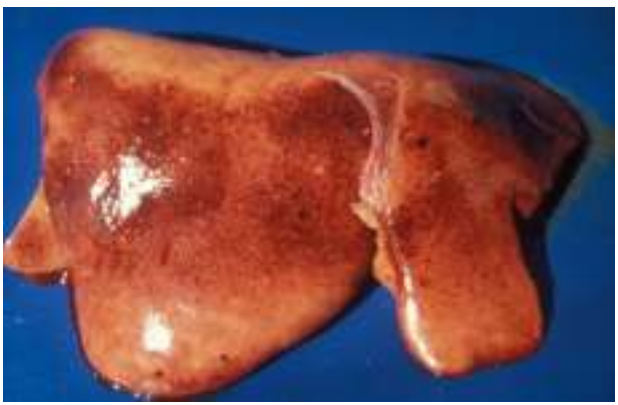
Picture source: https://veterinaryresearch.biomedcentral.com/articles/10.1186/1297-9716-44-78#Fig2

RVF infection in a cow placenta with oedema and haemorrhages (hydramnion) and aborted emphysematous foetus with 'meconium staining (left)



Small intestine of a cow (Left) with marked mesenteric and serosal oedema and New-born Lamb intestines (Right) with haemorrhages in the intestine

Picture source: https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0006353



Rift Valley fever (RVF) markedly enlarged ruminant liver, yellow-brown discolouration of the organ, and congested patches scattered throughout the organ

Picture source: https://repository.up.ac.za/handle/2263/32685

BRUCELLOSIS – AKIYEC / EDEKE AKIRING KA NG'AKILE

Other names: Contagious abortion, Bang's disease in livestock and "undulant fever", "Mediterranean fever" or "Malta fever" in humans

Definition

- Zoonosis transmitted by direct or indirect contact with infected animals or their products. It affects people of all age groups and of both sexes.
- Duration of human illness and its long convalescence means that brucellosis is an important economic problem for the patient because of time lost from normal activities. In many underserved regions the delay in diagnosis and endemic status of the disease in livestock means that the disease remains a constant threat to human welfare, particularly for those in the most vulnerable socioeconomic sections of the population.
- Brucellosis is primarily a disease of animals, especially domesticated livestock, with humans being accidental host.

Causative agent:

- Brucella organisms are gram-negative coccobacilli and may be present in high concentration in blood cultures drawn early in the disease process.
- Six main specie": B. abortus, B. suis, B. melitensis, B. neotomae, B. ovis, B. canis. Brucella melitensis and Brucella abortus are the most important species in Kenya.

Occurrence

- Disease occurs worldwide, except in countries where bovine brucellosis (B. abortus) has been eradicated. This is usually defined as the absence of any reported cases for at least five years. These countries include Australia, Canada, Cyprus, Denmark, Finland, the Netherlands, New Zealand, Norway, Sweden and the United Kingdom (WOAH Reports).
- Mediterranean countries of Europe, northern and eastern Africa, Near East countries, India, Central Asia, Mexico and Central and South America are especially affected. While B. melitensis has never been detected in some countries, there are no reliable reports that it has ever been eradicated from small ruminants.
- The sources of infection for humans and the species of Brucella spp. found vary according to geographical region. It is usually either an occupational or a food-borne infection. Both sporadic cases and epidemics occur in humans, but often the disease or infection is either unrecognized, or, if diagnosed, not reported to the public health authorities.
- Brucellosis is a notifiable disease and must be reported to the Kenya DVS who then reports to WOAH.

