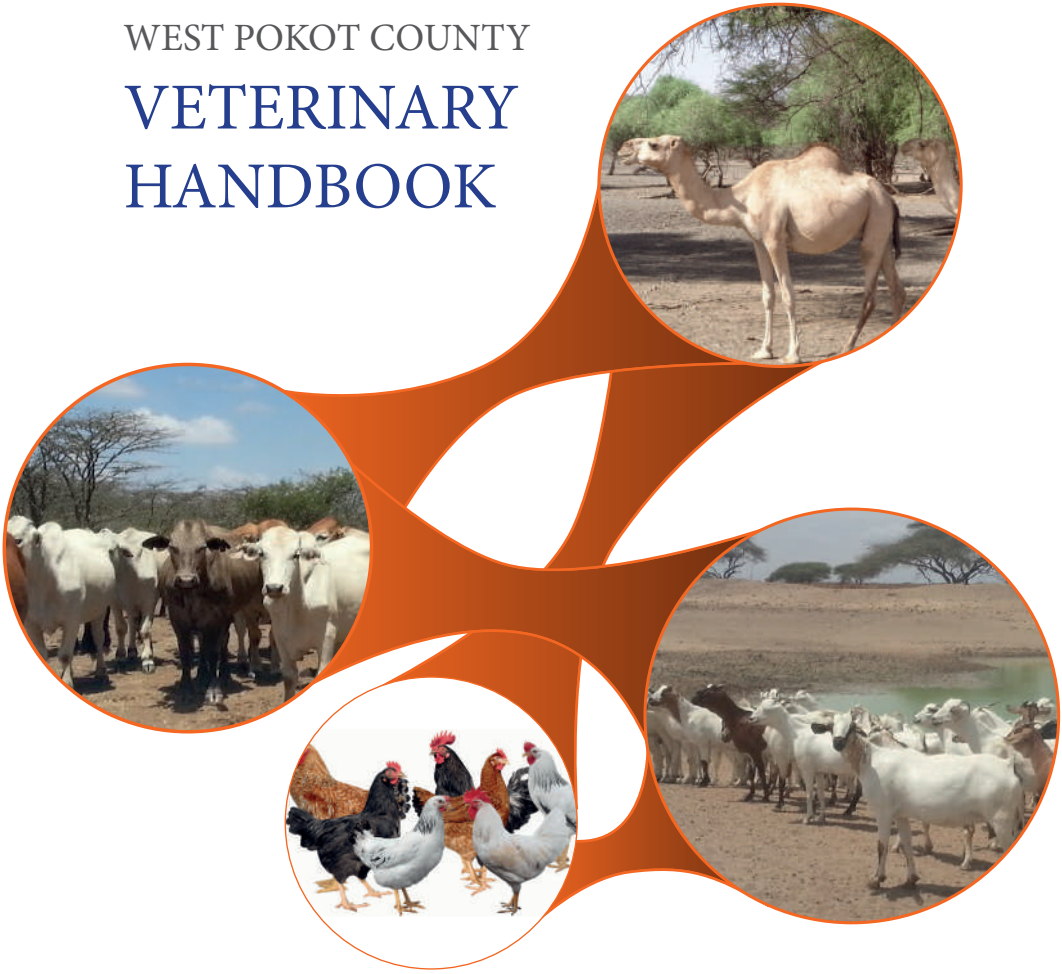
WEST POKOT COUNTY

VETERINARY HANDBOOK



USAID
FROM THE AMERICAN PEOPLE

ACTED

COUNTY GOVERNMENT OF WEST POKOT

FOR PRIORITY CAMEL, CATTLE, SHEEP, GOAT & CHICKEN DISEASES

Common 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 WEST POKOT COUNTY MANAGE ENDEMIC PRIORITY LIVESTOCK DISEASES·
THE HANDBOOK SUMMARISES CONVENTIONAL VETERINARY KNOWLEDGE AND INTEGRATES LOCAL DISEASE NAMES AND ETHNOVETERINARY KNOWLEDGE & PRACTICE·

PRACTITIONERS NEED TO BE AWARE OF ALL THE LAWS AND REGULATIONS THAT APPLY TO THE PRACTICE OF VETERINARY MEDICINE IN KENYA AS THE HANDBOOK DOES NOT EXPLICITLY COVER VETERINARY JURISPRUDENCE· SIMILARLY, 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, UNDERSTOOD BEFORE ANY DRUG IS ADMINISTERED OR PRESCRIBED·
THE PRACTITIONERS SHOULD ALWAYS ENSURE THE DRUG WITHDRAWAL PERIODS FOR MILK, MEAT AND EGGS ARE UNDERSTOOD BY THE LIVESTOCK KEEPERS·

Table of Contents

List of Figures

Preface

The veterinary technical handbook aims at harmonising the identification, diagnosis and control of priority livestock diseases in West Pokot County. The handbook is an initiative of the Agency for technical Cooperation for Development (ACTED) with funding support from the USAID Bureau for Humanitarian Assistance (BHA). The identified priority diseases are as a result of a participatory mapping study conducted between 18th and 21st May, 2021. The study conducted sixteen (16) Focus Group Discussions (FGDs) with livestock keepers across 4 sub counties, 6 wards and 7 villages. A total of 160 livestock keepers participated in the FGDs, of these, 107 (67%) were male and 53 (33%) were female. The validation workshop was the main data verification method used to collaborate FGD findings. The workshop was held on 23rd June 2021 and had a total of 41 participants composed of 35 males and 6 females. Seventeen (17) key informant interviews were also conducted. The KIIs respondents were drawn from the public and private sector as well as from the community leadership structures.

The handbook provides a simple and concise description of the priority diseases as well as contextualises the diseases to the local setting through provision of local names of disease syndromes and ethnoveterinary practices. The West Pokot Animal Health Service Providers (AHSPs) are urged to use the handbook alongside the set national and OIE guidelines for treatment and control of diseases. In addition, harmful treatment and ethnoveterinary practices by livestock keepers that are highlighted in red should be discouraged through sustained extension messaging. There is need to conduct more ethnobotanical surveys in the county so as to update and document the ethnoveterinary knowledge as well as to determine the pharmacological properties and efficacy of the medicinal plants.

It is my hope that the handbook serves as a quick reference companion for animal health practitioners serving in West Pokot County.

Dr. Pauline Njoki Gitonga
Animal Health Consultant
BVM, MSc, PhD.

Chapter 1:

Clinical Examination Guidelines

1.1 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

1.2 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 - Vaginal examination

Rectal examination and female udder and male external genitalia.

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).

1.3 Techniques used to conduct a physical examination

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

1.4 Normal body parameters of ruminant livestock

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

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

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

* 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

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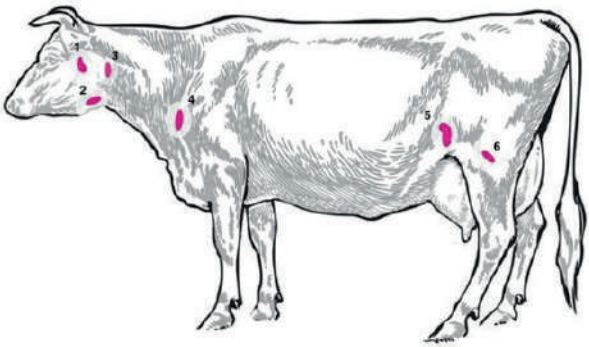
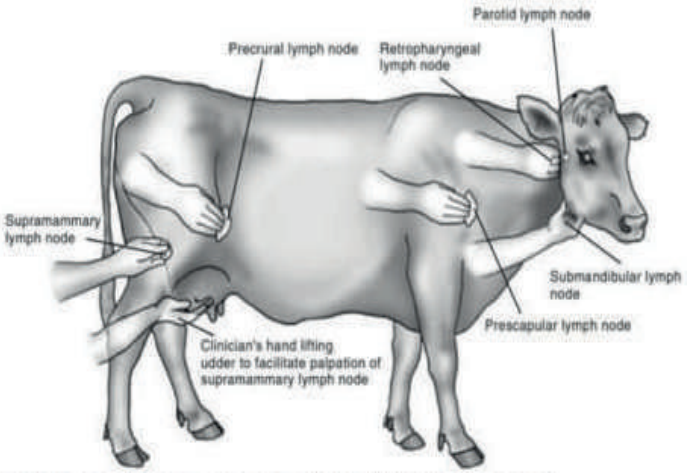
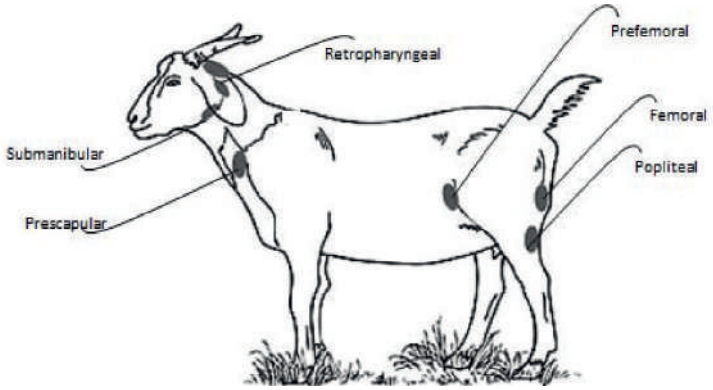
3. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam_reports/21

1.5 | Location of palpable superficial lymphnodes of ruminant livestock



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

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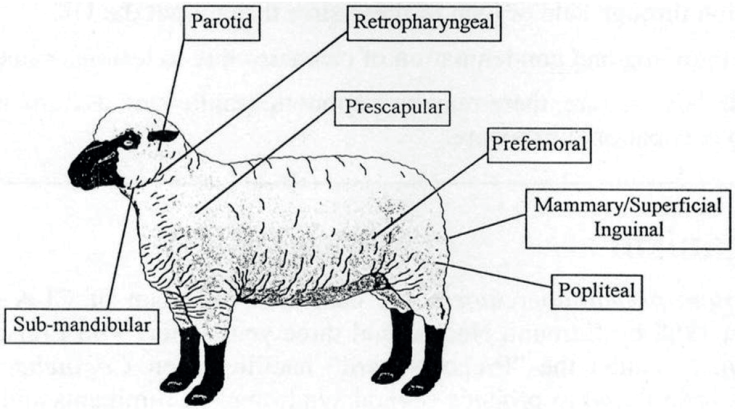
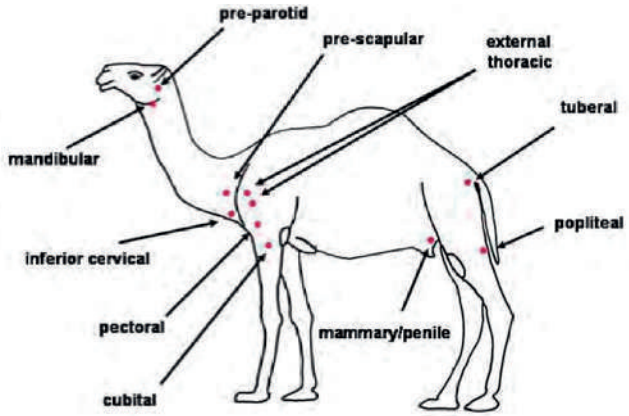
Prefemoral
Femoral
Popliteal
Submanibular
Prescapular


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

1.6 | Traditional names and clinical syndromes for priority livestock diseases in West Pokot county as described by Livestock Keepers

Cattle diseases	Main clinical signs
Lumpy Skin Disease (Lomolokoo)	• Nodular skin lesions (Lomolokoo) • Swelling of one fore limb (Kutu Kel) • Fever (Kilay)
Foot and Mouth Disease (Ng'orion)	• Oral lesions (Moyoi po-kuut), • Foot lesions (Moyoi po-kelien) • Salivation (Sumpaletat) • Lameness (Ngwalat) • Abortion (Torunogh) • Fever (Kilay)
East Coast Fever (Yit/ Lokit)	• Respiratory distress (Kissumat / Kirumat) • Enlarged lymphnodes (Nguriel), • Diarrhoea (Yitoy), • Terminal Bloody diarrhoea (Yitoy kison ta k'ma) • Brown ear tick infestation (Kinam Tilis) • Fever (Kilay) • Anaemia (Ngorungun po kison)
Anaplasmosis (Saak/ Toroy)	• Constipation/Hard dung- (Timatamuu / Kutunata muu/ Kiyomo s'ko) • Blue tick infestation (Kinam Talusia)
Contagious Bovine Pleuropneumonia (Luokoi/Psosoy)	• Coughing (Rolei) • Lacrimation (Lokio Konyin) • Respiratory distress (Kissmat/ Kirumat) • Emaciation (Nyasat /Changulat)
Primary Bloat (Musarer)	• Abnormal distension of abdomen (Musarer)
Black Quarter (Tiompo Parak Tiompo Yim Tiompo Ogha)	• Sudden death (Chemutei) • Swollen hind limb with crepitus of muscles • affected (Keloslos) • Swollen abdomen (Kutu Muu) • Recumbency (Ruwoy ngwiny)
Mastitis (Semewo Kitin)	• Swollen udder (Kutu krisio) • Mastitis milk/milk that has changed colour (Prochcho/ Marelach nye cho)

Cattle diseases	Main clinical signs
Plant poisoning (Ngwono)	Sudden death (Chemutei) Diarrhoea (Yitoy) Bloating (Musarer)
Heart water (Chemiloy / Chepirpirmot)	• Circling (Kirimot) • Recumbency (Kinyomot) with opisthotonus and leg • peddling (Kityorsho kelyon) • Diarrhoea (Yitoy)
Trypanosomiasis (Plis/Taperwak)	• Emaciation (Changulat / Nyasat) • Slight swelling of lymph nodes (Kutu Nguriel) • Abortion (Torunogh) • Presence of Tsetse flies (Psosoghion)
Photosensitisation (Melmel)	• Alopecia (Ngetata put) • Skin wounds (Moyoi po por) of light coloured areas of • the hair coat (Perperoy). • Pruritus (Ngworoi kegh)
Dermatophilosis / Streptothricosis (Ongeryo menyon)	• Initial mutting /clumping of hair coat (Kisupoto put) • Later formation of skin wounds (Moyoi po por) that form • crusts (Ongeryo moyoy) • Pruritus (Ngworoi kegh)
Babesiosis (Pkison)	• Red urine (Pkison/ Sikororei kison) • Icterus /yellow mucus membranes (Koloswo)

Camel diseases	Main clinical signs
Pneumonia (Luokoi/ Psosoy)	• Respiratory distress (Kisumat / Kirumat) • Nasal discharge (Pkono tingo sorun)
Trypanosomiasis (Plis / Taperwak)	• Emaciation (Changulat / Nyasat) • Offensive milk odour (Ngutoi Cho) • Offensive body odour (Ngutoi Por)

Camel diseases	Main clinical signs
Mange (Simpirion/ Lopadakaa/ Lomitina)	• Alopecia (Ngetata put) • Pruritus/Itching (Ngworoi kegh)
Camel pox (Mokoyon)	• Pox skin lesions (Mokoyon)
Haemorrhagic septicaemia (Saperkut)	• Respiratory distress (Kisumat / Kirumat) • Enlarged Lymph nodes (Kutu nguriel) • Nasal discharge (Pkono tingo sorunoy) • Swollen head (Kutu mot)
Camel orf (Ngirimen/ Ngurmen)	• Mouth (lips) skin lesions (Ngirimen/Ngurmen)
Brucellosis (Somewoche cho)	• Abortion (Torunogh)
Mastitis (Somewo Kitin)	• Swollen udder (Kutukrisio) • Change in milk colour (Marelach nye cho)
Mineral deficiency (Konyomoi)	• Recumbency (Kinyomot)

Goat diseases	Main clinical signs
Helminthiasis (Mochontu muu/ Ngutianta muu / Chepturu)	• Diarrhoea (Kiyitagh) • Submandibular oedema (Kalaplap) • Pot belly (Surtuyo muu)
Infectious Keratoconjunctivitis (pink eye) - (Semewo Konyin)	• Lacrimation (Lokyo Konyin) • Corneal opacity (Terwa)
Peste des Petits Ruminants (Losir)	• Diarrhoea (Kiyitagh) • Nasal discharge (Chakataa Sorun/ • Roponchot Sorunoy) • Salivation (Sumboleloi) • High mortality of young (Wow meghat • nyo po wara) • Fever (Kilay po porr)
Coenurosis (Kapir/ Lopira)	• Circling (Witwitoy/ Poroy marya/ Kirimoto mot)

Goat diseases	Main clinical signs
Contagious Caprine Pleuropneumonia (Luokoi / Psosoy)	• Coughing (Rolsho / Ralat) • Respiratory distress (Kitumot) • Abortion (Toru /Torunot) • Nasal discharge (Chakataa Sorun/ • Roponchot Sorunoy) • Fever (Kilay po porr)
Sheep and Goat Pox (Mokoyon/ Moyoi/ Lomolokoo)	• Skin pox lesions (Kitul-tul Porr) • Fever (Kilay po porr) • Abortion (Turunot/ Torunogh)
Foot rot (Chepkelien)	• Wounds on feet (Moyoi po-Kelion) • Foul smelling feet (Ngutoi Kelion) • Lameness (Ngwoloy/ Ngwalat)
Emaciation and diarrhoea syndrome (Chepkiyiy)	• Diarrhoea Kiyitagh • Emaciation (Nyosotyo / Nyasanat/ • Nyasate)
Mange (Simpirion/ Chesulet)	• Alopecia (Ngetata Put) • Pruritus/itching (Kingwarat)
Contagious ecthyma (orf) (Ngirimen)	• Mouth (lip) lesions (Ngirimen/ Moyoi po kut)

Sheep diseases	Main clinical signs
Enterotoxaemia (Lotuler)	• Sudden death (Meghat Nyopo Lawel/ Muto) • Bloat (Musarer)
Helminthiasis (Pokot- Mochonto muu/Ngutianta muu/Chepturu/ (Magargarek -Sengwer)	• Diarrhoea (Kiyitagh) • Submandibular oedema (Kalaplap)
Pneumonia (Somewo Kaghtan / Psosoy)	• Respiratory distress (Kitumot) • Nasal discharge (Chakataa Sorun/ Roponchot • Sorunoy) • Coughing (Rolsho / Ralat) • Fever (Kilay po porr)
Black Quarter (Tiompo Parak Tiompo Yim Tiompo Ogha)	• Sudden death (Meghat Nyopo Lawel/Muto) • Recumbency (Ruwoy ngwiny)

Sheep diseases	Main clinical signs
Heart water (Chemiloy/ Chepirpirmot)	• Circling (Witwitoy Maria / Poroy Maria) • Blindness (Korotio) • Staggering gait (Wetey lo tapaktapak) • Fever (Kilay po porr)
Peste des Petits Ruminants (Losir)	• Diarrhoea (Kiyitagh) • Nasal discharge (Chakataa Sorun/ Koroponcho • Sorunoy) • Salivation (Sumboleloi) • High mortality of young (Wow meghat nyo po • waraa) • Fever (Kilay po porr)
Plant poisoning (Kawatian/ Ngwono)	• Sudden death (Meghat Nyopo Lawel/ Muto) • Bloat (Musarer)
Abortion syndrome (Turunogh)	• Emaciation (Nyosotyo / Nyasanat / Nyasate) • Abortion (Toru / Torunot)
Foot rot (Chepkelien)	• Wounds on feet (Moyoi Po-Kelion) • Foul smelling feet (Ngutoi Kelion) • Lameness (Ngwoloi / Ngwalat)
Foot and Mouth Diseases (Ng'orion)	• Oral lesions (Moyoi po-kuut), • Foot lesions (Moyoi po-kelien) • Salivation (Sumpalelat) • Lameness (Ngwalat) • Abortion (Toru/Torunogh) • Fever (Kilay po porr)
Mange (Chesulet/ Simpirion)	• Alopecia (Kanget Puto) • Pruritus/itching (Kingwarat)
Bloat (Musarer)	• Abnormal distension of abdomen • (Kutnagh po muu / Lesanat)

Chicken diseases	Main clinical signs
Infectious Coryza (Tasus po konyin)	Swollen head (Kutunagh po Mot) Eye wounds (Moyoi po Konyin)
Fowl pox (Moyoi /Kupey-po Kokoroch)	Red nodules on head and wattle (Kupoy- po kokoroch) Body Wounds (Moyoi po Porr) Itching (King'warat)

Chicken diseases	Main clinical signs
Helminthiasis (Mochontu muu/ Ngutianta muu / Chepturu)	Paleness of wattle/ Anaemia (Kipaytagh po koturionto kokoroch) Presence of worms in droppings (Rumono sko mochonto/ Ngutianta muu/ Chepturu)
Coccidiosis / Bloody diarrhoea (Somewo Kiyitagh/ Kmun / Kaputi Chepareria /Loyita)	Bloody diarrhoea (Yitoy Kison) Sudden death (Muto/ Meghat ta lawel) Drooping wings (Kekatuta /Chundoy Kopepoy/ Lokoy kaputi)
Chronic Respiratory Disease (CRD) (Cheptakat)	• Respiratory distress (Cheptakat / Wiwoy takat) • Coughing (Rolyon / Rolsho) • Sneezing (Kiryonot) • Nasal discharge (Tingon /Tinyon)
New Castle Disease (Konukoi/ Knuch/ Konuktoi/ Ptongoroy)	• Drooping wings • (Kekatuta / Chundoy Kopepoy/ Lokoy kaputi) • High mortality (Wow Meghat) • Greenish diarrhoea (Yitoy cho nyoriloch) • Respiratory distress (Cheptakat/ Wiwoy • takat) • Sneezing (Kiryonot) • Nasal discharge (Tingon / Tinyon) • Sudden death (Muto/ Meghat ta lawel)
Ectoparasite infestation (Kiyominto onto/ Kiyominto menyon) Fleas (Kmit) Lice (Sirr) Ticks (Cheptapalal) Body Mites (Pititi) Leg mites (Ng'atwoi)	• Itching (King'warat) • Paleness of wattle/ Anemia (Kipaytagh po • koturionto kokoroch) • Presence of black dirt on feathers near • anal region (Nyroy sumyon nyo tuw wolo letorongwo)

Chapter 2:

Cattle Diseases

2.1 | Lumpy Skin Disease (LSD)- Lomolokoo

Definition

- LSD is a vector-borne viral disease of domestic cattle and is characterised by sudden appearance of skin nodules especially after the heavy rainy season that is usually accompanied by the presence of large number of biting insects.

Distribution (Epidemiology)

- LSD is endemic in West Pokot county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus)
- LSD in West Pokot has an apparent morbidity rate of 17%, mortality rate of 8% and case fatality rate of 47%
- LSD incidences are more common during the dry season locally known as Komoi (January-March).

Causative agent (Aetiology)

- Lumpy skin disease virus (LSDV) is a large double stranded DNA virus from the family Poxviridae making it stable with very little genetic variability (mutations).
- LSDV shares the same genus with sheep pox virus (SPPV) and goat pox virus (GTPV),
- LSDV is resistant to inactivation and can survive for long periods at ambient temperature in dried scabs for up to 35 days, in air-dried hides for 18 days and in dark contaminated animal sheds for months
- The virus is susceptible to sunlight and detergents containing lipid solvents.

Transmission and Pathophysiology

- Mechanical transmission through blood feeding vectors is the main mode of transmission. The vectors include; biting flies, mosquitoes and ticks.
- The main vector species involved are flies such as Stomoxys, Glossina, Muscidae and Tabanidae. Mosquitoes like Aedes and ticks like Rhipicephalus and Amblyomma.
- Direct contact with saliva and nasal discharge of viremic animals (fever due to virus in the blood) is rare and is considered an inefficient route of transmission.
- Indirect contact with oral and nasal discharge that contaminates feed and water is also a rare and also an inefficient route of transmission.
- Virus can persist in the semen of infected bulls meaning that natural mating or artificial insemination may be a source of infection for females.
- Infected pregnant cows can transmit the virus to the foetus and deliver calves with skin lesions. The virus may be transmitted to suckling calves through infected milk or from skin lesions in the teats.

Long distance transmission

- Unregulated movement of cattle between regions.
- Iatrogenic (intra- or inter-herd transmission) occurs via contaminated needles during vaccination or treatment, if hypodermic needles are not changed between animals or herds.

Affected group

- All age groups affected.
- Exotic breeds (Bos Taurus) more susceptible and develop a more severe disease.
- Young calves, lactating and malnourished cattle develop more severe clinical disease.

Main clinical signs

- Incubation period is 2 to 4 weeks.
- High fever (>40.5^{o}C) that persists for a week or more.
- Enlarged superficial lymph nodes (pre-scapular and pre-crural).
- Sudden appearance of skin and mucosa nodules that are 0.5 to 7 cm in diameter.
- Nodules numbers can range from a few to several hundred.

- Nodules affect the full skin thickness, they involve the epidermis, dermis, subcutaneous tissue and sometimes the underlying muscle.
- Nodules also affect the nasal, oral, ocular and genital mucosa.
- Increased nasal and oropharyngeal secretions is associated with the development of lesions on the muzzle and in the mouth.
- Skin nodules either resolve rapidly, or indurate and persist as hard lumps ('sitfasts') or become sequestrated to leave deep ulcers partly filled with granulation tissue, which often suppurates (discharge pus).
- Systemic reactions such as depression, anorexia, lameness, agalactia, temporary infertility or emaciation also occurs.

Post mortem lesions

- Pox lesions throughout the entire digestive and respiratory tracts and on the surface of almost any internal organ.

Seasonality of occurrence

- LSD outbreaks occur in epidemics several years' apart, outbreak drivers include introduction of sick animal, presence of naïve (not immune) animals and presence of high number of blood feeding arthropod vectors.

Diagnosis

Presumptive diagnosis

- Clinical signs of characteristic skin nodular lesions

Definitive diagnosis

- Laboratory samples to be taken should include- skin lesions and scabs, saliva or nasal swabs and blood in Ethylene Diamine Tetra Acetic acid (EDTA) anticoagulant tubes and clotted blood in Red-top tubes with clot activator.

Differential Diagnosis (DDx)

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

- Pseudo lumpy skin disease/Bovine herpes mammillitis;
- 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 disease has no specific treatment.
- Supportive treatment with broad spectrum antibiotic to prevent secondary bacterial infections.

Active ingredient	Indication	Dosage* and Route*
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM in the hind leg or neck muscles q48 hours PRD
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM PRD Calf:6-10 mL IV or IM PRD
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 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

- Recently recovered animals are immune for only 3 months.
- There is no carrier state - all animals' clear infection.
- Movement restrictions during outbreaks.
- Annual vaccination before outbreak season (Pengat season (June-August) in West Pokot. LUMPIVAX™- a freeze-dried, live attenuated vaccine is the most commonly used vaccine in Kenya. Vaccine confers immunity for 3 years but annual vaccination encouraged due to young immune naïve animals in the herd.
- Conduct regular (quarterly) active surveillance using PDS techniques.

Zoonotic potential

- None (does not affect humans).

Notifiable disease

- Yes, this is a World Animal Health Organisation (OIE) listed disease.
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS).

Further Reading

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http://www.fao.org/documents/card/en/c/1fcf63b0-80e9-4f8e-825f-10ea6e998479/

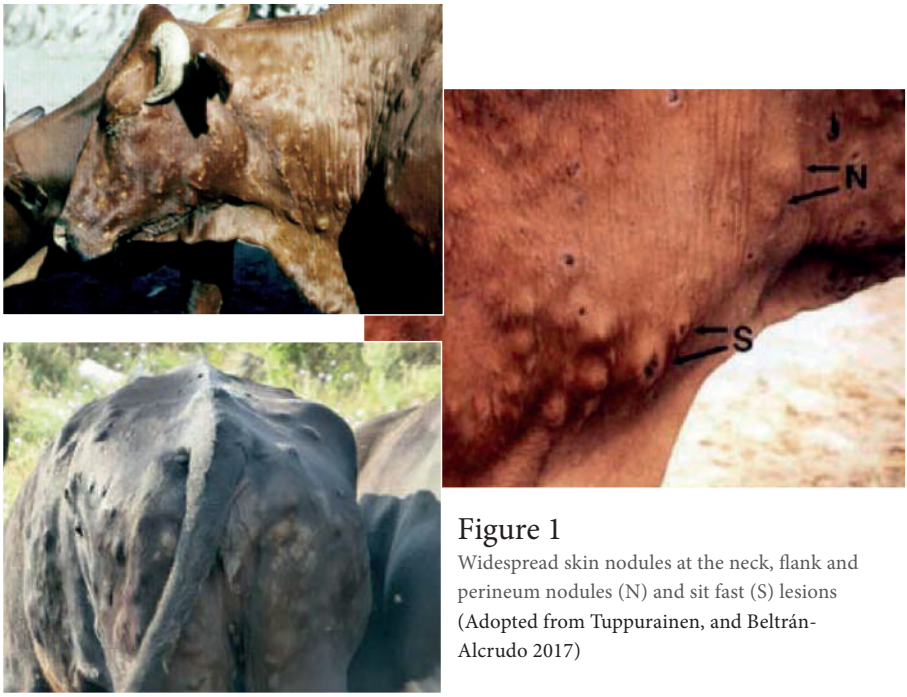

Figure 1
Widespread skin nodules at the neck, flank and perineum nodules (N) and sit fast (S) lesions (Adopted from Tuppurainen, and Beltrán-Alcrudo 2017)

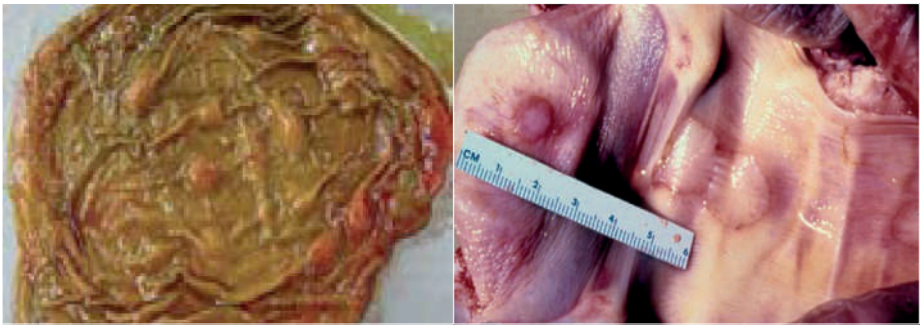

Figure 2
Nodules in the internal organs, bladder (Left) and epiglottis and trachea (Right) (Adopted from Tuppurainen, and Beltrán-Alcrudo 2017)

2.2 | Foot and Mouth Disease (FMD)- Ng'orion

Definition:
- Highly contagious viral disease of cattle, swine, sheep, goats and other cloven-hoofed ruminants.
- Characterised by fever and blister-like sores on the tongue and muzzle, teats and between the hooves.

Distribution
- FMD is endemic in West Pokot County with most incidences reported during the Kitokot light rainfall season that occur during the months of September to December.
- In West Pokot the apparent morbidity rate is 35%, mortality rate 10% and case fatality rate is 29%.

Causative agent/ Aetiology
- Foot-and-mouth disease virus (FMDV) is a single-stranded RNA virus. that belongs to the family Picornaviridae and genus Aphthovirus.
- 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 pH conditions ranges of >6.0 or >9.0.

Transmission and Pathophysiology
Source of infection
- Incubating and clinically affected animals, breath, saliva, faeces, urine, milk and semen (up to 4 days before clinical signs).
- Meat and meat by-products in which pH has remained above 6.0.
- Carrier, recovered or vaccinated animals in which FMDV persists in the oropharynx for more than 28 days. Rate of carriers in cattle vary from 15–50%.

Transmission modes
- Direct contact between infected and susceptible animals.
- Direct contact of susceptible animals with contaminated inanimate objects (hands, footwear, clothing and vehicles)
- Ingestion of contaminated milk (by calves)
- Artificial insemination with contaminated semen
- Inhalation of infectious aerosols- especially in temperate zones
- 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 in 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 and they later rupture within 48 hours. Once the vesicles rupture the healing process begins.
- More than 50% of ruminants that recover from the disease and also those that are vaccinated and have then been exposed to the virus can

become carriers, that is, they have a low level of infectious virus in their pharyngeal region.
- Carrier state can last for up to 3.5 years in cattle, 9 months in sheep, and over 5 years in the African buffalo, which is the main maintenance host of SAT serotypes.
- The risk posed by these carrier animals is low (but not zero) because it has not been possible to transmit the disease (under controlled conditions) from carrier cattle to naive cattle by close contact for extended periods of time. However, transmission of the disease has been achieved from carrier buffalo to cattle and also by direct transfer of pharyngeal fluid from carrier cattle to naive cattle.

Affected group
- All age groups and all breeds of cattle are affected

Clinical signs
- Incubation period of FMDV 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.
- Animal develop high fever of 40° to 41° C.
- They exhibit smacking of lips and careful chewing or grinding teeth due to stomatitis.
- They have excessive salivation with saliva hanging in long, rope-like strings (Figure 5).
- Mouth lesions appear in form of vesicles (1 to 2 cm in diameter) on the buccal mucosa, dental pad and tongue.
- Vesicles are thin walled and easily rupture after 24 hours releasing fluid of watery consistency that is straw-coloured.
- The rupture leaves a raw painful surface that heals in 1 week.
- Feet lesions also appear at the interdigital space and heel of the claws as well as on the coronet band. The vesicle rupture causes 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 from myocardial damage, even when typical vesicular lesions are absent in the mouth or feet.
- Recovery is after 8 to 15 days.

Complications
- Tongue erosions, superinfection of lesions, hoof deformation, mastitis and permanent impairment of milk production, abortion and permanent loss of weight may occur.
- 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."
- Hoof deformation is a common side effect of FMD and may be the cause of the overgrown hoof syndrome reported in West Pokot.

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.

Diagnosis

Presumptive diagnosis
- Clinical signs clinical signs of FMD are indistinguishable from those of vesicular stomatitis in cattle.
- Laboratory confirmation is essential for diagnosis of FMD and should be performed in specialized laboratories like OIE FMD reference laboratory located in Embakasi, Nairobi
- Aging of lesions is an important part of epidemiologic investigation of an FMD outbreak.

Definitive diagnosis
- Laboratory samples to be taken should include-vesicular epithelium or fluid. At least 1 g of epithelium should be placed in a transport

medium of phosphate-buffered saline (PBS) or equal parts glycerol and phosphate buffer with pH 7.2–7.6.
- Samples should be refrigerated or transported on ice.
- If vesicles are not present, oropharyngeal fluid can be collected via probang cup or pharyngeal swab.
- Blood in Ethylene Diamine Tetra Acetic acid (EDTA) anticoagulant tubes and clotted blood in Red-top tubes with clot activator should also be collected.

Differential Diagnosis (DDx)

- Clinically indistinguishable- Vesicular stomatitis

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

Treatment

- Viral disease has no treatment.
- Supportive treatment with broad spectrum antibiotics to prevent 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 hind leg or neck muscles q48 hours PRD
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM PRD Calf: 6-10 mL IV or IM PRD
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 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. Products with this pH range include; sodium hydroxide (2%), sodium carbonate (4%), citric acid (0.2%), acetic acid (2%) and sodium hypochlorite (3%). The products should be used to disinfect contaminated areas;
- Avoid coming into contact with livestock for 3-5 days if one has been exposed to FMD affected herds as humans can harbour FMDV in their respiratory tract for 24–48 hours
- Isolate sick animals and impose movement quarantine in affected areas
- Take samples and type the FMDV serotype involved and vaccinate.
- KEVEVAPI has two types of vaccines
  - Purified oil based FOTIVAX®- annual vaccine
  - FOTIVAX TM- requires 2 or 4 annual boosters to be effective
- Do not purchase new animals during FMD outbreak (wait 6 months)

Zoonotic potential

- 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.
- In West Pokot county, Focus Group Discussions (FGDs) indicated that children under 1 year when they drink milk from a cow with FMD they developed skin lesions on the scalp.

Notifiable disease

1. Yes, OIE listed disease- Report to county and national DVS using ND1 form and enter outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS)

Further Reading

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Figure 3

Profuse salivation (saliva hanging in long ropy strings up to the ground)

Source: https://agritech.tnau.ac.in/expert_system/cattlebuffalo/Foot%20and%20Mouth%20Disease.html

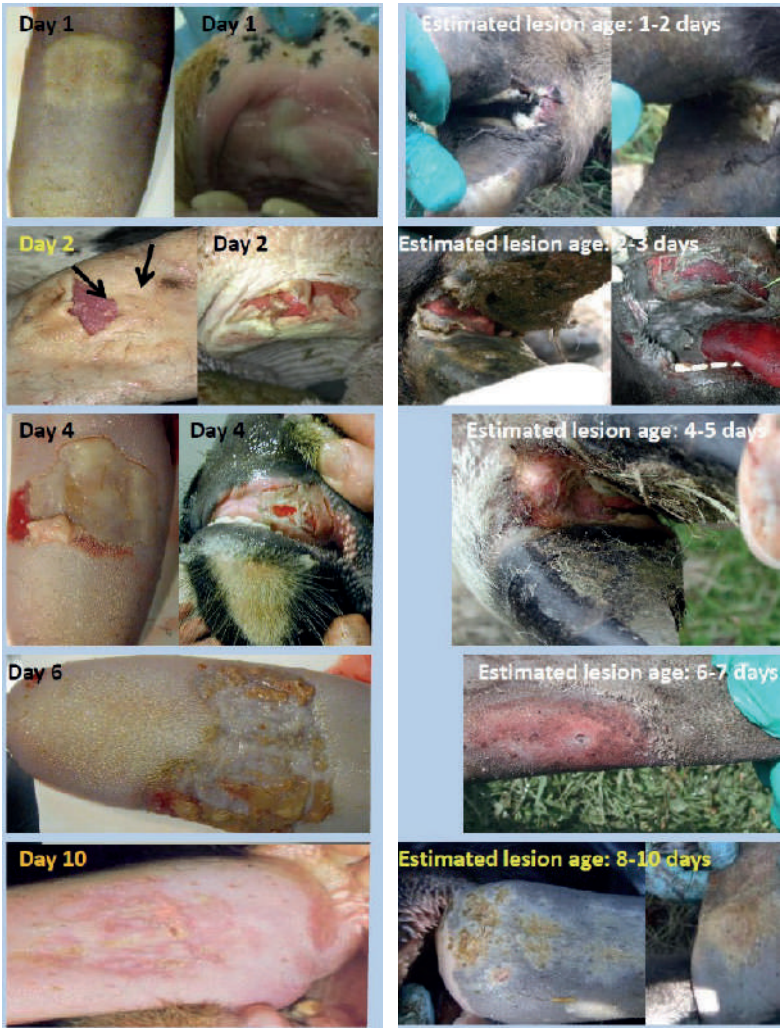

Figure 4

A pictorial guide to estimate the age of FMD lesions

Sourced from: http://www.fao.org/fileadmin/user_upload/eufmd/docs/training/EnglLesionageing.pdf

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.

2.3 | East Coast Fever (ECF)- Yit/ Lokit

Other names: Lokit in Pokot North, Corridor disease, Fortuna disease, January disease, and theileriosis.

Definition:

- ECF is an acute disease of cattle, characterized by high fever, swelling of the superficial lymph nodes, dyspnoea and high mortality.

Causative agent/ Aetiology

- ECF is cause by a protozoan parasite called Theileria parva, an obligate intracellular protozoan parasite that has many strains that vary in virulence.

Distribution

- ECF is endemic in West Pokot county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus)
- ECF in West Pokot has an apparent morbidity rate of 46%, mortality rate of 25% and case fatality rate of 54%
- LSD incidences are more common during the dry season locally known as Komoi (January-March).

Mode of transmission and Pathophysiology

Source of infection

- Bite of an infected Rhipicepalus appendiculatus, the brown ear tick. The saliva carries the infective form of the parasite, the Sporozoites that are found in the salivary glands of infected ticks.
- 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 cattle. When the larva hatches from the egg, it attaches to one animal, feeds (engorges) and drops off the animal onto the ground. It then buries itself and moults into the nymph stage. The nymph finds another animal, feeds on it and again drops off to moult into an adult. The adult attaches to a third animal, feeds, drops off after a few days and lays a large number of eggs on or under the ground. When these hatch into larvae, the whole cycle is repeated.
- 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 infect cattle.
- 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.

Transmission and Pathophysiology

- The brown ear tick picks piroplasms from erythrocytes of infected cattle, in the tick gut lumen lysis of erythrocytes occurs and piroplasms are released.
- Piroplasms differentiate into male and female gametes and undergo syngamy to form a Zygote.
- The Zygote enters gut epithelial cells and develops into motile Kinetes which invade cells of the tick salivary glands and form Sporonts.
- Sporonts undergoes meiosis (sporogony) when the tick feeds on a new host to form Sporozoites (haploid) Sporozoites enter mammalian host through tick saliva.
- Sporozoites then enter the lymphocyte cells and changes the cell from a small round cell with only a little cytoplasm to a bigger cell called the lymphoblast. This causes the lymph node to enlarge, and is said to be “active”. Lymphoblasts differ from lymphocytes as they

repeatedly divide and if a Giemsa stained lymph node smear is made there are large numbers of lymphoblasts. Enlarged parotid lymph nodes are often the first recognizable sign that an animal is infected with ECF.

- The host lymphoblasts enter the circulatory system and burst to release Micromerozoites that enter the red blood cells where they form piroplasms. The percentage of the red blood cells infected is described as the "percent parasitaemia"
- When a tick attaches to the infected cow and sucks blood, the tick becomes infected and the transmission cycle continues (Figure 7).
- 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.
- The contents of the broken cells, which include various enzymes and other chemicals, attack healthy tissues, causing them to break down. This is seen most clearly in the lungs, which leak fluid into the airways. The first symptom of this is that the animal begins to cough. In the advanced stages of ECF the lungs become full of fluid, breathing becomes very difficult (dyspnoea), and finally froth fills the airways and the animal dies of a mixture of choking and drowning.

Affected group

- All age groups and all breeds of cattle are affected.
- Calves rarely develop a 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) (Figure 8).
- Systemic signs that manifest as anorexia that is accompanied by rapid loss of body condition.
- Lacrimation, increased nasal discharge and coughing.
- Terminally there is marked dyspnoea.
- 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 (Figure 9).
- 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.

