SAMBURU COUNTY

VETERINARY HANDBOOK

USAID FROM THE AMERICAN PEOPLE

ACTED

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

Common Diagnosis, Treatment and Control Guidelines for Animal Health Service Providers

Acknowledgements

The ASAL eXtension veterinary team lead by Dr. Pauline Gitonga and supported by Dr. Viola Chemis, Dr. Lilian Ooko and Dr. Jacquiline Kisianan would like to express their sincere gratitude to the ACTED team. We are indebted to Shalom Magoma, Jillo Elema, Rebecca Chepkorir, Agnes Chepunton, Emmanuel Tinja and Michael. We acknowledge the significant role played by the community members who participated in the Focus Group Discussions. Their p atience a nd w illingness t o s hare t heir k nowledge a nd experience greatly enriched the technical handbook. Lastly, we are grateful to the county veterinary services director Dr. Boreya Lekenit and his entire team including the community disease reporters who supported data collection and validation of findings. Lastly, we recognise the generous support of the American people through the United States Agency for International Development (USAID) - Bureau for Humanitarian Assistance (BHA) that funded the I-CREATE project which supported the development of the veterinary handbook.

ASAL eXtension

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Disclaimer

THIS HANDBOOK IS INTENDED TO GUIDE ANIMAL HEALTH PRACTITIONERS WORKING IN SAMBURU 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 Samburu county veterinary technical handbook aims at harmonising the identification, diagnosis and control of priority livestock diseases. 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 28th to 31st May, 2021. The study gathered information from seventeen (17) Focus Group Discussions (FGDs) with livestock keepers across 3 sub counties, 8 wards and 13 villages. A total of 166 livestock keepers participated in the FGDs, of these, 109 (66%) were male and 57 (34%) were female. The validation workshop was the main data verification method used to collaborate FGD findings. The workshop was held on 25th June 2021 at Maralal. Twenty-Four (24) Key Informant Interviews were also conducted. The KII respondents were drawn from the public and private sector as well as from the community leadership structures.

The manual provides a simple and concise description of the priority livestock diseases as well as contextualises the diseases to the local setting through provision of local names of disease syndromes and ethnoveterinary practices. The Samburu County Animal Health Service Providers (AHSPs) are urged to use the handbook alongside the set national and OIE guidelines. 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 Samburu County.

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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

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

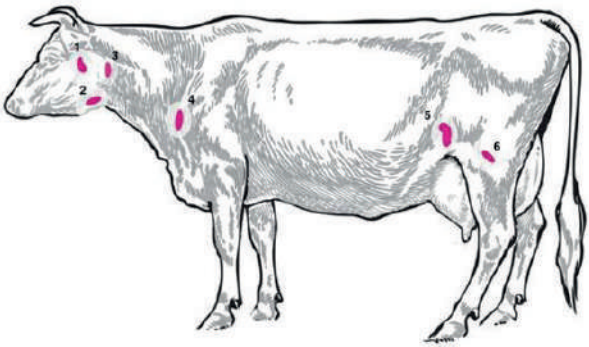
Specie	Range (°C)
Camel*	34.0- 41.0
Cattle- Beef	36.7- 39.1
Cattle-Dairy	38.0-39.3
Goat	38.5-39.7
Sheep	38.3- 39.9