Transmission and Predisposing factors

- Infection spreads rapidly and causes many abortions in unvaccinated herds especially in cattle.
- In endemic areas infected animals typically aborts only once after exposure; subsequent gestations and lactations appear normal. After exposure, animal become bacteraemic for a short period and spontaneously recover.
- The incubation period is variable and is inversely related to stage of gestation at time of exposure. Organisms are shed in milk and

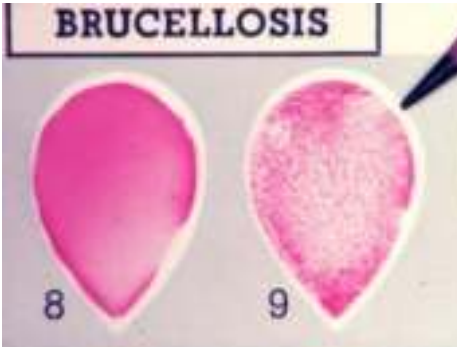
uterine discharges, and the cow may become temporarily infertile. Bacteria may be found in the uterus during pregnancy, uterine involution, and infrequently, for a prolonged time in the non-gravid uterus.
- Shedding from the vagina largely disappears with the cessation of fluids after parturition.
- Some infected cows that previously aborted shed Brucella from the uterus at subsequent normal parturitions.
- Organisms are shed in milk for a variable length of time—in most cattle for life. B abortus can frequently be isolated from secretions of nonlactating udders.

Main clinical signs
- Abortion
- Stillborn
- Weak calf born
- Retention of foetal membranes
- Swollen testicles in bulls
- General health of animal is not impaired in uncomplicated abortions or orchitis
- Testicular abscesses may occur. Longstanding infections may result in arthritic joints in some cattle

Differential Diagnosis
- Rift valley Fever (RVF)- abortion in animals and fever in humans
- Q fever- abortion in animals and fever in humans
- Leptospirosis- abortion in livestock
- Listeriosis- abortion in livestock
- Campylobacteriosis and chlamydia infection - Infertility and orchitis in livestock
- Blue tongue for small ruminants- abortion

Diagnosis
- Culture is the hold standard for confirmatory diagnosis. Culture requires a BSL laboratory. I
- ELISA or PCR can also be used as confirmatory tests.

Control
- The search for ideal brucellosis vaccines remains active today. Currently, no licensed human or canine anti-brucellosis vaccines are available. In bovines, the most successful vaccine (S19) and is only used in calves, as adult vaccination results in orchitis in male, prolonged infection, and possible abortion complications in pregnant female cattle. Another widely deployed vaccine (RB51) has a low protective efficacy. In Kenya, there is no vaccine approved for use by the DVS.
- Methods of prevention include health education to reduce occupational and food-borne risks, including pasteurization of all dairy products and handling aborted foetuses with protective gloves.



Rose Bengal or card test showing negative (left) and positive (right) reactions

Rapid Brucella antibody chromatography test

Further reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/
2. Peter J. Fernández & William R. White (2010). Atlas of Transboundary Animal Diseases https://WOAHbulletin.com/?panorama=atlas-of-transboundary-animal-diseases
3. United Nations Environment Programme and International Livestock Research Institute (2020). Preventing the Next Pandemic: Zoonotic diseases and how to break the chain of transmission. Nairobi, Kenya. https://www.unep.org/resources/report/preventing-future-zoonotic-disease-outbreaks-protecting-environment-animals-and

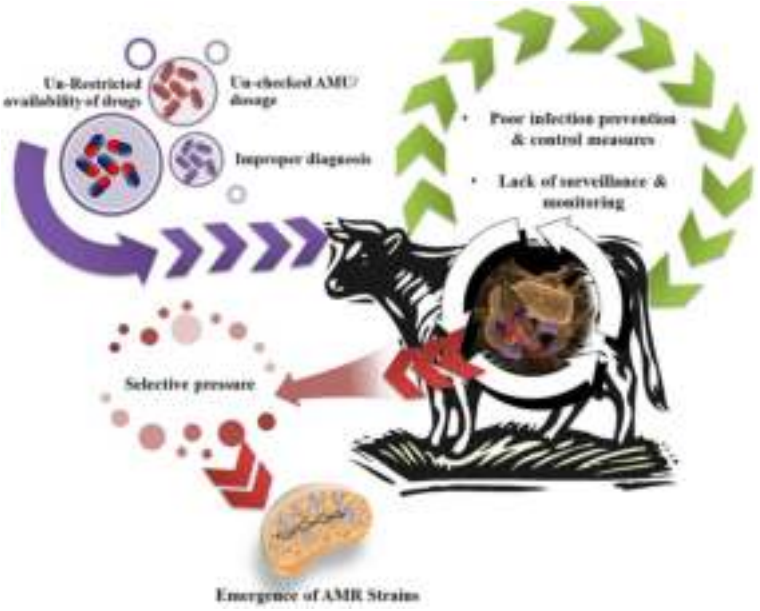
Annex 3:

Antimicrobial Resistance

Definition

- Antimicrobial resistance (AMR) is the ability of bacteria, fungi, viruses and parasites to develop biological and propagatable ability to resist the effects antimicrobial agents (antibiotics, antifungals, antivirals, antimalarial, and anthelmintics). As a result, the medicines become ineffective and infections persist in the body, increasing the risk of spread to others.
- Resistant diseases agents have the ability to multiply or persist in the presence of increased level of an antimicrobial agent relative to their susceptible counterpart of the same species.
- Antimicrobial resistance (AMR), is described as a silent pandemic at both national and global levels that is threatening the advancement of antibiotics use in managing infectious diseases in both humans and livestock.
- Emergence of antibiotic resistant bacterial strains are a product of both continuous evolution and un-checked antimicrobial usage (AMU).
- Significant emphasis has been given to AMR in humans, however, trends of AMU in livestock is often not emphasized.
- There is need for global co-operative efforts at individual, community, local, regional, national, and international level to address AMR.
- In order to meet this global goal, a Tripartite Alliance was formed between the WHO, FAO, and WOAH with One Health approach. The tripartite alliance published the Global Action Plan on AMR in 2015. Likewise, FAO also launched its AMR Strategy in 2016 to back the proper execution of the WHO Global Action Plan in food and agricultural sectors. The WHO Global Action Plan emphasizes on increased awareness and understanding on AMU and associated AMR; build up knowledge regarding AMR through proper surveillance and research; optimal and rational use of antibiotics; lowering the incidence of infectious diseases; and organizing resources, research, and development for proper integrated prevention and containment of antibiotic resistance.

Factors leading to antimicrobial resistance (AMR) in livestock



Conceptual representation of possible movement of antibiotic-resistant bacterial strains/genes between different

Critically important antibiotics for human use based on WHO

Critically Important		Highly Important	Important	Currently not used in humans
Highest priority	High Priority			
• Cephalosporins (3rd, 4th, 5th Gen.) • Fluro And Other Quinolones • Glycopeptides • Macrolides And Ketolides • Polymyxins	• Aminoglycosides • Ansamycins • Carbapenems And Other Penems • Glycylcylines • Lipopeptides • Monobactams • Oxazolidinones • Penicillins (Natural Aminopenicillins & Antipseudomonal) • Phosphonic Acid Derivertives • Tuberculosis And Other Mycobacterial Drugs	• Amphenicals • Cephalosporins (1st & 2nd Gen.) • Lincosamides • Penicillins (Anti-Staphylococcal) • Pseudomonic Acid • Riminofenazines • Steroids Antibacterials • Streptogrmins • Sulfonamides • Sulfones • Tertacyclines	• Aminocyclitols • Cyclic Polypeptides • Nitrofurantoin • Nitromidazoles • Pleuromitilins	• Bambermycins • Quinoxalines • Ionophores • Orthosomycins

Info source: https://www.frontiersin.org/articles/10.3389/fvets.2017.00237/full

Antibiotics Mode of Action

- Understanding the mechanism of action of antibiotics by animal health service providers is a first step in mitigating AMR.
- Ability of an antimicrobial drug to arrest the growth of or kill bacteria is dependent upon its mechanism of action and the concentration that the drug attains at the infection site. When a drug is introduced into the body, it is rapidly carried through the bloodstream to the liver, kidneys, and other organs that can chemically change or reduce its antibacterial activity and promote its excretion.
- The pharmacokinetic process (movement of drugs within the body) include;

(1) absorption from its site of administration
(2) distribution throughout the body and its elimination by (3) biochemical metabolism, and (4) excretion through the urine, bile, or other routes are collectively given the acronym ADME.