Diagnosis

- Presumptive diagnosis is based on clinical signs and post mortem lesions as well as history of previous outbreak in the herd, and knowledge vector distribution.
- Laboratory samples include lymph node smear, peripheral blood smear, blood in Ethylene Diamine Tetra Acetic acid (EDTA) anticoagulant tubes 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 (Figure 10).

Differential Diagnosis (DDx)

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

Treatment

Active ingredient	Indication	Dosage* and Route*
Parvaquone or Buparvaquone	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

- Tetracycline though widely used by some livestock keepers in West Pokot County has limited value in treating clinical ECF cases. Tetracycline is mainly used during ECF vaccination to suppress early schizogony multiplication.

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 have low burden of tick infestation. This is especially important for calves.
- 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.

Zoonotic potential

- No

Notifiable disease

- No

Further Reading

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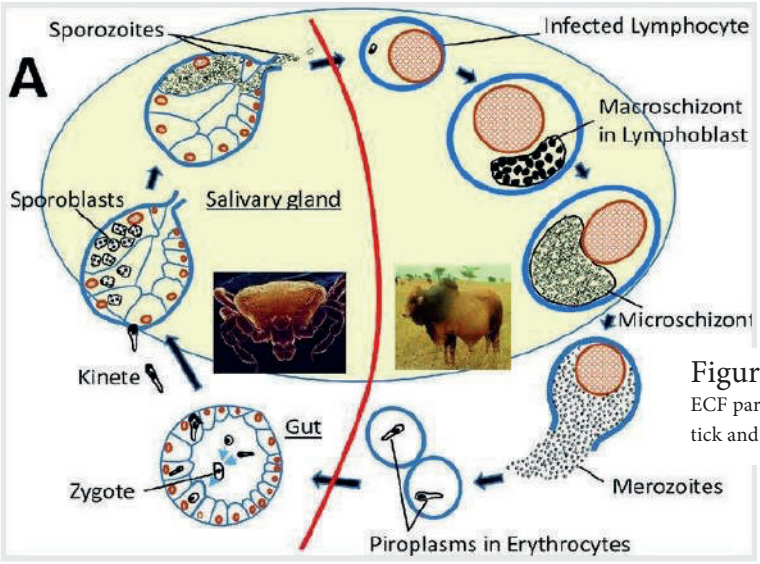

Figure 5
ECF parasite life cycle in the tick and cattle

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

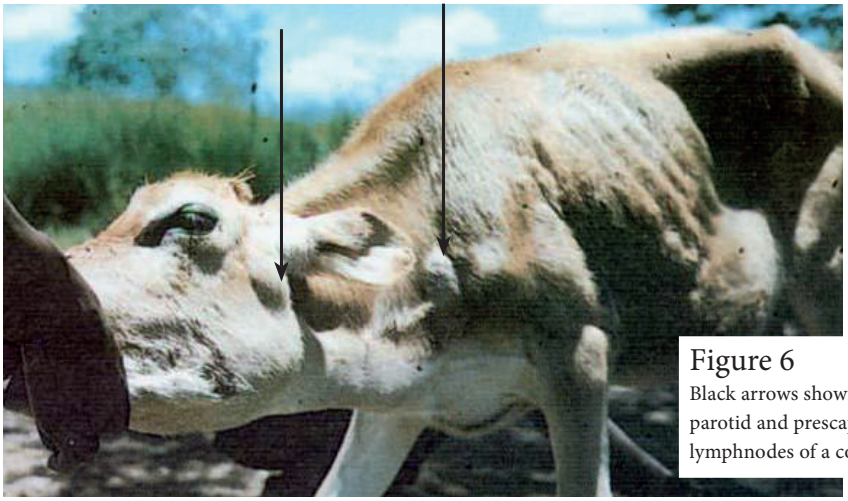

Figure 6
Black arrows showing enlarged parotid and prescapular lymphnodes of a cow with ECF

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

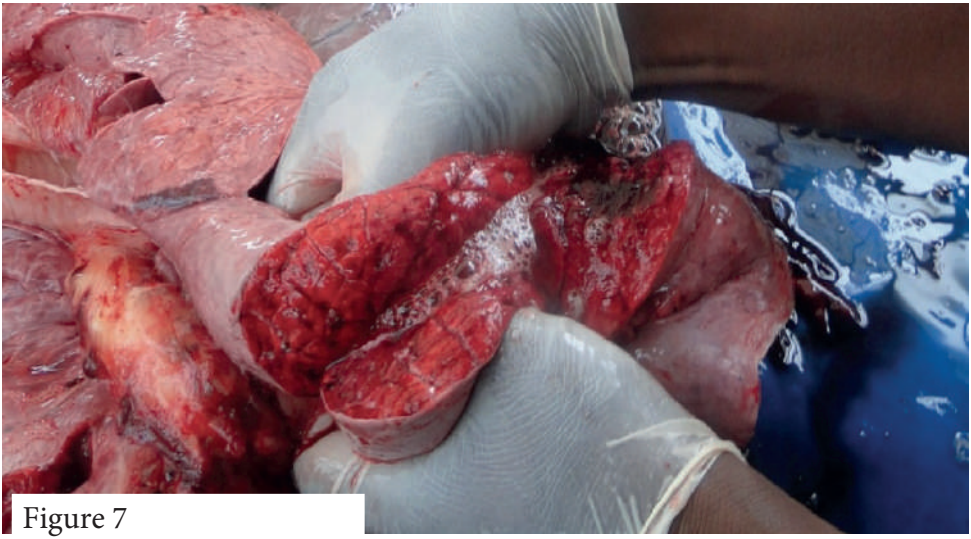

Figure 7

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

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

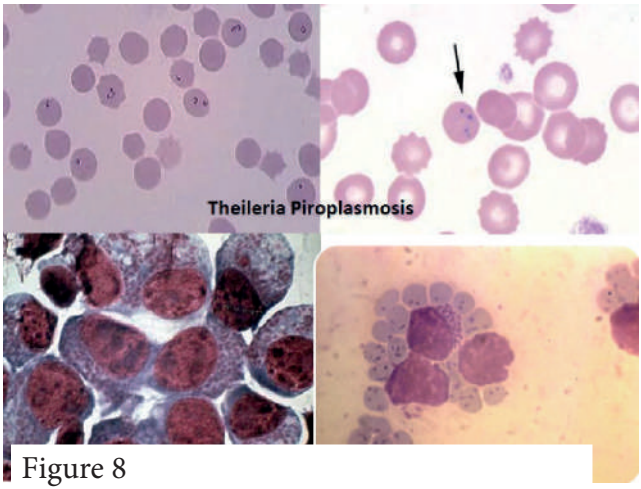

Figure 8

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

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

2.4 | Anaplasmosis- Saak / Toroy

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

Definition

- Anaplasmosis is a disease of ruminants caused by an obligate intraerythrocytic bacteria called Anaplasma. It infects red blood cells of cattle, sheep, goats, buffalo, and some wild ruminants.

Distribution (Epidemiology)

- Anaplasmosis is endemic in West Pokot county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus)
- In West Pokot the disease has an apparent morbidity rate of 19%, mortality rate of 6% and case fatality rate of 32%
- Incidences are more common during the heavy rainfall season locally known as Pengat (June to August).

Causative agent/ Aetiology

- Anaplasma bacteria is from the order Rickettsiales, family Anaplasmataceae, genus Anaplasma. Clinical 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.

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, 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.
- The Boophilus specie is a one host tick, only engorged adult female tick drop off from the host. At any time, the larvae and nymph stages are more numerous than the adult ticks but they are too small to be visible to the livestock keeper.
- 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.

Prepatent Period – is the time between infection with a parasite and the production of eggs by a female; equivalent to the incubation period of microbial infections, but biologically different because the parasite is going through developmental stages in the host.

–

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.
- Depression and anorexia become more obvious as the disease progresses.
- 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.

Extra notes on clinical presentation

- In calves under (<) 1-year-old Anaplasmosis is usually subclinical.
- In yearlings and 2-year-olds the disease is moderately severe.
- In older cattle it is severe and often fatal.
- The main clinical sign is progressive anaemia due to extravascular destruction of infected and uninfected erythrocytes.
- The prepatent period of A marginale is directly related to the infective dose and typically ranges from 15–36 days (although it may be as long as 100 days).
- Rapid bounding pulse or heart rate is usually evident in the late stages of disease
- Urine brown colour contrasts that of blood in urine seen in Babesiosis infections. This is because the infected red blood cells are being destroyed in the spleen. In Babesiosis, red blood cells rupture/haemolyse while in circulation hence the bloody urine.
- Pregnant cows may abort.
- Surviving cattle convalesce over several weeks, during which hematologic parameters gradually return to normal.
- Anaplasmosis frequently occurs at the same time as 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 ECF infection is also present. Whereas, animals with high Anaplasmosis parasitaemia of between 10% and 70% may show milder disease.

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 (Figure 11).
- Liver is enlarged with a yellow-orange discolouration.
- Gall bladder is distended and contains thick brown or green bile.
- Fluid (serous) effusions n the body cavity.
- Petechiae haemorrhages in the epi and endocardium.
- Spleen is enlarged with reddish-brown pulp and enlarged splenic follicles.

Diagnosis

- Samples to be collected include blood in anticoagulant tubes (EDTA) 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 should be taken from the tip of either the ear or the tail of the sick animal.

- Confirmatory diagnosis is made through making thin blood smears stained in Giemsa. Anaplasma species appear as dense, homogeneously staining blue-purple inclusions in the red blood cells that are located towards the margin of the infected erythrocyte (Figure 12).
- Rickettsemia approximately doubles every 24 hours, generally, 10%–30% of erythrocytes are infected at peak rickettsemia,
- Other blood parameters that indicate anaemia like PCV (Normal range 24-46) are severely reduced.

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 like Anaplasma

Active ingredient	Indication	Dosage* and Route*
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg body weight q48 hours PRD
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. A marginale grown in tick cell cultures are being investigated as an alternative to produce live vaccine.
- Sustained and stringent tick control using acaricides is the most common strategy. This is the most common prevention and control strategy used in West Pokot.

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

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).
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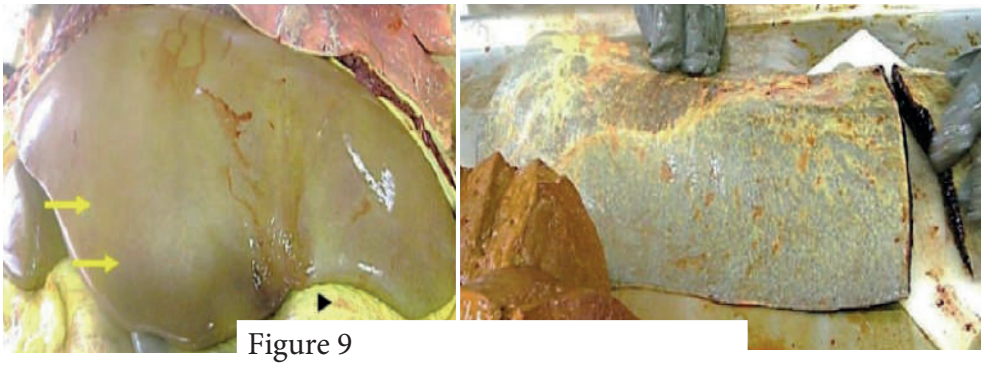

Figure 9
Post mortem Anaplasmosis lesions: Enlarged and jaundices liver (right) and spleen (left)

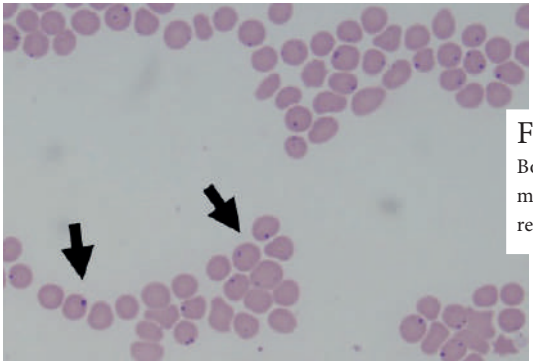

Figure 10
Bovine blood smear showing Anaplasma marginale parasite (black arrows) in the red blood cells

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

2.5 | Contagions Bovine Pleuropneumonia (CBPP)- Luokoi/ Psosoy

Other name: Lung plague

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 West Pokot county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus)
- CBPP in West Pokot has an apparent morbidity rate of 24%, mortality rate of 15% and case fatality rate of 63%
- CBPP incidences are more common during the light rainfall season locally known as Sarngatat (April- May).

Causative agent/ Aetiology

- Bacteria called Mycoplasma mycoides mycoides, small-colony type (MmmSC)
- Mycoplasma bacteria lack cell walls, are 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.

Mode of transmission and Pathophysiology

Source of infection

- Aerosol droplets from cough.
- organism can also be found in saliva, urine, foetal membranes and uterine discharges.
- Fomites are not a major source of transmission.
- 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 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 as CBPP organisms are 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 world animal health (OIE) 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);

Pleomorphic - ability of a microorganism to alter their morphology, biological functions or reproductive modes in response to environmental conditions.

–

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 that is abducted (Figure 13). 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 from 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 both appetite and weight.
- Infected calves under 6 months present primarily with polyarthritis that is seen as swelling of joints and lameness.
- 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. They are often referred to as 'lungers' and are believed to play a role in initiating new outbreaks when they are moved into susceptible herds.
- Infected calves present with poly-arthritis with swelling of carpal and tarsal joints.

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 (Figure 14).
- 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 (Figure 15 and 16).
- 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

- Post mortem lesions are so characteristic and slaughterhouse monitoring is a powerful tool to use in detecting introduction and spread of the disease.
- Co-existence of pulmonary signs in adults and arthritis in young animals should alert the clinician to tentatively suspect 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 claves with arthritis.
- Samples should be shipped cool but may be frozen if transport to the laboratory is delayed.
- 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.

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 twice a day for six days and danofloxacin for 3 consecutive days.
- In West Pokot county, most livestock keepers indicated they used the following antibiotics Penstrep® (Penicillin and Streptomycin), Tylosin and Oxytetracycline. Based on the nature of the pathogen the penicillin preparation will not be effective.
- Multivitamin injection (rarely used in West Pokot county but recommended).
- Antihistamine injection due to severe pneumonia complications (rarely used in West Pokot county but recommended).

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 country is high (> 60%) of animals covered.

Zoonotic potential
- No

Notifiable disease
3. Yes, OIE listed disease
4. Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS).

Further Reading

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2. KEVEVAPI portal - https://kevevapi.or.ke/shop/
3. OIE (2017). Technical Disease Card https://www.oie.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
4. MSD Veterinary Manual - https://www.msdvetmanual.com/

Figure 11
Typical stance of an animal affected by CBPP

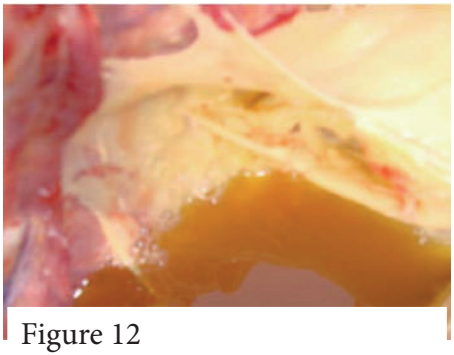

Figure 12
Post-mortem finding of yellow turbid fluid in the thoracic cavity with thick yellow fibrin

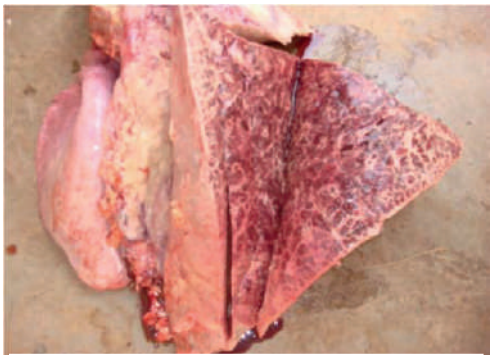

Figure 13
Typical mottled appearance of cut surface of the lung tissue of an animal infected with CBPP

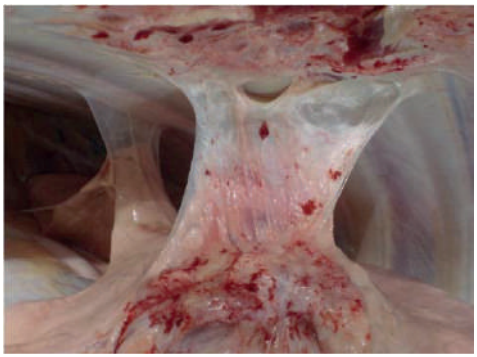

Figure 14
Adherence of lung tissue to the thoracic wall due to CBPP infection

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

2.6 | Bloat – Musarer

Other name: Ruminal Tympany

Definition

- Bloat is a form of indigestion marked by excessive accumulation of gas in the rumen and reticulum forestomaches.
- The gases of fermentation are either in the form of a persistent foam mixed with the ruminal contents (primary or frothy bloat) or in the form of free gas separated from the ingesta, called secondary or free-gas bloat.
- Predominantly a disorder of cattle but may occur in sheep and goats.
- Susceptibility of individual cattle varies and is genetically determined.

Distribution (Epidemiology)

- Bloat in West Pokot has an apparent morbidity rate of 8%, mortality rate of 6% and case fatality rate of 75%
- Bloat incidences are more common during the light rainfall season locally known as Sarngatat (April- May).

Pathophysiology

Primary ruminal tympany or frothy bloat

- Caused by entrapment of the normal gases of fermentation in a stable foam. Coalescence of the small gas bubbles is inhibited, and intraruminal pressure increases because eructation cannot occur.
- Several factors, both animal and plant, influence the formation of a stable foam. Soluble leaf proteins, saponins and hemicelluloses are believed to be the primary foaming agents and to form a monomolecular layer around gas rumen bubbles that has its greatest stability at about pH 6.
- Salivary mucin is an antifoaming agent, but saliva production is reduced with succulent forages.
- Bloat-producing pastures are more rapidly digested and may release a greater amount of small chloroplast particles that trap gas bubbles and prevent their coalescence.
- The major factor that determines whether bloat will occur is the nature of the ruminal contents, specifically protein content and rates of digestion and ruminal passage. All these determine the forage's potential for causing bloat. Over a 24-hr period, the bloat-causing forage and unknown animal factors combine to maintain an increased concentration of small feed particles and enhance the susceptibility to bloat.
- Bloat is common in animals grazing newly sprouted lush pastures and legume or legume-dominant pastures, such as, alfalfa, red and white clovers.

Secondary ruminal tympany, or free-gas bloat,

- Caused by physical obstruction of eructation. Commonly due to oesophageal obstruction due to a foreign body, stenosis, or pressure from enlargement outside the oesophagus as seen in lymphadenopathy. Interference with oesophageal groove function in vagal indigestion and diaphragmatic hernia may cause chronic ruminal tympany.
- Unusual postures, particularly lateral recumbency, are commonly associated with secondary tympany. Ruminants may die of bloat if they become accidentally cast in dorsal recumbency or other restrictive positions in handling facilities and crowded transportation vehicles.

Affected group
- All breeds, however there is genetic susceptibility of individual animals
- Ages of cattle that are grazing.

Clinical signs
- Distended left abdomen (Figure 17) is the most obvious sign and is usually associated with pain, discomfort, and bellowing and frequent urination.
- Bloat commonly begins within 1 hr after being turned onto a bloat-producing pasture. Bloat may develop on the first day after being placed on the pasture but more commonly develops on the second or third day.
- Death can occur within 15 minutes after the development of bloat.
- Sudden death may be the only sign seen.

Post mortem lesions
Characteristic lesions
- Congestion and haemorrhage of the lymph nodes of the head and neck, epicardium, and upper respiratory tract are marked.
- Lungs are compressed, and intrabronchial haemorrhage may be present.
- Cervical oesophagus is congested and haemorrhagic, but the thoracic portion of the oesophagus is pale and blanched (this demarcation is known as the "bloat line" of the oesophagus (Figure 18).
- Rumen is distended, but the contents usually are much less frothy than before death.
- Liver is pale because of expulsion of blood from it.

Diagnosis
- Clinical signs and history of access to lush pasture.
- Passing a stomach tube will distinguish between gassy and frothy bloat. If it's gassy bloat a stomach tube passed into the rumen will allow the gas build-up to escape through the tube.

Differential Diagnosis (DDx)
- The causes of secondary bloat must be ascertained by clinical examination to determine the cause of the failure of eructation.

Treatment
- Passing a stomach tube is the best treatment for gas bloat. Once the gas has been released, the cause of the obstruction should be looked for.
- In a few cases a trochar and cannula punched through the side into the rumen will relieve gasy bloat when a stomach tube has not worked. But such cases are rare, and as the trochar provides a tremendous opportunity for introduction of infection, it should only be used as a last resort.
- The main treatment for frothy bloat is using antifoaming agents that disperse the foam. This should be given via a stomach tube.
- If an outbreak of frothy bloat occurs all cattle on that pasture should be removed immediately and put onto a high fibre diet (hay or straw), and any cows showing bloating signs treated with an anti-foaming agent. The pasture should not be grazed for at least ten days.
- In West Pokot county, antifoaming agents are not used. The use of this agents like Stop bloat™ (Simethicone 10%) should be recommended as most bloat is due to frothy bloat.

Prevention/control

- If possible, avoid using known high-risk pastures at high-risk times (beginning of rainy season when the grass is wet).
- If you have to use high-risk pastures, introduce the cattle to them slowly. In some cases, restricting access to as little as ten minutes per day at the start may be necessary to prevent bloat or fill animals with dry hay or grass pasture before beginning to graze high bloat-potential pastures.
- Administer anti-foaming agents daily if bloat is a severe problem.
- Remove high-risk animals. Some animals have recurrent bloat despite prevention and treatment.
- Practice good pasture establishment and management by mixing legume and grass with legumes providing no more than 50 percent of the available forage.

Zoonotic potential - No
Notifiable disease - No

Further Reading

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

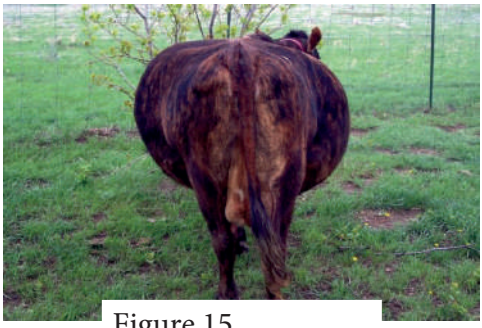

Figure 15
Marked distension of the left abdomen in a cow with bloat

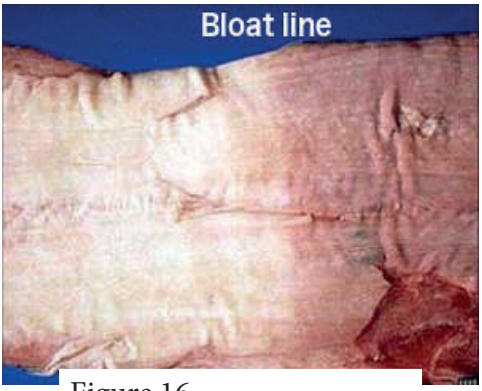

Figure 16
Characteristic oesophagus bloat line necropsy lesion in an animal that died of bloat.

Sourced from:
http://www.vivo.colostate.edu/hbooks/pathphys/digestion/herbivores/tympany.html

2.7 | Black Quarter (BQ)- Tiompo Parak / Tiompo Yim / Tiompo Ogha

Other name: Clostridial myositis; Black leg; Quarter evil

Definition

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

Distribution (Epidemiology)

- BQ in West Pokot has an apparent morbidity rate of 7%, mortality rate of 7% and case fatality rate of 100%
- BQ incidences are more common during the heavy rainfall season locally known as Pengat (June- August).

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 such as alpha toxin in favourable conditions.

Mode of transmission and Pathophysiology

- Cattle and sheep are susceptible as 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 have occurred in cattle on farms in which recent excavations have occurred or after flooding.
- 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

- The animals that contract blackleg are mainly of the beef breed that are in excellent body condition.
- Outbreaks occur in which a few new cases are found each day, sometimes for several days. Most cases are seen in 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 with crepitant swellings. Most common muscles involved are hip, shoulder, chest, back and neck. In some very rare cases, the tongue can be affected and may protrude.
- 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, with decreased blood supply to affected areas.
- Prostration and tremors before death within 12–48 hours.

- In some cattle, the lesions are restricted to the myocardium and the diaphragm. Clinical signs include abnormal breathing and pericardial friction rub.

Post mortem lesions

- Red–brown water leaks from the swellings and it has a rancid smell.
- The affected muscle when it is cut open, looks like a bubbly, black sponge.

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 danger 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 thereafter sets in.

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

2. No

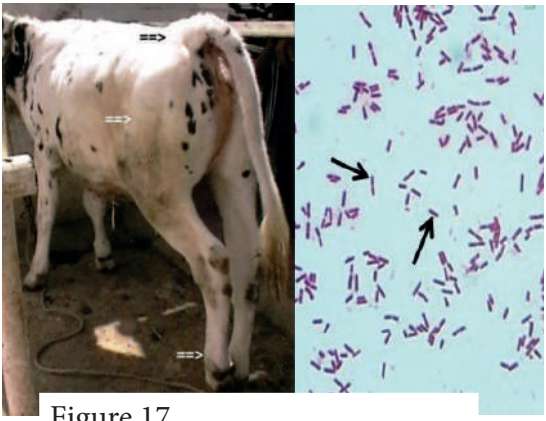

Figure 17

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)

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

Further Reading

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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5074928/#__ffn_sectitle
2. MSD Veterinary Manual - https://www.msdvetmanual.com/

2.8 | Mastitis- Semewo Kitin

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.

Distribution (Epidemiology)
- Mastitis affects both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus), but dairy breeds are more prone to mastitis.
- Mastitis in West Pokot has an apparent morbidity rate of 9% with no fatality reported.
- Mastitis incidences are more common during the light rainfall season locally known as Kitokot (September-December).

Causative agent/ Aetiology
- Most infections are caused by various species of streptococci (or similar gram-positive cocci), 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, contagious spread of pathogens from the cow's environment.

Mode of transmission and Pathophysiology
Source of infection
- Dairy cows are more prone to mastitis because of the larger mammary gland tissue and the frequent handling of their udders thus they are more exposed to more pathogens than that found in a calf's mouth.

Transmission and Pathophysiology
- 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 lose milk production from that quarter due to scar tissue.

Affected group
- All breeds affected however dairy breeds and older beef breeds due to their higher milk production and loosening of the suspensory udder ligaments makes the udder more pendulous hence prone to mastitis.

Clinical signs

- Clinical mastitis main symptom is udder swelling, heat, 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.
- Reduced milk yield and increases 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 the test principles are shown in table below.

Common tests used for mastitis diagnosis

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 is preferred. 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
- In west Pokot county livestock keepers indicated they used Penstrep® (Penicillin and Streptomycin) injection and Milking Salve (Cetridine and Lanolin) to manage mastitis cases.

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

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https://doi.org/10.1155/2020/8831172
2. 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
3. MSD Veterinary Manual https://www.msdvetmanual.com/

2.9 | Plant poisoning- Ngwono/Kawatian

Definition and pathophysiology

- Most poisonous range plants fall into two categories

- Indigenous to the range and increase with heavy grazing and
- Invasive to the range that invade after overgrazing or disturbance of the land.

- Poisonous plants are always present in most range plant communities, so proper range management is important.
- Animals which have been bred in a particular area usually know which plants are edible and which are not. Poisoning in such animals is rare and may only occur if there is hunger due to drought and they have no choice but to consume the plant.
- Most plant poisoning cases in cattle occur when animals are introduced into new grazing areas. Animals will then eat toxic species which they are unfamiliar with.
- Plant poisoning is usually difficult to diagnose. Different plants act in different ways and therefore produce different symptoms. It should be noted that most plants with yellow flowering heads are toxic to stock but, unless the grazing is very sparse, they will avoid eating them.
- Some plants that are generally eaten by animals are poisonous at certain stage of growth and not at others or are only poisonous when eaten in large quantities.

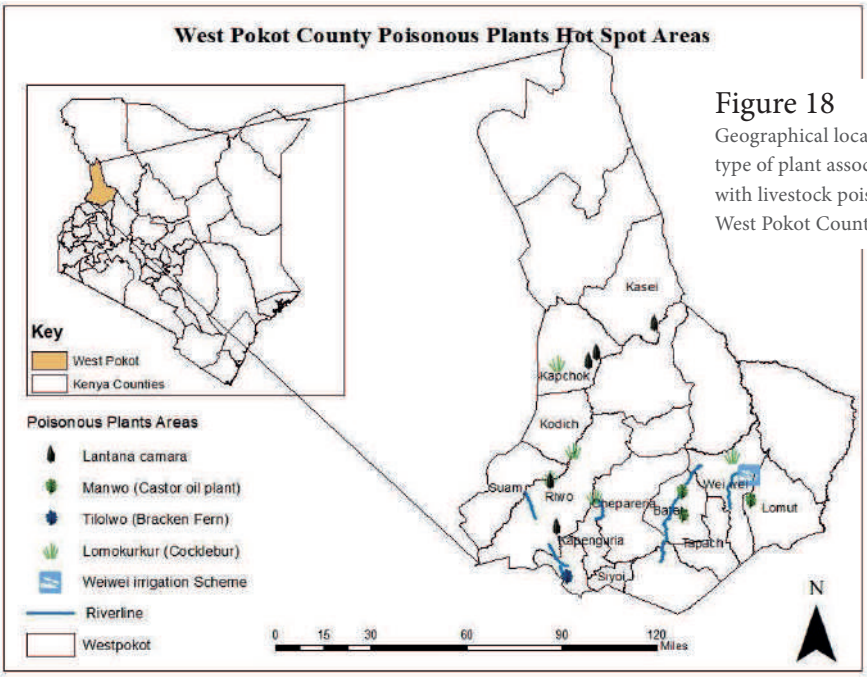


Figure 18
Geographical location and type of plant associated with livestock poisoning in West Pokot County

Distribution (Epidemiology)

- Plant poisoning in West Pokot county is common during the Sarngatat season that occurs between the months of April to May when there is light rainfall.
- In West Pokot county, plant poisoning has an apparent morbidity rate of 14%, mortality rate of 11% and case fatality rate of 79%.

Diagnosis

- Making a definitive diagnosis of plant poisoning is difficult. It is important to be familiar with the poisonous plants growing in the specific area and the conditions under which animals may be poisoned.
- If plant poisoning is suspected often there are signs of gastrointestinal involvement. The gut should be inspected to see if any pieces of plant can be identified.
- The sudden death of newly introduced animals may indicate suspicion of plant poisoning.
- Inspection of grazing areas (transect walk) reveal known toxic plants when several animals are taken ill at the same time.
- In West Pokot county, a tentative diagnosis is made when cattle exhibit clinical signs linked to plant poisoning while grazing in a known area with poisonous plants or when the light rains have caused sprouting of plants that are only poisonous when young or when cattle are introduced to a new grazing area with plants they are unfamiliar with. The main clinical signs relied for diagnosis are sudden death (Chemutei), diarrhoea (Yitoy) and bloating (Musarer).

Treatment

- Oral drench of a purgative, such as Epsom's salt. This may help to remove the toxin from the gut. Dosage is 100grams Epsom salt in half litre of water to sheep and goats and up to 500 grams Epsom salt in 1 litre of water for cattle. Epsom salt makes more water go into the intestines inducing diarrhoea so most of the poison in the intestines mixes with water and comes out as diarrhoea.
- Oral drench of kaolin mixed with water. For cattle give 200grams Kaolin for sheep and goats give 10 grams. Give every day for a 3 days.
- Oral drench of activated charcoal or charcoal powder mixed with water
- Animals which are excited or have muscle spasms may benefit from sedation medication.
- Drug label dosage should be followed to avoid adverse fatal reactions.

Prevention and Control

- Clearing or burning areas invaded by the poisonous plants
- During dry season herder should be more vigilant to ensure animals do not graze on plants that are ever green and poisonous

Clinical signs of common poisonous plants in Kenya

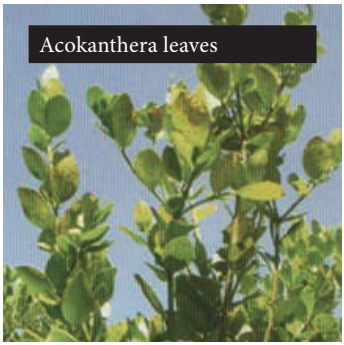
Literature and pictures sourced from: https://infonet-biovision.org/AnimalHealth/Plant-and-Other-Poisoning

1. Acokanthera Schimperi common name arrow poison tree

Used to make arrow poisons and is an ever green tree hence attracts livestock during dry or drought conditions.

Symptoms

Sudden death, some animals show rapid shallow breathing with grunts, diarrhoea, muscular spasms, teeth grinding, profuse, clear nasal discharge. Post mortem reveals gastroenteritis, pale pink lungs distended with air and haemorrhages within the heart.



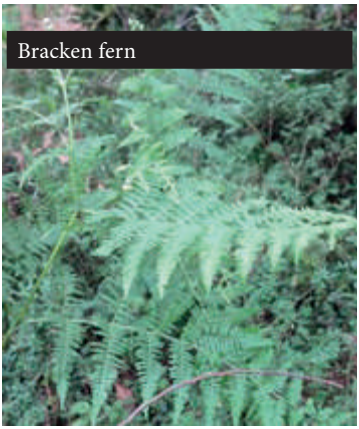
Acokanthera leaves

Acokanthera fruits

Acokanthera flowers

2. Pteridium aquilinum – common name Bracken fern Pokot Name-Tilolwo

Common in high altitude areas. Main symptoms in cattle is cumulative and may require up to three months to develop and sometimes animals have already been moved to a different pasture when symptoms develop. Both the leaves and the roots of the bracken fern are toxic. Symptoms are those of an acute haemorrhagic syndrome, with clots of blood in the faeces, bleeding from body orifices, failure of blood to clot, high temperature, loss of weight, and death with multiple haemorrhages throughout the carcass. One characteristic clinical picture in bracken poisoning in cattle is chronic enzootic haematuria with tumours and haemorrhages in the urinary bladder which ultimately causes death from anaemia.



Bracken fern

3. Ricinus communis- common name castor oil plant: Pokot Name: Manor

Soft woody shrub or small tree up to 20 feet tall, with large leaves- up to 2 feet long and wide- divided into 5 -11 lobes shaped like the fingers of a hand, with small yellowish flowers and seeds shaped like beans of various colours - red, brown, cream and black. Symptoms in cattle include bloody diarrhoea

Castor oil plant
(Ricinus communis)

4. Cestrum auranticum-common name orange or yellow cestrum


Cestrum plant

Flowering shrub/tree with golden yellow pendant blooms and white berries. Its importance lies in the fact that poisoning produces symptoms similar to those of Rabies.
Main symptoms are hypersensitivity to any external stimuli, animals charge with wide open eyes at any moving object. Prior to death there may be frequent bellowing followed by paralysis starting at the hindquarters. Post mortem lesions mainly located in the rumen where there is erosion and detachment of the rumen lining.

5. Datura stramonium- common name night shade Jimson weed, moon flower, mad apple or thorn apple


Datura plant

A shrubby herb, 6 inches to 6 feet tall, with almost hairless stems. It has white trumpet like flowers and black, kidney-shaped seeds. The fresh plant is unpleasantly scented, particularly when bruised, and is therefore avoided by stock.
Signs of poisoning in cattle include paralysis, rapid heart rate and death.

6. Lantana camara –common and Pokot name Lantana


Lantana (Lantana camara)

Flowering shrub with coarse, branched stems and flower heads of various colours, including red, yellow and white. It has an unpalatable taste and is avoided by livestock but during dry season it can be ingested causing photosensitization and diarrhoea.

7. Xanthium Strimariam- common name Cocklebur Pokot name-Lomokurkur


Xanthium Strimariam

Invasive species in West Pokot associated with the recent death of cattle in Konyao, Nakwapa Ngotut, Alakas and Kopeyon.
Clinical signs include sudden death, depression, weakness, anorexia, reluctance to move, opisthotonus, ataxia, hyper excitability, spasmodic muscular activity, and an unusual gait with erect ears and head held high.

Further Reading

1. Infonet biovision website https://infonet-biovision.org/AnimalHealth/Plant-and-Other-Poisoning
2. MSD Veterinary Manual https://www.msdvetmanual.com/

2.10 | Heart water – Chepirpirmot or Chemloi

Other name: Cowdriosis

Definition
- Tick borne rickettsial disease of ruminants common in areas infested by ticks of the genus Amblyomma.

Distribution (Epidemiology)
- Herat water is endemic in West Pokot county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus).
- In West Pokot county, the disease has an apparent morbidity rate of 9%, mortality rate of 5% and case fatality rate of 56%.
- Heart water incidences are more common during the Kitokot light rainfall season that occurs during the months of September to December.

Causative agent/ Aetiology
- Gram negative, pleomorphic obligate intracellular bacterium. Previously known as Cowdria ruminantium now classified as Ehrlichia ruminantium.
- E ruminantium is transmitted by the bite of the Amblyomma three host tick.

Mode of transmission and Pathophysiology
Source of infection
- Tick infects the host via bacterial organisms in the saliva or in regurgitated gut content while feeding.
- Whole blood or plasma of vertebrate host during the febrile reaction, but highest levels of agent occur during the second or third day of fever.
- Colostrum containing infected cells can be passed to new born animals.

Transmission and Pathophysiology
- Transtadial transmission.
- 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 in the body. In domestic ruminants, there is a predilection for endothelial cells of the brain.
- The bont tick is a three-host tick, where every stage (larvae, nymph, adult) of the tick feeds on a different animal. The bont tick get infected when it feeds on infected or carrier animals and then transmits the disease to a susceptible animals with their next feeding on second- or third host.
- Wild animals play a role as reservoir hosts.

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

Clinical signs
- Heart water occurs in four clinical forms 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.
- Acute cases have persistent high fever (41°C), 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 of infection

Post mortem lesions

Due to vascular permeability due to infection of endothelial cells
- Gross lesions in cattle, sheep, and goats are very similar.
- Heart water derives its name from one of the prominent lesions observed in the disease hydropericardium. 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.

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 at necropsy, or in the blood of live animals from just before the onset of the fever to a few days after recovery
- Heartwater can also be diagnosed by observing E. ruminantium colonies in stained (Giemsa) smears from the brain or intima of blood vessels at necropsy.
- 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. Traditionally, this is done with "squash" smears of cerebral or cerebellar gray matter stained with Romanowsky-type stains. Low concentration Giemsa stain developed for 30 min gives the best colour differentiation and batch-to-batch consistency. Organisms in autolyzed material lose their stainability, and diagnosis then becomes difficult.

Differential Diagnosis (DDx)
- Plant poisoning

Treatment
- Oxytetracycline at 10 mg/kg/day, IM, or doxycycline at 2 mg/kg/day . A higher dosage of oxytetracycline (20 mg/kg) is 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 doxycycline, short- or long-acting oxytetracycline, and sulphonamides must

be observed based on local regulations.
- Corticosteroids have been used as supportive therapy (prednisolone 1 mg/kg, IM), although there is debate as to the effectiveness and rationale for their use.
- Diazepam may be required to control convulsions.

Prevention/control

- Vaccination can help with the control of heart water; however, it is neither easily administered nor monitored and gives variable to no cross-protection to the various E ruminantium stocks. The "infection and treatment method" 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 (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). Young calves (<4 wk old), lambs, and kids (<1 wk old) have an innate age-related resistance to heart water, so if challenged by natural or induced infections within this time period, most recover spontaneously and develop a reasonable immunity.
- Control of tick infestation is a useful preventive measure in some instances but may be difficult and expensive to maintain in others. Excessive reduction of tick numbers, however, interferes with the maintenance of adequate immunity through regular field challenge in endemic areas and may periodically result in heavy losses.
- Chemoprophylaxis involves a series of oxytetracycline injections to protect susceptible animals from contracting heart water when introduced into endemic areas while also allowing them to develop a natural immunity.

Zoonotic potential

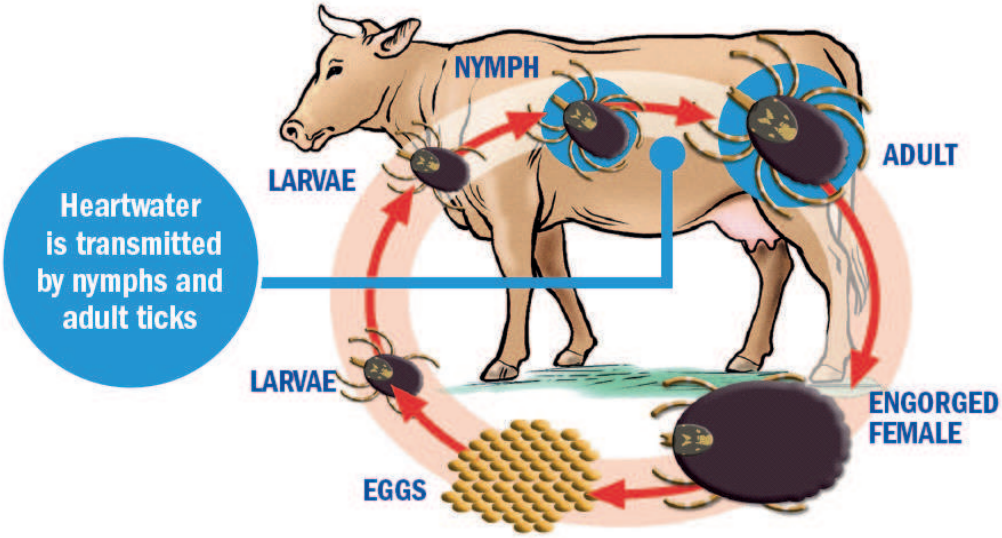
- No

Notifiable disease

- Yes, OIE listed disease
- Report to county and national DVS using ND1 form. Upload outbreak event on KABS

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

Figure 19
Role of Amblyomma tick in Heart water transmission



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

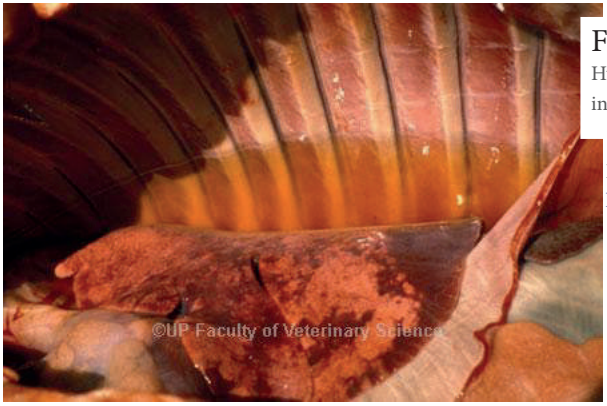


Figure 20
Hydrothorax due to heart water infection in a cattle

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

Figure 21
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

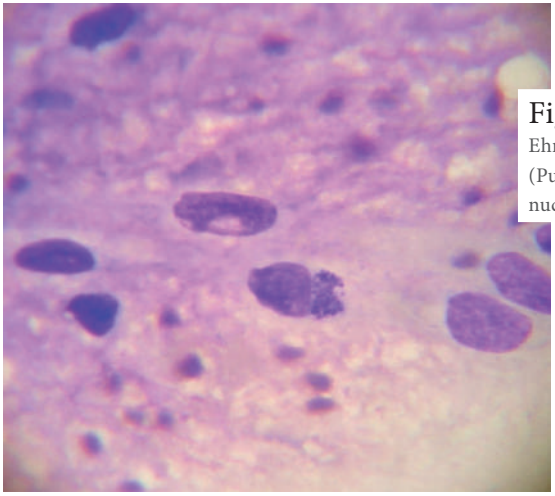


Figure 22
Ehrlichia ruminantium parasite colonies (Purplish staining) near the endothelial cell nucleus

2.11 | Trypanosomiasis- Plis/ Taperwak

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

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 from anaemia, loss of condition and effects on reproduction.
- In West Pokot county, Trypanosomiasis cases are more during the Sarngatat light rainfall season that falls within the months of April to May. The apparent morbidity rate due to Trypanosomiasis in the county is 30% with a mortality rate of 8% and case fatality rate of 27%.

Distribution (Epidemiology)		
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. T. brucei may remain alive for a few hours in blood, and for up to a few days if it is refrigerated under certain conditions (e.g., in blood with adequate oxygen).

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 their lower exposure to tsetse bites.
- Amongst Pokot community herds, trypanosomiasis in cattle is common during the light rainfall season (Sarngatat- April- May).

Morbidity and Mortality rates

- Study estimate a morbidity rate of between 30 and 50% and mortality rate of 6 to 30%.
- In West Pokot county an apparent morbidity rate of 30%, mortality rate of 2% and case fatality rate of 7% has been estimated.

Clinical signs

- The incubation period is usually 1–4 weeks 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 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.

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)

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)
Sourced: https://www.msdvetmanual.com/circulatory-system/blood-parasites/trypanosomiasis#v3258185

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 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

Further Reading

1. Garoma DesaSamson LetaSamson LetaShibiru Debeda Sisay GetachewSisay Getachew (2016). Prevalence of Bovine Trypanosomosis in Gari Settlement Area of Eastern Wollega Zone, Ethiopia. Global Veterinaria 17(2):169-174. https://www.researchgate.net/publication/306066825_Prevalence_of_Bovine_Trypanosomosis_in_Gari_Settlement_Area_of_Eastern_Wollega_Zone_Ethiopia/references

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

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

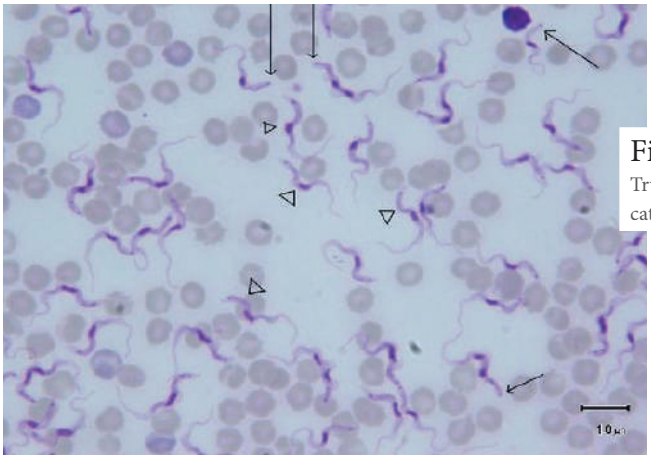


Figure 23
Trypanosome evansi in cattle blood smear

Picture source:
https://www.hindawi.com/journals/bmri/2013/194176/

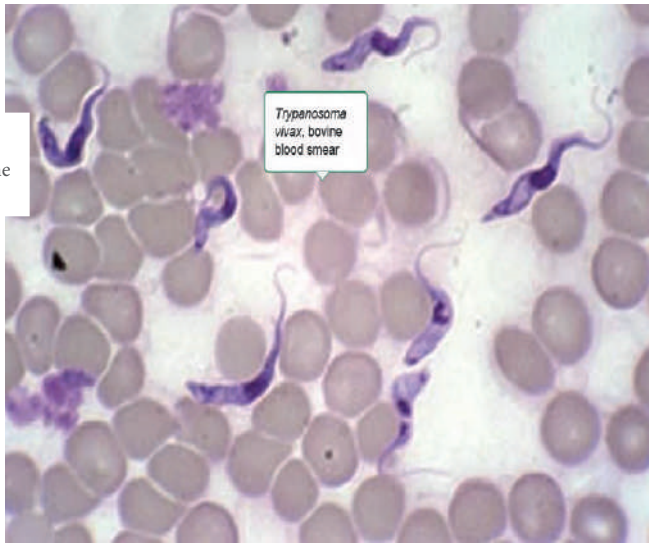


Figure 24
Trypanosome Vivax in bovine blood smear

Picture source:
https://www.msdvetmanual.com/circulatory-system/blood-parasites/trypanosomiasis#v3258185

2.12 | Photosensitisation- Melmel

Definition and pathophysiology

- Photosensitization is a serious skin and sometimes liver condition characterized by "sunburned," crusty white or non-pigmented skin on cattle.
- Caused by photo-reactive plant pigments that the cow has eaten.
- The skin problem does not appear until the animal is exposed to the ultraviolet (UV) rays in sunlight, causing the pigments to damage the non-pigmented skin, which dies and sloughs away.
- There are two general types of photosensitivity problems — primary photosensitization, in which plants contain unique photo-reactive pigments that, when eaten by the animal, are absorbed from the digestive tract into the bloodstream and react in non-pigmented skin areas (white skin areas) when exposed to UV light. This causes cell damage and sloughing of the white-skinned areas,
- Secondary photosensitization When liver damage occurs, however, the problem becomes more severe. "Plant or algae toxins sometimes cause liver damage," says Knight. Once it is damaged by the plant toxins (pyrrolizidine alkaloids), the liver is unable to excrete the various products of chlorophyll metabolism, which include phylloerythrin. Green forage plants generally contain a lot of chlorophyll. "In the healthy animal, phylloerlythrin is absorbed in the circulation that goes through the liver. If the liver is doing its job, it conjugates the phylloerythrin and excretes it in bile, routing it back into the intestines for elimination. This is a much more serious form of photosensitization because [the skin damage] is secondary to severe liver damage. The liver damage or failure can potentially kill the animals even if they don't show photosensitization especially in dark-coloured animals with pigmented skin.

Distribution (Epidemiology)

- In West Pokot county incidences of plant poisoning have an apparent morbidity rate of 14%, mortality rate of 11% and case fatality rate of 79%.
- Plant poisoning incidences are more common during the light rainfall season locally known as Sarngatat (April-May).

Clinical signs

- Non-pigmented skin affected
- Hair loss, reddening, peeling of skin or sloughing off
- Ulceration of skin
- Crusting, bleeding

Diagnosis

- On the clinical signs described above
- Laboratory investigation to rule out liver failure

Treatment

- Removal to cool shaded housing
- Fly control
- Supportive therapy- Antihistamine preparations-
- Treatment of liver failure (if present)

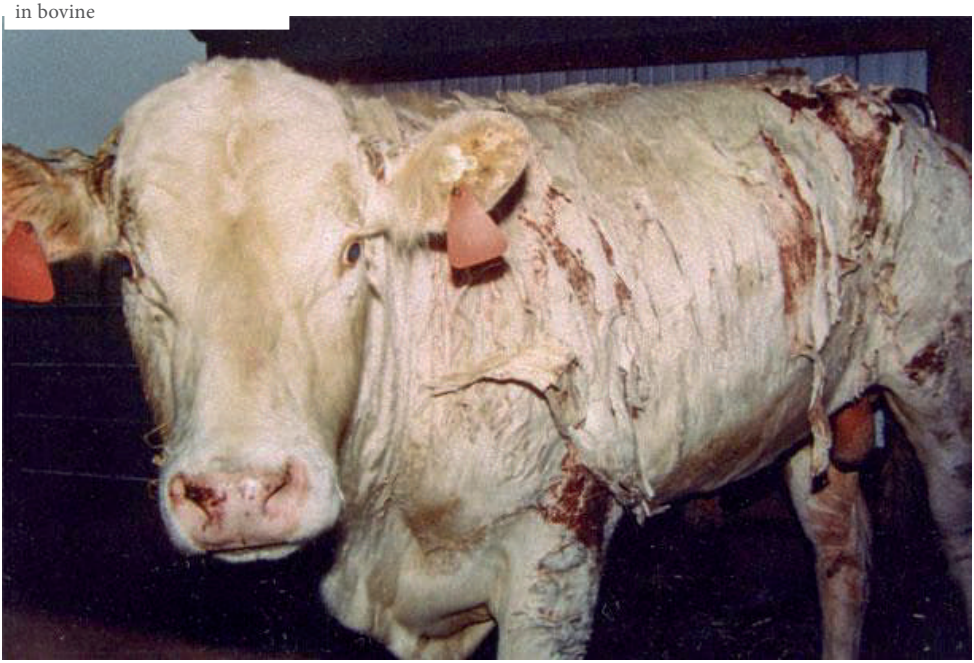
Prevention

- Do not breed from animals with photosensitisation due to a genetic defect Identify
- Remove possible plant sources of photosensitising chemicals

Further Reading

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

Figure 25
Photosensitisation skin lesions in bovine



Picture source:
https://www.progressivecattle.com/topics/herd-health/photosensitization-in-cattle-and-risks-to-the-skin

2.13 | Dermatophilosis- Ongoryo menyon

Other name: Lumpy wool, cutaneous streptothrichosis, rain scald and strawberry footrot

Definition
- Dermatophilosis is a skin disease caused by a bacterium called Dermatophilus congolensis
- Common in young or immunosuppressed animals or in animals chronically exposed to wet condition

Distribution (Epidemiology)
- In West Pokot county dermatophilosis is associated with an apparent morbidity rate of 10%, mortality rate of 1% and case fatality rate of 10%. Most cases of the disease are reported during the dry season locally known as Komoi (January- March).

Causative agent/ Aetiology
- D congolensis is a gram-positive, non-acid-fast, facultative anaerobic actinomycete. It is the only currently accepted species in the genus, but a variety of strains can be present within a group of animals during an outbreak. It has two characteristic morphologic forms: filamentous hyphae and motile zoospores. The hyphae are characterized by branching filaments (1–5 mcm in diameter) that ultimately fragment by both transverse and longitudinal septation into packets of coccoid cells. The coccoid cells mature into flagellated ovoid zoospores (0.6–1 mcm in diameter).
- It is believed to be spread by direct contact between animals, through contaminated environments, or possibly via biting insects.
- It has been isolated only from the integument of various animals and is restricted to the living layers of the epidermis.

Mode of transmission and Pathophysiology
Source of infection
- Asymptomatic, chronically infected animals are considered the primary reservoir
- Factors such as prolonged wetting by rain, high humidity, high temperature, and various ectoparasites that reduce or permeate the natural barriers of the integument influence the development, prevalence, seasonal incidence, and transmission of dermatophilosis. Ticks are major predisposing factors in cattle.
- Introducing an infected animal into a herd.

Transmission and Pathophysiology
- The organism can exist in a quiescent form within the epidermis until infection is exacerbated by climatic conditions. Epidemics usually occur during the rainy season. Moisture facilitates release of zoospores from pre-existing lesions and their subsequent penetration of the epidermis and establishment of new foci of infection.
- To cause infection, D congolensis zoospores must reach a skin site where the normal protective barriers are reduced or deficient. The respiratory efflux of low concentrations of carbon dioxide from the skin attracts the motile zoospores to susceptible areas on the skin surface. Zoospores germinate to produce hyphae, which penetrate into the living epidermis and subsequently spread in all directions from the initial focus. Hyphal penetration causes an acute inflammatory reaction.

Affected group
- All breeds and age groups are affected most common in young calves

Clinical signs

In cattle, lesions observed in three stages:

Stage 1 - Hairs matted together as paintbrush lesions

Stage 2 - Crust or scab formation as the initial lesions coalesce

Stage 3 - Accumulations of cutaneous keratinized material forming wart-like lesions 0.5–2 cm in diameter.

- Typical lesions consist of raised, matted tufts of hair. Lesions distributed over the head, dorsal surfaces of the neck and body, and upper lateral surfaces of the neck and chest.

Diagnosis

- Cytologic examination of fresh lesions or and/or impression smears of the underside of freshly avulsed lesions. Fresh crusts are minced on a glass microscope slide with a sterile scalpel blade in several drops of sterile saline. The slide is allowed to air dry and is then stained with a fast Giemsa stain. The organisms are seen under oil immersion as 2–6 parallel rows of gram-positive cocci that look like railroad tracks
- Confirmatory diagnosis is through Isolation via culture
- An indirect fluorescent antibody technique and a single dilution ELISA test have been developed for large serologic and epidemiologic surveys.

Differential Diagnosis (DDx)

- Warts
- Lumpy skin disease
- Dermatophytosis

Treatment

- Organisms are susceptible to a wide range of antimicrobials: erythromycin, spiramycin, penicillin G, ampicillin, chloramphenicol, streptomycin, amoxicillin, tetracyclines, and novobiocin.
- In food-producing animals, topical applications of lime sulphur are a cost-effective adjuvant to antibacterial therapy.
- Insecticides applied externally are frequently used to control biting insects

Prevention/control

- Keeping animal dry is the main control strategy-
- Isolating clinically affected animals, culling affected animals, and controlling ectoparasites are methods used to break the infective cycle.
- Zinc levels should be checked in the feed of cattle, because outbreaks have been associated with zinc deficiencies.
- No vaccine available

Figure 26
Dermatophilosis skin lesions in an adult cow

Cow with skin lesions on the neck and perineal areas (A &B); clinically ill cow with active skin lesions, focal alopecia and dermatitis on the face and neck (C); matted hair resembling paint brush pulled from the skin lesions (D & E); slightly haemorrhagic and eroded skin surface following the removal of the matted hair (F).

Further Reading

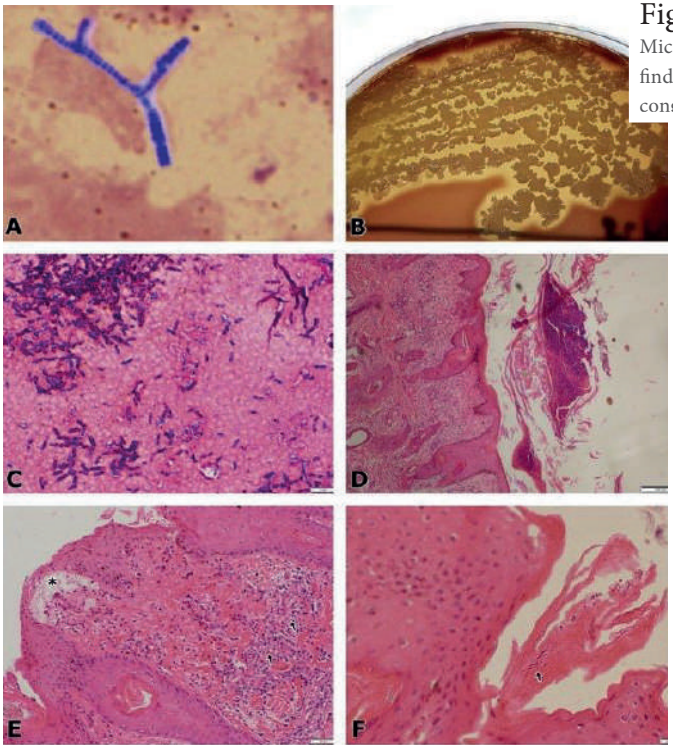
1. MSD Veterinary Manual https://www.msdvetmanual.com/

Zoonotic potential

- Yes, Dermatophilosis can be transmitted to people. Direct contact with an infected animal can lead to infections on the hands and arms. Affected animals should be handled with gloves, and thorough handwashing with an antibacterial soap is recommended after contact with an infected animal.

Notifiable disease

- No



Picture source:
https://www.researchgate.net/publication/339148207_First_report_of_Dermatophilus_congolensis_infection_in_native_Omani_cattle/figures

Figure 27

Microscopic, culture and histological findings in Dermatophilus congolensis infections in bovine

Direct smear from the skin lesions stained with gram stain: gram positive bacteria showing the typical train track morphology of the D. congolensis.
1000x (A); Cultured smear from the skin lesion showing irregular, yellowish-grey , beta hemolytic colonies on sheep blood agar (B); gram positive D. congolensis from culture smear 48 hours post culture (C); skin epidermis showing a multilaminated crust that are heavily infiltrated with neutrophil (D); skin dermis showing micro-abscess with clear fluid and neutrophil infiltrations (asterisk) and mononuclear cells (arrows) (E); skin epidermis showing branching filaments of D. congolensis bacteria arranged in parallel rows (arrow)

2.15 | Bovine Babesiosis (BB)- Pkison

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

Definition

- Babesiosis is caused by intraerythrocytic protozoan parasites of the genus Babesia. Transmitted by ticks. In cattle it is an economically important disease.
- Babesiosis affects a wide range of domestic and wild animals and occasionally people.

Distribution (Epidemiology)

- Babesiosis in West Pokot county has an apparent morbidity rate of 9%, mortality rate of 4% and case fatality rate of 44%. The disease is most common during the dry season locally known as Komoi (January- March).

Causative agent/ Aetiology

- Bovine babesiosis (BB) caused by protozoan parasites of the genus Babesia, order Piroplasmida, phylum Apicomplexa. The principal species of Babesia that causes BB are: Babesia bovis, Babesia bigemina and Babesia divergens. Two important species in cattle—B bigemina and B bovis.
- Agent does not survive outside its hosts and is transmitted mainly through a tick vector.

Mode of transmission and Pathophysiology

Source of infection

- Transmitted by tick vectors Rhipicephalus microplus (formerly Boophilus microplus) and Rhipicephalus annulatus (formerly Boophilus annulatus) where transmission occurs transovarially. Although the parasites can be readily transmitted experimentally by blood inoculation, mechanical transmission by insects or during surgical procedures these routes have no practical significance. Intrauterine infection has also been reported but is rare.

Transmission and Pathophysiology

- In endemic areas, three features are important in determining the risk of clinical disease: 1) calves 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), and 3) the susceptibility of cattle 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. At high levels of tick transmission, virtually all calves become infected with Babesia by 6 months of age and show few if any clinical signs, and subsequently become immune. This situation can be upset by either a natural (climatic) or artificial (acaricide treatment or changing breed composition of herd) reduction in tick numbers to levels such that tick transmission of Babesia to calves is insufficient to ensure all are infected during this critical early period. Other circumstances that can lead to clinical outbreaks include the introduction of susceptible cattle to endemic areas and the incursion of Babesia-infected ticks into previously tick-free areas. Strain variation in immunity has been demonstrated but is not significance.

Affected group

- Cattle from 2 years of age in endemic areas or any age or breed in non-endemic areas.

Clinical signs

- Incubation period is often 2–3 weeks or longer after tick infestation
- B bovis is a much more virulent organism than

B bigemina. With most strains of B bigemina pathogenic effects related more to erythrocyte destruction. However, virulent strains of 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) persists throughout, and is accompanied by muscle tremors, anaemia, jaundice, and weight loss; haemoglobinemia and haemoglobinuria occur in the final stages. CNS involvement 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
- In acute cases: maximum parasitaemia (percentage of infected erythrocytes) in circulating blood is often less than 1%

Babesia bigemina

- Fever
- Haemoglobinuria and anaemia
- Production of dark red or brown-coloured urine
- Nervous signs minimal or non-existent as intravascular sequestration of infected erythrocytes does not occur
- Parasitaemia often exceeds 10% and may be as high as 30%

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

Diagnosis

- Confirmation of 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.
- Other serologic tests to describe low parasitaemia or carrier animals include have been indirect fluorescent antibody test and ELISA for detection of antibodies to Babesia. PCR and real-time PCR assays capable of detecting extremely low parasitaemia, as occur in carrier animals.

Differential Diagnosis (DDx)

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

Treatment

Active ingredient	Indication	Dosage* and Route*
Diminazene aceturate (Berenil™)	Antiprotozoal effective in the treatment of Babesiosis, Trypanosomiasis and Theileriosis	Deep IM or subcutaneous at the side of the neck. 5-10 mL/100 kg body BWT
Imidocarb dipropionate (Imizol™)	Antiprotozoal Effective to offer protection for newly introduced animals for 4 weeks in endemic areas	Strictly IM or SC at the side of the neck 1 ML per 100kg BWT Mixed Anaplasma & Babesia 2.5 ML per 100kg BWT Prophylaxis of Babesiosis 2.5 ML/100kg BWT
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM PRD Calf: 6-10 mL IV or IM PRD
Multivitamin	Boost immunity and red blood components formation to manage the anaemia	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 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

- 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 a 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, OIE listed disease and should be reported to county and national director of veterinary services as well as upload the event in KABS.

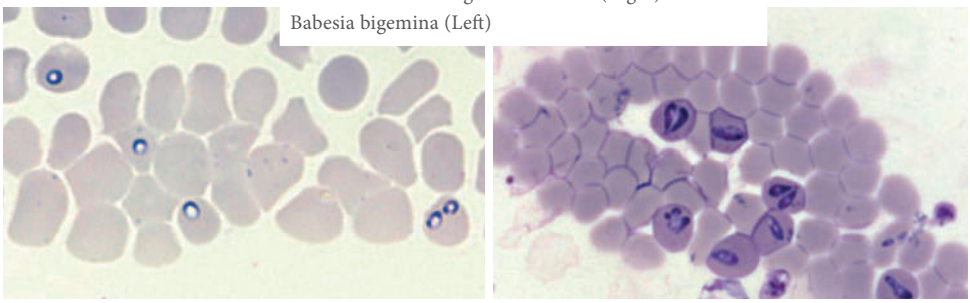
Figure 28

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

Picture source:
https://www.researchgate.net/publication/329416657_A_study_on_bovine_babesiosis_and_treatment_with_reference_to_hematobiochemical_and_molecular_diagnosis/figures?lo=1

Figure 29

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



Picture source:
Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. OIE (World Organisation for Animal Health) ISBN 978-92-9044-804-4

Chapter 3:

Camel Diseases

3.1 | Haemorrhagic Septicaemia (HS) – Saperkut

Other names: Pasteurellosis, Pneumonic Head

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 to move from being normal flora to pathogenic organisms. The bacteria have been isolated from the respiratory tract of healthy and sick animals.

Distribution (Epidemiology)

- HS outbreaks in West Pokot County are common during the heavy rainy season locally known as Pengat between the months of June and August. The apparent morbidity rate for HS in camel herds in the county is 19% with a 7% mortality rate and 37% case fatality rate.

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 serious 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 is primarily found when the resistance of the body is lowered by environmental influences such as transportation over long distances, deficiencies of dietary vitamins and minerals, heavy parasitic infestation (Trypanosomiasis) and sudden changes in weather.
- In other domestic animals, parainfluenza-3 virus are well established as the cause of haemorrhagic septicaemia. In camels, the role of the PI-3 virus is not clear, however, 6.5% seroprevalence of PI-3 virus infection has been reported in camels.
- P. multocida is transmitted by direct contact with infected animals and indirectly through contact with contaminated fomites
- Incubation period vary from 2 to 5 days depending upon the virulence of the organism and the degree of immunosuppression in 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 seldom recover, and death usually occurs within 2-3 days.
- Biting arthropods are not significant vectors.
- Carrier or sick camels can be a source of infection to other animals and in young calves particularly. The introduction of new camels to an existing herd may also spread the disease, but mostly, the disease spreads through droplet infection, or following the ingestion of the organism in contaminated water or feed.

Affected group

- All age groups are equally affected

Clinical signs

- Three different clinical forms in camels peracute, acute, and abdominal forms.
- 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 between the mandibles.
- Mandibular and cervical lymph nodes also become enlarged and painful. Affected camels also show signs of dilated nostrils and open-mouthed breathing. In some cases, there is a 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, cause bronchopneumonia and the lung cut surface oozes exudate. The lung tissue does not collapse when cavity is open

Diagnosis

- Presence of gelatinous fluid in the throat and neck, and fibrinous pneumonia and pulmonary oedema are some of the characteristic lesions used for making a diagnosis.
- Confirmation is to demonstrate the organism; P. 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.
- Blood is collected from the heart within a few hours of death. Other samples include nasal swab. long bone marrow and spleen
- Diagnosis is through bacterial culture and identification of the organism by biochemical, serological and molecular methods.
- Culture of bacteria should be on blood agar, the bacteria grows as moderate sized, round, raised, grayish, nonhaemolytic colonies with a sweetish odour after 24 hours of incubation. The bacterial isolates are not able to grow on MacConkey agar.
- Gram staining of the isolates reveals Gram-negative non-acid-fast coccobacilli.
- HS bacteria is non-motile, urease-negative and catalase-positive. On Triple Sugar Iron (TSI) slopes, the HS bacteria forms a yellow slant and TSI butt has no gas or hydrogen sulphide production. The bacteria also ferment mannitol and sorbitol but is unable to ferment maltose and dulcitol.

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 sets in and recumbency.
- 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.

Treatment

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

- During outbreaks mass medication of affected herd.
- Minimized stress factors.
- Immunization (vaccination) of all camels in the endemic area. 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

- No

Notifiable disease

No

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. MSD Veterinary Manual - https://www.msdvetmanual.com/
3. 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 30

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

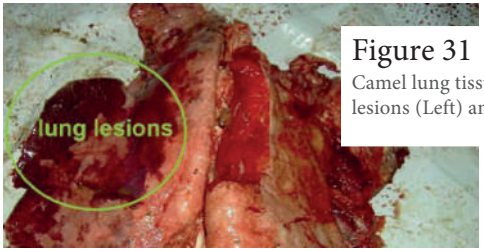
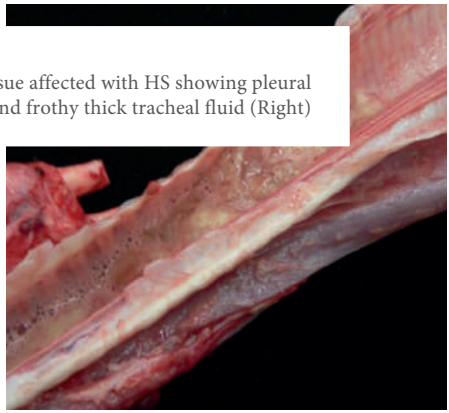


Figure 31

Camel lung tissue affected with HS showing pleural lesions (Left) and frothy thick tracheal fluid (Right)



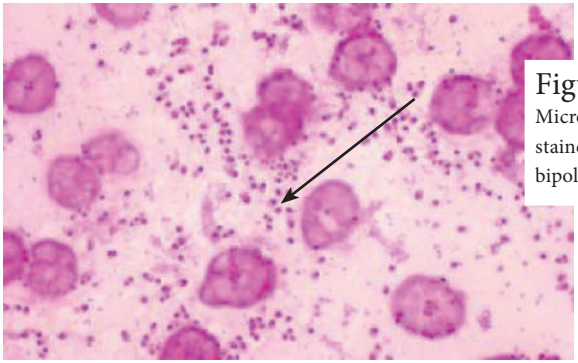


Figure 32

Microscopic analysis HS inflammatory fluid 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

3.2 | Camel Pneumonia- Psosoy

Definition
- Lower respiratory tract infections or pneumonia are considered as emerging health problems in dromedary camels.

Distribution (Epidemiology)
- Camel Pneumonia in West Pokot county is more common during the dry season locally known as Komoi (January- March). The disease has an apparent morbidity rate of 5 %, mortality rate of 2% and case fatality rate of 40%

Causative agent/ Aetiology
- Most common pathogenic bacteria are Staphylococcus aureus, Corynebacterium pyogenes, Streptococcus pyogenes, Escherichia coli, Klebsiella pneumonia, Pseudomonas aeruginosa, Trueperella pyogenes (formerly Arcanobacterium pyogenes), Mannheimia haemolytica and Pasteurella multocida.
- Most common viruses associated with pneumonia in the dromedary camel include parainfluenza 3, adenovirus, respiratory syncytial virus (RSV), infectious bovine rhinotracheitis (IBR; bovine herpes virus-1) and pestivirus or bovine viral diarrhoea virus (BVD).

Mode of transmission and pathophysiology and affected group
- Samples that yield bacterial isolates are nasal, nasopharyngeal, tracheal swabs and lung tissues.
- Samples that yielded viral agents are nasal swabs and lung tissues.
- Studies have shown that the most commonly reported risk factors for bacterial pneumonia are age and season. The highest incidence of pneumonia are 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 more frequent in younger camels (6 months to 4 years of age).

Clinical signs
Bacterial pneumonia
- Moist painful harsh cough, Rhinitis, Congested mucous membranes, Serous or mucoid nasal discharges. Increased respiratory and pulse rates, elevated rectal temperature, Depression, Ruminal atony, Ataxia and decreased milk production

Viral pneumonia
- Fever (41.5^{o}C), Anorexia, Listlessness, Dyspnoea, Hyperaemia of the nasal mucosa, - Nasal and ocular serous discharge

Post mortem lesions
- Lesions in acute pneumonia include- fibrinous bronchopneumonia, oedema and congestion while lesions in chronic pneumonia are characterised by fibrosis, proliferative bronchopneumonia, pleuropneumonia and abscessation. Fibrinous bronchopneumonia usually appears as a gray and red hepatisation. Suppurative bronchopneumonia is characterised by the presence of suppurative exudates in the lumen of bronchioles and peribronchiolar tissues with partial replacement of the bronchiolar wall. Purulent exudates may accumulate focally to form variable sized abscesses.

Diagnosis

- 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.

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 sets in and recumbency.
- 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.
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

- Use of inactivated vaccines: -Vaccination is routinely practiced in endemic Areas. 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 month
- 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

Further Reading
1. M. S. Abubakar, M. Y. Fatihu, N. D. G. Ibrahim, S. B. Oladele and M. B. Abubakar (2010). Camel pneumonia in Nigeria: Epidemiology and bacterial flora in normal and diseased lung. African Journal of Microbiology Research Vol. 4(23), pp. 2479-2483.
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
3. Zuhair Bani Ismail (2010). Pneumonia in dromedary camels (camelus dromedarius): A review of clinico-pathological and etiological characteristics. Journal of Camel Practice and Research. https://www.indianjournals.com/ijor.aspx?target=ijor:jcpr&volume=24&issue=1&article=007

Recent outbreak of Acute Respiratory Disease in Kenya
Clinical signs
- Outbreak started in April 2020, in Camels of all ages and in good body condition. Disease reported in all camel keeping counties in Kenya.
- The outbreak 'spread like bush fire' affecting many herds in an area at the same time.
- First signs were sneezing, mucus discharge from nostrils (first discharge was watery then turned mucopurulent and thick after some days)
- Coughing and laboured breathing.
- Sternal recumbency and death in 3 to 4 days.
- Response to treatment was only successful if administered in the first day of manifestation of clinical signs. Most cases responded to high dose of Penicillin Streptomycin injection.

Post mortem lesions
- Post mortem lesions revealed hydrothorax with straw like fluid, lung adhesions to the rib cage, lung distended with fluid that had mucopurulent consistency.

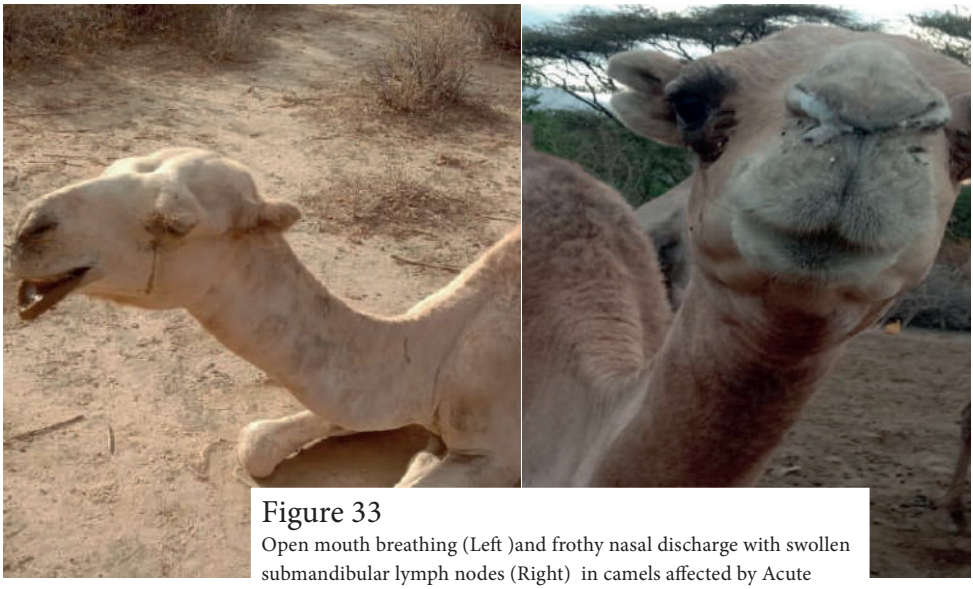


Figure 33
Open mouth breathing (Left)and frothy nasal discharge with swollen submandibular lymph nodes (Right) in camels affected by Acute Respiratory disease syndrome (ARDS) in Samburu County, April 2020

Figure 34
Sternal Recumbency and nasal discharge in a camel affected by ARDS in Samburu County, April 2020

Figure 35
Picture showing hydrothorax and lung adhesions to the rib cage (Left) and exudate of mucopurulent discharge from cut lung surface in a camel affected by ARDS in Samburu County, April 2020

Picture source:
Emmanuel Lesiantam Government Animal Health Assistant based in South Horr, Samburu County-

3.3 | Trypanosomiasis – Plis / Taperwak

Other names: Surra

Definition

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

Distribution (Epidemiology)

- Trypanosomiasis incidences in West Pokot county are common throughout the year but more cases are reported during the Pengat, heavy rainfall season between the months of June and August.
- The disease in the county has an apparent morbidity rate of 28%, mortality rate of 8% and case fatality rate of 29%

Causative agent/ Aetiology

- T. evansi, types A and B,
- T. evansi is morphologically related to T. equiperdum—a derivative of T. brucei.
- During its evolution it lost the mitochondrial (kinetoplast) DNA thus making it lose its ability to infect invertebrate vector and establishing subsequent life-stages like other Trypanosoma 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 (horseflies) 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 haemopoietin system. This exceeds the replenishment of RBC by the bone marrow hence the severe anaemia.
- 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.
- The herd eventually reaches an endemic disease situation. Some animals may carry trypanosomes for years whereas 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
- Any age of camel can get the disease. However, high incidence of infection 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. Later, the appetite is relatively unimpaired and the temperature may become normal or slightly elevated.
- The animal develops a drooping hump and 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 of the base of the neck, ventrum and eyelids, anaemia, intermittent fever, poor milk production, and late term abortion or calving of premature calves.
- Animals subjected to stress, like malnutrition, pregnancy, and physical labour, are more susceptible to disease causing mortality of 3%.

Post mortem lesions
- Carcass is very thin (emaciated) and tissues are often pale (anaemia).
- Subcutaneous oedema is present of the feet, brisket, underbelly and eyelids.
- Lymph nodes and spleen are enlarged.
- Pericardial effusion.

Diagnosis
- Direct examination of the blood by light microscopy is the most readily applied method for diagnosis of trypanosomiasis, this technique can be easily applied in the field. The basic technique, involves examination of fresh or stained blood films and has been modified to improve diagnostic sensitivity by concentrating the blood through centrifugation in a haematocrit tube- haematocrit centrifuge technique (HCT).
- The direct methods of trypanosome detection, utilise a wet blood film, a stained thick drop of blood, and a thin blood smear. The wet blood film is used to detect live parasite through observing parasite movement and 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.
- In chronic cases, antibody detection ELISA is useful for large-scale surveys. Card agglutination and Latex agglutination test are useful antibody testing that can be used in the

field as pen side tests.
- In chronic cases, Polymerase chain reaction (PCR) has also proven useful in detecting trypanosomes in infected blood or tissues.

Differential Diagnosis (DDx)
- Camels other tsetse-transmitted trypanosomosis,
- Anthrax,
- Chronic parasitism- helminthiasis

Treatment
- Two drugs are recommended for the treatment of evansi infections in dromedaries: Quinapyramine, as a sulphate salt and Trypamidium Samorin®. Quinapyramine is marketed as Triquin® . Both drugs are available in Kenya.
- Most of the drugs for cattle trypanosomiasis are either not curative (homidium bromide = Ethidium;pyrithidium bromide = Prothidium) or are toxic at high doses for camels (diminazene aceturate = Berenil).
- However, for the indicated drugs in camels there are numerous documented incidences of resistance to the drugs; Trypamidium Samorin recommended dosage is 12 mg/kg or about 5 g per adult camel, given by slow intravenous injection. Application of drug perivascular (outside jugular vein) results in severe inflammation of vein site (phlebitis). The drug is excreted slowly, which results in some prophylactic drug cover for 12 weeks depending on the dosage and the infection challenge.
- Quinapyramine is easier to use as it is subcutaneously administered. methylsulphate is used as a curative drug, whereas as a mixture of two salts, quinapyramine methylsulphate and quinapyramine chloride at a ratio of 3:2, for prophylactic purposes. Prophylactic cover lasts for 4 to 6 months. Severe overdose causes salivation, muscle tremors, stiffness and collapse or death.
- Supportive treatment with multivitamin and improving nutrition will give the animals a better chance of recovery and survival.

Prevention/control
- No vaccines are available because of the ability of trypanosomes to rapidly change their surface glycoproteins to avoid the immune response.
- Control measures are aimed at the host rather than vector, unlike in Nagana and 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 ons especially during the rainy season when the biting flies' population is high.

Zoonotic potential
- No, not known to have a zoonotic potential.

Notifiable disease
- No in Kenya, recognised by OIE as a camel disease of economic importance.

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.
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2. World Animal Health Organisation (OIE) technical disease cards
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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

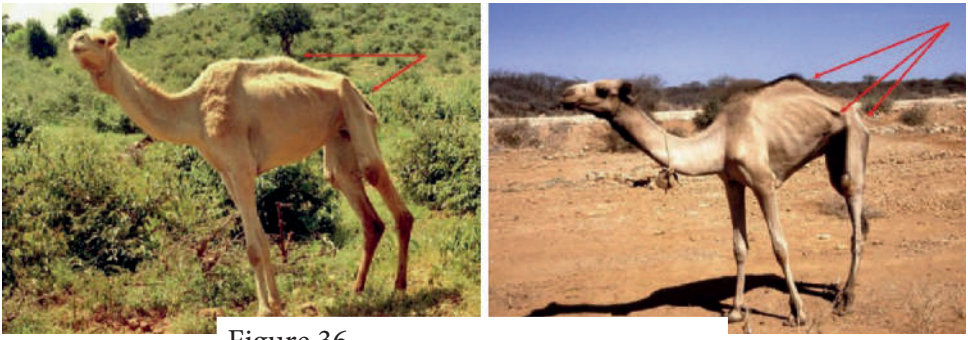

Figure 36
Emaciated camels showing lack of muscle mass, drooping hump, prominent and visible ribs, spine and hip bones (red arrows)

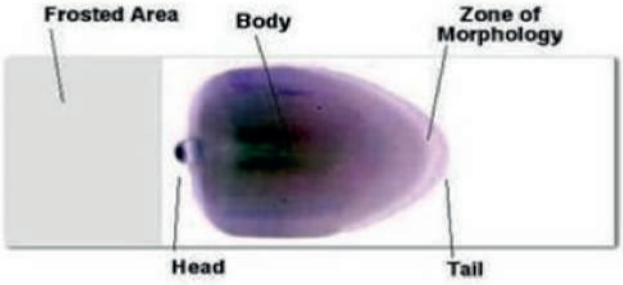

Figure 37
A good blood smear has a head, body and tail

T. evansi has a small sub terminal kinetoplast at the pointed posterior end, a long free flagellum and a well-developed undulating membrane

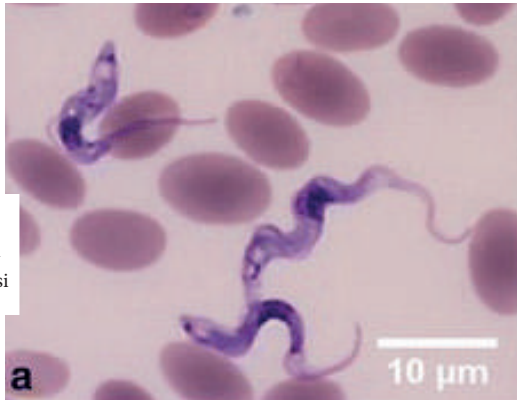

Figure 38
Giemsa-stained blood smears from camel showing Trypanosoma evansi

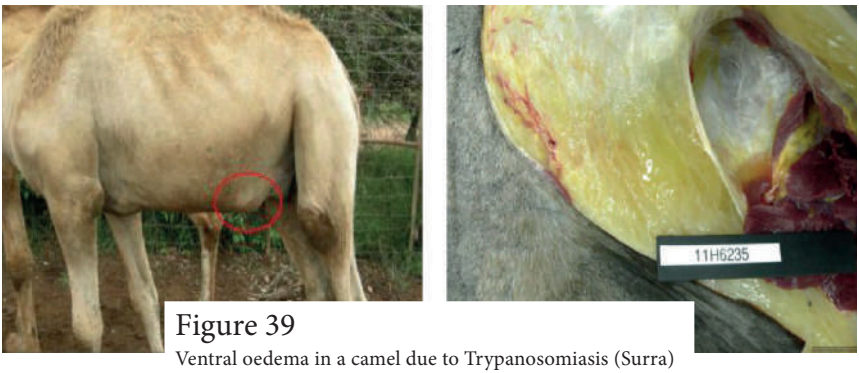

Figure 39

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

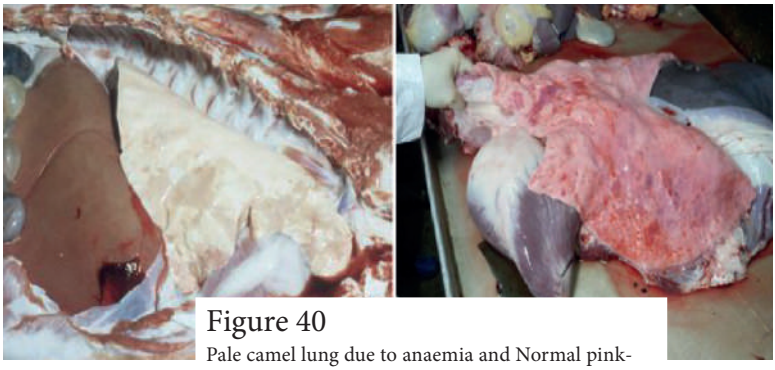

Figure 40

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

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

3.4 | Camel Brucellosis- Somewo cho

Definition
- Brucellosis in camels is an insidious disease that occurs in all camel rearing countries with exception of Australia.