- Bactericidal Vs. Bacteriostatic- Antibiotics can be divided to two groups on the basis of their effect on microbial cells through two main mechanisms, which are either bactericidal or bacteriostatic. Bactericidal antibiotics kill the bacteria and bacteriostatic antibiotics suppress the growth of bacteria (keep them in the stationary phase of growth).
- Bactericidal antibiotics cause cell death by preventing cell wall synthesis of the bacterial cell. This reduces the number of bacterial cells present.

- Bacteriostatic antibiotics prevent further cell growth. This is achieved by inhibiting protein synthesis, DNA replication or other cellular metabolic actions, although it does not cause bacterial cell death. This effect is reversible, unlike the action of bactericidal drugs. The amount of bacterial cells, therefore, remains the same, although in a static phase. The bacteriostatic drugs rely on the host immune system to overcome or clear the bacteria held in static phase.
- Clinical outcome for antibiotics is affected by factors that include infecting bacterial density, host immune response, underlying disease or the site of infection for example if infection is in the organs with physiological barriers like the brain or infection has caused formation of thick granulomatous capsule that is not penetrable.
- Efficacy- bactericidal antibiotics are effective against dividing cells its efficacy may be reduced if treatment is paired with the slower growth caused by bacteriostatic drugs. This is why practitioners should not mix bactericidal and bacteriostatic drugs.
- Side effects- bactericidal drugs have a rapid mode of action and can stimulate severe inflammation due to the release of bacterial cell contents upon cell death, which can result in toxic shock syndrome. This means that in certain diseases caused by bacteria that release toxins, bacteriostatic drugs are often preferred so as to prevent fatal toxaemia syndrome. Bacteriostatic drugs have no side effects other than preventing bacterial growth, although if removed from the system their effects can be reversed and the bacteria can start multiplying (Main reason why dosage regime and length should be followed).

Further Reading

1. Sharma C, Rokana N, Chandra M, Singh BP, Gulhane RD, Gill JPS, Ray P, Puniya AK and Panwar H (2018) Antimicrobial Resistance: Its Surveillance, Impact, and Alternative Management Strategies in Dairy Animals. Front. Vet. Sci. 4:237. https://doi.org/10.3389/fvets.2017.00237

2. Difference between bactericidal and bacteriostatic antibiotics http://www.differencebetween.net/science/difference-between-bactericidal-and-bacteriostatic/#ixzz74urGEVrM

3. WHO guidelines on use of medically important antimicrobials in food-producing animals. Geneva: World Health Organization; 2017. Licence: CC BY-NC-SA 3.0 IGO.
https://apps.who.int/iris/bitstream/handle/10665/258970/9789241550130-eng.pdf