Distribution (Epidemiology)
- Brucellosis in West Pokot county has an apparent morbidity rate of 5% with case fatalities. Livestock keepers indicated it occurs at any time of the year.

Causative agent/ Aetiology
- Brucellosis is a contagious disease caused by bacteria of the genus Brucella.
- Brucella bacteria are Gram-negative coccobacilli that are non-motile and nonspore-forming.
- Camels are highly susceptible to brucellosis caused by Brucella melitensis and Brucella abortus.

Mode of transmission and Pathophysiology
Source of infection
- The infection occurs via the mucous membranes, including oral nasopharyngeal, conjunctival and genital mucosa, and also through cutaneous abrasions. Animals become infected through feed, water, colostrum, contaminated milk and, especially, by licking or sniffing at placentas and aborted foetuses. The spread of brucellosis during sexual activity plays a major role.
- The primary shedding routes of Brucella organisms remain uterine fluids (lochia) and placenta expelled from infected animals.
- Survival of the organisms in the environment is enhanced by cool temperatures and humidity; however, reports indicated in hot dusty environments dust particles mixed with uterine or placental fluids can be carried over short distances (500 metres) and this can be a source of infection.
- Many placental mammals, including herbivores, practice placentophagy, with camelids as a noted exception this may further contribute to the spread of Brucella bacteria through wind.
- In general, abortions occur mainly during the first pregnancy with infected camels having no symptoms.
- Once the bacteraemia phase is over, the bacteria are phagocytised and are found intracellular in mononuclear phagocytes, in which they also multiply. In pregnant camels, the bacteria localise in the placenta, mammary glands and associated lymphnodes.
- Brucella melitensis and/or B. abortus organisms have been isolated from camel milk, aborted foetuses, the placenta, foetal stomach fluid, lymph nodes, vaginal swabs, testes and hygromas during a period of two years.
- Humans are at risk through consumption of unpasteurised milk or through handling Brucella-positive animals. Main clinical signs in man are arthralgia and fever and positive blood culture for Brucella and high serum agglutination titres of 1:60.

Affected group
- Breeding female and male animals, once infected and abortion occurs the animal sheds the organisms without obvious signs in subsequent parturition.

Clinical signs
- History of abortion, presence of hygromas or testicular lesions should indicate Brucella infection in herds.

- Brucellosis is characterised by abortion and to a lesser extent by orchitis and infection of the accessory sex glands in males.
- Infection in breeding camelids causes fewer abortions than it does in bovines and small ruminants
- Infections may cause stillborn calves, retained placenta, foetal death mummification and reduced milk yield.
- It may also cause delayed service age and conception.
- Unlike in bovine and small ruminants retained placenta is rare in Camels. This may be a result of the difference in the placental attachment. Camelids possess a placenta diffusa like the horse and not a cotyledonary placenta.

Post mortem lesions

- Little is known about the pathological changes caused by Brucella organisms in camelids. These bacteria have a predilection for the pregnant uterus, udder, testicles, accessory male sex glands, lymph nodes, joint capsules and in the joint hygromas.
- Studies have found in the non-pregnant dromedaries, there is inflammation of the uterus lining with reddening, oedema and necrotic foci in the uterus epithelium, as well as fibrosis of the endometrium and atrophy of the uterine glands. The is also increased number of ovario bursal adhesions.

Seasonality of occurrence

- Most common during the breeding season.

Diagnosis

- Bacterial culture is the confirmatory test for Brucella infections, however, the organisms grows anaerobically and certain biotypes need a 5% to 10% carbon dioxide atmosphere. Brucella organisms grow slowly, but can be enhanced by using enriched media, such as Ferrell's media supplemented with 5% horse serum and antibiotics.
- Commonly used serological diagnostic tests are Rose Bengal test (RBT), serum agglutination test (SAT) and enzyme-linked immunosorbent assay (ELISA) tests
- PCR has also been used.

Differential Diagnosis (DDx)

- Salmonellosis, trypanosomosis, or infections with Campylobacter or Trichomonas foetus
- An incorrect diagnosis of brucellosis may occur when based on serology alone Some proteins of Brucella are responsible for serological cross-reactions between Brucella spp. and other bacterial species.

Treatment

- Brucella organisms are Gram-negative coccobacilli and are sensitive to many broad-spectrum antibiotics, but the use of antibiotics is forbidden in many countries because of the uncertainty related to the infective status of the treated animals and because of the spread of antibiotic resistance.
- Treatment is unlikely to be cost-efficient or therapeutically effective because of the intracellular sequestration of the organisms, mainly in the lymph nodes.
- Antibiotic regime is not realistic and the only course of action for infected animals is culling out of the herd.

Prevention/control

- Vaccination is the main prevention and control strategy.
- Use of both inactivated and attenuated Brucella vaccines have been done successfully in camels.
- Most vaccine efficacy research has been done on cattle and small ruminants. In cattle, the optimum age for vaccination is between four and eight months of age. Cattle vaccination is recommended only in young stock. Vaccination of bulls with S19 is of no value because it often results in the development of orchitis and the presence of strain S19 in the semen.
- Very little is known about the optimal vaccination age in camels and their serological response. Before vaccination is started in dromedaries, thorough investigations are paramount in order to find if animals are naturally infected by B. abortus or B. melitensis, this can only be determined by culture or PCR. Brucellosis melitensis Rev 1 is an attenuated vaccine and must be carefully used otherwise infections of considerable virulence may occur in both vaccinated and in-contact humans. Vaccination of pregnant goats and sheep may result in abortion and excretion of live B. melitensis vaccine bacteria in milk and vaginal discharge. The situation in dromedaries is unknown.

Zoonotic potential

- Yes, Brucella is the oldest zoonosis known to man.

Notifiable disease

- No in Kenya, however Camel brucellosis is recognised as an important camel disease by OIE.

Further Reading

1. Abdirahman, Dahir B (2020). Camel Brucellosis: Sero-prevalence and Pathological Lesions at Slaughterhouses in Garissa County, Kenya. Master Thesis in the department of veterinary pathology, microbiology and parasitology, Faculty of Veterinary Medicine, University of Nairobi. http://erepository.uonbi.ac.ke/handle/11295/153058

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

3.5 | Camel Mange- Simpirion/ Lopadakaa/ Lomitina

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

Clinical signs
The mite burrows through the skin producing irritation and intense pruritus, causing the camel to itch/scratch. 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.

Distribution (Epidemiology)
Mange in West Pokot county occurs throughout the year but more cases are reported during the heavy rainfall season, Pengat (June to August). The disease has an apparent morbidity rate of 21%, mortality rate of 5% and case fatality rate of 24%

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.

Diagnosis
Mange is easy to diagnose clinically from the pruritus, depilation and encrusted plaques. Only the invasive stage may be difficult to diagnose, but the intense pruritus is characteristic of this parasitosis. The hyperkeratotic stage is easy to recognise by large areas devoid of hair, with thickened skin and folds around the joints, affecting the hind limbs and neck.

Differential diagnosis
Other skin diseases that cause thickening of the skin such as massive tick infestation, mineral deficiencies, recovered pox lesions and ringworm infections.

Laboratory diagnosis
Involving detection of the mite is often difficult. Direct microscopy of a skin scraping (done until the skin starts to bleed) may not always demonstrate the parasite. Better results are obtained by treating the specimen with a heated 10% solution of potassium hydroxide and centrifugation and examination of the deposit for mites.

Zoonotic potential
Yes there have been a few reports as man can be an accidental host. Close contact with infected camels may (rarely) result in the spread of disease to caretakers.

Notifiable disease

No in Kenya.

Treatment

Mange is treated effectively with acaricides like organochlorine (Amitraz®, Tix fix®), organophosphates like Malathion. These are applied by brushing or as a spray, although brushing is used only on the worst and least accessible lesions. Spray treatment must be applied thoroughly to all parts of the body in order to reach mites within galleries burrowed into the epidermis. Treatment should be repeated after an interval of 8-15 days. Also wash fomites like saddles with the acaricide.

• Ivermectin, a macrocyclic lactone is effective against mange two subcutaneous injections, 2 weeks apart combined with acaricide wash results in clinical resolution of signs.

Prevention

From the prophylactic standpoint, it would be desirable to treat the entire herd but this may be impracticable. Priority should be given then to young camels and breeding males.

Further Reading

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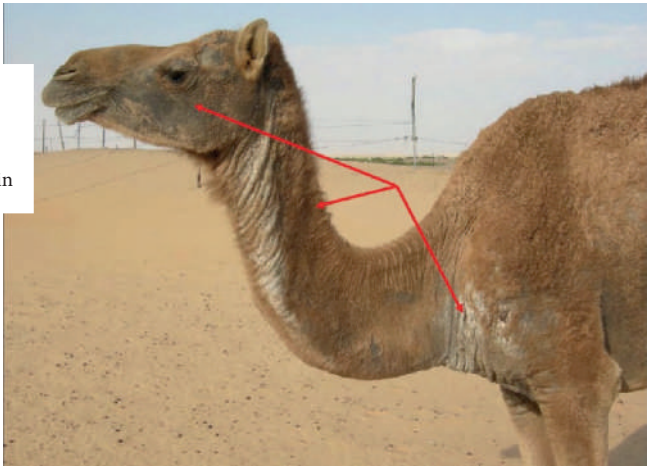

Figure 41

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

Figure 42

Sarcoptic mange Skin scraping showing Sarcoptes scabiei mite

Picture source:
https://doi.org/10.5455/JAVAR.2015.B109

3.6 | Camel Pox- Mokoyon

Definition
Camel pox is an OIE notifiable skin disease. It is highly contagious and has sporadic occurrence.

Distribution (Epidemiology)
Camel pox in West Pokot county is common during the light rainfall Kitokot season during the months of September to December. In the county the disease has an apparent morbidity rate of 9%, mortality rate of 2% and case fatality rate of 22%.

Causative agent
Camel pox virus (CMLV)- CMLV genus Orthopoxvirus (OPV), is genetically similar to variola virus (VARV), 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 Somalia and India in camel handlers who had not received small pox vaccination.

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 contact - 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.

Season of occurrence
Disease occurs frequently during rainy season due to suspected arthropod vectors ticks and biting flies' presence.

Main 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), Urticaria- skin reaction to insect bites.
- Camel Pox and Contagious Ecthyma are contagious viral diseases that affect young

camels. They cause raised crusting skin lesions commonly on the lips and nostrils and often cause oedema and enlargement of regional lymph nodes due to secondary bacterial infection.
- 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 and are not associated with oedema or regional lymph node enlargement.

Post mortem lesions

- In the generalised form the animal external skin nodules and internal pox lesions on the mucous membranes of the mouth, respiratory and digestive tracts;
- Lung tissue pox lesions vary in diameter between 0.5 and 1.3 cm, occasionally up to 4–5 cm. Smaller lesions may have haemorrhagic centres.

Diagnosis

- In the field, clinical signs of pox lesions in all age groups
- Sample Collection- Skin scrapings, thick skin papule biopsies, scabs, aspirates of lymph nodes, vesicular fluids, Whole blood (in anticoagulant) and serum.
- In dead animals, skin lesions, lung lesions (including normal tissue), lymph nodes and other organs with pox lesions.
- Samples must be chilled and transported to the laboratory as soon as possible. Samples must be kept cool at 4°C if stored for a few days or frozen at or below –20°C for a longer period. If there are no ice packs preserve tissues in 10% glycerol/PBS or saline.
- Confirmatory diagnostic tests include antibody detection using ELISA and virus identification through egg or cell culture, molecular techniques (PCR) and virus agent visualisation using a Transmission electron microscopy (TEM) can also be used.

Treatment and Control

- Viral disease there is no specific treatment
- Supportive antimicrobial and multivitamin therapy
- 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 confreres immunity of up to 6 years. However, a booster vaccination is recommended in young animals vaccinated below 6 months as there will be maternal antibody interference.

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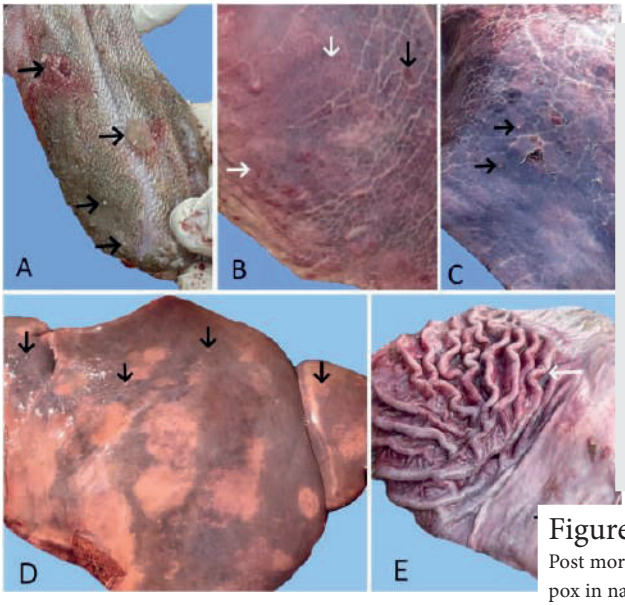
Figure 43

Generalised camel pox virus lesions

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



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 44

Post mortem lesions of systemic form of camel pox in naturally infected adult male dromedary camels in India

Picture source:

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

3.7 | Camel Contagious Ecthyma (CCE/ Camel orf)- Ngurmen / Ngirimen

Other names: scabby mouth, pustular dermatitis

Definition:

- Viral disease characterized clinically by pustule formation on the lips, gums and tongue.

Aetiology, Distribution/epidemiology:

- Caused by pox virus of the genus parapoxvirus (PPV), subfamily Chordopoxvirinae of the family Poxviridae.
- The age group at risk are those less than one year of age including one month-old camel calves.
- Recovered animals do not contract the disease again, which indicates lifelong immunity. The disease is highly contagious, but mortality is rare.
- Orf virus transmitted directly through contact with the scab lesions or indirectly by skin wounded by tree thorns. Other fomites, including contaminated tools, cloth, and animal attendants are also responsible for spread of the virus.
- In West Pokot county, Orf in camels is associated with an apparent morbidity rate of 23%, mortality rate of 12% and case fatality rate of 52%. The disease occurs throughout the year but most cases are seen during the heavy rainfall season locally known as Pengat (June to August).

Zoonotic potential

- ORF virus can infect man through direct contact with infected fomites. It appears as ulcerative lesions on the fingers, limbs, or face of the affected person.

Clinical signs

- Affects mainly immature young camels, producing lesions on the mouth and nostrils.
- However, these lesions may spread to other parts of body.
- In most cases, the disease caused no mortality, but when camel calves are severely affected the lesions interferes with the calves' ability to suckle or graze and extends to eyelids leading to blindness. The camel calves may die due to an inability to graze and suckle their dam.
- Clinically, the pox-lesions first appear on the lips of affected animals as small papules that progressively developed into scabs on the lips, muzzle, nares and eyelids culminating into fissured crusts on the lips.
- Swelling of the head and sometimes the neck has been observed in the field.
- The lesions are proliferative and highly vascularized and may extend into gum, palate and tongue.
- The disease is generally mild and self-limiting. However, in some cases the clinical signs are severe. Clinically, localized lesions at the commissures of the mouth and nostrils characterize the disease; however, generalized lesions have been observed which are similar to camel pox. The affected camel calves become weak and emaciated. Acute disease is characterized by swelling of the head with proliferative nodular lesions around lips, with occasional extension into the mucosa of the mouth and nostrils. The lesions soon convert into pustules and fissured crusts, and then become haemorrhagic and ulcerated due to rubbing and scratching against hard objects and other body parts.
- Papules progress to pustules before becoming encrusted. Finally, the scabs become dark brown and drop off after 6-10 weeks.
- In young camels, lesions sometimes have a tendency to generalize. In this case, lesions

occur all over the body, with prevalence on the distal area of the leg, inner thighs, and the vaginal area. Superficial lymph nodes of the head region such as parotid, submaxillary, and cervical lymph nodes, become enlarged.

Post mortem lesions

- The papule, pustule and scabs are found around mouth, nostrils and on eyelids. Due to intense pruritus, scratching and rubbing of affected parts against hard object results in haemorrhages and extensive skin excoriation. Enlargement of superficial parotid, submaxillary and cervical lymph nodes have been reported due to ecthyma virus.
- In the mouth, mucosa also becomes haemorrhagic and ulcerated.

Diagnosis

- Clinical signs and lesions present on 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.

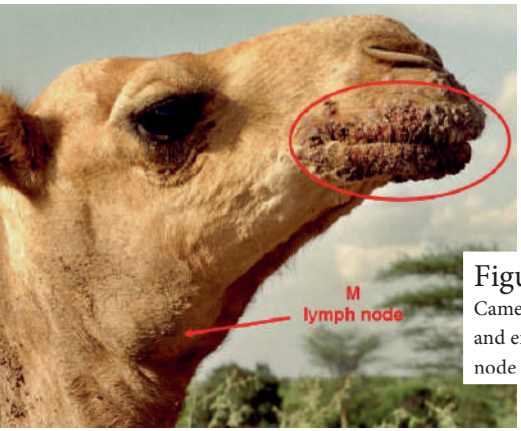


Figure 45
Camel orf oral lesions (red oval) and enlarged sub-mandibular lymph node (red arrow)

Differential diagnosis

- Camel pox
- Mange
- Fungal infections

Treatment

- No treatment since it is viral.
- Local application of antiseptic solution such as providone iodine or iodine ointment on the affected areas is recommended.
- Antibiotic therapy is useful in control of secondary bacterial infection. Anti-inflammatory drugs such as flunixin meglumine are recommended.
- Supportive multivitamin therapy may be required.

Prevention and control:

- Affected animals must be segregated to stop the further spread of the disease.
- Cleaning and disinfecting all contaminated materials and the carcasses of infected animals should be burned.
- 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 for camels.
- A recombinant vaccine in camel poxvirus vector is under preparation as a bivalent vaccine for both contagious ecthyma and camel pox.

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3.8 | Mineral deficiency- Konyomoi

Introduction

- There are few published information on the mineral status of camels in Kenya.
- Macro and micro minerals are essential elements for animal functioning and health. Trace elements such as cobalt, selenium, copper, zinc and iron are integral components of some enzymes and are also involved in many physiological activities.
- Deficiency of trace or micro elements leads to a wide variety of pathological and metabolic defects. On the other hand, macro elements such as calcium, phosphorus, magnesium are important structural components and their deficiency weakens the body immunity and function.
- Clinical mineral deficiencies in camels and their incidence and importance tend to be underestimated because most manifest as sub-clinical diseases that often go unnoticed for extended periods.

Distribution (Epidemiology)

- In West Pokot county, mineral deficiency in camels manifest mainly during the dry season locally known as Komoi (January- March). The apparent morbidity rate due to these deficiencies is 5% with a 1% mortality and a 20% case fatality rate.

Diagnosis

- Serum samples from camel are the preferred samples for mineral analysis. Analysis is done using the Atomic Absorption Spectroscopy (AAS) method, particularly for potassium, sodium, calcium, phosphorus, magnesium, zinc, cobalt and copper while Se, Chrome (Cr) and Mo were analysed using graphite furnace.

Clinical signs

- A 2011 study involving 15,000 camels (1250 camel herds) in different districts of Abu Dhabi Emirate showed that the deficiencies of vitamins (A, D, E, and Vitamin B1) and minerals (selenium, calcium. Phosphorus, copper, iron and iodine) were the most common nutritional diseases of camels in UAE. Racing and young camels were highly susceptible to vitamin deficiency as compared to dairy camels.

Type of Mineral	Deficiency Syndrome
Vitamin A	Presents in three forms Congenital form: Calves are born blind or have multiple congenital deformities likes hydrocephalus or anophthalmos. Postnatal form: Calves poor appetite, reduced the growth rate, true xerophthalmia, (with thickening and clouding of the cornea) and discharge of thin serous mucoid from the eyes. Subclinical form: Mainly in adult camels, characterized by night blindness and loss of reproductive function in both males and females.
Vitamin D	• Osteodystrophic diseases (Rickets and Osteomalacia) • Weakness.
Vitamin E	Two forms of the deficiency were observed. • Acute form: Occurs in calves' and is characterized by sudden death without showing any obvious symptoms. • Subclinical form: Mainly in adult camels. This form is characterized by impaired fertility and a gradual reduction in muscular activity, especially in race camels.
Vitamin B1	Variable signs seen • Disorientation with aimless walking with a high stepping gait due to blindness, • Opisthotonus or head retraction (star gazing), • Muscle tremor and convulsion followed by recumbency with paddling movement and death. • Vitamin B1 (Thiamine deficiency) higher incidence seen racing camels and camels within the age range of 2-4 years.
Selenium	• Poor racing performance, • Stiff gait, lethargy, heart and respiratory disturbances • Reduced fertility in adult camels
Calcium and Phosphorous	• Calves (one year) shows stiff gait, reluctance to move, increase in the size of the joints especially the fore limbs, lameness and sometimes arched back. • Later on and as the condition become more progressive calves show abnormal curvature of the shift of the long bones with an abnormal increase in the depth and width of the epiphyseal plates of particularly the long bones. • General weakness, emaciation and sudden recumbency are the main signs observed in adult camels

Type of Mineral	Deficiency Syndrome
Copper	Two forms seen; Clinical form: • Signs progress gradually and mostly seen in young calves (4-6 months) and are characterized by ataxia and incoordination of the legs, particularly fore legs, during movement of the animal and softening of the bones specially the bones of the foot, followed by leg deformities and poor growth. Subclinical form: • Adult camels affected with variable signs that include general weakness, low milk production, anaemia, temporary infertility. Some animals have hair coat that is rough and has depigmentation.
Iodine	Clinical and subclinical) forms; were diagnosed. • Young camels are usually prone to the clinical form more than adult camels. In this form, calves are either born with goitre or the disease appears in calves 1-2 months old. • Characteristic findings are birth of stillborn or weakness in new-born calves with gross palpable enlargement of the thyroid gland. • Adult camels have subclinical form which is manifested clinically by loss of libido in the male, failure to express oestrus in the female, and high incidence of aborted, stillborn or weak calves.
Iron	• Low serum iron levels (40 ug/100ml) was observed in association with ruminal lactic acidosis, haemorrhagic disease, severe mange, heavy tick infestation, diseases accompanied by fever and trypanosomiasis

Treatment and Prevention

- Provide green or dry ration rich in beta carotene and selenium, especially in the later stage of pregnancy.
- Regular oral doses of copper oxide (4 grams) should be provided for pregnant camels between the fifth and eight month of pregnancy and repeated every two months.
- Provide balanced diets of phosphorus and calcium in the late stage of pregnancy and after delivery.
- Regular supplement of camel farms with mineral blocks.
- Early intravenous treatment with supplement of thiamine is considered as the most important method for the early diagnosis of the disease.

Tincture of iodine should be applied on the flank skin of pregnant camels at least once every two weeks, especially during the last three months of pregnancy.

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Chapter 4:

Sheep Diseases

4.1 | Enterotoxaemia (Pulpy Kidney disease) - Lotuler

Definition:

- Enterotoxaemia also known as pulpy kidney disease due to post-mortem kidney lesions, is a toxaemia caused by abnormal proliferation of a normal flora gut bacteria called Clostridium perfringens Type D.
- Other names – over eating disease and milk colic
- The disease in West Pokot has a morbidity and mortality rate of 23% with 100% case fatality rate. The disease is more common during the Sarngatat light rain season (April to May).

Causative agent/ aetiology

- Clostridium perfringens Type D is a Gram-positive rod shaped bacterium
- Bacteria normally found in the soil and as part of the normal microflora in the gastrointestinal tract of healthy sheep and goats.
- Under specific predisposing conditions, the bacteria can rapidly reproduce in the animal's intestine, producing large quantities of the epsilon toxin.

Mode of transmission and predisposing factors

Source of infection

- Adult sheep carry strains of C perfringens type D as part of their normal intestinal microflora, which is the main source of organisms that infects the new-born lambs.

Predisposing factors include;

- Feeding on green lush pasture or excessive consumption of milk in young lambs especially single lambs not twin (most likely cause in West Pokot)
-Excessive consumption feed with high grain concentration
- Waning of natural immunity seen when animals are ill or recovering from an illness or stressed
- Heavy gastrointestinal parasitism with nematodes, cestodes (tapeworms) and coccidia Can affect gut motility and reduces ingesta movement

Affected group

- Mainly affects sheep of any age and breed. However, the most susceptible age group are nursing and weaned lambs
- Although adult animals are also susceptible to enterotoxaemia, they develop immunity due to frequent exposure to low doses of the toxins

Clinical signs and Pathophysiology

- C. perfringens bacteria proliferates and produces the epsilon toxin in the small intestine.
- The epsilon toxin causes increased vascular permeability and development of oedema.
- Oedema in the brain is associated with neurological sigs.
- Sudden death in lambs that are in the best body condition is the first indicator of enterotoxaemia, these lambs may be found dead or observed to suddenly jump in the air, fall to the ground in a convulsive seizure and die.
- Neurological signs include excitement with increased vocalisation (bleating), running blindly, incoordination, seizures, opisthotonus, circling, and pushing the head against fixed objects.
- Abdominal discomfort manifests as animal repeatedly lying down and getting up, vocalising, and kicking at their abdomen. There

abdomen may also look bloated
- Watery diarrhoea that may be bloody can also occur.
- Elevated glucose in urine (Glucosuria) is strongly suggestive of the disease.

Post-Mortem lesions
- Rapid autolysis of the kidneys has led to the popular term pulpy kidney disease, however, pulpy kidneys are not always found in affected lambs
- Bilateral pulmonary oedema and congestion mainly seen in older animals, but not young lambs (Figure).
- There is hydro pericardium, the increased fluid in the heart sac is clear and has strands of fibrin. The outer surface of the heart (epicardium) and lining of the heart chambers (endocardium) has streaks of pinpoint haemorrhages (Figure).
- A large section of the small intestine can appear dark red to purple (Figure) The intestinal contents are often bloody and full of fibrin clots and necrotic debris.
- The liver is Congested and friable.
- In both young and older animals, the rumen and abomasum contains an abundance of milk or feed. There is also undigested milk or feed in the ileum of the small intestine.
- If helminth burden is the cause, there will be clumps of the helminth in the intestinal tract.
- The season of occurrence is associated with the lambing period and presence of lush green grass.

Diagnosis
- Presumptive diagnosis based on clinical signs of seizures and sudden deaths of nursing or weaned lambs that are in good body condition and post-mortem lesions.

Laboratory
- Make smears of intestinal contents and stain with Giemsa stain to reveal many short, thick, gram-positive rods.
- Confirmation requires detection of epsilon toxin in the small-intestinal fluid. Do not collect ingesta only intestinal fluid. The fluid should be collected in a sterile vial within a few hours after death and sent refrigerated or in a cold pack to a laboratory for toxin identification. If available, Chloroform, added at 1 drop for each 10 mL of intestinal fluid, stabilizes any toxin present.
- Enzyme-linked immunosorbent assay (ELISA) tests are available but studies have found they have inconsistent results.
- A more definitive diagnostic test is the Polymerase Chain Reaction (PCR) assay for detection of epsilon toxin gene in bacteria in intestinal or faecal samples.
- Use of urine dip stick to check for high levels of glucose in the urine (glucosuria) before death and immediately after death. If necropsy is delayed, glucose is rapidly fermented by bacteria in urine and will not be elevated.

Differential Diagnosis (DDx)
- Plant poisoning
- Anthrax
- Black quarter

Treatment
- Treatment of the animals is usually ineffective, due to the rapid progression of the disease in the animal.

Prevention/control

- Vaccination of breeding ewes in their first year of breeding, initial course of two vaccines 4-6 weeks apart when they enter breeding flock.
- Thereafter, annual booster injection done 4 to 6 weeks before which provides passive protection of lambs up to 16 weeks.
- Lambs born to vaccinated ewes should receive a sensitizer dose at 8-12 weeks then second booster at 4 weeks
- KEVEVAPI in 2019 developed a vaccine (Clostivax) that is in the process of being made commercially available
- When enterotoxaemia develops in lambs of nursing ewes on lush pasture immediately restrict ewe access to lush grass to limit the excess milk production and provide grass that is dry and has more roughage. One can also restrict grazing time by letting out the animals to pasture later in the morning.
- During an outbreak restrict the suckling period of lambs especially single born lambs

Zoonotic potential

- No documentation of C. perfringens type D causing diseases in humans
- However, due to the systemic widespread effect of the toxins meat from the carcass of dead animals should not be consumed

Notifiable disease

- No, not listed in OIE

Further Reading

1. KEVEVAPI news
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3. Nazki S, Wani SA, Parveen R, Ahangar SA, Kashoo ZA,Hamid S, Dar ZA, Dar TA, Dar PA (2017) Isolation, molecular characterization and prevalence of Clostridium perfringens in sheep and goats of Kashmir Himalayas, India, Veterinary World, 10(12):1501-1507.
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Enterotoxaemia in Sheep Photo Gallery

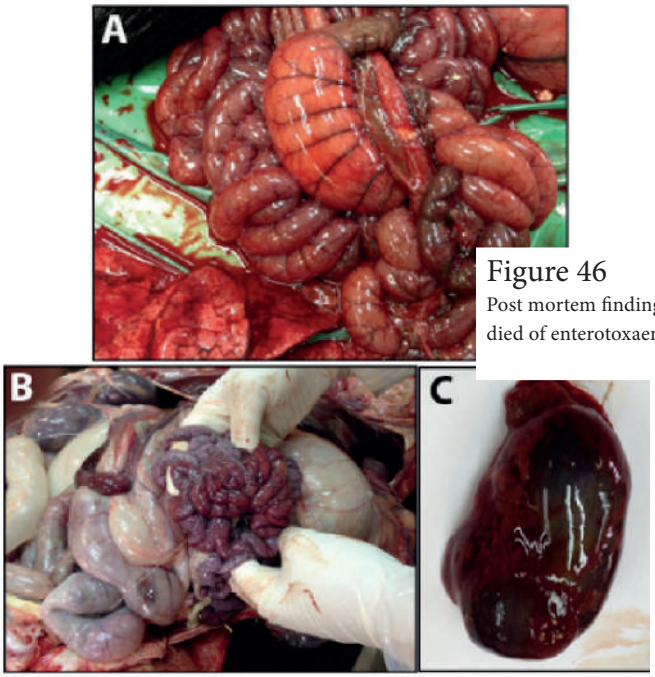


Figure 46

Post mortem findings of sheep that died of enterotoxaemia

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).

Picture source:
http://www.sciencepub.net/nature/ns1108/003_19105ns1108_15_21.pdf

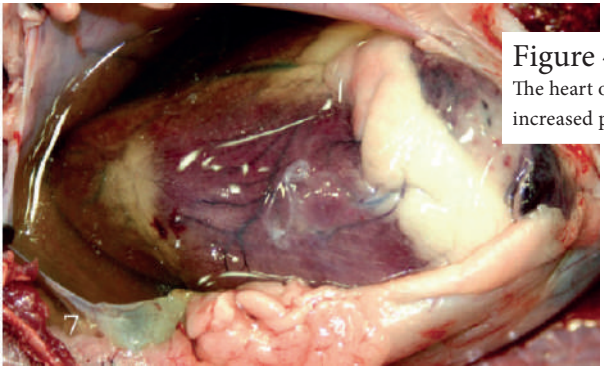


Figure 47

The heart of a lamb showing increased pericardial sac fluid with

Picture source:
https://doi.org/10.1177%2F104063870802000301

4.2 | Helminthiasis - Kawatianta muu/ Ngutianta muu/ Mochonto mu Chepturu (Pokot) Magargarek (Sengwer)

Definition:

- Helminthiasis also known as Internal parasitism or gastrointestinal parasitism is a major cause of diarrhoea and poor growth in sheep production systems.
- The apparent morbidity rate in West Pokot county for helminthiasis is 20% with mortality rate of 11% and a case fatality rate of 55%. The incidence of helminthiasis occur throughout the year but more cases are reported during the Pengat heavy rainy season in the months of June to August.

Causative agent/ aetiology

- Helminths in sheep can be divided into nematodes (round worms), trematodes (flukes) and cestodes (tape worms).
- Nematodes include: Haemonchus contortus, Nematodirus species, Bunostomum species, Teladorsagia (formerly Ostertagia) circumcincta and Trichostrongylus species
- Cestodes include: Monenzia expanza and Trematodes include: Fasciola hepatica (liver fluke)
- In Kenya, helminths species that cause the greatest production losses are Trichostrongylus, Haemonchus, Cooperia, Oesophagostomum, Teladorsagia (formerly Ostertagia) and Fasciola hepatica.
- Haemonchus contortus are important nematode species as they are ferocious blood suckers. The barber pole worm is the largest and deadliest stomach worm.

Mode of transmission
Source of infection

- Consumption of infective helminth larvae during grazing, the larvae on pasture are vulnerable to drying and will not survive long in dry, hot conditions. In contrast, in cool wet conditions, the larvae can survive for longer periods and if suitable conditions persist the larvae can survive for up to six months even after pasture has been rested and animals have not used the area.
- Haemonchus Contortus, better known as the "barber pole" or wire worm is a blood-sucking parasite that pierces the lining of the abomasum causing blood plasma and protein loss to the sheep. A female barber pole worm can produce 5,000 to 10,000 eggs per day. The barber pole worm is also capable of going into a "hypobiotic" or arrested state when environmental conditions are not conducive to its development and resuming its life cycle once environmental conditions improve.

Transmission and pathophysiology

- Nematodes are the most economically important helminth species and most belong to the order Strongylida and family Trichostrongylidae, meaning that the life cycles are similar (Figure)
- The nematode lifecycles are simple and direct with majority having no intermediate hosts, the most of the species eggs are also similar in shape and size
- The eggs are passed in faeces and start to develop (embryonate) immediately at suitable

temperatures (>26°C) and in the presence of sufficient moisture and oxygen will develop and hatch into first-stage larvae (L1) within 24 hours

- The larva moults four times before adulthood, with the free-living stages (L1 to L3) on pasture and the L4 and L5 being parasitic. The cuticle is shed and replaced at each moult except for L3 in which the sheath is generally retained and moulting occurs in the sheep. The larvae are infective to sheep only at the L3 stage.
- Cestodes (tapeworms) and trematodes (flukes) have an indirect life-cycle. This means that there are two animals involved in the development of the infection. The animal harbouring the adult tapeworm is known as the final host, while that harbouring the juvenile stages is the intermediate host for cestodes this is the mite (Figure) and for trematodes the snail (Figure)
- The trematode life cycle begins when the liver fluke eggs are passed in the bile and later in the faeces of affected animal. The eggs hatch in 2–3 weeks to form the free-swimming Miracidia. The Miracidia penetrates the body of an intermediate host (usually freshwater snails) and develop through several stages to form the Cercariae. The Cercariae leaves the intermediate host and swims to grassy vegetation where it forms cysts called Metacercariae, the cyst remain in a dormant stage for 6 months or longer until ingested by a ruminant. Once ingested the Metacercariae activate and penetrate the small-intestinal wall, migrate through the abdominal cavity to the liver where they cause extensive damage to the liver tissue until they reach the bile ducts. Here they mature into adult flukes and start to lay eggs and the life cycle begins again. It takes 10-12 weeks from infestation until eggs start to be laid and shed with the faeces. The liver flukes can remain in the animal for up to 4 years.

Affected group

- All age groups are affected, however young lambs under 1 year are more susceptible as they have not developed immunity

Clinical signs and Pathophysiology
Pathophysiology

- Sheep are more susceptible to helminthiasis when compared to other livestock species because;

- They have small faecal pellets that disintegrate easily thus releasing the worm larvae onto pastures.
- They graze close to the soil surface and thus ingest infective larvae more readily
- The lambs acquire immunity to parasites more slowly as they take 10 to 12 months
- Breeding ewes suffer a temporary loss of immunity around the time of lambing resulting in periparturient egg rise

Clinical signs

- Anaemia- characterized by pale mucous membranes, especially in the lower eye lid; and "bottle jaw," an accumulation (or swelling) of fluid under the jaw especially due to Haemonchus
- Enteritis with a distinctive dark-coloured diarrhoea, Trichostrongylus is probably more often important in combination with Haemonchus and other nematodes in a general parasitic gastroenteritis. Diarrhoea (scours)
- This worm lives in the large intestine where it causes formation of nodules. Both the nodules and the worms themselves are large enough to be seen at slaughter, but the occurrence of large numbers of hardened nodules is diagnostic. Apart from the loss of value in matumbo, large infections of Oesophagostomum can be a cause of scouring.

- Helminths of sheep result in varying degrees of morbidity. Of pathological importance are hemonchosis caused by Haemonchus contortus (a roundworm) and liver flukes caused by Fasciola hepatica.

Haemonchosis
Pathophysiology
H. contortus is transmitted horizontally through grazing by third stage (L3) larvae. It has a two-phase life cycle, a free-living phase and a parasitic phase within the abomasum of the host. The eggs reach the ground through faeces of infested ruminants, infecting herbage. The eggs then evolve to first stage larvae (L1), continuing to second stage larvae (L2), then to its parasitic form, third stage larvae (L3). After L3 larvae reach the ruminant, they migrate to the abomasum, their predilection site, where they become adult nematode parasites, expressing their blood-sucking activity, which is the primary cause of anemia and occasionally, consequentially death. The parasites also produce a hemolytic factor that causes distinct changes on the surface of affected erythrocytes.

Clinical signs
Helminths in sheep cause varying degrees of morbidity depending on the infecting species, host age and host immunological status. The disease is more severe in lambs than in adult animals.
Heavy helminth infestation causes enteritis resulting in hemorrhage of the intestinal mucosa, diarrhea and dehydration. The key features of hemonchosis are sub-mandibular oedema and anaemia.

Liver fluke disease
Pathophysiology
Acute liver fluke disease is related to the damage caused by the migration of immature flukes which leads to liver inflammation, hemorrhage, necrosis, and fibrosis. Chronic disease may result from fluke-induced physical damage to the bile ducts and cholangiohepatitis. Blood loss into the bile may lead to anemia and hypoproteinemia.

Clinical signs
Severe liver fluke infestation results in anaemia, sub-mandibular edema and may be fatal. Other signs of liver fluke disease include anorexia, weight loss, unthriftiness, edema, and ascites.

Zoonotic potential
- No however, finding of a current study confirms the zoonotic aspect of Trichostrongylus species and T. circumcincta. The occurrence of natural human infection by T. circumcincta has been reported for in Iran.

Notifiable disease
- No

Diagnosis	Treatment and control
Laboratory sample to be collected: Faecal sample worm egg count, identification of worm eggs and larvae	The choice of which anthelmintic and when they should be used is a question of balance between the necessity for treatment or prevention, the cost in terms of economics and the potential for the development of anthelmintic resistance. Broad spectrum anthelmintics (benzimidazoles, levamisole, ivermectin) may be used for treatment of helminthiasis in sheep. Strategic deworming which involves deworming sheep with high worm loads and annual rotation of dewormers, i.e, changing the class of dewormer used every year to prevent development of resistance. Grazing management, specifically, rotational grazing is also an effective method of controlling helminthiasis.

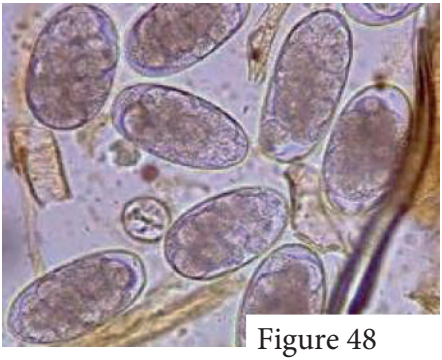


Figure 48
Strongyle egg species

Figure 49
Nematodirus egg species

Figure 50
Trichuris egg species

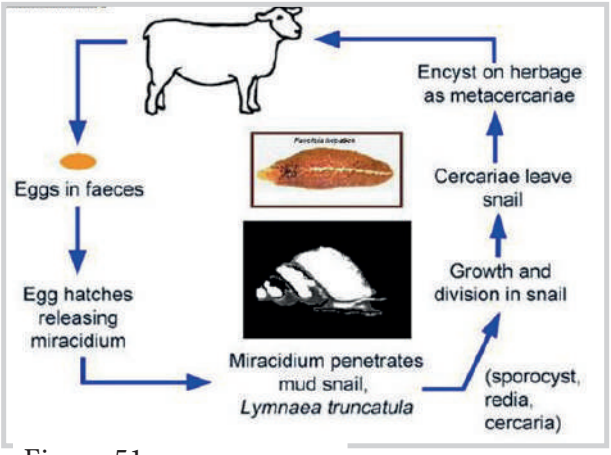


Figure 51
Lifecycle of Fasciola hepatica

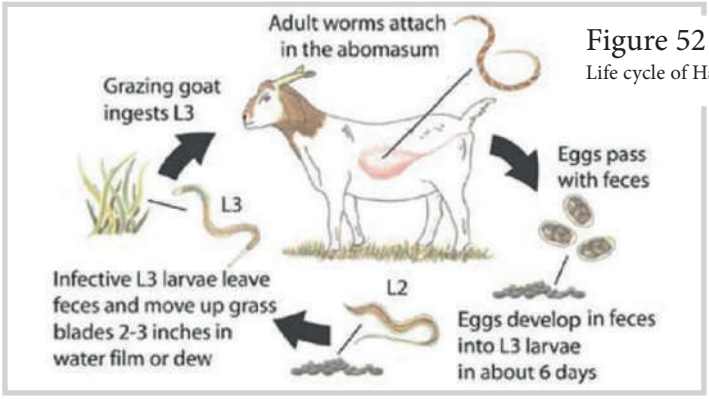


Figure 52
Life cycle of Haemonchus contortus

Picture source:
https://infonet-biovision.org/AnimalHealth/Worms

4.3 | Heart water (Cowdriosis)- Chemiloy / Chepirpirmot

Aetiology

- An infectious, noncontagious tickborne disease of ruminants caused by Cowdria ruminantium and transmitted by the ticks, Amblyomma variegatum.
- In West Pokot county the apparent morbidity rate is 7%, mortality rate 6% and case fatality rate of 86%. The highest incidence of the disease is during the Pengat long rain season (June-August).

Physiopathology

- Once in the host, the organisms may 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 does seem to be a predilection for endothelial cells of the brain. Organisms can often be found in colonies (commonly but mistakenly referred to as morulas) 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; "pedaling," "thrashing," or stiffening of the limbs; and convulsions are seen.
- Diarrhea is seen occasionally.

In subacute cases;

- The signs are less marked and CNS involvement is inconsistent.

Treatment

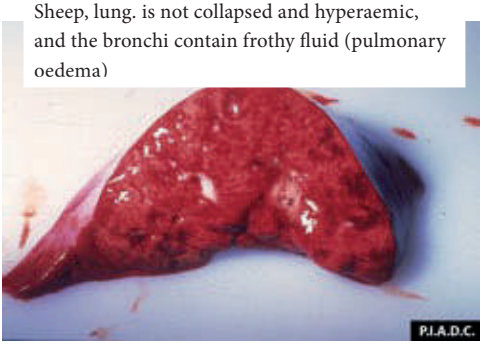
- Oxytetracycline at 10 mg/kg/day, IM, or doxycycline at 2 mg/kg/day will usually effect a cure if administered early in the course of heartwater infection.
- A higher dosage of oxytetracycline (20 mg/kg) is usually required if treatment begins late during the febrile reaction or when clinical signs 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 sulfonamide at 15 mg/kg/day, IM, has been successful. The withdrawal times for milk and meat after treatment with doxycycline, short- or long-acting oxytetracycline, and sulfonamides 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

Figure 53
Amblyomma variegatum ticks feeding on a small ruminant

Figure 54
Sheep, lung. Interlobular septa are distended with oedema fluid (above) and the bellow picture the Sheep, lung. is not collapsed and hyperaemic, and the bronchi contain frothy fluid (pulmonary oedema)



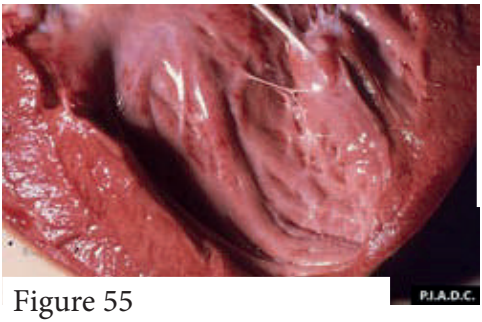


Figure 55
Heart of a small ruminant with haemorrhages on the endocardial surfacel

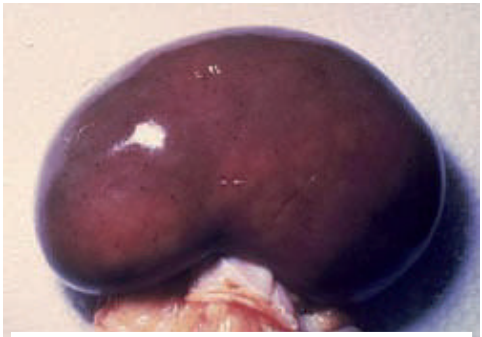
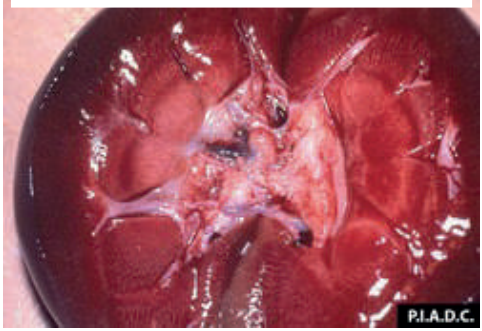


Figure 56
Sheep, kidney with multiple petechiae on the cortical surface (above picture) and bellow cut surface numerous haemorrhages



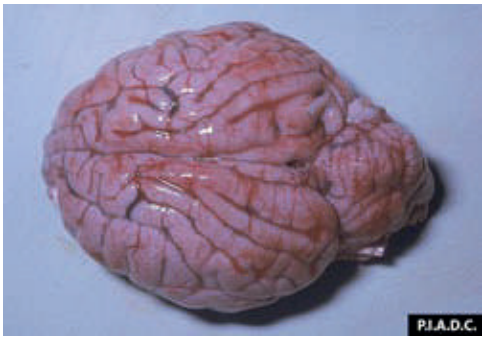
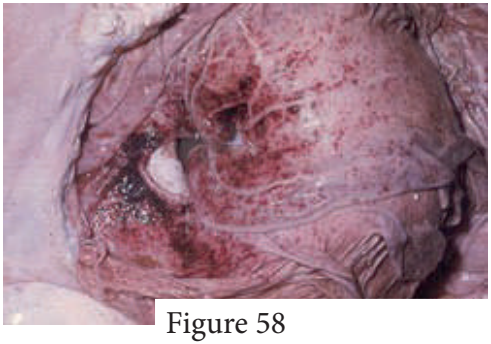


Figure 57
Sheep, brain. The leptomeninges are congested and contain many small hemorrhages. Gyri are flattened (cerebral edema)

Picture source:
https://www.cfsph.iastate.edu/diseaseinfo/disease-images/?disease=heartwater



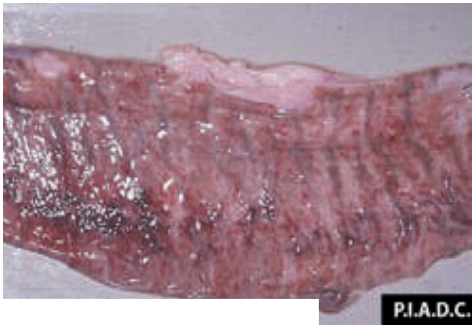


Figure 58

Small ruminant, abomasum (right) and small intestine (left) the mucosa contains disseminated petechiae and coalescing ecchymotic haemorrhages

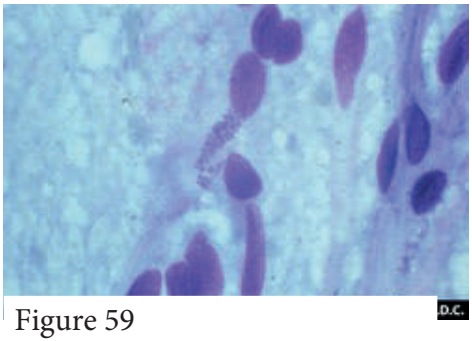


Figure 59

Goat, brain smear. An endothelial cell contains a morula (cluster) of Ehrlichia ruminantium

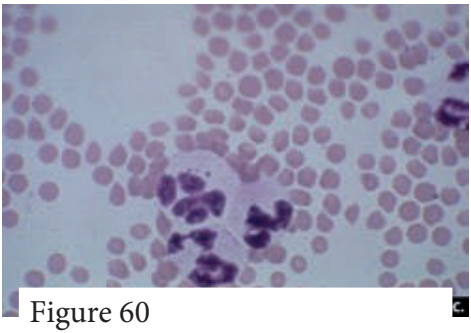
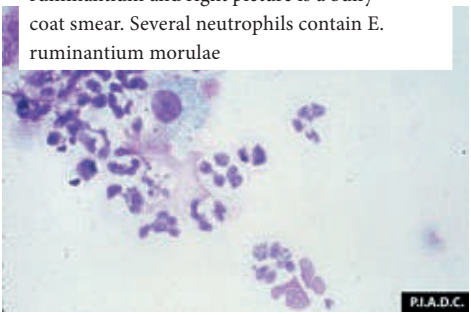


Figure 60

Small ruminant peripheral blood smea (left) the neutrophil contains a few Ehrlichia ruminantium and right picture is a buffy coat smear. Several neutrophils contain E. ruminantium morulae



Picture source:
https://www.cfsph.iastate.edu/diseaseinfo/disease-images/?disease=heartwater

4.4 | Pneumonia - Psosoy

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 West Pokot county, pneumonia cases are common during the heavy rain season, locally known as Pengat (June to August).
- The apparent morbidity rate in the county is 16% with a 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 spp is mediated by the action of several factors, including endotoxin, leukotoxin, and capsular polysaccharide. The leukotoxin 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.
- Fever of above 40.6oC and as high as 42oC.
- Respiratory distress and purulent discharge from the nostrils.

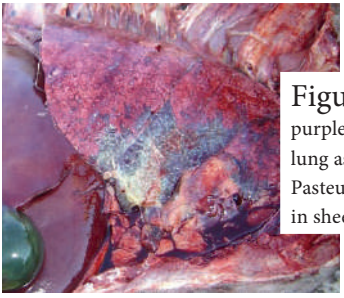


Figure 61
purple-red lesions on lung associated with Pasteurella pneumonia in sheep.

Picture credit: Bruce Watt

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 4th generation cephaloporins (cefquinom).	The ultimate control of pasteurella pneumonia in sheep is by vaccinationation –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.

4.5 | Mange- Chesulet

Aetiology and epidemiology

The most important parasitic mite species of sheep are:

1. Psoroptes ovis that causes psoroptic mange, also called sheep scab occurs worldwide.
2. Sarcoptes scabiei var. ovis that causes sarcoptic mange, also called scabies occurs worldwide.
3. Chorioptes ovis that causes chorioptic mange, also called leg mite, foot scab occurs worldwide.
4. Demodex ovis, responsible for sheep demodectic mange can have local importance. In most cases it causes no clinical symptoms and has little or no economic impact on sheep flocks.

- In West Pokot county the apparent morbidity rate is 11% with a 3% mortality rate and 27% case fatality rate.
- In the county mite infestations are common during the dry season locally known as Komoi (January to March).

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 scrappings 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 (doramectin, ivermectin, moxidectin) 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.

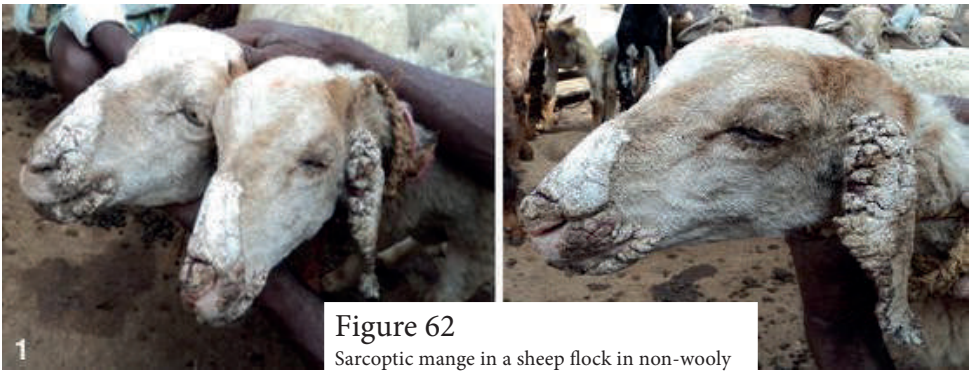


Figure 62
Sarcoptic mange in a sheep flock in non-wooly skin and frequently start on the head

Picture source:
https://link.springer.com/article/10.1007/s12639-012-0157-5

4.6 | Black quarter (BQ)- Tiompoparak/ Tiompoyum/ Tiompoogha

Aetiology

- Black quarter disease is a bacterial disease caused by Clostridium chauvoei.
- The apparent morbidity rate in West Pokot county is 7% with a 100% case fatality rate.
- BQ is most common during the dry season Komoi (January to march).

Predisposing factors

- Mostly as a result of wound infection, often follows some form of injury such as shearing, docking, castration or crutching.

Pathophysiology

- C. chauvoei may be present in the gut, liver and spleen of apparently healthy animals and disease only occurs when conditions become favourable for spore formation. Spores are resistant to heat and disinfectants and can persist in soil, water reservoirs and pasture for many years. dead animals may be sources of spores to the environment. Infection of animals occurs via ingestion of feed and water. Spores may also enter the body through broken skin. Ingested spores are absorbed through the intestinal wall and are carried via blood and lymphatic vessels to muscles and other tissues where they lie dormant. When muscles are bruised or necrotized, the dormant spores germinate and produce alpha, delta, beta and gamma toxins. The alpha toxin is a necrotizing and lethal histotoxin which causes necrotizing myositis, and its absorption via muscles leads to toxaemia and death. The beta toxin destroys the nuclei of muscle cells.

Clinical signs

- Often sudden onset and dead without premonitory signs.
- It has a high case fatality rate, approaching 100%.
- Oedematous and crepitant swellings on the hip, shoulder, chest, back, neck, or elsewhere
- Depression, prostration and tremors
- Death usually occurs in 24 to 48 hours and is characterized by very rapid bloating and carcass decomposition

Diagnosis	Treatment	Control
Diagnosis is often reached at post-mortem. Gross lesions are: Characteristic necrotizing myositis and gas bubble formation Bloated carcass Bloody edema in the sub-cutaneous tissues and around the lesion	Treatment is usually unsuccessful as the disease is rapidly fatal and the animals are usually found dead without prior signs	Black quarter disease is managed by control. During an outbreak, all animals are treated prophylactically using penicillin to prevent new cases. Control is by vaccination. Naive ewes should be vaccinated twice 1 month before lambing and then with yearly boosters. Carcasses should be destroyed by burning or buried deeply in a fenced-off area to limit heavy spore contamination of the soil.

4.7 | Peste de Petits Ruminants (PPR)- Losir

Aetiology

- PPR is a highly infectious viral disease cause by PPRV (Peste de Petits Ruminants Virus) of the morbillivirus genus.

Geographical distribution

- PPR is distributed in sub-Saharan belt of Africa, and goes northwards through Sudan and Egypt into the Middle East, the Arabian Peninsula and then eastwards through Afghanistan, Iran, Pakistan, India as far as Bangladesh.
- PPR is endemic in West Pokot county and disease occurs throughout the year with peak incidences reported during the dry season, Komoi (January to March).
- The apparent morbidity rate in the county is 20% with a 15% mortality rate and 75% case fatality rate.

Predisposing factors

- The disease spreads when animals are crowded together.
- Livestock markets are linked to large-scale outbreaks due to purchase of incubating animals.
- Similarly, communal grazing may spread the disease between flocks belonging to the same village as well as trekking animals over longer distances. The nomadic flocks represent a source of infection and livestock keepers residing near migration routes frequently associate outbreaks of PPR with migrating animals.

Pathophysiology

- 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.

Clinical signs

- Necrotic stomatitis characterized by shallow erosions, halitosis and increased salivation. The lips become swollen and a thin line of necrosis develops along the muco-cutaneous junction.
- Profuse watery, foul smelling diarrhoea.
- Nasal discharge that begins as watery discharge then progresses to purulent that later encrusts blocking nostrils
- Ocular discharge which is water/serous then progresses to purulent and sticky causing the eyelids to be stuck together.
- Pregnant animals may abort. Morbidity and mortality rates are higher in young animals than in adults.

Diagnosis	Treatment	Control
Laboratory: Specimen - lymph nodes, tonsils, spleen, and whole lung for antigen isolation and identification. Whole blood for serum antibody analysis. Necropsy: The main lesions seen in the gastro-intestinal tract are: Small streaks of haemorrhages in small intestines Severe ulceration of Peyer's patches Zebra stripping at ileo-cecal valve, colon and rectum. Severe congestion and enlargement of spleen and regional lymph nodes	Treatment is symptomatic. Broad spectrum antibiotics such as penicillins, streptomycin, sulphonamides may be used to treat stomatitis, enteritis and secondary pneumonia. Sulphonamides are particularly recommended as they are effective against a wide range of bacteria affecting the enteric system.	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 PESTEVAX

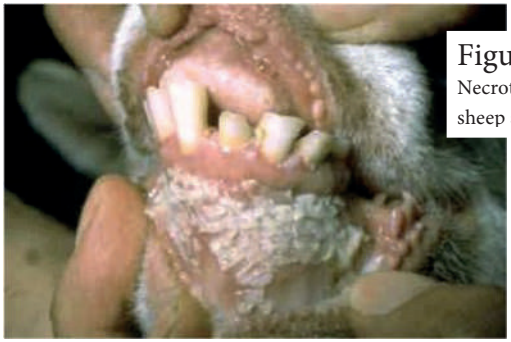


Figure 63
Necrotic Stomatitis in a sheep affected by PPR

Picture source:
http://www.fao.org/3/x1703e/x1703e00.htm

4.8 | Foot rot – Chepkelien

Aetiology

- Contagious foot rot is a common infection of sheep and goats caused by bacteria that live in the soil and are easily carried onto a farm on the feet of infected animals or on shoe soles.
- Two types of bacteria are commonly associated with this condition, Dichelobacter nodosus and Fusobacterium necrophorum.
- Both thrive in moist soil conditions and are difficult to control or eliminate once the soil is contaminated and sheep and goats are kept on the property.
- In West Pokot county the apparent morbidity rate is 14% with a 2% mortality rate and 14% case fatality rate. Foot rot is most common during the heavy rainy season locally known as Pengat (January to March).

Clinical signs

- Foot rot is a more aggressive progression of foot scald, 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 to foot rot infection include; overgrown, cracked or damaged hooves; poor diets deficient in certain minerals also predispose animals to poor hoof health and secondary infections.

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.

Control

- Do not purchase lame animals. Thoroughly inspect feet before purchase. Observe herd of origin for lameness of other animals.
- Quarantine all herd additions for at least 30 days.
- Trim feet and treat feet with foot dip solutions.
- 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. Raise them to ensure drainage.
- Ensure regular hoof care and management and always check feet of limping goats,

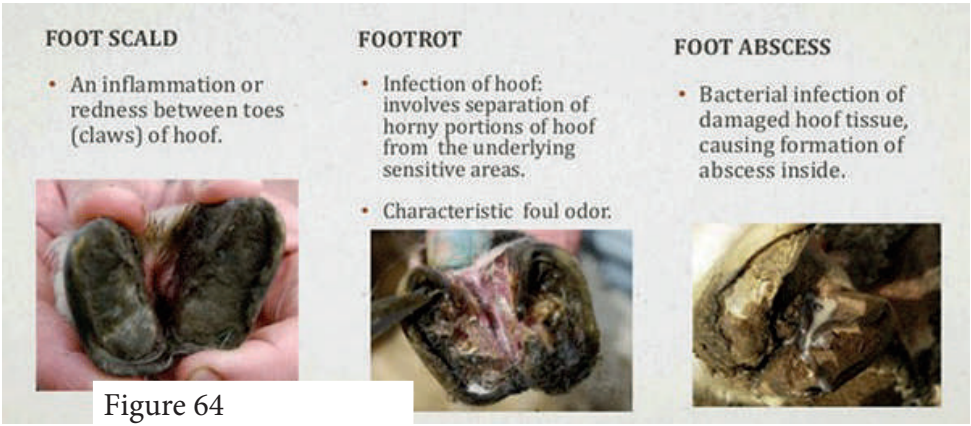


Figure 64
Common hoof conditions in goats

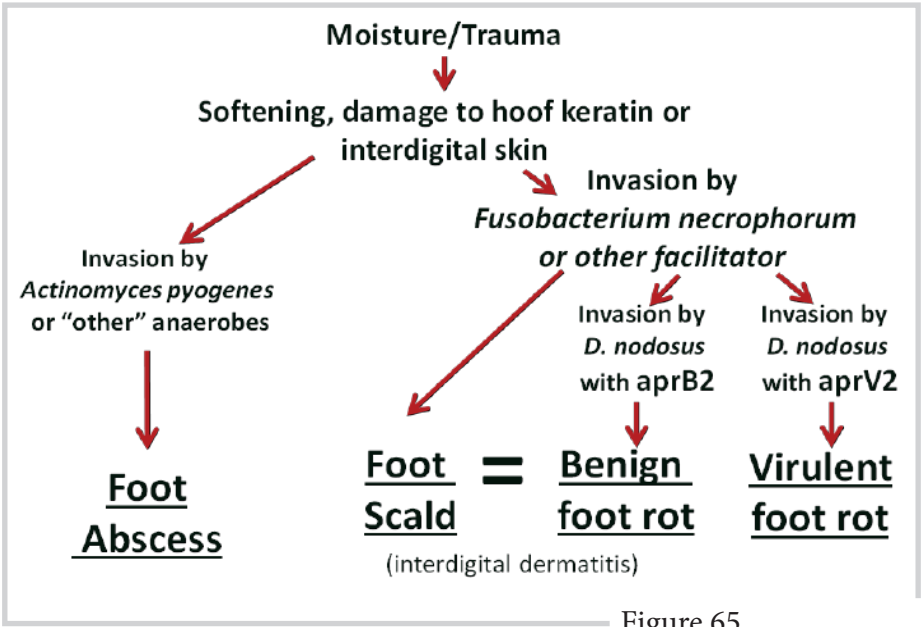


Figure 65
differences of foot abscess, foot scald and benign or virulent foot rot

Foot rot caused by two bacteria soil borne bacteria Fusobacterium necrophorum in combination with benign or virulent strains of Dichelobacter nodosus (formerly Bacteroides nodosus, a Gram-negative, obligate anaerobe)

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

4.9 | Foot and mouth disease (FMD)- Ng'orion

Aetiology

- FMD is a highly infectious disease caused by a virus belonging to the genus Apthovirus of the Piconarviridae family. It has 7 major serotypes namely, A, O, C, SAT 1, SAT 2, SAT 3 and Asia 1. Its spread occurs by direct contact between infected and susceptible livestock, by mechanical carriage by people, vehicles or formites, or by the airborne route.
- In West Pokot county the apparent morbidity rate is 16%, mortality rate 8% and case fatality rate 50%.
- In West Pokot, FMD is common amongst West Pokot community flocks during the long rains season, Ltumuren (November – December). Amongst the Turkana community it is common during the long rainy season, Akiporo in the months of March to June.

Predisposing factors

- Spread is enhanced when sheep are kept in close contact as in markets
- The movement of sheep in large numbers also enhances spread.

Pathophysiology

- The virus is transmitted via ingestion or inhalation leading to oropharyngeal and nasopharyngeal infection. Proliferation leads to viremia leading to the virus localizing in epidermal cells such as the epithelium of the mouth, feet and teats and in various other tissues including lymph nodes, tonsils, pharyngeal mucosa and heart muscle. In young animals, especially neonates, the virus causes necrotizing myocarditis.

Clinical signs

- The first sign observed in a flock of sheep is usually lameness that rapidly spreads among the animals.
- Mouth lesions comprise of erosions on the gum, hard palate, lips and tongue. Occasionally, they may occur on the nostrils.
- Fluid filled vesicles may also be seen. Foot lesions are seen as vesicles in the interdigital space, at the bulb of the heel and along the coronary band. They may rupture leading to secondary infection. The feet feel warm and painful to touch.

Diagnosis	Treatment	Control
Laboratory: Samples – serum sample for detection of FMD immunoglobulins using ELISA Necropsy: In young lambs, there may be extra epithelial replication in the myocardium leading to necrosis of the cardiac muscle "tiger heart"	There is no effective treatment of FMD and supportive/ symptomatic care may be given to affected animals. Self-recovery may occur in two weeks.	Control and prevention of FMD is mainly by vaccination of the herd. Fotivax™ (KEVAVAPI) for prevention of FMD serotypes A, O, SAT 1 and SAT 2. Isolation, quarantine and movement control during outbreaks and practicing biosecurity measures including disinfection, hygiene and sanitation controls the transmission and spread.

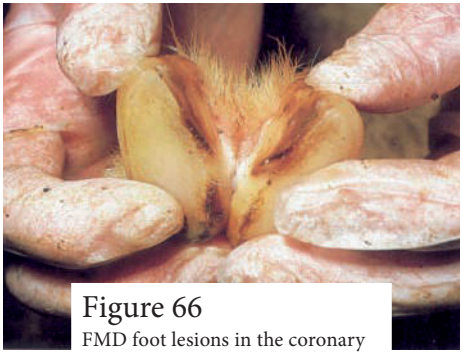


Figure 66
FMD foot lesions in the coronary band and interdigital space.

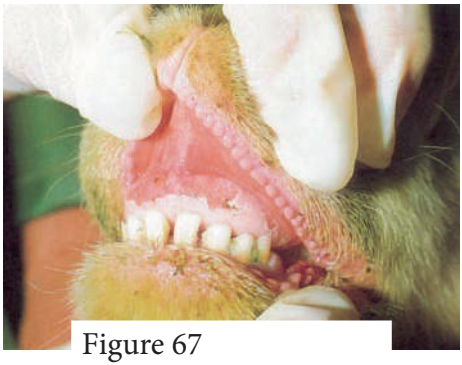


Figure 67
FMD Gum lesions

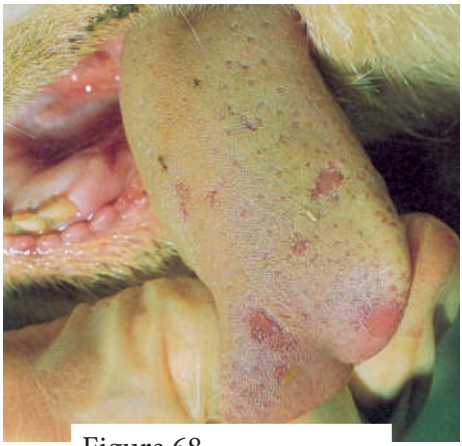


Figure 68
FMD tongue lesions

4.10 | Bloat- Musarer

Two types of bloat can occur in sheep:
1. Frothy bloat (primary ruminal tympany)
2. Free gas bloat (secondary ruminal tympany)

Aetiology and pathogenesis

- Bloat in West Pokot county has an apparent morbidity rate of 9% and mortality rate of 2% with a case fatality rate of 22%.
- Bloat in the county occurs mainly in the heavy rainy season locally known as Pengat (June to August),

Frothy bloat occurs due to overeating lush, legume-dominant pastures in the vegetative and early budding stage. These provide readily available nutrients that are rapidly broken down once ingested, leading to swift production of gas in the rumen. This gas production produces thick foam and causes a quick change in the pH, increasing the viscosity (stickiness) of the rumen fluid. The small bubbles in the foam cannot merge due to this increased viscosity. This prevents larger bubbles from forming and breaking off from the foam to be released through belching. As the amount of gas trapped in foam increases, the rumen will expand, primarily on the left side. As the rumen expands, it displaces internal organs, restricts blood flow and interferes with the animal's ability to breathe. If there is no intervention, the animal will die from respiratory or circulatory failure.

Free-gas bloat, also called grain bloat, occurs when the animal cannot belch, whether it is because of an obstruction, posture or functional problems. Obstructions from foreign bodies, abscesses or tumors can prevent eructation, allowing a build-up of gas in the rumen. Posture-induced bloat happens because ruminants absolutely cannot belch while on their backs. Therefore, bloat will occur rapidly if an animal falls into a position where it cannot right itself. Functional problems from grain overload, internal damage or certain diseases can also prevent animals from belching. Grain overload, or grain bloat, occurs when animals suddenly eat large amounts of grain, like maize, before the rumen has had a chance to adapt to the high digestibility. As the grain is broken down, it ferments as usual, but because the rumen is not adapted to the high digestibility, the pH drops, resulting in reduced ruminal contractions. Without the contractions of the rumen, gas cannot be expelled and accumulates, distending the rumen.

Clinical signs

- Often the first sign is sudden death unless animals are under close surveillance.
- Main signs are distended left abdomen, respiratory distress, abdominal kicking and death.
- Usually, free gas bloat is seen in one or two animals but frothy bloat affects a number of animals in the flock.

Diagnosis	Treatment	Control
• Abdominal distension is highly suggestive of the disease. Passing a stomach tube will distinguish between free-gas and frothy bloat.	• Treatment should be prompt before the animal suffocates and dies. The use of a stomach tube is the most effective treatment for free-gas bloat. Once you place the tube, the gas in the rumen will escape and relieve the pressure. • If no gas escapes and a foamy substance trickles out of the tube, then the animal has frothy bloat. • For frothy bloat, the stomach tube should remain in place and antifoaming agents such as vegetable oils (eg, peanut, corn, soybean) and mineral oils (paraffins), at doses of 250–500 mL administered. Follow dosing directions for your chosen antifoaming agent. If relief does not occur almost immediately, then the animal should be closely monitored for at least the next hour to determine if treatment was successful. • In extreme cases of bloat, the last resort is to use a trocar and cannula to relieve rumen gas.	Prevention is by limiting access to legume-dominant pastures during vegetative and early bud stages. • Only leave animals on pasture for a couple of hours in the afternoon. • Do not turn livestock onto pasture moist with dew, rain or irrigation water. The moisture can increase the chances of bloat. Observe livestock closely the first several days they are on pastures with bloat potential and remove any animals showing signs of bloat.

4.11 | Plant poisoning- Kawatian / Ngwono

- Occurs suddenly and results in heavy losses.
- In West Pokot, plant poisoning is common during the light rain season locally known as Sarngatat (April to May).
- In West Pokot county the apparent morbidity rate is 10% with a 5% mortality rate and 50% case fatality rate.
- Animals which have been bred in a certain area usually know which plants are edible and which are not. Poisoning in such animals may, however, occur when grazing is scarce, or when they are taken to a new area of grazing on the same farm. Animals newly introduced to an area will frequently eat toxic species with which they are unfamiliar.

Diagnosis	Treatment	Control	Community practices
• Difficult to diagnose as different plants have different mechanisms of action and produce different symptoms. • Diagnosis depends on history. The fact that animals had access to a particular plant or been seen to be eating it, the presence of identifiable fragments in the stomach and the fact animals were new to the area should be considered	• Treatment of plant poisoning is usually supportive and symptomatic as different poisons produce different effects	• Weeding out poisonous plants • Relocation of herd • Fencing off grazing area to avoid animals grazing where poisonous plants are	Traditional treatments/ practices for treatment of plant poisoning in West Pokot • Oral drench of milk and sugar • Oral drench of honey • Warm salty water oral drench • Oral drench of crushed charcoal and ashes • Oral drench of raw eggs • Activated charcoal

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Chapter 5:
Goat diseases

5.1 | Coccidiosis – Emaciation and diarrhoea syndrome - Chepkiyiy

Aetiology
- 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 West Pokot county the apparent morbidity rate is 8% with a mortality rate of 4% and a case fatality rate of 50%. The disease has an increased incidence rate during the Komoi dry season during the months of January and March.

Clinical Signs
- Coccidia are host-specific and each animal species is susceptible to infection with various coccidia species. In goats, 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.

Treatment
- Oral powder 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 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.

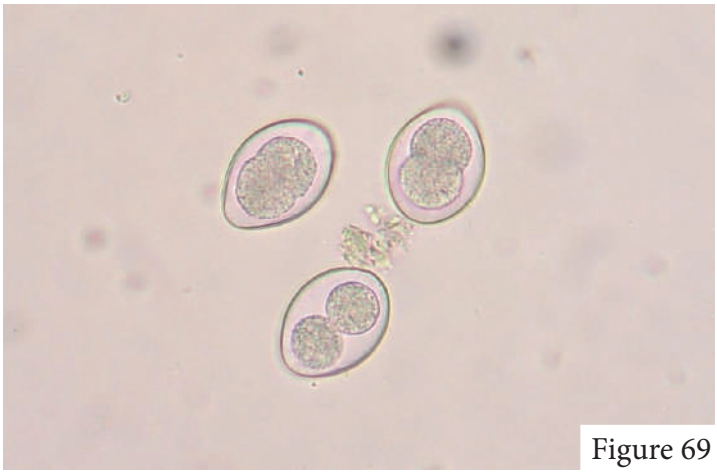


Figure 69
Eimeria species unsporulated oocysts in goat kid faecal sample

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

Figure 70
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/

5.2 | Infectious Keratoconjunctivitis (Pink eye)- Semewo Konyin

Aetiology

- In goats, pinkeye is primarily caused by the microorganisms Mycoplasma conjunctivae and Chlamydia. These are not the same microorganisms that cause pinkeye in cattle (Moxarella bovis), so the vaccine used to prevent pinkeye in cattle is not effective in goats.
- In West Pokot county pink eye has an apparent morbidity rate of 4% with no case fatality rate. The disease occurs throughout the year but is more common during the Pengat heavy rainy season between the months of June and August.

Clinical Signs

- Pinkeye progresses rapidly once a 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, 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.

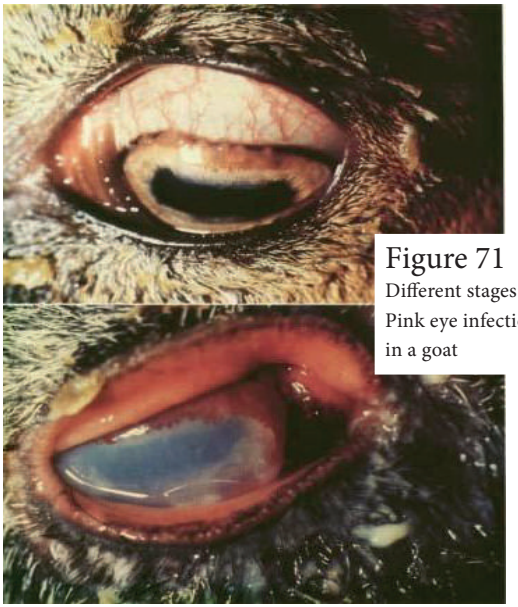


Figure 71
Different stages of Pink eye infection in a goat

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

5.3 | Contagious Caprine Pleuropneumonia (CCPP)- Luokoi

Causative agent
- CCPP is caused by bacterium Mycoplasma capricolum subspecies capripneumoniae.

Distribution
- Occurs in many countries in Africa, Asia and the Middle East.
- In West Pokot county CCPP is endemic in and is more common in the months of June to August during the Pengat heavy rain season.

Morbidity and Mortality rates
- In naive herds, the morbidity rate reaches 100% and the mortality rate can be as high as 80%.
- In West Pokot county the apparent morbidity rate is 35% with a 11% mortality and 31% case fatality rate.

Transmission
- Transmitted during close contact and by inhalation of respiratory droplets.

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

Figure 72
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

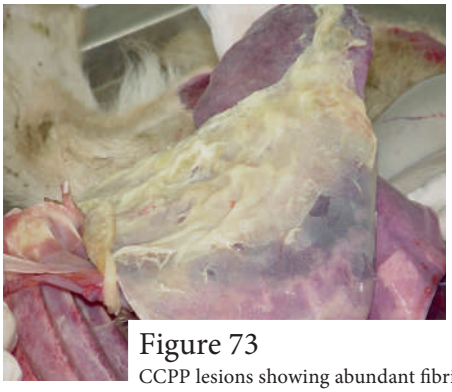


Figure 73
CCPP lesions showing abundant fibrin on lung lobes (Left) and large quantities of pleural fluid and "port wine"–coloured hepatized lung lobes in a goat

Picture source:
https://www.msdvetmanual.com/respiratory-system/respiratory-diseases-of-sheep-and-goats/contagious-caprine-pleuropneumonia

5.4 | Peste des Petits Ruminants (PPR) - Losir

- PPR is an acute or sub-acute viral disease of goats and sheep caused by Mobillivirus in the family of Paramyxoviridae.
- It was first reported in Cote d'Ivoire in 1942 and subsequently in other parts of West Africa. PPR has now been reported in all parts of Africa except for southern Tip, the Middle East, and the entire Indian Sub-continent.
- PPR has rapidly expanded within Africa and to large parts of Central Asia, South Asia, and East Asia. Goats and sheep appear to be equally susceptible to the virus, but goats exhibit more severe clinical disease. The virus also affects several wild small ruminant species.
- In West Pokot county, PPR apparent morbidity rate is 31% with a mortality rate of 23% and case fatality rate of 74%.
- In the county PPR incidences in goats occurs throughout the year but more incidences are common during the Pengat season which is the heavy rain season (June to August).

Aetiology

- PPR is a viral disease caused by Mobillivirus in the family of Paramyxoviridae

Pathophysiology

- The virus enters the body of animals through their oral and nasal passages and multiplies first in the oropharynx and local lymphoid tissues. All of the immune cells (lymphocytes, macrophages, reticular cells) can be a target for virus multiplication. The newly formed virions spread throughout the host's organs and tissues with a preference for digestive, pulmonary, and respiratory mucosa and the immune system

Clinical signs

- Fever (40 °C – 41.3 °C).
- Serous Nasal discharge that becomes mucopurulent.
- Halitosis with necrotic stomatitis on the lower lip and gum.
- Coughing may develop at late stages of the disease
- Morbidity and mortality rates are higher in young animals than in adults

Diagnosis

- Presumptive diagnosis is based on clinical, pathologic and epidemiological findings and may be confirmed by viral isolation and identification.
- Antigen capture ELISA and reverse transcription-PCR are the preferred laboratory tests for confirmation of the virus.
- For antibody detection, competitive ELISA and virus neutralization are the OIE-recommended tests
- The specimens required are lymph nodes, tonsil, spleen, and whole lung for antigen or nucleic acid detection, and serum for antibody detection.

Treatment

- No treatment is available but treatment for bacterial and parasitic complications decreases mortality in affected flocks/herd.
- Treatment with Tylosin at 10 mg/kg, intramuscular for 3 days.
- 10% Oxytetracycline at 1 ML/10 kg body weight for 3 days.

Control

- Attenuated PPR vaccine prepared in vero cell culture is available and gives protection from natural disease for >1 year.
- Isolation of sick animals from the flock

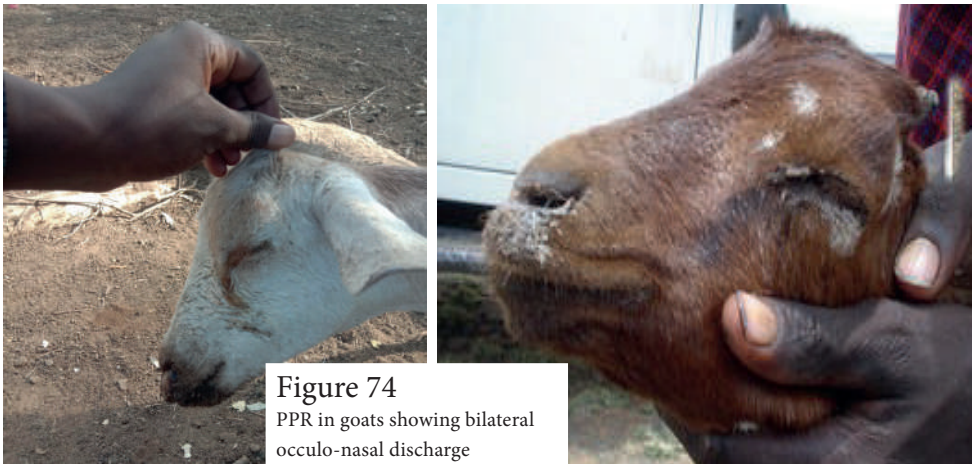


Figure 74
PPR in goats showing bilateral occulo-nasal discharge

Picture source:
Emmanuel Lesiantam – Samburu County (Left) and Jonas Johansson Wensman (Right) https://www.researchgate.net/profile/Jonas-Wensman

Figure 75
PPR in a goat kid showing bilateral mucopurulent nasal discharge and hypersalivation due to oral lesions

Picture source:
Emmanuel Lesiantam – Samburu County

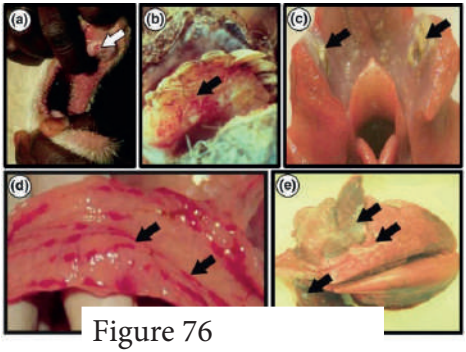


Figure 76
Post-mortem lesions in goats infected with PPR

a- Necrotic lesions on dental pad; b-lesions on gum and oral cavity; c-oro-pharyngeal sections showing necrotic lesions on palatine tonsils and small fibrin deposits on the base of tongue; d-zebra striping in large intestine and e-consolidated lungs leading to pneumonia

5.5 | Helminthiasis -Mochontu muu/ Ngutianta muu/ Chepturu

Aetiology

- The most common helminth species involved are Haemonchus contortus, Ostertagia, circumcincta, Trichostrongylus axei, Trichostrongylus, Nematodirus species, Bunostomus trigonocephalum, Oesophagostomum columbianum, Bunostomum phlebotomum (hookworm), Dictyocaulus filarial and Muellerius capillaris.
- In West Pokot county the apparent morbidity rate is 14% with a 5% mortality and 36% case fatality.
- Most helminth infestations in the county occur throughout the year but incidences are more common during the Kitokot light rain season (September and December).