Examples of commonly used veterinary antibiotic and mode of action

Class of antibiotic	Mode of action	Spectrum of activity	Veterinary use in ruminants
Aminoglycosides Gentamicin, tobramycin, amikacin, streptomycin, kanamycin	Bactericidal – inhibit protein synthesis	Broad spectrum except anaerobic bacteria	Enterobacteriaceae. Infections and severe sepsis cause by Gram-negative aerobes.
ß-Lactam Penicillins: penicillin G, ampicillin, amoxicillin, carbenicillin 1st, 2nd, 3rd, 4th Generation Cephalosporins*	Bactericidal- Inhibit of cell wall synthesis.	Broad spectrum for 2nd, 3rd, and 4th generation cephalosporins Narrow spectrum (Gram positive mainly) for penicillins and 1st generation cephalosporins	Anthrax, listeriosis, leptospirosis, clostridial and corynebacterial infections; streptococcal mastitis, keratoconjuntivitis
Fluoroquinolones Enrofloxacin, ciprofloxacin,	Bactericidal- Inhibition of nucleic acid synthesis	Broad spectrum: 3rd-generation fluoroquinolones Narrow spectrum: other fluoroquinolones	Acute respiratory disease, infections with E. coli, Salmonella, Mycoplasma, mastitis, metritis, conjunctivitis
Macrolides Erythromycin, Tylosin, Spiramycin, Tilmicosin, Tulathromycin	Bacteriostatic- inhibit protein synthesis- can be bactericidal at high concentrations or if there is low numbers of susceptible bacterial	Narrow spectrum	Erythromycin: drug of choice against Campylobacter jejuni. alternative to penicillin in penicillin-allergic animals and second choice for anaerobic infections. Tylosin against Mannheimia, Actinobaciullus, Pasteurella, Mycoplasma.
Sulphonamides Sulfadiazine, sulfamethoxazole, sulfadoxine	Bacteriostatic- inhibit folic acid synthesis	Broad-spectrum; affects Gram-positive, Gram-negative bacteria, toxoplasma and protozoal agents	First line of drugs used in ruminants and poultry to manage diarrhoea and coccidiosis Also used for infections due to E. coli Haemophilus Salmonella Pasteurella Staphyloccocus Streptococcus
Diaminopyrimidines Trimethoprim,	Diaminopyrimidines Trimethoprim,	Broad spectrum; affects Gram-positive and many Gram-negative bacteria Act synergistically (and becomes bactericidal) in combination with sulphonamides	
Tetracyclines Chlortetracycline, oxytetracycline, doxycycline, minocycline	Bacteriostatic – Inhibits protein synthesis	Broad spectrum. Gram-positive, Gram-negative bacteria, atypical organisms such as chlamydiae, mycoplasmas, rickettsiae, and protozoan parasites.	Ehrlichiosis, leptospirosis, listeriosis, rickettsiosis, Anaplasmosis, Heart water, ECF vaccination

Annex 4:

Calculating drug dosage

The Volume of injectable veterinary drug to be administered can be calculated as follows (Nair & Jacob, 2016);

$$\text{Injection Volume (ml)} = \frac{\text{Dosage (mg/kg) X Body weight (kg)}}{\text{Concentration (mg/ml)}}$$

Where;

Volume is measured in ml

Dosage is measured in mg/kg body weight- usually stated in the drug label or insert kit

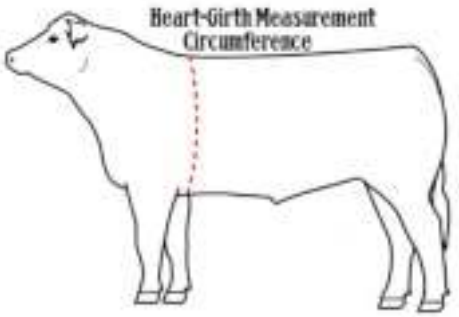
Body weight is measured in kg

Concentration is measured in mg/ml- usually stated in the drug label or insert kit

Therefore: To calculate the volume to inject a 150 kg cow for the different oxytetracycline preparation if given a recommended dosage of 10mg/kg will be;

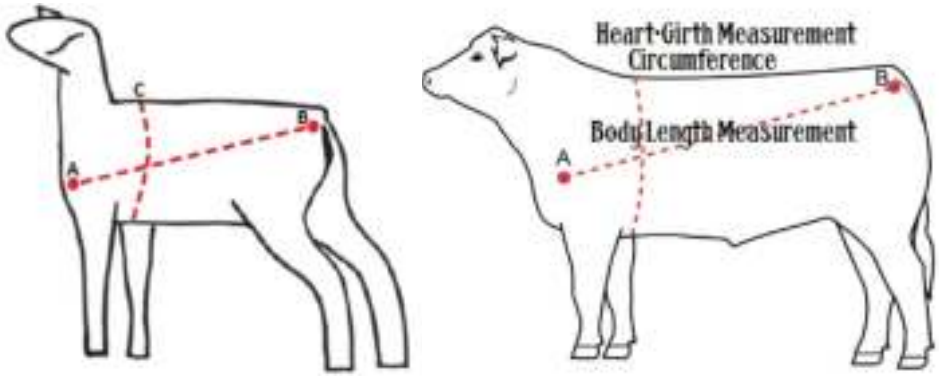
- 5% preparation = (10× 150) ÷ 50 = 30 mls
- 10% preparation= (10× 150) ÷ 100 = 15 mls
- 20% preparation= (10× 150) ÷ 200 = 7.5 mls
- 30% preparation= (10× 150) ÷ 300 = 1 ml