Clinical signs

- Coughing and Dyspnoea
- Depression
- Loss of condition
- Diarrhoea, Dehydration,
- Anaemia and bottle jaw
- Presence of adult worms in faeces

Diagnosis

- Clinical signs
- Presence of adult worms in faeces
- Microscopic evaluation of single or pooled faecal samples

Treatment

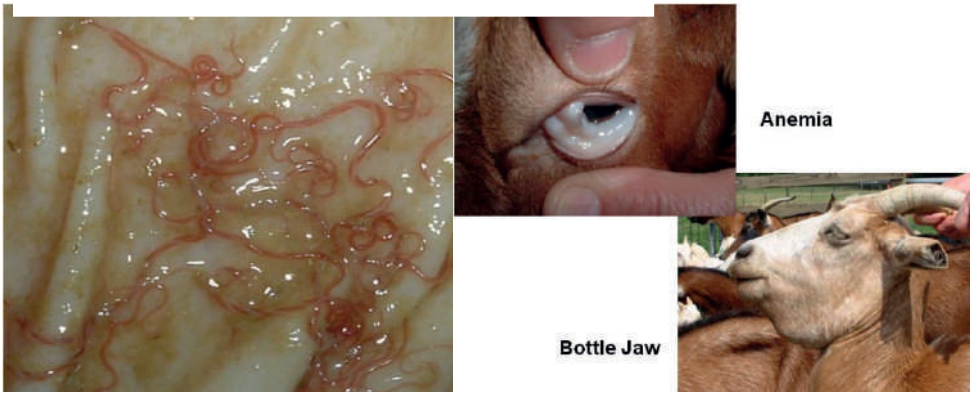
- Lungworms- Benzimidazoles (fenbendazole, oxfendazole and albendazole), 5mg/kg body weight, orally.
- Macrocyclic lactones (ivermectin, doramectin, eprinomectin and moxidectin), 0.4 mg/kg orally.
- Levamisoles and repeat two weeks later
- Multivitamin supplementation

Control

- Regular deworming with broad-spectrum anthelmintics 2 weeks after the onset of rainy seasons and at the beginning of the dry season.
- Broadspectrum anthelmintics currently available belong to five different chemical groups:
  Benzimidazoles; effective against most of the major helminth parasites of ruminants have varying levels of activity against inhibited larvae.
- Imidazothiazoles (levamisole, morantel, pyrantel); highly effective, safe broad-spectrum anthelmintics but has little activity against larval stages of worms.
- Macrocyclic lactones (avermectins and milbemycins) highly effective against adult and larval stages, including inhibited larvae of all the common GI nematodes of ruminants and some important ectoparasites.

Figure 77

Haemonchus contortus adult worms in the abomasal lumen in a goat
(left) anaemia and bottle jaw in a goat affected by Hemonchosis (right)



Picture source:
https://www.apsc.vt.edu/content/dam/apsc_vt_edu/extension/sheep/programs/shepherds-symposium/2012/12_symposium_getz_famacha2.pdf

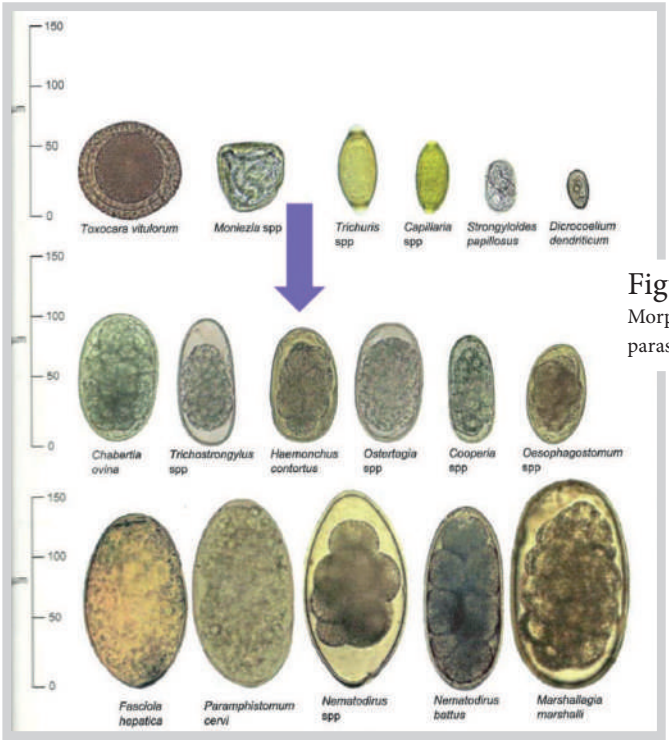


Figure 78

Morphology of ruminant parasitic eggs

Picture source:
https://www.vasci.umass.edu/sites/vasci/files/control_of_gastrointestinal_parasites_in_camelids_sheep_and_goats_0.pdf

5.6 | Sheep and Goat Pox (SGP) – Mokoyon, Moyoi and Lomolokoo

Aetiology

- Sheep and goat pox is caused by a virus.
- Transmission usually by the aerosol route, but can be spread mechanically by insect bite or iatrogenic through hypodermic injection.
- Pox lesions usually occur all over the skin including mucus membranes of the cheeks, lips and nostrils as well as the tongue and gum.
- In West Pokot county, SGP is endemic and common during the heavy rain Pengat season that occurs in the months of June to August.
- The apparent SGP morbidity rate in goats in the West Pokot county is 9% with a mortality rate of 6% and a case fatality rate of 67%.

Clinical signs

- Characteristic raised skin lesions on the skin, lesions more widespread in the skin of the groin, axilla and perineum.
- Fever of 40oC or higher.

Diagnosis	Treatment	Control
Laboratory: • Capripoxviruses, their antigens and nucleic acids can be detected in skin lesions. oral, nasal and ocular secretions; blood; lymph node aspirates; and tissue samples from external or internal lesions collected at necropsy. Samples for virus isolation and for some antigen-detection tests should be collected during Blood samples should be taken as early as possible; virus isolation is unlikely to be successful after generalized lesions have been present for more than a few days. Necropsy: • On post-mortem pox lesions will be present on the tongue, rumen, kidney cortex, liver and lungs.	• There is no effective treatment and treatment is usually supportive by administration of broad spectrum antibiotics such as Penstrep® and oxytetracycline and fly control. • Vaccination- KEVEVAPI has a vaccine called S&G Vax.	• Prevention heavily relies on vaccination. Quarantines, movement control and rapid stamping out followed by thorough disinfection is critical in control. New animals being introduced to a herd should always be quarantined prior to adding them to existing flocks.

Figure 79
Morphology of ruminant parasitic eggs

Picture source:
https://www.pirbright.ac.uk/news/2018/04/tackling-sheep-and-goat-pox-nigeria
https://www.msdvetmanual.com/integumentary-system/pox-diseases/sheeppox-and-goatpox

5.7 | Foot rot - Chepkelien

Aetiology

- Contagious foot rot is a common infection of sheep and goats caused by bacteria that live in the soil and are easily carried onto a farm on the feet of infected animals or on shoe soles.
- Two types of bacteria are commonly associated with this condition, Dichelobacter nodosus and Fusobacterium necrophorum.
- Both thrive in moist soil conditions and are difficult to control or eliminate once the soil is contaminated and sheep and goats are kept on the property.
- In West Pokot county the apparent morbidity rate is 14% with a mortality rate of 4% and case fatality rate of 29%. Foot rot is most common during the heavy rainy season locally known as Pengat (June to August).

Clinical signs

- Foot rot is a more aggressive progression of foot scald, 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 to foot rot infection include; overgrown, cracked or damaged hooves; poor diets deficient in certain minerals also predispose animals to poor hoof health and secondary infections.

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.

Control

- Do not purchase lame animals. Thoroughly inspect feet before purchase. Observe herd of origin for lameness of other animals.
- Quarantine all herd additions for at least 30 days.
- Trim feet and treat feet with foot dip solutions.
- 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. Raise them to ensure drainage.
- Ensure regular hoof care and management and always check feet of limping goats,

Figure 80

Common hoof conditions in goats

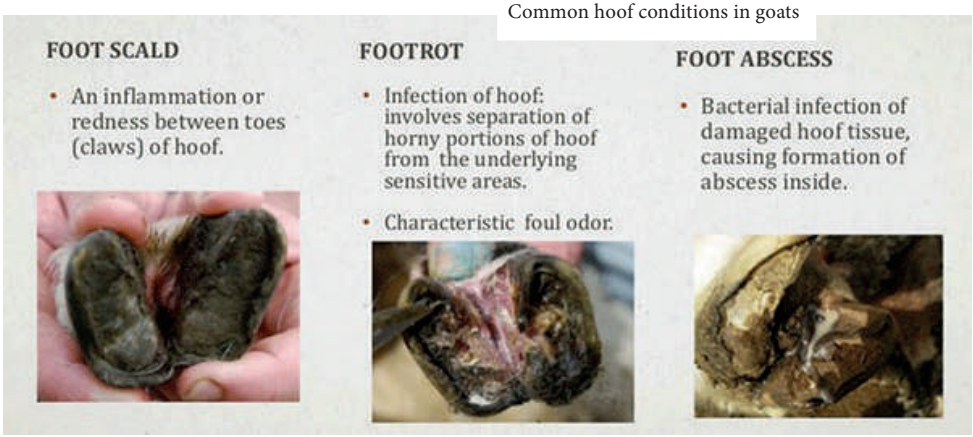
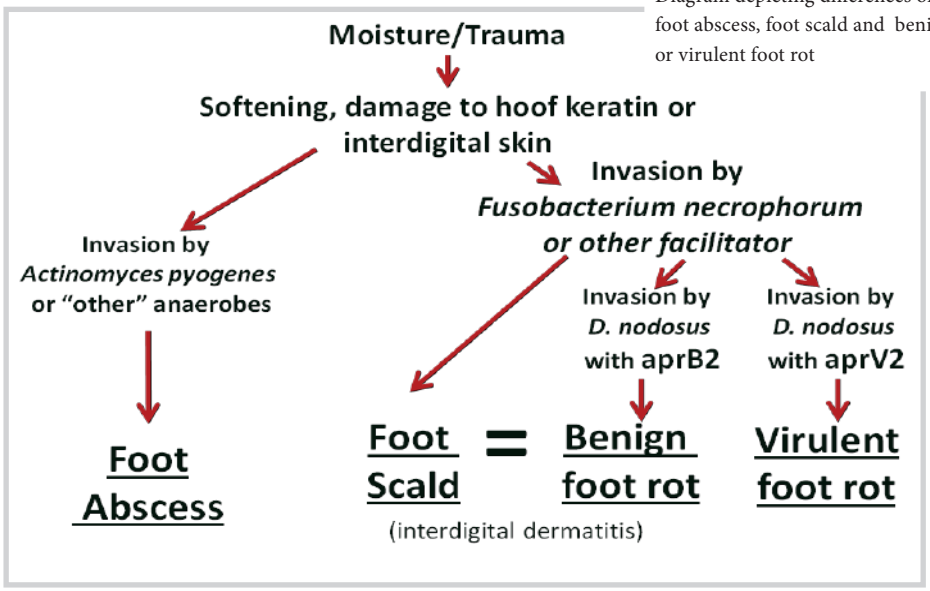


Figure 81

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



Foot rot caused by two bacteria soil borne bacteria Fusobacterium necrophorum in combination with benign or virulent strains of Dichelobacter nodosus (formerly Bacteroides nodosus, a Gram-negative, obligate anaerobe)

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

5.8 | Mange in goats- Simpirion

- In West Pokot county the apparent morbidity rate of goat mange is 6% with a mortality rate of 2% and case fatality rate of 33%.
- Mange occurs throughout the year but most incidences of goat mange in the county are reported during the Pengat rain season (June to August).

Sarcoptic Mange:

- S scabiei var caprae infests 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 is zoonotic. Consistent with other animal variants of Sarcoptes, zoonoses are initiated from direct contact with infected animals but are self-limiting infestations.

Chorioptic Mange:

- Chorioptes bovis infests goats.
- Prevalence of C bovis on goats is fairly common.
- Distribution of lesions is the same as that in cattle, with papules and crusts seen on the feet and legs.

Demodectic Mange (Caprine Demodicosis):

- Demodex caprae infests goats.
- D caprae are relatively common in goats. Lesions are similar to those in cattle. In goats, nonpruritic papules and nodules develop, especially over the face, neck, shoulders, and sides or udder.
- Demodectic mange in goats occurs commonly in kids, pregnant does, and dairy goats. The nodules contain a thick, waxy, grayish material that can be easily expressed; mites can be found in this exudate. The disease can become chronic.

Treatment and Control

- Hot lime sulfur 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 pyrethrins or organochlorines (Amitraz) are labelled for mange in sheep and goats. As with cattle, they are not considered the compound of choicebut 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.

Figure 82
Sarcoptic Mange in a goat showing alopecia 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

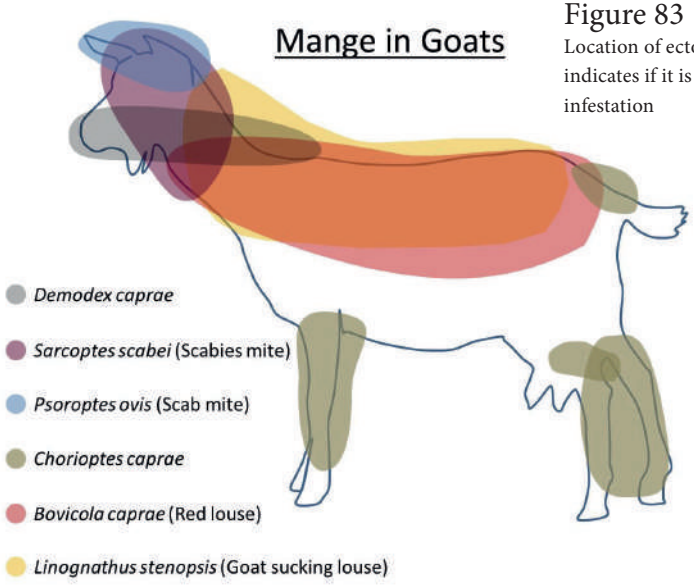


Figure 83
Location of ectoparasite in goats indicates if it is a mite or flea infestation

Picture source:
https://www.farmhealthonline.com/US/disease-management/goat-diseases/mange-in-goats/

5.9 | Coenurosis - Kapir / Lopira

Aetiology

- The causative agent is Coenurus cerebralis, the larval stage of Taenia multiceps, a tapeworm that infests the small intestine of carnivores.
- Contamination of pastures with dog faeces result in larval invasion of the central nervous system and clinical disease. The life cycle is completed when the carnivorous definitive host ingests the infested goat's brain.
- In West Pokot county the apparent morbidity rate in goats is 3% with a 3% mortality rate and 100% case fatality rate.
- Coenurosis manifestation is more common during the long dry season locally known as Lameioodo (June to September) in West Pokot language and Akaamu nakoyen (July to October) in Turkana language.

Pathophysiology

- The adult tapeworm, Taenia multiceps, inhabits the dog guts where it produces parasite 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.

Clinical signs

- Coenurosis is characterized by signs ranging from pyrexia, listlessness and head aversion to convulsions and death within 4–5 days. Circling is observed in affected animals.

Diagnosis	Treatment	Control
Diagnosis is confirmed on necropsy by the presence of Coenurus cerebralis cysts in the brain of goat.	Treatment with medication is not usually successful, farmers resort into culling affected animals	Control is done by breakage of the goat-dog cycle by proper disposal of carcasses and deworming of dogs with praziquantel

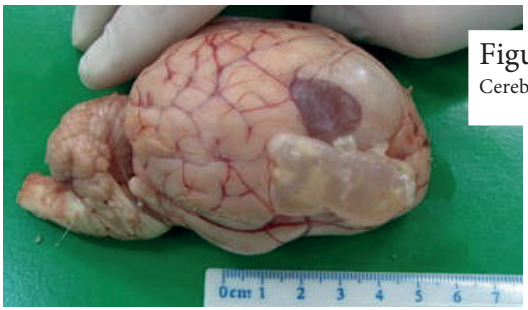


Figure 84
Cerebral Coenurosis in a goat

Picture source:
https://link.springer.com/article/10.1007/s00580-008-0742-2

5.10 | Contagious ecthyma (orf)- Ngirimen/ Ngurumen

Aetiology

- Caused by a virus from the parapoxvirus genus in the Poxvirus family.
- Virus primarily causes an infection in sheep and goats, although it can be transmitted to humans (Zoonosis).
- Orf virus infection in animals is commonly referred to as sore mouth, scabby mouth, or contagious ecthyma.
- Animals infected with orf virus typically develop scabby sores (lesions) around their lips, muzzle, and in their mouth.
- Humans infected typically develop ulcerative lesions or nodules on their hands.
- In West Pokot county the apparent morbidity rate is 7%, mortality rate 2% and case fatality rate of 29%.
- Orf in the county is common during the heavy rainy season locally known as Pengat (June to August).

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.

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.
- Severely affected kids will require good nursing care to ensure that they are eating and drinking.
- Does and ewes may require one treatment of long acting antibiotic either oxytetracycline or amoxicillin based if they develop mastitis.

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.

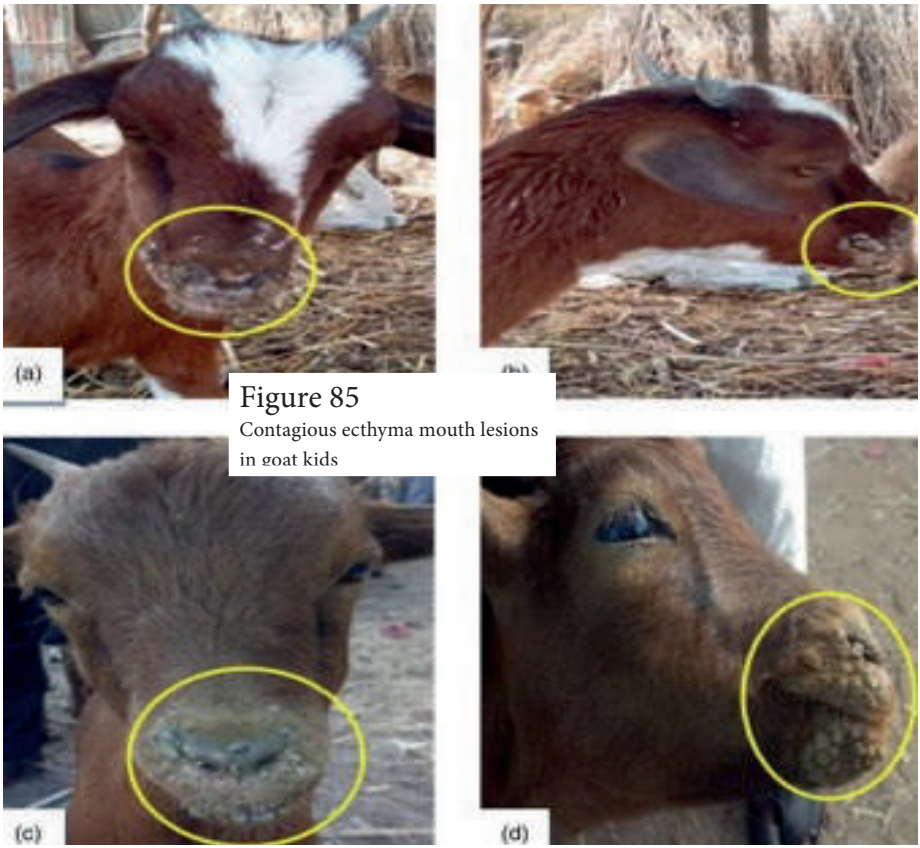


Figure 85
Contagious ecthyma mouth lesions in goat kids

Further Reading

1. Merck Veterinary Manual (Online Version)
2. Firdaus, Faez & Hambali, Idris & Abba, Yusuf & Sadiq, Muhammad Abubakar & Bitrus, Asinamai & Chung, Eric Lim Teik & Mohd Lila, Mohd & Haron, Abd Wahid & Saharee, Abdul. (2016). Therapeutic Options in Management of a Clinical Case of Chorioptic Mange in a Goat. Research Journal for Veterinary Practitioners. 4. 39-41. 10.14737/journal.rjvp/2016/4.2.39.41.
3. Besier, Brown & Kahn, L.P. & Sargison, N.D. & Van Wyk, Jan. (2016). Diagnosis, Treatment and Management of Haemonchus contortus in Small Ruminants. 10.1016/bs.apar.2016.02.024

Chapter 6:

Chicken Diseases

6.1 | New Castle Disease - Konukoi/ Tongoroi/ Knuch/ Konuktoi

Definition

- Other names Avian pneumoencephalitis, Exotic or velogenic Newcastle disease and rarely Ranikhet disease;
- Newcastle disease (ND) is a severe and highly contagious disease of wild birds and domestic poultry.
- ND is endemic in West Pokot county and has been identified as the main disease limiting chicken production in the county.

Distribution (Epidemiology)

- Purchase of new flock additions from chicken markets in the county serve as an important source of ND infection.
- Other domestic bird species like ducks and geese that are normally not vaccinated also serve as an important source of infection.
- In West Pokot county the disease has been estimated to have an apparent morbidity rate of 61% with a mortality rate of 53% and case fatality rate of 87%.
- The disease in West Pokot county occurs throughout the year but most outbreaks are seen during the months of June to August during the heavy rainfall season locally known as Pengat.

Causative agent (Aetiology)

- Newcastle disease virus (NDV) also known as avian paramyxovirus (APMV-1) is an RNA
- NDV has three virulent forms virulent (velogenic), moderately virulent (mesogenic), or low virulence (lentogenic). virulent NDV (vNDV) is the cause of Newcastle disease and is an OIE reportable infection while the low virulence NDV (loNDV) is widely used as live vaccines.

Mode of transmission

- Direct contact with virus shed by infected or carrier birds (domestic and wild) in exhaled air, respiratory discharges, faeces, contaminated water or food and in carcasses.
- Indirectly through movement of contaminated equipment, people or litter
- Predisposing factors:
- Viral survival is prolonged by presence of faeces, for example on eggshells and cool environments.

Affected group

- All ages of birds

Main clinical signs and Post-mortem (PM) lesions

- Acute respiratory distress manifested as - gasping, coughing, sneezing, rales;
- Severe depression;
- Nervous manifestations – tremors, paralyzed wings and legs, twisted necks, circling, spasms and paralysis;
- Diarrhoea;
- Partial or complete drop in egg production;
- Eggs if produced have abnormal colour, shape or surface with watery albumen.
- PM Gross lesions are not pathognomonic- several birds must be examined to determine

a tentative diagnosis and final diagnosis must await virus isolation and identification.

Only velogenic strains produce significant gross lesions that include;

- 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

- Clinical signs and gross lesions are not specific to ND for confirmatory diagnosis collect samples for laboratory investigation;

Samples collection should be from recently dead birds (less than 8 hours), moribund birds that have been killed humanely or live birds;

- Dead birds: oral-nasal swabs; lung, kidneys, intestine (including contents), caecal tonsils, spleen, brain, liver and heart tissues, separately or as a pool;
- Live birds: tracheal or oropharyngeal and cloacael swabs (visibly coated with faecal material) or collect pooled fresh faeces;
- Clotted blood for serum harvesting in both live and dead birds

Main laboratory test principle –Virus isolation and antibody detection through;

- Virus isolation- Inoculation of embryonated specified pathogen free (SPF) eggs and tested for haemagglutination (HA) activity or use of Molecular methods (PCR);
- Serological tests- Haemagglutination and haemagglutination inhibition tests or Enzyme-linked immunosorbent assay (ELISA

Differential Diagnosis (DDx)

- Fowl cholera
- Highly pathogenic avian influenza
- Fowl pox (diphtheritic form)
- Mycoplasmosis
- Infectious bronchitis

Treatment

- Viral disease - no treatment
- Supportive- use combined multivitamin and antibiotic oral medication in drinking water

Prevention/control

- Prompt disposal of dead birds through burning.
- Vaccination - thermostable live vaccine produced by KEVEVAPI is called AVIVAX-L. Vaccine remains viable for 8 weeks at room temperature. It is administered orally in drinking water. Withdraw drinking water for at least 3 hours prior to vaccination. Reconstitute the vaccine using water free from chlorine and detergents. The vaccine is sold in doses of 200 – 500 vials. Reconstitute the vaccine as per the guide below:

Age of chicken	200 dose pack (For 200 birds)	500 dose pack (For 500 birds)
4 days to 3 weeks	1-2 litres of water	2.5 to 5 litres of water
4 to 8 weeks	4 litres of water	10 litres of water
Over 8 weeks	8 litres of water	20 litres of water

- Vaccinate at chicks at 4 days old, repeat at 4 and 16 weeks of age;
- If one acquires older birds give the first primary vaccine, then repeat the second one six weeks later;
- AVIVAX-L does not give poultry lifelong protection and must be repeated every four months;
- Vaccinate new birds before introducing them to the flock;
- In West Pokot county chicken are reared in the free range system- biosecurity measures include building night housing structures and regular removal of litter;
- After outbreak disinfect chicken house and wait 21 days before bringing in new birds.

Zoonotic potential

- Yes, considered a minor zoonosis
- NDV can be transmitted to humans causing conjunctivitis which is mild and self-limiting, hence considered a minor zoonosis

Notifiable disease

- OIE listed disease
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio surveillance System (KABS)

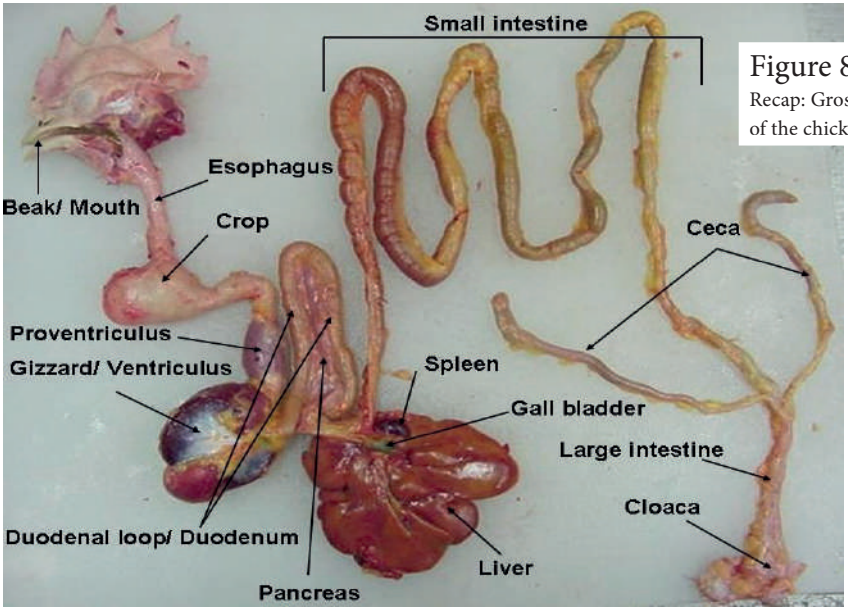


Figure 86
Recap: Gross anatomy of the chicken

Picture source:
https://www.itpnews.com/uploads/2017/03/color%20atlas%20of%20poultry%20disease%20-%2001.pdf?direct_access_media=1

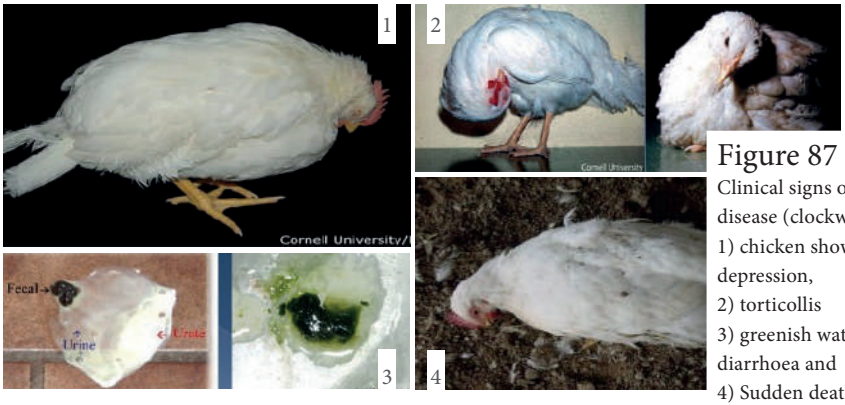


Figure 87

Clinical signs of Newcastle disease (clockwise order):
1) chicken showing severe depression,
2) torticollis
3) greenish watery diarrhoea and
4) Sudden death

Picture source:
Vegad J.L. (2007). A colour atlas of poultry diseases: an aid to farmers and poultry professionals. published by international book distributing co. (publishing division). ISBN: 978-81-8189-130-3

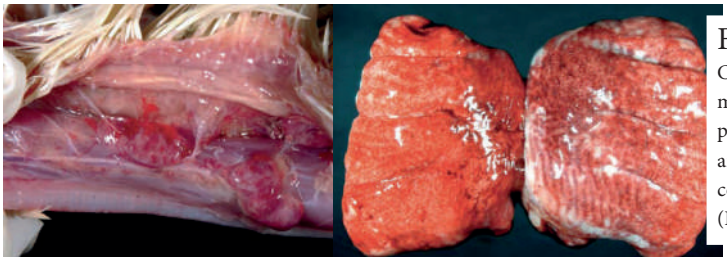


Figure 88

Chicken, neck with marked subcutaneous and peritracheal oedema (Left) and Marked pulmonary congestion and oedema (Right)

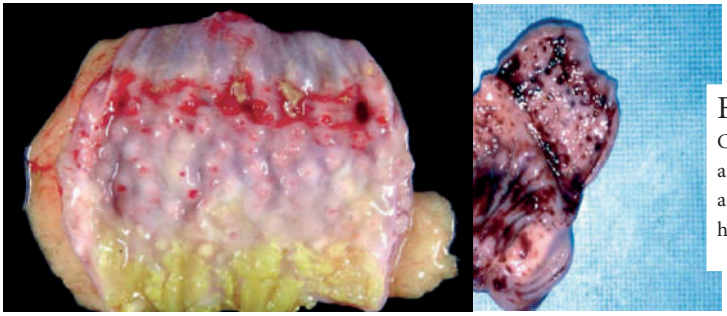


Figure 89

Chicken proventriculus and ventriculus. Severe, acute multifocal mucosal haemorrhages

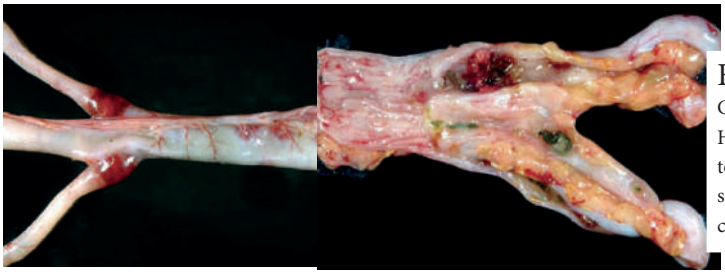


Figure 90

Chicken, cecal tonsils. Haemorrhage of the cecal tonsils visible through the serosa (Left) and necrosis of cecal tonsil (Right)

6.2 | Coccidiosis – Somewo Kiyitagh/ Kmun (Pokot) Loyita/ Kaputi (Sengwer)

Other Name: Coxy

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 don not infect turkeys and vice versa. Their life cycle is complex and takes about 7 to 9 days.

Transmission:
The sporulated oocyst is the infective stage of the life-cycle. Infected, recovered chickens shed oocysts.

- 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.
- Coccidiosis in West Pokot county occurs throughout the year but peak disease incidences are seen during Pengat, the heavy rainfall season that occurs in the months of June to August.
- The apparent morbidity rate of coccidiosis in West Pokot County is estimated to be 43% with a 23% mortality rate and 53% case fatality rate.

Predisposing factors

- Wet houses with litter moisture content exceeding 30% due to rain or leaking waterers.
- Immunosuppression (due to other infections like Marek's disease, IBD, mycotoxins) or low immunity which is common in young birds because of the immaturity of their immune systems
- Environmental and managemental stress such as overstocking, inadequate ventilation.
- Sub-optimal inclusion of anticoccidials or incomplete distribution (poor mixing) in feed.

Main Clinical Signs:
It occurs in two forms:
Caecal coccidiosis is caused by Eimeria tenella. Disease is severe and signs include:

- Bloody diarrhoea
- Reduced weight gain
- High mortality
- Unthriftiness and emaciation

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 per 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: develops in the anterior part of the small intestine. In severe infections, E. mivati may cause reddening of the duodenum because of denuding of the villi. 1-2mm areas of hemorrhage interspersed with white foci 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. In severe cases, the intestinal wall is thickened, and the infected area dilated to 2–2.5 times the normal diameter. The lumen may be filled with blood, mucus, and fluid. Fluid loss may result in marked dehydration. Although the damage is in the small intestine, the sexual phase of the life cycle is completed in the ceca and oocysts may be found only in the ceca. Because of concurrent infections, oocysts of other species may be found in the area of major lesions, thus misleading in diagnosis.
- E. maxima: develops in the small intestine, where it causes dilatation and thickening of the wall; petechial hemorrhage; 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: hemorrhagic 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.
- E. brunetti: is found in the lower small intestine, rectum, ceca, and cloaca. There may be hemorrhages of the mucosa of the distal jejunum and colon. In moderate infections, the mucosa is pale and disrupted but lacking in discrete foci, and may be thickened. In severe infections, coagulative necrosis and sloughing of the mucosa occurs throughout most of the small intestine.
- E. mitis can be distinguished from E. brunetti by finding small, round oocysts associated with the lesion.
- E. praecox: infects the upper small intestine, does not cause distinct lesions. The oocysts are larger than those of E. acervulina and are numerous in affected areas. The intestinal contents may be watery. It is considered to be of less economic importance than the other species.
- E. hagani and E. mivati develops in the anterior part of the small intestine. The lesions of E. hagani are indistinct and difficult to characterize.

Diagnosis:

- Tentative diagnosis is based on flock history, clinical signs and lesions.
- Observe 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

and confirming microscopic stages on necropsy of typical birds from the flock, rather than from culls.

Prevention and Control:

- Prevention is mainly with anti-coccidial drugs, good hygiene, disinfection and sanitation of premises and providing clean feed and water.
- Early emphasis in chemotherapy is centered on the treatment of outbreaks as soon as signs of infection are apparent.
- Prophylactic use is preferred, because most of the damage occurs before signs become apparent and because drugs cannot completely stop an outbreak.
- Antibiotics and increased levels of vitamins A and K are sometimes used in the ration to improve rate of recovery and prevent secondary infections.
- Medication used include: amprolium, clopidol and quinolines (eg, decoquinate, methylbenzoquate), folic acid antagonists like the sulfonamides, 2,4-diaminopyrimidines, and ethopabate, halofuginone hydrobromide (though coccidia may become resistant after extended exposure, ionophores, nitrobenzamides (eg, dinitolmide), robenidine, roxarsone, diclazuril 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 91

Pictorial description of sites parasitized by Eimeria tenella (A), E. necatrix (B), E acervuline (C), E brunetti (D), E maxima (E), and E mitis (F). (Illustration by Dr. Gheorghe Constantinescu).

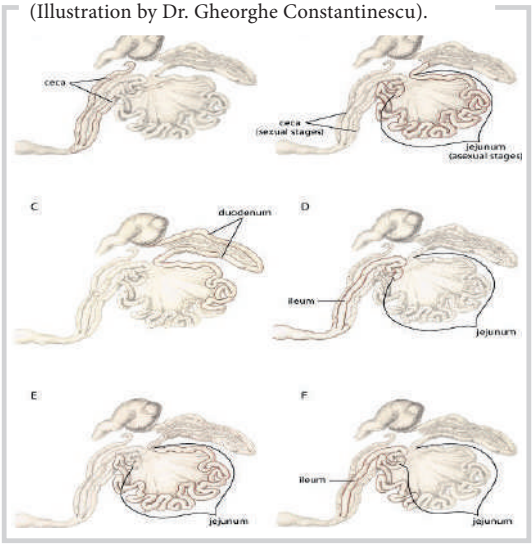
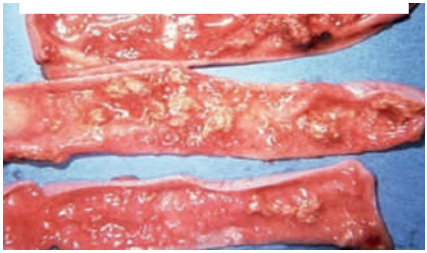


Figure 92

Coagulative necrosis and sloughing of intestinal mucosa as a result of severe infection with Eimeria brunetti



Picture source:

All Pictures sourced from: A colour atlas of poultry diseases: An Aid to Farmers and Poultry Professionals by J.L. VEGAD, 2007

6.3 | Fowl pox –Moyoi/ Kupoy-po kokoroch

Other Names: 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 West Pokot County the disease has an estimated apparent morbidity rate is 22% with a mortality rate of 12% and case fatality rate of 55%.
- The disease in the county occurs throughout the year but it is most common during the months of June to August October which is the heavy rainy season locally known as Pengat.

Predisposing factors:
- Presence of mosquitoes, hence common during rainy seasons.
- Contaminated environment; remember virus can survive in dried scabs for months or several years

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. Most lesions are found in the mouth, but may also be present in the larynx, trachea, and oesophagus. 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 dies suddenly from asphyxiation (lack of oxygen). Lesions in the nares (nose) give rise to nasal discharge, while those on the conjunctiva to eye discharge.

Fowl pox results in poor weight gain resulting in retarded growth in young chicken. Egg production drops in layers. Mortality is low with the cutaneous form but may be slightly higher with the diphtheritic form.

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.
- In the diphtheritic form, lesions develop on the

mucous membranes of the mouth, oesophagus, pharynx, larynx, and trachea. Caseous patches firmly adherent to the mucosa of the larynx and mouth or proliferative masses may develop. Mouth lesions interfere with feeding.
- Tracheal lesions cause difficulty in respiration. In cases of systemic infection caused by virulent fowl pox virus strains, lesions may be seen in internal organs. More than one form of the disease, that is cutaneous, diphtheritic, and/or systemic, may be seen in a single bird.

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.
- Histological examination of affected tissue to confirm presence of intracytoplasmic inclusions (Bollinger bodies) in the respiratory mucosa and skin.

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" indicates lack of potency of vaccine, passive or acquired immunity, or improper vaccination. Revaccination with another serial lot of vaccine may be indicated.
- Isolation of sick birds, good sanitation and hygienic conditions at all times.

Figure 93
Cutaneous form of fowl pox showing (white arrows) scab-like lesions on the combs of chicken

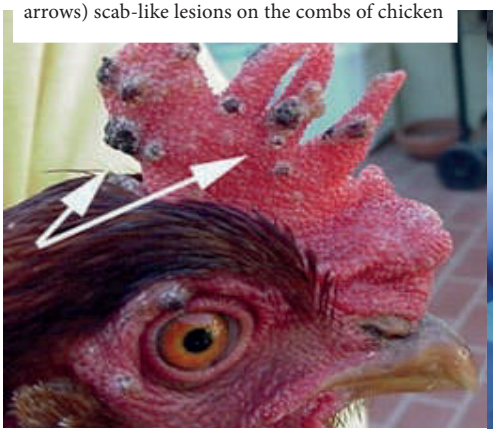
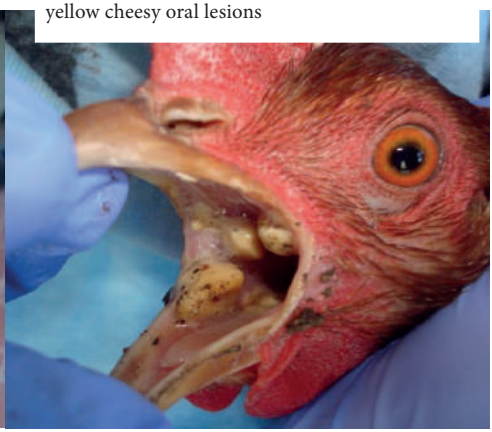


Figure 94
Diphtheric form of fowl pox infection showing yellow cheesy oral lesions



6.4 | Helminthiasis- Mochonto muu/ Ngutianta muu/ Chepturu

Causative agents:
- Mainly nematodes (roundworms) and cestodes (tapeworms) Ascarids are the main round worms that infect chicken. Gapeworms live in the trachea, capillaria live in the intesines, heterakis lives in the ceca, trichostrongyles and tapeworms live in intestines while gizzard worms live in gizzard.

Transmission:
- Direct transmission from bird to bird by ingestion of infective eggs or larvae.
- Indirectly through an intermediate host (insect, snail or slug).
- In West Pokot county the apparent morbidity rate due to helminths is 5% with a mortality rate of 3% and case fatality rate of 60%.
- Helminths impact in the county are mostly seen during the months of January to December which is the dry season locally known as Komoi.

Predisposing factors:
- Poor hygienic conditions leading to pile up of litter
- Lack of treatment or development of resistance to medications

Main Clinical Signs:
- General unthriftiness and inactivity
- Depressed appetite
- Suppressed growth
- In severe cases, may result in death
- Gapeworms cause chickens to gasp with throat extension

Diagnosis:
- Identification of parasites by their morphology or molecular biological methods. Specific recognition of the parasite allows meaningful recommendations for flock therapy and management. Put worms observed during post-mortem in a saline solution and examine under a microscope.
- 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.
- Apply insecticides to soil and litter when premises are unoccupied to interrupt the life cycle of the parasite by destroying its intermediate host.