Estimating weight of animal



Weight measurement using heart girth in cattle (top) and goat (down)

Picture sources:
http://bairnsley.com/Weight%20by%20Girth.htm (Cattle)
http://www.infovets.com/books/smrm/C/C098.htm (Goat)



Estimating live-weight of goats (top) and cattle (bottom) using weight estimation formul

- MEASURE: (A-B) body length measured from point of shoulder to point-of-rump or pin bone.
- MEASURE: (C) heart girth circumference, around the body behind the shoulder, over the heart area and very close to the forelegs. When measuring a sheep, compress the sheep's wool to obtain a circumference that reflects the body and not the body plus the wool.
- Using these 2 measurements, calculate body weight estimate with this formula:

o (Heart girth (inches) X heart girth (inches) X body length (inches) ÷ 300 = approx. animal weight (pounds).

- Convert to kilograms using formula- 1 kilogram is equivalent to 2.205 so divide answer by 2.2

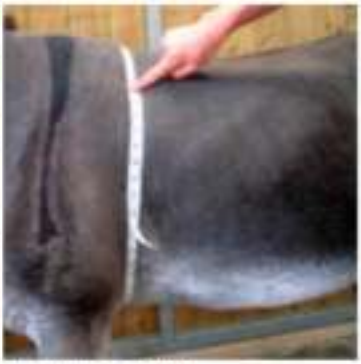
Example; if a sheep has a heart girth equal to 35 inches and a body length equal to 30 inches, the calculation would be (35 x 35 x 30) / 300 = 122 lbs

converted to kgs 122÷2.2= 55.45 kgs.

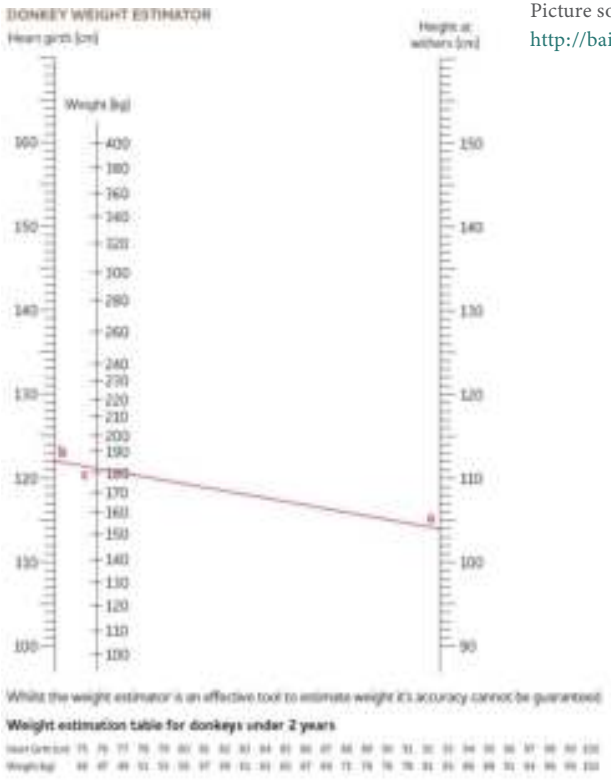
DONKEY WEIGHT ESTIMATOR

To estimate a donkey's weight using the diagram below mark the height and heart girth measurements on the correct axis. Then draw a line between the two. The donkey's weight is indicated by where the line crosses the weight. For example, a donkey 104cm tall (a) and with a heart girth 122cm (b) should weigh 181kg (c).