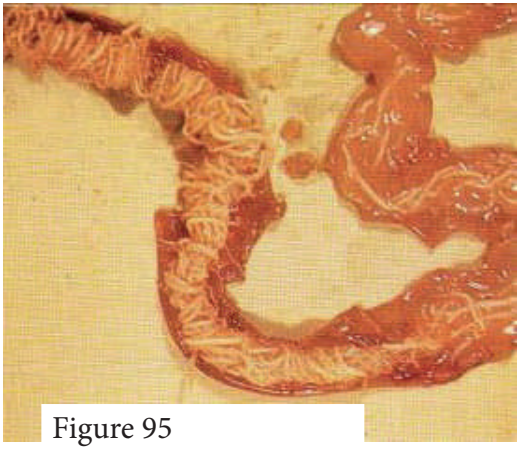


Figure 95
Chicken intestinal tract impacted by Ascarid worms

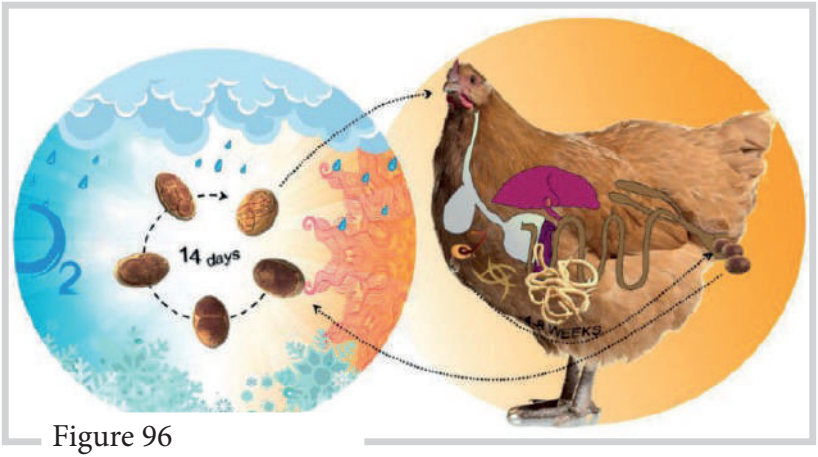


Figure 96
Life cycle of the large round worm of chicken Ascarid galli

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

6.5 | Ectoparasites- Kiyominto onto / Kiyominto menyo

There are a number of ecto-parasites that infest chicken as shown in the table:

English name	Local name	Causative agent
Fleas	Kmit	The main flea pest is the sticktight flea; Echidnophaga gallinacean. Attach on the skin of the head
Lice	Sirr	Chewing lice; Order Mallophaga. They feed on feathers, feather debris, skin scales and other dermal scurf.
Ticks	Cheptapalal	Argasid Ticks (soft-shelled ticks) are most common. Argasid ticks are nocturnal feeders and favour the soft un-feathered skin beneath the wings
Body or Scaly leg Mites	Ng'atwoi	Ornithonyssus spp remain on chickens permanently; while Dermanyssus mites parasitize chickens nocturnally Knemidocoptes mutans- scaly leg mites

- In West Pokot county the apparent morbidity rate due to external parasites in chicken is estimated to be 29% with morbidity rates of 14% and case fatality rates of 48%.
- External parasitism in chicken flocks of West Pokot county occurs throughout the year with seasonal peaks seen during the dry season (Komoi) during the months of January to March.

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 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 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. Ensure to apply to base of feathers in case of mite infestation.
- 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 shouls also be dusted with the acaricide powder.
- Quarantine all new birds and apply acaricide powder before allowing them to mix with the flock.
- 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 solution Ectomin solution. Always follow manufacturer's instructions on use to ensure birds are not poisoned.

Figure 97

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

6.6 | Infectious coryza- Tasus po konyin

Other name: Fowl Coryza

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 West Pokot county infectious coryza has the apparent highest morbidity rate at 9% with a mortality rate of 3% and case fatality rate of 33%.
- The disease occurs throughout the year but incidence peaks are seen during the dry season locally known as Komoi during the months of January to March.

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. Sulfonamides, 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

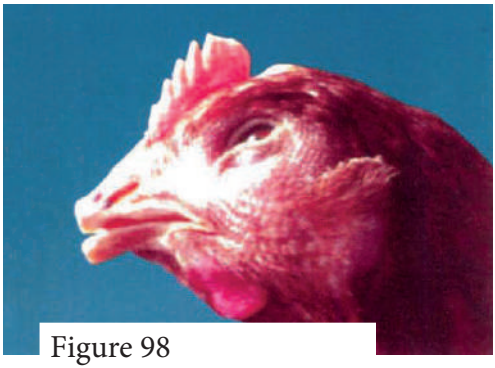


Figure 98
Chicken with swollen facial tissue due to infectious coryza

6.7 | Fowl Cholera- Tuw mot/ Cheptuw mot

Other names: Avian cholera, Avian Pasteurellosis, or Avian Haemorrhagic Septicaemia

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.
- Direct transmission can also occur through the nose, eye and through wounds.
- The bacteria can also be spread indirectly by humans – clothing or footwear.

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
- 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.

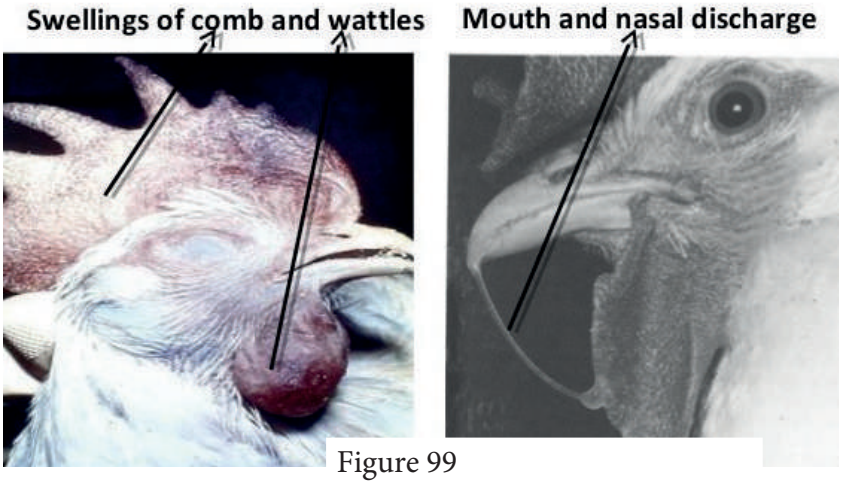


Figure 99
Chicken infected with fowl cholera

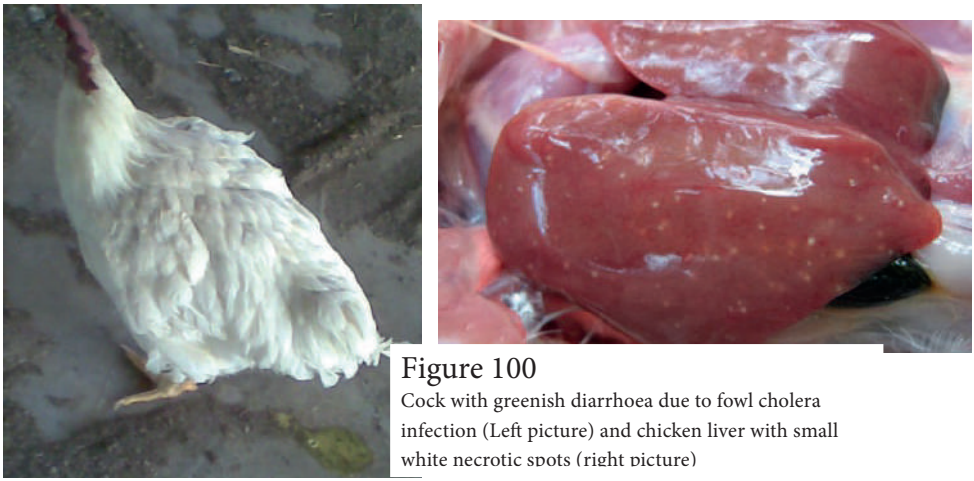


Figure 100
Cock with greenish diarrhoea due to fowl cholera infection (Left picture) and chicken liver with small white necrotic spots (right picture)

Picture source:
https://www.veterinariadigital.com/en/articulos/main-avian-diseases-found-in-india/

6.8 | Chronic Respiratory Disease (CRD)- Cheptakat

Other names: Avian cholera, Avian Pasteurellosis, or Avian Haemorrhagic Septicaemia

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.
- In West Pokot county the apparent morbidity rate is 18% with a mortality rate of 10%and case fatality rate of 56%.
- CRD in the county is common during the heavy rainfall season Pengat that occurs during the months of June to August.

Clinical Signs

- Sniffling, sneezing, coughing and other signs of respiratory distress.
- 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.

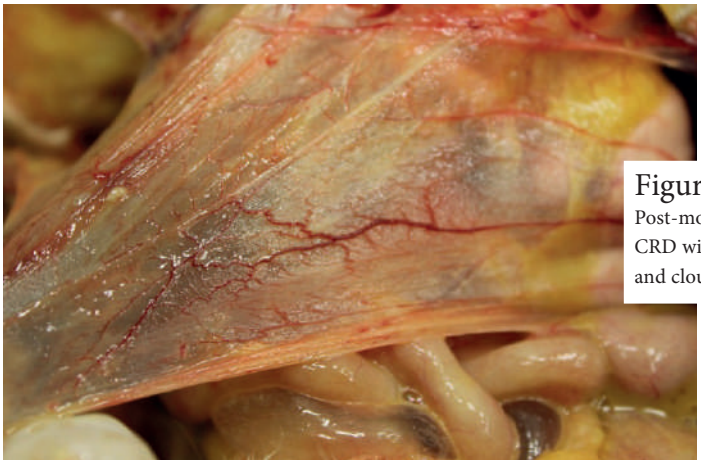


Figure 101
Post-mortem lesions of a bird with CRD with abdominal sac thickened and cloudy with blood vessels visible

There are many causes of airsacculitis in chickens and turkeys, but M gallisepticum should always be considered as a likely differential diagnosis.

6.9 | Infectious bronchitis (IB)- Cheptakat

Aetiology

- Infectious bronchitis virus (IBV) is an avian gammacoronavirus that only causes disease in chickens,
- The virus has many antigenic types that can circulate in a given region. Some IBV types are widespread, whereas others are regional.
- IB is an acute, highly contagious upper respiratory tract disease in chickens. In addition to respiratory signs there is decreased egg production and egg quality. Some virus strains can also cause nephritis.
- This virus causes fibrinous and haemorrhagic infiltration of the respiratory tract, so the presence of blood in the trachea, sinuses and oral cavity makes breathing difficult.

Transmission

- IBV is shed by infected chickens in respiratory discharges and faeces, and it can be spread by aerosol, ingestion of contaminated feed and water, and contact with contaminated equipment and clothing. Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks after infection. The incubation period is generally 24–48 hours, with the peak in excretion of virus from the respiratory tract lasting 3–5 days after infection.

Clinical signs

The severity of disease and the body systems involved are influenced by:

- Strain of the virus, age, immune status, diet, cold stress and presence or absence of coinfection with Mycoplasma gallisepticum, M synoviae, Escherichia coli, and/or Avibacterium paragallinarum that can exacerbate the disease.
- Morbidity for flocks affected by infectious bronchitis is typically 100%.
- Chicks may cough, sneeze, and have tracheal rales for 10–14 days. Conjunctivitis, dyspnoea and facial swelling may occur if there is concurrent bacterial infection of the sinuses. Chicks may appear depressed and huddle under heat lamps. Feed consumption and weight gain are reduced. Infection with nephropathogenic strains can cause initial respiratory signs, then later depression, ruffled feathers, wet droppings, greater water intake and death.
- In layers, egg production may drop by as much as 70%, and eggs are often misshapen, with thin, soft, wrinkled, rough, and/or pale shells, and can be smaller and have watery albumen. Egg production and egg quality can return to normal, but this may take up to 8 weeks.
- In most outbreaks, mortality is approximately 5%, although mortality rates can be as high as 60% when disease is complicated by concurrent

bacterial infection or when nephropathogenic strains induce interstitial nephritis in chicks. Infection of chicks may cause permanent damage to the oviduct, resulting in layers or breeders that never reach normal levels of production (false layer syndrome).

Post-mortem lesions

- The trachea, sinuses, and nasal passages contain serous, catarrhal, or caseous exudates, and the air sacs have a foamy exudate initially, progressing to cloudy thickening. If complicated by infection with E coli, there may be caseous airsacculitis, perihepatitis, and pericarditis.
- Young birds will have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and regression of the ovaries.
- Infection with nephropathogenic strains results in swollen, pale kidneys, with the tubules and ureters distended with urates; in birds with urolithiasis, the ureters may be distended with urates and contain uroliths, and the kidneys may be atrophied.

Diagnosis

- Detection of rising antibody titers by ELISA or HI testing and virus detection and typing using RT-PCR and sequencing.
- Laboratory confirmation is required for diagnosis of respiratory forms of infectious bronchitis because of similarities to mild forms of disease caused by agents such as Newcastle disease virus, infectious laryngotracheitis virus , mycoplasmas , A paragallinarum, and Ornithobacterium rhinotracheale.

Treatment and Prevention

- 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

Figure 102
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

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.

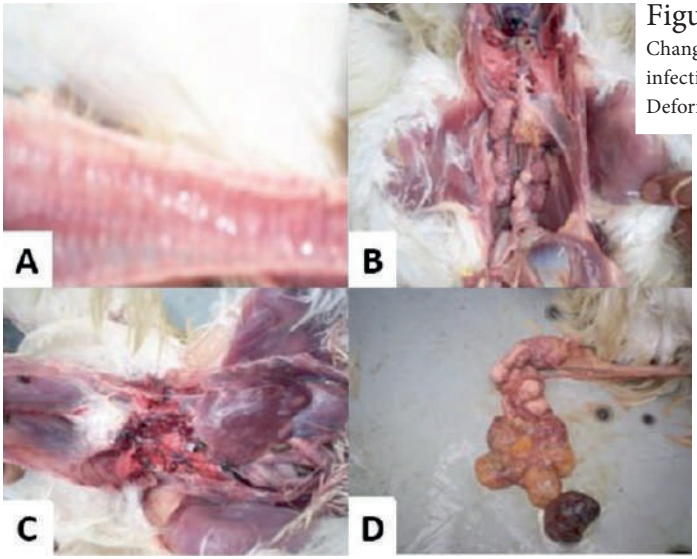


Figure 103

Changes in Laid eggs due to IBV infection N: Normal Egg; D: Deformed Egg; SL: Shell less Egg

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

6.10 | Gumboro disease (Infectious Bursal Disease –IBD)- Pchokek

Aetiology and Transmission

- Acute viral infection, caused by a birna virus that mainly affects the Bursa of Fabricius, destroying the immature B lymphocytes of young chicks.
- IBD is common in birds between 3 and 6 weeks old.
- infectious bursal disease virus; IBDV) is shed in the droppings and transferred from house to house by fomites.
- The virus is very stable and difficult to eradicate from premises.

Clinical signs

- Infections before 3 weeks of age are usually subclinical. Chickens are most susceptible to clinical disease at 3–6 weeks of age when immature B cells populate the bursa and maternal immunity has waned.
- Early subclinical infections cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen. The humoral (B cell) immune response is most severely affected; the cell-mediated (T cell) immune response is affected to a lesser extent. Chickens immunosuppressed by early IBDV infections do not respond well to vaccination and are predisposed to infections with normally non-pathogenic viruses and bacteria
- Chickens exhibit severe prostration, incoordination, watery diarrhoea, soiled vent feathers, vent picking, and inflammation of the cloaca.
- Flock morbidity is typically 100%, and mortality can range from 5% to greater than 60% depending on the strain of virus and breed of chicken.
- Mortality is typically higher in layer breeds compared with broiler chickens. Recovery occurs in <1 week, and broiler weight gain is delayed by 3–5 days. The presence of maternal antibody will modify the clinical course of the disease.

Post-mortem lesions

- Cloacal bursa is swollen, oedematous, yellowish, and occasionally haemorrhagic.
- Congestion and haemorrhage of the pectoral and leg muscles can also occur.
- Chickens that have recovered from IBDV infections have small, atrophied, cloacal bursas due to the destruction and lack of regeneration of the bursal follicles.

Diagnosis

- Gross lesions in the cloacal bursa
- Confirmed by microscopic analysis of the bursa for lymphocyte depletion in the follicles.
- Molecular diagnostic assays are often used to identify IBDV genome strains in bursa tissue.

Treatment and control

- There is no treatment as it is a viral disease.
- Rigorous disinfection of contaminated farms after depopulation has achieved limited success.
- Live vaccines of chicken embryo or cell-culture origin and of varying low pathogenicity can be administered by eye drop, drinking water, or SC routes at 1–21 days of age. Replication of these vaccines and thus the immune response can be altered by maternal antibody, although the more virulent vaccine strains can override higher levels of maternal antibody.

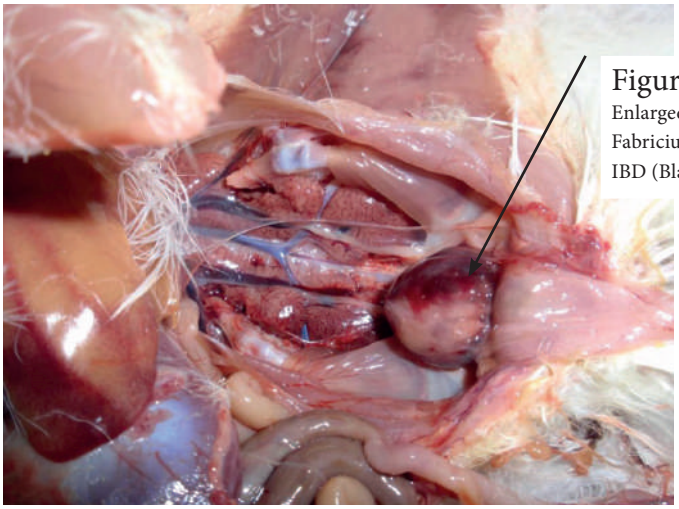


Figure 104
Enlarged, haemorrhagic bursa of Fabricius in a chicken infected with IBD (Black Arrow)

Further Reading:

1. A colour atlas of poultry diseases: An Aid to Farmers and Poultry Professionals by J.L. VEGAD, 2007
2. ASA Handbook on Poultry Diseases 2nd Edition by Simon M. Shane, 2005
3. Diseases of Poultry, 13th ed. / editor-in-chief, David E. Swayne; associate editors, John R. Glisson [et al.]., 2013
4. https://www.extension.iastate.edu/smallfarms/take-action-against-external-parasites-and-pests-poultry
5. https://farminence.com/internal-parasites-in-chickens/
6. https://www.nadis.org.uk/disease-a-z/poultry/diseases-of-farmyard-poultry/part-4-external-and-internal-parasites-of-chickens/
7. https://www.slideshare.net/VetAbdulrhmanSubhi/newcastle-disease-31243174?next_slideshow=1
8. MSD Manual: Veterinary Manual online: https://www.msdvetmanual.com/
9. Poultry Disease Manual by Michael A. Davis, The Texas A&M University System

Annex:

1: Vaccination schedule for Indigenous chicken

Date of Hatching:_____

INDIGENOUS CHICKEN VACCINATION PROGRAM

AGE	VACCINE	MODE OF ADMINISTRATION	REMARKS
Day old	Mareks	Subcutaneous	Mainly for commercial hatcheries
Day 10	Gumboro (1st dose)	Drinking water	
Day 18	Gumboro (2nd dose)	Drinking water	
3 Weeks	Newcastle disease (1st dose)	Eye drop or Drinking water	
3 Weeks (In hot spot areas) 6 Weeks (Other areas)	Fowl pox	Wing web stab	
8 Weeks	Newcastle disease (2nd dose)	Eye drop or Drinking water	
	Fowl typhoid	Intramuscular injection	
18 Weeks	Newcastle disease (3rd dose at point of lay)	Eye drop or Drinking water	Repeat every 3 months
19 Weeks	De-worming	Drinking water	Repeat every 3 months

Notes:

- **NEVER vaccinate sick chicken**
- **Consult your veterinary/livestock staff for detailed vaccination programs in your area**

Annex:

2: West Pokot Livestock Vaccination calendar

Key: Cattle, Camel, Goats, Sheep, Chicken

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sept	Oct	Nov	Dec	Notes
CBPP													Annual booster CONTAVAX TM
ECF													Infection and Treatment Vaccine Confers lifelong immunity
FMD													FOTIVAX TM- Twice a year or
BQ													Annual booster; Vaccinate calves 2 months old and naïve ewes twice, 4 weeks apart. The 2nd dose for ewes be done 2 months before lambing
LSD													Annual booster- LUMPIVAXTM (KEVEVAPI)
Haemorrhagic Septicaemia													No Vaccine available for Camel in Kenya only for Sheep and Cattle (KEVEVAPI)- PASTREVAXTM
Camel Pox													Not available in Kenya
Camel Orf													Not available in Kenya
CCPP													Goats over 3 months old, repeat every 6 months CAPRIVAX (KEVEVAPI)
PPR													Annual booster for animals 4 months & older with PESTEVAX (KEVEVAPI)
SGP													For sheep/goats over 5 weeks of age; unless in outbreak up to 3 weeks old. Annual vaccination recommended S & G VAX (KEVEVAPI)
ORF													For does and ewes at least 7 weeks before kidding or lambing or kids and lambs 6 weeks & older; annual vaccination with ORFVAX (KEVEVAPI)
Enterotoxaemia													Target ewes in 3rd gestation; booster after 4 weeks
Newcastle disease													Vaccinate at day 4, repeat week 4, week 16, then every 4 months
Fowl pox													Vaccinate early in life, repeat after 12-16 weeks
Infectious coryza													No vaccine available in Kenya

NB: Follow manufacturer's instructions on vaccine use and for acceptable vaccine combinations

Annex:

3: Zoonotic diseases- Somewut cho kinomtoy

- A zoonosis (zoonotic disease or zoonoses -plural) is an infectious disease that is transmitted between species from 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- Tiompo ogha/ Tiompo parak/ Tiompo yum

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.
- Conduct Participatory disease surveillance (PDS) to search for active disease and identify the hot spot areas and community at highest risk of disease. PDS also allows passing of extension health messages that stop practices that are spreading the disease during outbreaks.
- 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.

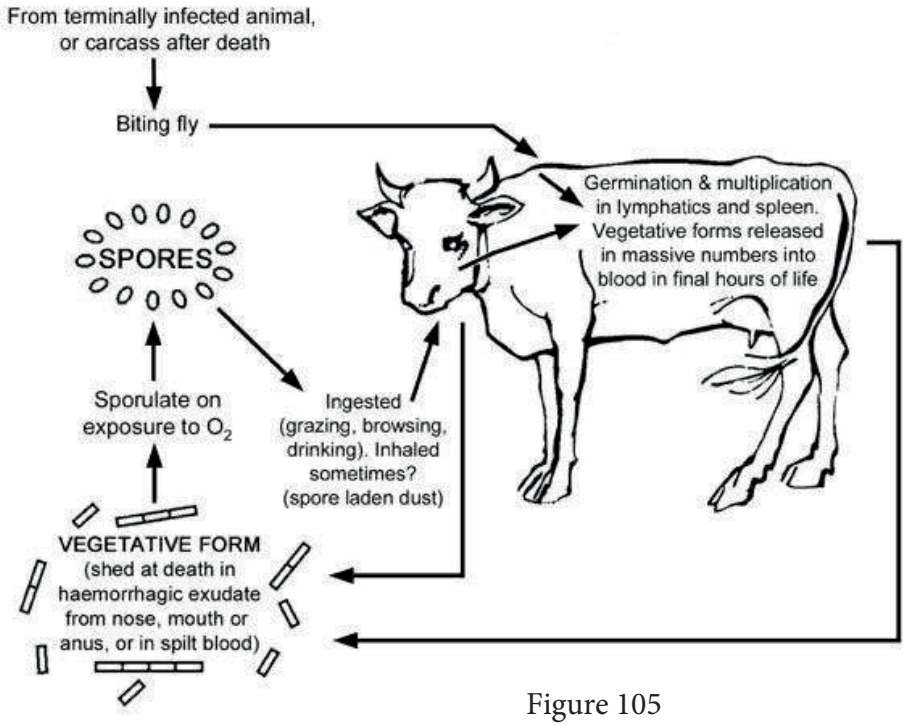


Figure 105
Anthrax Transmission cycle in cattle

Picture source:
https://infonet-biovision.org/AnimalHealth/Diseases-Killing-very-fast-Killer-diseases

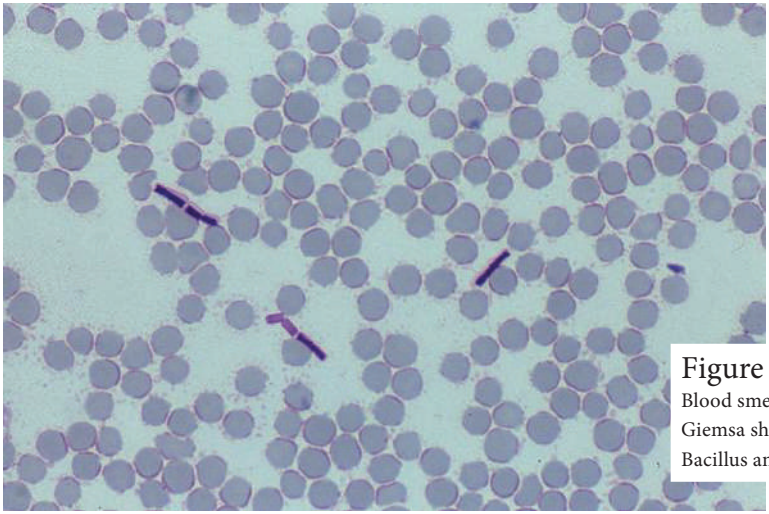


Figure 106
Blood smear stained with Giemsa showing characteristic Bacillus anthracis bacteria

Picture source:
https://repository.up.ac.za/handle/2263/32656

Rabies- T9/Hoy po Kukui/ Chepoiynono kukui

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.

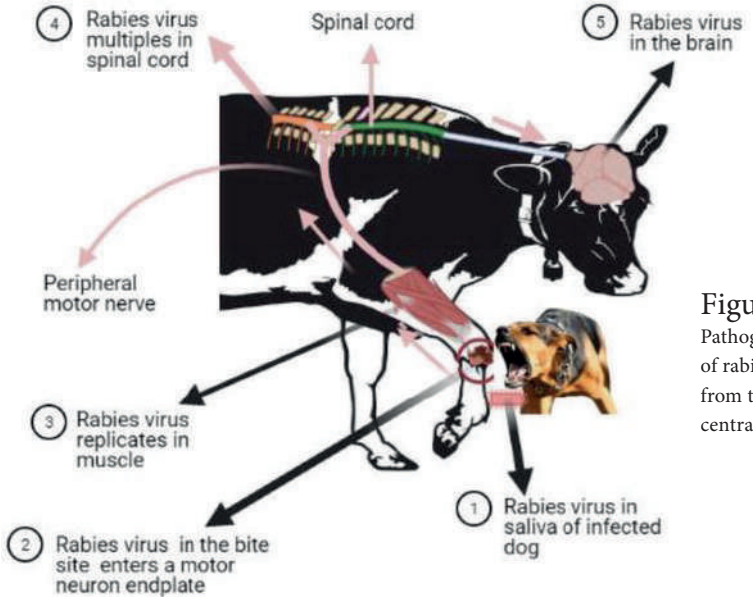


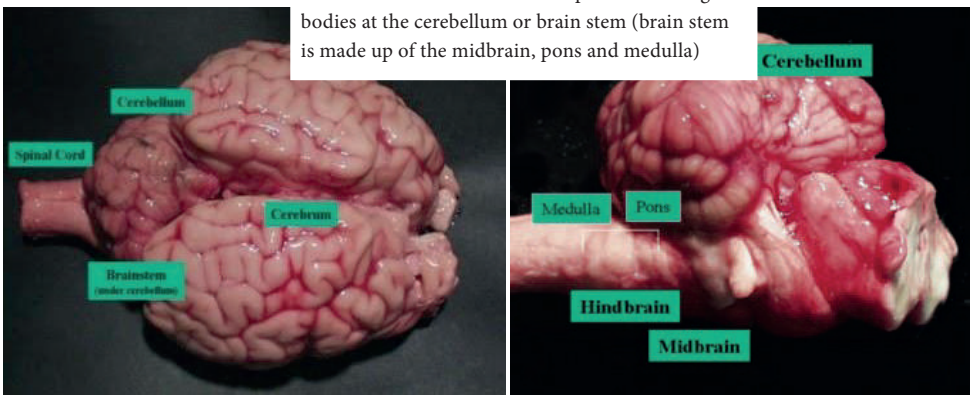
Figure 107

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

Figure 108

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)



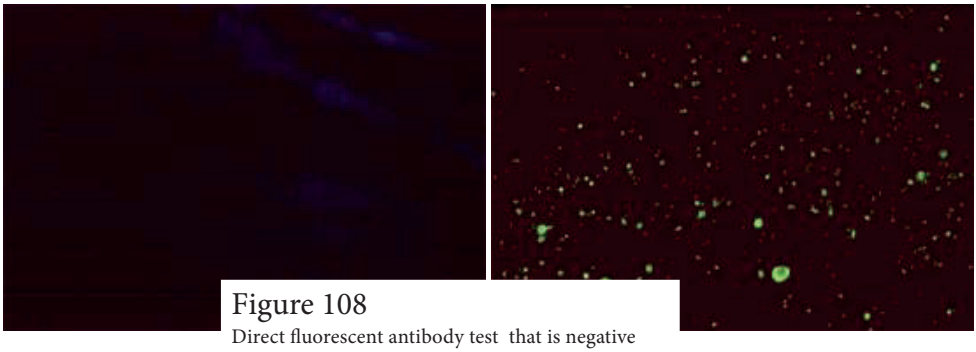


Figure 108
Direct fluorescent antibody test that is negative (Left) and positive (Right) for the rabies virus

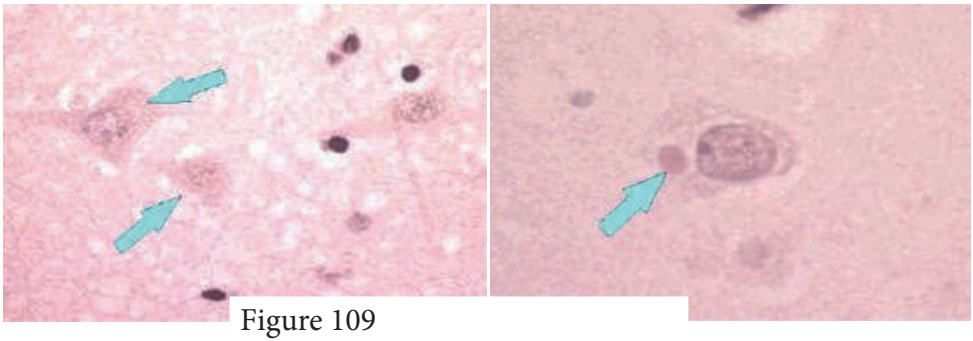


Figure 109
Histology brain tissue with neuron without Negri bodies (right) and with Negri bodies (left)

Picture source:
http://uwrfbio353koob.blogspot.com/p/histology.html

Rift Valley Fever- Kilay po mpoghogh

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 interepidemic 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 interepidemic survival strategy of 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 serious 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

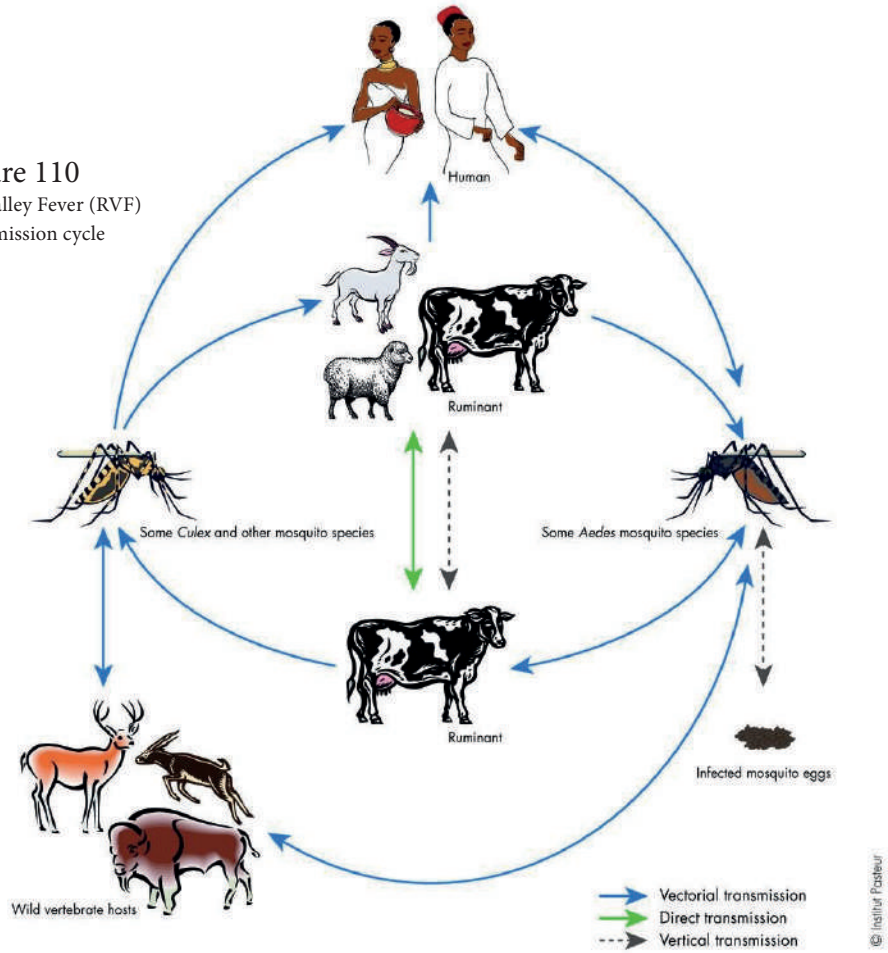
- There is no treatment of sick animals, supportive therapy to animals with clinical signs. Care should be taken not to spread infection through re-use of hypodermic needles or self-infection during treatment of the sick animals.
- Conduct Participatory disease surveillance (PDS) Surveillance during 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.

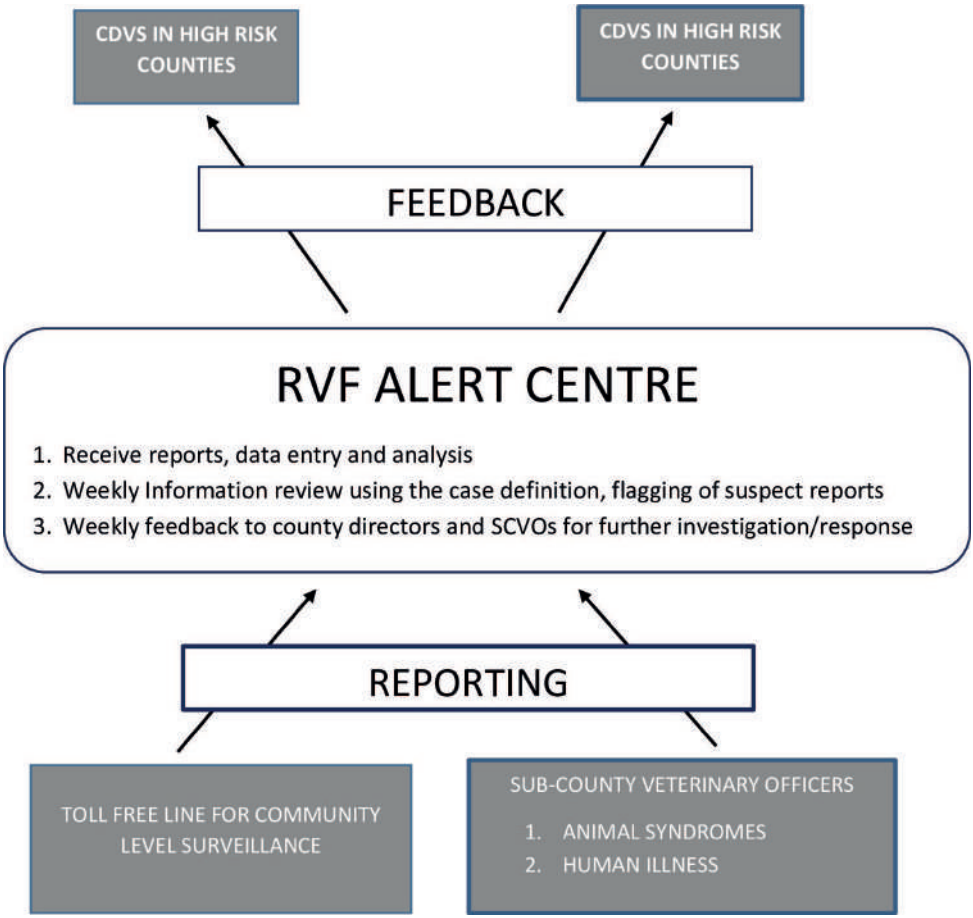
Figure 110
Rift Valley Fever (RVF)
Transmission cycle



Picture source:
https://veterinaryresearch.biomedcentral.com/articles/10.1186/1297-9716-44-78#Fig2

Figure 111

Illustration of RVF surveillance system conducted in Kenya between November 2015 and February 2016.



CDVS = County Director of Veterinary Services, RVF = Rift Valley Fever, SCVO = Sub-County Veterinary Officers

Picture source:
https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0006353

Figure 112

RVF infection in a cow (Left) aborted foetus and placenta with oedema and haemorrhages (hydramnion) and aborted emphysematous foetus with 'meconium staining (left)

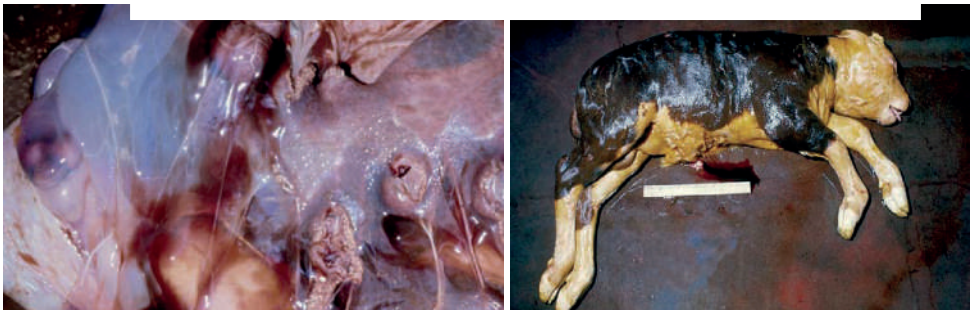
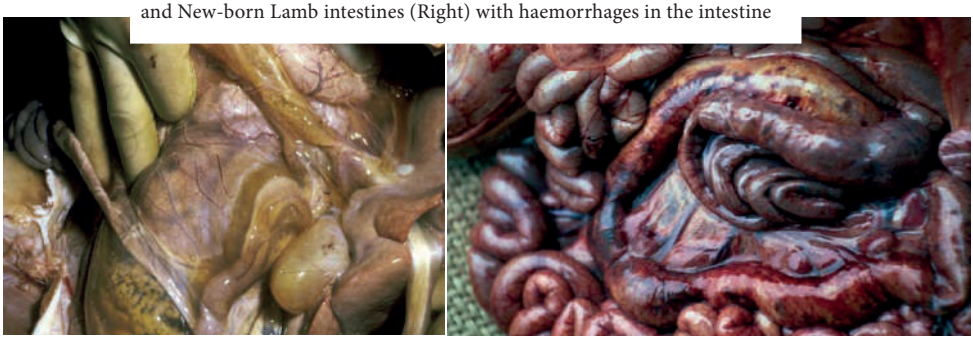


Figure 113

Small intestine of a cow (Left) with marked mesenteric and serosal oedema and New-born Lamb intestines (Right) with haemorrhages in the intestine



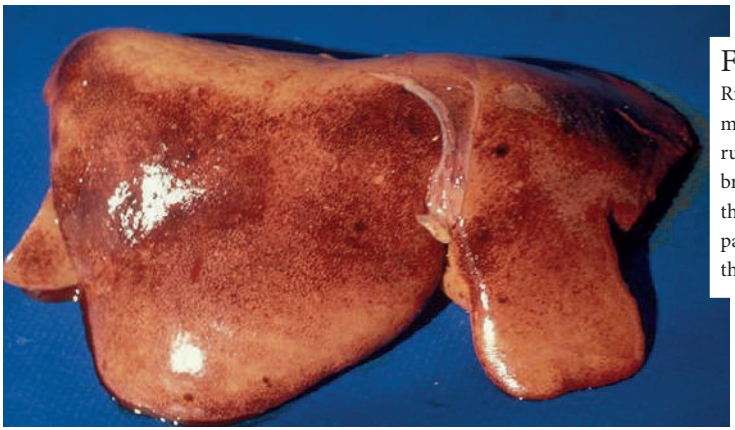


Figure 114

Rift Valley fever (RVF) markedly enlarged ruminant liver, yellow-brown discolouration of the organ, and congested patches scattered throughout the organ

Picture source:
https://veterinaryresearch.biomedcentral.com/articles/10.1186/1297-9716-44-78#Fig2

Brucellosis - Somewo cho

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 (OIE 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 OIE.

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.
- Natural transmission occurs by ingestion of organisms, which are present in large numbers in aborted foetuses, foetal membranes, and uterine discharges. Cattle may ingest contaminated feed and water or may lick contaminated genitals of other animals. Venereal transmission by infected bulls to susceptible cows appears to be rare. Transmission may occur by artificial

insemination when Brucella-contaminated semen is deposited in the uterus but, reportedly, not when deposited in the midcervix. Brucella may enter the body through mucous membranes, conjunctivae, wounds, or intact skin in both people and animals.
- Brucella have been recovered from foetuses and from manure that has remained in a cool environment for >2 mo. Exposure to direct sunlight kills the organisms within a few hours.