Measuring height (cm)



Measuring girth (cm)



Picture source:
http://bairnsley.com/Weight%20by%20Girth.htm

Annex 5:

Photo Gallery of Medicinal and Poisonous plants in Turkana County

The livestock keepers identified a total of 19 medicinal plants that are used to manage the priority livestock diseases in Turkana county. The photo identification of these plants are highlighted below.

Etaba- Chewing Tobacco (Nicotiana tabacum)
Photo credit: Gosh Kwanyang (CDR ILLeret, Marsabit County)

Egis- Cissus quadrangularis
Photo credit: iNaturalist https://inaturalist.nz/photos/2231433

Emus- Euphorbia kalisinia or E.uhligiana
Photo credit: Dr. Pauline Gitonga,
Turkana county

Echuchuka- Aloe turkanensis
Picture source: https://davesgarden.com/guides/pf/showimage/172698/

Mwarubaini /Neem tree- Azadirachta indica
Picture source: Paukwa https://paukwa.or.ke/mwarubaini-the-plant-that-can-cure-40-diseases/

Ereng- Cadaba farinose
Picture credit: Feedipedia https://www.feedipedia.org/node/174

Ekwang'a –Dalechampia scandens
Picture credit: https://wildflowersearch.org/search?&tsn=28307

Elamach- Balanites pedicellaris

Ebei- Balanites rotundifolia

Photos credit: https://powo.science.kew.org/taxon/urn:lsid:ipni.org:names:873118-1

Edung- Boscia coriacea

Etirae- Prosopis Julliflora
Pictures credit: https://powo.science.kew.org/taxon/urn:lsid:ipni.org:names:77214011-1

Etirae- Prosopis Julliflora
Pictures credit: https://powo.science.kew.org/taxon/urn:lsid:ipni.org:names:77214011-1

Picture credit: https://www.tandfonline.com/doi/full/10.1080/23311932.2020.1853867

Picture credit: Plants of the world https://powo.science.kew.org/taxon/urn:lsid:ipni.org:names:303822-1

Ereng- Maerua crassifolia

Picture credit: Feedipedia https://www.feedipedia.org/node/130

Esekon – Salvadora persica

Etesuro- Calatropis procera

Pictures credit: Feedipedia https://www.feedipedia.org/node/130

Ekore (seeds) - Gardenia ternifolia

Pictures credit: Useful Tropical Plants http://tropical.theferns.info/viewtropical.php?id=Gardenia+ternifolia

Eligoi – Sarcostemma viminale

Pictures credit: https://www.flickr.com/photos/48991563@N06/5779510983

Egeles- Adenium obesum (Desert rose)

Pictures credit: https://www.istockphoto.com/photo/desert-rose-or-adenium-in-turkana-kenya-gm117483539-7740585

Sorghum is associated with cyanide like poisoning

Picture source: Dr Jacquiline Kisianan in Turkana County

Lomuge ang'ikadong'oi, or Lomarindae - Xanthium Strimariam commonly known as Cocklebur a poisonous plant found near the river banks

Lomuge ang'ikadong'oi means brown cattle bell and Lomarindae means women petticoats. The plant is poisonous when it has just sprouted or during the dry season when livestock eat the seeds.

Traditional practice- Hot iron or stone cauterisation- Akimad emachar kori amour

The most common traditional practice was Akimad also known as Emacher which is hot iron cauterisation of body part afflicted with the disease as shown in the photos below.

Emacher/Akimad- Hot iron cauterisation of a goat with Contagious Caprine Pleuropneumonia (CCPP)

Picture credit: Dr. Jacquiline in Turkana County

Emacher/Akimad- Hot stone cauterisation to treat a sick camel

Picture credit: Kangiro Evans (Plant village One Health Scout) Turkana county

NOTES

TURKANA COUNTY

VETERINARY HANDBOOK

FOR PRIORITY LIVESTOCK DISEASES

Diagnosis, Treatment and Control Guidelines
For Animal Health Service Providers

ASAL eXtension

www.asalextension.com

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www.whitespace.co.ke