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
- Diagnosis is based on bacteriology or serology. B abortus can be recovered from the placenta but more conveniently in pure culture from the stomach and lungs of an aborted foetus. Most cows cease shedding organisms from the genital tract when uterine involution is complete. Foci of infection remain in some parts of the reticuloendothelial system, especially supramammary lymph nodes, and in the udder. Udder secretions are the preferred specimens for culture from a live cow.
- Serum agglutination tests have been the standard diagnostic method. Agglutination tests may also detect antibodies in milk, whey, and semen. An ELISA has been developed to detect antibodies in milk and serum. When the standard plate or tube serum agglutination test is used, complete agglutination at dilutions of 1:100 or more in serum samples of nonvaccinated animals, and of 1:200 of animals vaccinated at 4–12 months of age, are considered positive, and the animals are classified as reactors. Other tests that may be used are complement fixation, rivanol precipitation, and acidified antigen procedures.

Screening Tests for eradication process:
- The Brucella milk ring test (BRT) has effectively located infected dairy herds, but there are many false-positives. The brucellosis status of dairy herds in any area can be monitored by implementing the BRT at 3- to 4-months intervals. Milk samples from individual herds are collected at the farm or milk processing plant. Cows in herds with a positive BRT are individually blood tested, and seropositive cows are slaughtered.
- Non-dairy and dairy herds in an area may also be screened for brucellosis by testing serum samples collected from cattle destined for slaughter or replacements through intermediate and terminal markets, or at abattoirs. Reactors are traced to the herd of origin, and the herd is tested. The cost of identifying reactors by this method is minimal compared with that of testing cattle in all herds. Screening tests, including the brucellosis card (or rose bengal) test and plate test, may be used in markets and laboratories to identify presumptively infected animals, thus reducing the number of more expensive and laborious diagnostic tests.
- Brucellosis-free areas can be achieved and maintained, effectively and economically, by using the BRT on dairy herds and through market cattle testing.
- Another supplemental diagnostic procedure is to test milk samples from individual udder quarters by serial dilution BRT, which can be used to detect chronic infection in udders of cows that may have equivocal serum test reactions
- Herds must be tested at regular intervals until two or three successive tests are negative.

Control

- The greatest danger in brucellosis control is from replacement animals. Replacement animals should be vaccinated calves or non-pregnant heifers. If pregnant or fresh cows are added, they should originate from brucellosis-free areas or herds and be seronegative. Replacements should be isolated for 30 days and retested before being added to the herd.
- Vaccination of calves with B abortus Strain 19 or RB51 increases resistance to infection. Resistance may not be complete, and some vaccinated calves may become infected, depending on severity of exposure. A small percentage of vaccinated calves develop antibodies to Strain 19 that may persist for years and can confuse diagnostic test results. To minimize this problem, calves in the USA are mostly vaccinated with a vaccine of Strain RB51. It is a rough attenuated strain and does not cause production of antibodies, which are detected by most serologic tests.
- Whole-herd adult cattle vaccination using Strain 19 or RB51 has been practiced in certain high-incidence areas and selected herds in the USA with much success.

Vaccination as the sole means of disease control has been effective. Reduction in the number of reactors in a herd is directly related to the percentage of vaccinated animals.

In Kenya, livestock keepers do not vaccinate against Brucella because most only invest in adult animals not calves.

- Methods of prevention include health education to reduce occupational and food-borne risks, including pasteurization of all dairy products. However, education campaigns have never resulted in fully eliminating the risks of infection, and the ultimate prevention of human infection remains elimination of the infection among animals. This can be achieved by a combination of vaccination of all breeding animals to reduce the risks of abortion and raise herd immunity, followed by elimination of infected animals or herds by segregation and slaughter.

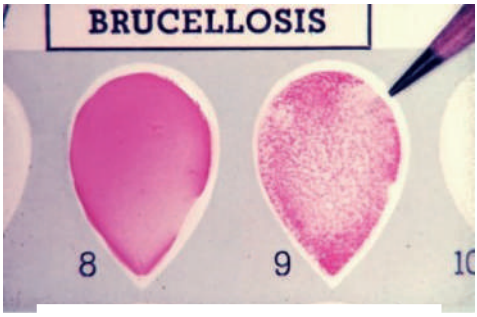


Figure 115
Rose Bengal or card test showing negative (left) and positive (right) reactions

Figure 116
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://oiebulletin.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:

4: 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 OIE 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.

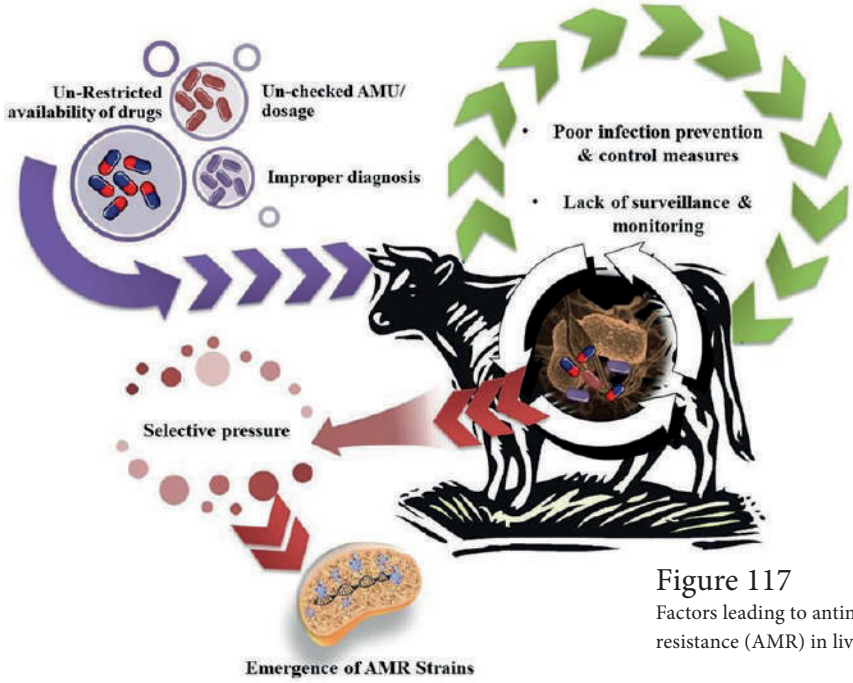


Figure 117
Factors leading to antimicrobial resistance (AMR) in livestock

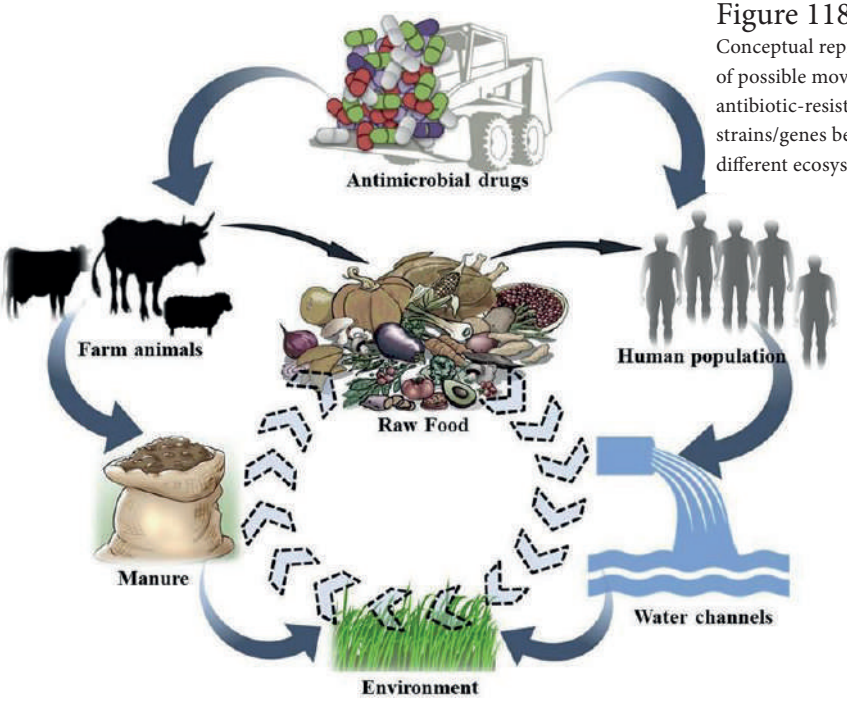


Figure 118
Conceptual representation of possible movement of antibiotic-resistant bacterial strains/genes between different ecosystems

Figure 119

Critically important antibiotics used in human medicine, their use are restricted or controlled in livestock

Critically Important		Highly Important	Important	Currently not used in humans
Highest Priority	**High Priority**			
Cephalosporins (third, fourth, fifth gen.)	Aminoglycosides	Amphenicols	Aminocyclitols	Bambermycins
Fluro and other quinolones	Ansamycins	Cephalosporins (first & second gen.)	Cyclic polypeptides	Quinoxalines
Glycopeptides	Carbapenems and other penems	Lincosamides	Nitrofurantoin	Ionophores
Macrolides and ketolides	Glycylcylines	Penicillins (anti-staphylococcal)	Nitroimidazoles	Orthosomycins
Polymyxins	Lipopeptides	Pseudomonic acids	Pleuromutilins	
	Monobactams	Riminofenazines		
	Oxazolidinones	Steroid antibacterials		
	Penicillins (natural aminopenicillins and antipseudomonal)	Streptogramins		
	Phosphonic acid derivatives	Sulfonamides		
	Tuberculosis and other mycobacterial drugs	Sulfones		
		Tertacyclines		

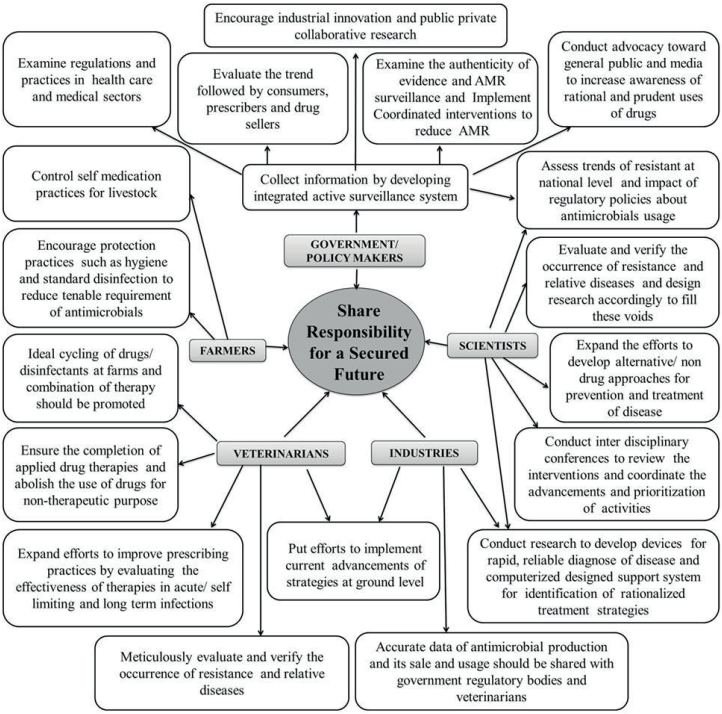


Figure 120

Framework for collaborative approaches to mitigate antimicrobial resistance (AMR)

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

Infographic source:
https://www.frontiersin.org/articles/10.3389/fvets.2017.00237/full

- 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) (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. These variables are dependent both on the patient and on the physicochemical features and other properties of the antimicrobial drug.
- This chemical and physiological processing by the body, as well as the lipid solubility and other chemical properties of the drug, affect the ability of the drug to penetrate infected tissues and make contact with pathogens that reside in interstitial fluids or host cells. The early exposure of pathogenic bacteria to effective drug concentrations for an optimum period of time is directly associated with the clinical success of antimicrobial drug therapy.
- 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). One of many factors to predict a favourable clinical outcome of the potential action of antimicrobial chemicals may be provided using in vitro bactericidal/bacteriostatic data. Bactericidal drugs have a minimum bactericidal concentration (MBC) necessary to kill bacteria, while bacteriostatic antibiotics need to comply with a minimum inhibitory concentration (MIC) in order to function effectively. 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 also differ since they function alongside 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 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. At low concentrations, the efficacy of bactericidal drugs may be reduced to merely bacteriostatic effects. On the other hand, bacteriostatic drugs can kill bacteria at high concentrations.
- 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 to limit 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 (Main reason why dosage regime and length should be followed).

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
Chloramphenicol **	Bacteriostatic- – inhibit protein synthesis	Broad spectrum	Not indicated for use in ruminants
Fluoroquinolones*** Enrofloxacin, ciprofloxacin, Danofloxacin, Difloxacin, Ibafloxacin, Marbofloxacin, Pradofloxacin, Orbifloxacin	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
Glycopeptides Vancomycin, teicoplanin, avoparcin	Bactericidal- Inhibition of cell wall synthesis	Bactericidal- Inhibition of cell wall synthesis	Vancomycin: "last resort" drug in human medicine should not be used in animals. Avoparcin: used extensively for growth promotion of chickens and pigs
Lincosamides Lincomycin, Clindamycin, and Pirlimycin	Both- Inhibits protein synthesis bactericidal or bacteriostatic, depending on the drug concentration, bacterial species and concentration of bacteria	Broad- active against Gram-positive bacteria, most anaerobic bacteria, and some mycoplasma Clindamycin has an excellent activity against anaerobes	Swine: Lincomycin used in prevention and treatment of dysentery and sometimes mycoplasma infections Cattle: mainly used as intramammary infusion in mastitis (pilrimycin) Poultry: control of mycoplasmosis and necrotic enteritis

Class of antibiotic	Mode of action	Spectrum of activity	Veterinary use in ruminants
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 and Spiramycin for Mycoplasma infections and growth promotants Tilmicosin: against Mannheimia, Actinobaciullus, Pasteurella, Mycoplasma.
Class of antibiotic	Mode of action	Spectrum of activity	Veterinary use in ruminants
Polymixins Polymixin B, colistin (Polymixin E)	Bactericidal- Inhibits cell membrane function	Narrow spectrum affecting primarily Gram-negative bacteria	Cattle colibacillosis and salmonellosis in calves, mastitis
Rifamycins Rifampin, Rifabutin, Rifapentine	Bactericidal- Inhibits nucleic acid synthesis	Broad spectrum; also has antiviral and antifungal activity	Rifampin is used as a first-line oral drug treatment for tuberculosis in humans- Not for use in animals
Sulfonamides Sulfadiazine, sulfamethoxazole, sulfadoxine	Bacteriostatic- inhibit folic acid synthesis	Broad-spectrum; affects Gram-positive, Gram-negative bacteria, toxoplasma and protozoal agents Act synergistically (and becomes bactericidal) in combination with diaminopyrimidines (trimethoprim)	First line of drugs used in ruminants and poultry to manage diarrhoea and coccidiosis
Diaminopyrimidines Trimethoprim, Aditoprim, Baquiloprim, Ormetoprim	Bacteriostatic- Inhibits folic acid pathway	Broad spectrum; affects Gram-positive and many Gram-negative bacteria Act synergistically (and becomes bactericidal) in combination with sulfonamides	

Class of antibiotic	Mode of action	Spectrum of activity	Veterinary use in ruminants
Tetracyclines Chlortetracycline, oxytetracycline, demethylchlortetracycline, rolitetracycline, limecycline, clomocycline, methacycline, doxycycline, minocycline	Bacteriostatic – Inhibits protein synthesis	Broad spectrum. Exhibits activity against a wide range of Gram-positive, Gram-negative bacteria, atypical organisms such as chlamydiae, mycoplasmas, rickettsiae, and protozoan parasites.	Ehrlichiosis, leptospirosis, listeriosis, rickettsiosis, used as part of ECF vaccination strategy Anaplasmosis Heart water
Streptogramins Virginiamycin	Both Group A or Group B - Bacteriostatic Group A and Group B - Bacteriocidal	Narrow spectrum; mainly Gram-positive bacteria	growth promotant for livestock and prevention of laminitis

Annex:

5: Calculating drug dosage

The Volume of injectable veterinary drug to be administered can be calculated as follows (Nair & Jacob, 2016);

Injection

$$\text{Volume (ml)} = \frac{\text{Dosage (mg/kg) X Body weight (kg)}}{\text{Concentration (mg/ml)}}$$

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

Dosage of Oxytetracycline antibiotics in livestock

- Oxytetracycline injectable is available in the following concentrations; 5% (50mg/ml), 10% (100mg/ml), 20% (200mg/ml) and 30% (300mg/ml) preparation. 5% and 10% are short acting preparations that need to be administered continuously for a minimum of 4 days while 20% and 30% are long acting preparations that can be administered once or repeated after 48 or 72 hours if clinical signs are not resolving.
- Dosage in camels – 10% preparation- 10mg/kg intravenous (IV) every (q) 12 to 24hours (h) and 20% preparation- 20mg/kg Intramuscular (IM) or Sub-cutaneous (SQ) q24-72h (NB: in camel this is an extra-label use that is using an approved drug in a way that isn't listed on the drug's labelling also called off label use).
- Dosage in cattle: - Oxytetracycline 20% preparation- label dose for pneumonia 20mg/kg SQ, IV, or IM and for other diseases 6.6-11mg/kg SQ, IV, or IM q24h for up to 4days.

- Oxytetracycline 300mg/mL label dose for pneumonia or pink eye 20-30mg/kg IM or SQ once and for other indications 6.6-11mg/kg IM, SQ, or IV (given slowly) q24h for no more than 4 days.

- Dosage in sheep and goats- 5% or 10% preparation 10mg/kg IV or IM q12-24h and Long-acting formulas 20%- 20mg/kg IM q48-72h.

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

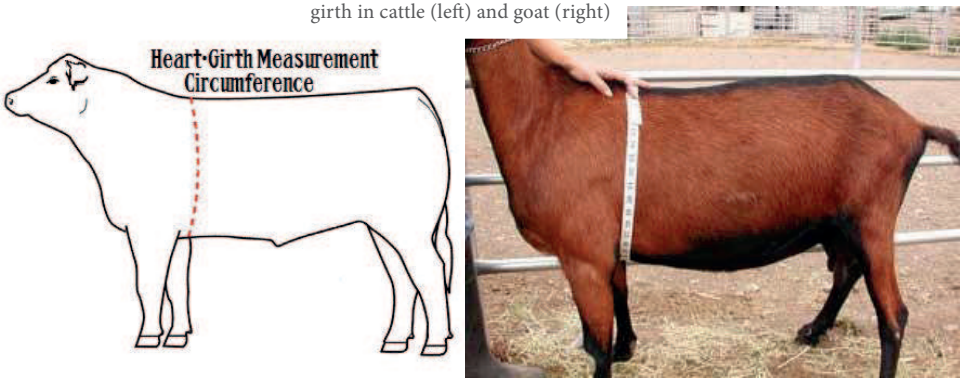
- Livestock owners need to know the weight of their animals in order to determine proper dosage of a medication, or to track how individual animals grow and use their feed. Approved and properly calibrated livestock scales are the most accurate and consistent method for determining body weight. However, they are expensive for most livestock keepers.
- Several methods can be used to determine an animal's weight when access to scales is not

possible: visual observation or guessing, weigh tapes, and weight estimation formulas.

- Visual observation is very inaccurate and not recommended for use when determining rations or medication dosages/.
- Weigh tapes are specially marked tapes used to measure the heart girth and convert that measurement to a fairly accurate estimate of the animal's body weight. Weigh tapes are easy to use and can be utilized effectively for monitoring purposes when used in a consistent manner by the same person. They can be purchased for camel, cattle, sheep and goats.
- Measurements are taken at narrowest part of the chest, immediately behind the front legs, and at the base of hump for Bos indicus breeds. Ensure animal's head is up, If head is down the chest expands causing an over estimation of the weight. If one tightens the tape too much (less common mistake) one will underestimate the weight. If one pulls the tape lightly (more common) one will overestimate the weight.

Figure 121

Weight measurement using heart girth in cattle (left) and goat (right)



Picture source:
http://bairnsley.com/Weight%20by%20Girth.htm (Cattle)
http://www.infovets.com/books/smrm/C/C098.htm (Goat)

Figure 122

Estimation of live-weight of cattle using chest girth measurements

Calves		Heifers		Cows	
Girth size (cm)	Live-weight (kg)	Girth size (cm)	Live-weight (kg)	Girth size (cm)	Live-weight (kg)
45	15	108	112	172	420
47	17	110	118	174	435
49	19	112	124	176	451
51	21	114	130	178	467
53	23	116	137	180	483
55	25	118	143	182	500
57	27	120	150	184	516
59	29	122	158	186	534
61	31	124	166	188	552
63	33	126	174	190	570
65	35	128	182	192	590
67	37	130	190	194	610
69	39	132	198	196	631
71	41	134	206	198	653
73	43	136	214	200	675
75	45	138	222	202	697
77	47	140	230	204	720
79	49	142	240	206	
81	51	144	248	208	
83	55	146	256	210	
85	59	148	264	212	
87	63	150	272	214	
89	67	152	280	216	
91	71	154	290	218	
93	75	156	301	220	
95	79	158	313	222	
97	83	160	325	224	
99	87	162	353	226	
101	92	164	366	228	
103	98	166	378	230	
104	103	168	392	232	
106	106	170	406	234	

Picture source:
https://infonet-biovision.org/taxonomy/term/12?page=8

Figure 123

Estimation of live-weight of goats using chest girth measurements

ESTIMATING BODY WEIGHT OF GOATS					
Centimeters →	Kilograms	Centimeters →	Kilograms	Centimeters →	Kilograms
27	2.3	50	13.2	73	35.4
29	2.5	51	14.1	74	36.7
30	2.7	53	15.0	76	38.1
31	3.0	54	15.9	77	39.5
32	3.2	55	16.8	78	40.8
34	3.6	57	17.7	79	42.2
35	4.1	58	19.1	81	44.0
36	4.5	59	20.4	82	45.8
38	5.0	60	21.8	83	47.6
39	5.4	62	23.1	85	49.9
40	5.9	63	24.5	86	52.2
41	6.8	64	25.9	87	54.4
43	7.7	66	27.2	88	56.7
44	8.6	67	28.6	90	59.0
45	9.5	68	29.9	91	61.2
46	10.4	69	31.3	92	63.5
48	11.3	71	32.7	93	65.8
49	12.2	72	34.0	95	68.1

Weight estimation formulas are more reliable than using weight tapes.

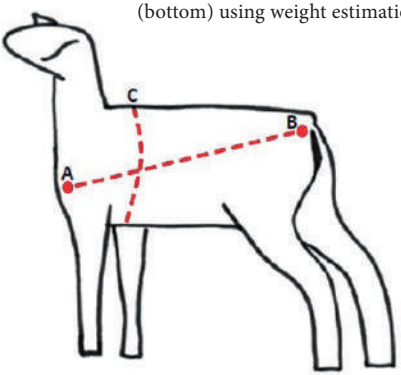
A common tape measure is used to determine the heart girth and body length measurement. These measurements are then used to calculate the animal's weight using the weight estimation formulas described below;

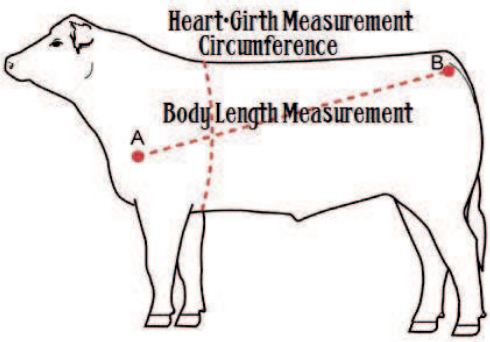
- MEASURE: (A-B) body length measured from point of shoulder to point-of-rump or pin bone.
- MEASURE: (C) heart girth circumference, around the body behind the shoulder, over the heart area and very close to the forelegs. When measuring a sheep, compress the sheep's wool to obtain a circumference that reflects the body and not the body plus the wool.
- Using these 2 measurements, calculate body weight estimate with this formula: (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.

Figure 124

Estimating live-weight of goats (top) and cattle (bottom) using weight estimation formula





Picture source:
http://bairnsley.com/Weight%20by%20Girth.htm

Further Reading

• Odadi W.O. (2018). Using heart girth to estimate live weight of heifers (Bos indicus) in pastoral rangelands of Northern Kenya. Livestock Research for Rural development 30:1
http://www.lrrd.org/lrrd30/1/cont3001.htm

Annex:

6: Ethnoveterinary Treatment and Prevention practices for priority livestock diseases in West Pokot County as described by livestock keepers

Cattle diseases	Ethnoveterinary Treatment and Control Practices
Lumpy Skin Disease (Lomolokoo)	Treatment - Topical application of used engine oil Control - Burn leaves or cow dung in the cattle boma in the evenings to chase away biting flies that could be on the animals - During the fly season graze animals very early in the morning or late in the evening
Foot and Mouth Disease (Ng'orion)	Treatment - topical application with one of the following; - Pariyan (Magadi soda-sodium chloride and Sodium bicarbonate) -Kipketin (Honey) -Sugary milk solution Control - Burn leaves or cow dung in the cattle boma in the evening to chase away biting flies that could be on the animals - Machey- Hot iron brand Marks made on the right side of the animal. Two or three horizontal lines running from the chest to the rump area to protect animals especially calves from contracting FMD - Restricting the calf from accessing milk from a sick cow by milking the cow often. It is believed the milk will make the calf sick with FMDV.
East Coast Fever (Yit/ Lokit)	Treatment with one of the following; - oral drench of Sokwon (Warburgia ugandensis) or Kaptolongwo or Sikowo Fruit and plant (Solanum aculeastrum). - Hot iron burning of swollen parotid and pre-scapular lymph nodes, oral drench of Kiripos (Kitchen Soot) and bleeding animal from Jugular vein Control - Hot iron burning of parotid and pre-scapular lymph nodes of cattle not affected by disease as a preventive measure - Machey (hot iron brand marks on the neck to protect animals during outbreaks
Anaplasmosis (Saak/ Toroy)	Treatment with one of the following; - Oral drench of Tamarind indicus or Porowo (Apodytes dimidiate) or Sikowo Fruit and plant (Solanum culeastrum) - Powder detergent given as an oral drench Control - Machey (hot iron brand marks at the rump and inguinal area of animals to protect animals at risk

Cattle diseases	Ethnoveterinary Treatment and Control Practices
Contagions Bovine Pleuropneumonia (Luokoi/ Psosoy)	Treatment - oral drench of Sokwon (Warburgia ugandensis) Control - Machey (hot iron brand marks of the chest (rib) area to protect animals at risk.
Primary Bloat (Musarer)	Treatment with one of the following; - oral drench of powder detergent - Pariyan given as an oral drench (Magadi soda-sodium chloride and Sodium bicarbonate) Control -prevent animals grazing lush pasture or plants that can cause bloat
Black Quarter (Tiompo Parak/ Tiompo ogha/ Tiompo Yim)	Treatment with one of the following; - oral drench of Kaptolongwo -topical application of Ash (Oriyon) - Machey (Hot iron branding of affected limb (three straight lines) Control -Avoid grazing in known outbreak areas
Mastitis (Semewo Kitin)	Treatment - Machey- Hot iron branding of the upper part of the hips in the shape of a diagonal cross Control -None
Plant poisoning (Ngwono/ Kawatian)	Treatment - oral dench of ash (oriyon) or ash mixed with fermented milk (Soyo) Control -Avoidance of area with poisonous plants -Clear through cutting or burning the poisonous plants
Heart water (Chemloi / Chepirpirmot)	Treatment with one of the following; - oral drench of Tapa (Tobacco) or Sokwon (Warburgia ugandensis) or Kaptolongwo leaves -oral drench of Kiripos (Kitchen roof and walls Soot) - Machey (hot branding of both sides of the base of the horns Control -None
Trypanosomiasis (Plis/ Taperwak)	Treatment with one of the following; - oral drench of Sokwon (Warburgia ugandensis) or Tolkos (Aloe species) - bleeding animal from Jugular vein - oral drench of fat from a tail of a sheep Control -Boost immunity ofanimals that are showing weakness (Kiturunogh) by giving an oral drench of pounded and cold soaked roots of Tunyunwo (Balanites aegyptiaca) or bleed animals from the Jugular vein to rejuvenate them - When cattle return in the evening to the boma, leaves or cow dung are burned to chase away biting flies that could be on the animals - During the fly season graze animals very early in the morning or late in the evening.

* Red highlights practices that should not be encouraged to continue as they go against animal welfare principles, promote drug resistance in both man and livestock or pose a risk of introduction of multispecies pathogens in the animals.

Cattle diseases	Ethnoveterinary Treatment and Control Practices
Photosensitisation (Melmel)	Treatment with one of the following; -topical application of Tapakapow (Euphorbia candelabrum) or Sikowo Fruit and plant (Solanum aculeastrum) - topical application of used engine oil Control -Avoid grazing animals in area with plants known to cause photosensitisation
Dermatophilosis / Streptothricosis (Ongoryo menyon)	Treatment with one of the following; - topical application of Tapakapow (Euphorbia candelabrum) - topical application of used engine oil Control -None
Babesiosis (Pkison)	Treatment - oral drench of fat from the tail of a sheep Control -None

Camel diseases	Ethnoveterinary Treatment and Control Practices
Haemorrhagic septicaemia (Saperkut)	Treatment with one of the following; - oral drench of Sokwon (Warburgia ugandensis) - Machey (Hot iron branding along the ribs) -Head of sheep with intact skin is burned over a fire until black then put in a cooking pot to make soup and given as an oral drench when cool. Control - During outbreaks Hot iron burning of head and neck lymph nodes to prevent animals from contracting the disease
Pneumonia (Psosoy)	Treatment with one the following; -oral drench Tolkos (Aloe species) - oral drench of fat from the tail of a sheep Control - Machey (hot iron brand marks at the bridge of the nose)
Trypanosomiasis (Plis / Taperwak)	Treatment with one of the following; -oral drench of Songowowo (Zanthoxylon chalybeum) or Asiokonyon (Salvadora persica) -oral drench of Lorit (type of soil) - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) given as a mineral block lick - oral drench of fat from the tail of a sheep - bleeding animal from Jugular vein Control - Boost immunity of animals that are showing weakness (Kiturunogh) by bleeding them from the Jugular vein so as to rejuvenate them. - During the fly season graze animals very early in the morning or late in the evening

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Camel diseases	Ethnoveterinary Treatment and Control Practices
Mange(Simpirion/ Lopadakaa/ Lomitina)	Treatment with one of the following; - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) given as a mineral block lick -Take animal to Kapedo area that has salty water and pasture - oral drench of fat from the tail of a sheep - topical application of used engine oil Control - Ensure animals access browse and water that has high salt content as this prevents skin conditions
Camel pox (Mokoyon)	Treatment with one of the following; - Machey (Hot iron branding) of neck, flank and rump area - oral drench of Lorit (type of soil) Control -None
Camel contagious ecthyma- orf (Ngirimen/ Ngurmen)	Treatment with one of the following; - Kinyotwo fruits (Ximenia Americana) boiled and pounded into a paste for topical application - Smear blood of a cow on mouth lesions - Smear fat from the tail of a sheep on mouth lesions Control - Machey (hot iron brand marks at the bridge of the nose)
Brucellosis (Somewo cho)	Treatment - Machey (Hot iron branding) of rump area to prevent uterine infections Control -None
Mastitis (Somewo Kitin)	Treatment with one of the following; - Asiokonyon (Salvadora persica) leaves, branches and barks burned in a container and used to fumigate the udder - Machey (hot iron branding of rump area/upper part if hip region with a diagonal cross) Control -None
Mineral deficiency(Konyomoi)	Treatment with one of the following; - Camels are moved to grazing areas with grass and water with high Salt content (Orusyion) Areas like Nginyang, Kapedo and Lomelo (near Turkana county) - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) given as a mineral block lick. Control - Ensure access to browse and water with high salt content

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Goat diseases	Ethnoveterinary Treatment and Control Practices
Contagious Caprine Pleuropneumonia (Luokoi / Psosoy)	Treatment with one of the following; -oral drench of Sokwon (Warburgia ugandensis) or Tolkos (Aloe species) - Dissolve in water Tetracycline capsules (human drug) and inject in between the ribs of sick animal Control -Avoid herds and areas with disease
Peste des Petits Ruminants (Losir)	Treatment with one of the following; - Machey (hot iron brand marks at the bridge of the nose, two or three lines) -Administer orally a little acaricide (dip wash) Control -Avoid herds and areas with disease
Helminthiasis (Pokot-Mochonto muu/ Ngutianta muu/ Chepturu) (Sengwer-Magargarek)	Treatment with one of the following; - Administer orally small quantity of used/waste engine oil -oral drench of Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) Control Delay removing animals in the morning to allow the dew on the grass blades to dry up
Sheep and Goat Pox (Mokoyon/ Moyoi/ Lomolokoo)	Treatment with one of the following; -topical application of used engine oil or Vaseline –used as a skin solid oil in humans Control - Provide mineral block licks of Pariyan (Magadi soda- sodium chloride and Sodium bicarbonate)
Foot rot (Chepkelien)	Treatment with one of the following; -topical application of used engine oil or oriyon (Ash) or utto (vegetable cooking oil Control -None
Mange (Simpirion)	Treatment with one of the following; - topical application of used engine oil - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) given as mineral block lick Control -None
Coenurosis (Kapir/ Lopira)	Treatment -none Control -None
Emaciation and scours syndrome (Chepkiyiy)	Treatment -none Control -none

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Goat diseases	Ethnoveterinary Treatment and Control Practices
Contagious ecthyma orf (Ngirimen/ Ngurumen)	Treatment with one of the following; - topical application of Kinyotwo fruits (Ximenia Americana) boiled and pounded to form a paste - topical application of used engine oil and utto (vegetable cooking oil) -Machey (hot iron branding of the bridge of the nose) Control -None
Infectious Keratoconjunctivitis-pink eye (Semewo Konyin)	Treatment -None Control -None
Sheep diseases	Ethnoveterinary Treatment and Control Practices
Enterotoxaemia (Lotuler)	Treatment -None Control -Avoid young lambs from taking too much milk by separating the dam and lamb at night.
Helminthiasis (Pokot-Mochonto muu/ Ngutianta muu/ Chepturu) (Sengwer-Magargarek)	Treatment -None Control - Provide mineral block licks of Pariyan (Magadi soda- sodium chloride and Sodium bicarbonate) - Delay removing animals in the morning to allow the dew on the grass blades to dry up
Pneumonia (Psosoy)	Pneumonia (Semewo Kaghtan / Psosoy)
Mange (Chesulet)	Treatment with one of the following; -topical application of used engine oil - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate) given as a mineral block lick. Control -None
Black Quarter (Tiompo parak / Tiompo yum / Tiompo ogha)	Treatment -None Control -Avoiding grazing in areas that had previous outbreak
Heart water (Chemiloy / Chepirpirmot_	Treatment with one of the following; -oral drench Tapa (Tobacco) - Machey (Hot iron branding cross marks on the head) Control -None
Peste des Petits Ruminants (Losir)	Treatment -None Control -Avoid herds or areas with disease

* Red highlights practices that should not be encouraged to continue as they go against animal welfare principles, promote drug resistance in both man and livestock or pose a risk of introduction of multispecies pathogens in the animals.

Sheep diseases	Ethnoveterinary Treatment and Control Practices
Plant poisoning (Kawatian/ Ngwono)	Treatment with one of the following; -oral drench of Milk and sugar solution or diluted Kipketin (Honey) or crushed charcoal and ash and or raw eggs Control -Avoid grazing with known poisonous plants -Clear plants from the range through cutting of burning
Abortion syndrome (Turunogh)	Treatment - Pariyan (Magadi soda sodium chloride and Sodium bicarbonate)-given as a salt lick block Control - Provide mineral block licks of Pariyan (Magadi soda- sodium chloride and Sodium bicarbonate)
Foot rot (Chepkelien)	Treatment with one of the following; - topical application of used engine oil, oriyon (Ash) and utto (vegetable cooking oil) Control -None
Foot and Mouth Disease (Ng'orion)	Treatment with one of the following; -topical application of Warm saline solution to wash mouth and feet lesions or Sugary milk solution or Kipketin (Honey) or Changaa decant (traditional beer) * - Animal fed on green banana (alkaline pH dries lesions) Control -Avoid herds with disease or outbreak areas -Migrate out of area with disease
Primary Bloat (Musarer)	Treatment - oral drench of Utto (vegetable cooking oil) - oral drench of used engine oil- small quantity Control -Avoid grazing on lush pastures

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Chicken diseases	Ethnoveterinary Treatment and Control Practices
Chronic Respiratory Disease (CRD) (Cheptakat)	Treatment with one of the following; - Paagha wao ground leaves and flower and given in drinking water - Lantana camara flowers ground and given in drinking water - Kiripos (Soot from kitchen roof) given in drinking water - Piriton® (Chlorphenamine) tablet crushed and given in drinking water - Amoxycillin capsule (Human preparation) powder in capsule mixed with water for oral use Control - Mix Aloe Vera sap in the drinking water to prevent diseases
New Castle Disease (Konukoi/ Tongoroi/ Knuch/ Konuktoi)	Treatment with one of the following; -Ground Pilipili seeds (Capsicum frutescens) –given in drinking water - Tolkos (Aloe species) crushed to extract sap –given in drinking water - Korkorwo (Erythrina abyssinica) leaves and bark pounded into powder and given in drinking water - Paagha wao ground leaves and flowers given in drinking water -Lantana camara flowers ground and given in drinking water - Raw milk given orally - Metronidazole tablet (human preparation) crushed and given in drinking water - Amoxycillin capsule (Human preparation) powder in capsule mixed with water given in drinking water - Panadol®(acetaminophen) human preparation- 1 tablet is crushed and dissolved in drinking water Control - Mix Aloe Vera sap in the drinking water to prevent diseases
Coccidiosis- Bloody diarrhoea syndrome (Pokot- Semewo Kiyitagh/ Kmun) (Sengwer- Loyita/ Kaputi)	Treatment with one of the following; -Ground Pilipili seeds (Capsicum frutescens) –given in drinking water -Tolkos (Aloe species) crushed to extract sap given in drinking water Korkorwo (Erythrina abyssinica) leaves and bark pounded into powder and given in drinking water - Paagha wao ground leaves and fruits given in drinking water - Lantana camara flowers ground and given in drinking water - Raw milk given orally - Metronidazole tablet (human preparation) crushed and given in drinking water - Kiripos (Soot from kitchen roof) given in drinking water - Amoxycillin capsule (Human preparation) powder in capsule mixed with water and given in the drinking water Control - Mix Aloe Vera sap in the drinking water to prevent diseases

* Red highlights practices that should not be encouraged to continue as they go against animal welfare principles, promote drug resistance in both man and livestock or pose a risk of introduction of multispecies pathogens in the animals.

Chicken diseases	Ethnoveterinary Treatment and Control Practices
Infectious Coryza (Tasus po konyin)	Treatment - Tolkos (Aloe species) crushed to extract sap given in drinking water Control - Mix Aloe Vera sap in the drinking water to prevent diseases
Fowl pox (Moyoi / Kupoy-po Kokoroch)	Treatment -topical application of used engine oil Control -None
Ectoparasite infestation (Kiyominto onto/ Kiyominto menyon) Fleas (Kmit) Lice (Sirr) Ticks (Cheptapalal) Body Mites (Pititi) Leg mites (Ng'atwoi)	Treatment -topical application of Paraffin or Used engine oil Control -Apply used engine oil in the walls of the chicken house - Pour ash on the earthen floor of the chicken house

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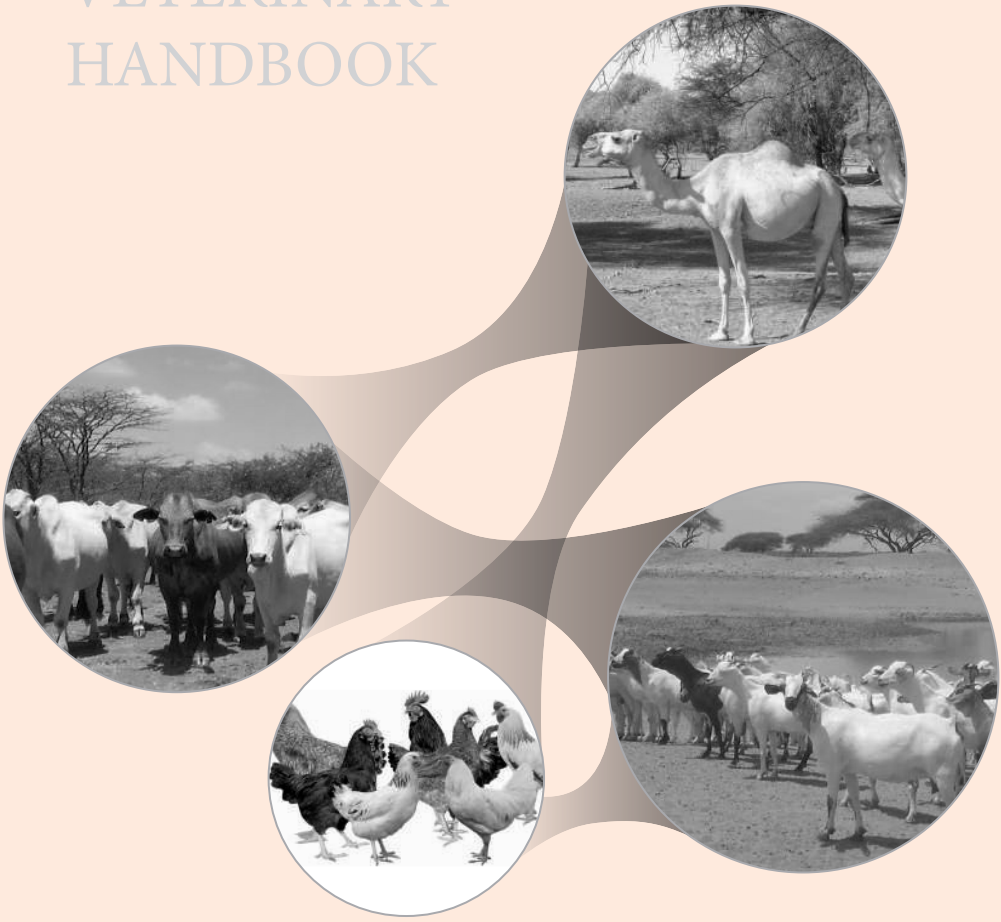
WEST POKOT COUNTY

VETERINARY HANDBOOK



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