* 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

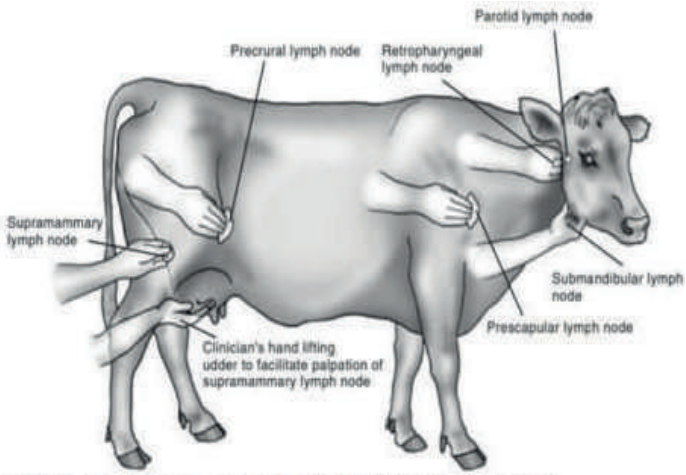
Reference

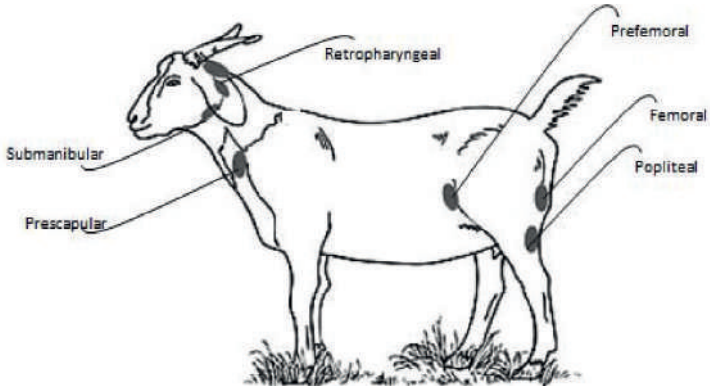
1. Clinical Examination of Farm Animals Peter G.G. Jackson, Peter D. Cockcroft Copyright © 2002 by Blackwell Science Ltd.
2. The MSD Veterinary Manual https://www.msdvetmanual.com/
3. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary 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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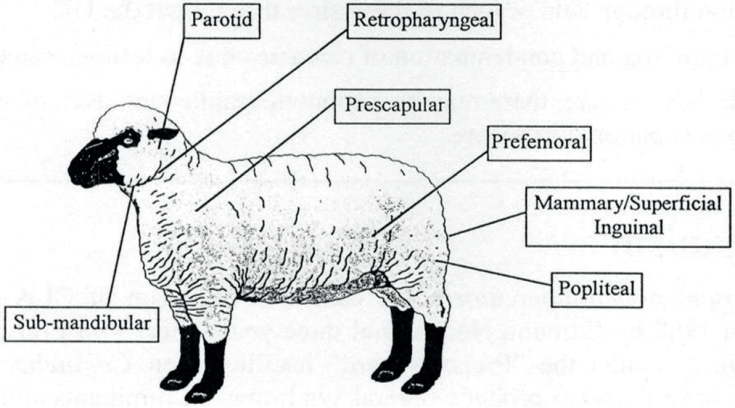
Prefemoral
Femoral
Submanibular
Popliteal
Prescapular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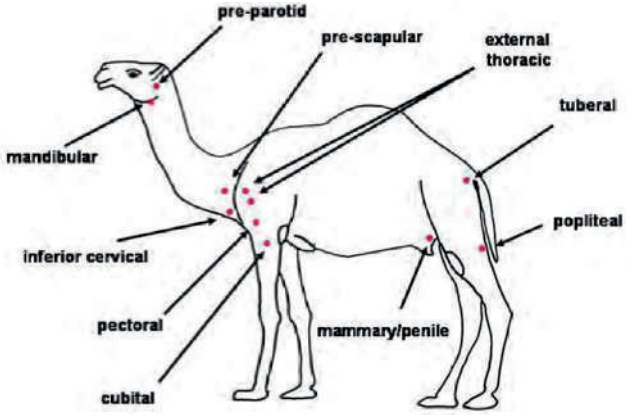
Retropharyngeal
Prescapular
Prefemoral
Mammary/Superficial Inguinal
Popliteal
Sub-mandibular


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

Reference
1. Clinical Examination of Farm Animals Peter G.G. Jackson, Peter D. Cockcroft Copyright © 2002 by Blackwell Science Ltd.
2. The MSD Veterinary Manual https://www.msdvetmanual.com/
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

1.6 | Traditional disease names and clinical syndromes for priority livestock diseases in Samburu county as described by livestock keepers

Camel Diseases	Samburu Name	Turkana Name
1. Camel Flu	Lchama	Lowola
2. Haemorrhagic septicemia	Nalngaringari	Longaruei / Lokou
3. Contagious Skin Necrosis (CSN)	Lmonkoi	Akitherit
4. Camel Mange	Lpepedo	Emitina Pepedo
5. Trypanosomiasis	Saar	Ekwakoit/ Saar/ Lokipi
6. Contagious ecthyma (orf)	Abitiro	Ng'iboruok
7. Abuth	Araar	Abuth

Camel Clinical signs English Name	Samburu Name	Turkana name
1. Coughing	Luata	Awala
2. Lacrimation	Keichir Nkonyek	Akugoro
3. Nasal discharge	Koruko Lchama (mucus consistensy) Koruko Nkumeshin (watery consistency)	Alakakin Ngithurma
4. Enlarged lymphnodes	Kejei Lngarngar	Ang'are
5. Fever	Keirewa sesen	Eyala
6. Swollen head	Kejei nkwe	Lokou
7. Skin lesion- wound	Ngoldonyot	Eparra
8. Skin lesion- abscess	Ntubui	Abuth
9. Pruritus	Ojo	Akoo
10. Alopecia	Aputo	Echudan/ Ekoikoi
11. Jaundice	Adarlenu	Lonyang
12. Anaemia	Meata lodo	Erogo
13. Diarrhoea	Nkiriata	Aremorit
14. Mastitis- swollen udder with clots/ blood in milk	Kejei Nyawa- swollen udder Kenyokuo kule- mastitis milk	Edeke aket- swollen udder Akile naang'akot- mastitis milk

Cattle Diseases	Samburu Name	Turkana Name
1. Foot and Mouth Disease (FMD)	Lkulup	Lokulup
2. Anaplasmosis	Ndis	Lonyang
3. Babesiosis	Nkula	Akul Narrengak
4. Lumpy Skin Disease (LSD)	Nariri Eenkishu	Tune
5. East Coast Fever (ECF)	Lipis	Lipis
6. Black Quarter	Nenkeju	Ngwaat
7. Fascioliasis (Liver Fluke)	Lkurui Lemonyua	Ngirtan
8. Pneumonia	Lkipei Loonkishu	Loukoi
9. Trypanosomiasis	Saara	Saar/ Ekwakoit/ Lokipi
10. Listeriosis	Mporoto	Mporroto

Cattle Clinical signs English Name	Samburu Name	Turkana name
1. Salivation	Sampulal	Angakumul
2. Lameness	Ng'ojini	Akichodo
3. Enlarged lymph nodes	Ajei Lngarngar	Ngangaruei
4. Blindness	Modoki	Amudukanu
5. Constipation	Aibok modio	Akiding
6. Red urine	Nkula naanyokuo	Akul narrengak
7. Distended abdomen	Mpangit	Akitebukin / Abwere alochin
8. Emaciation	Saso	Erogo
9. Coughing	Luata	Awalakin
10. Skin lesions-nodules	Ltukach	Abuth
11. Central nervous system signs-circling or peddling	Lmilo	Akirim
12. Mouth lesions	Kedanydanya nkutuk	Ngajamei aakituk
13. Foot lesions	Kedanydanya nkejek	Ebaibai
14. Sudden death	Lbus	Atular
15. Diarrhoea	Nkiriata	Aremorit
16. Swollen leg	Ajei Nkeju	Abuore akeju
17. Lacrimation	Keishir nkonyek	Akigoro ankonyen
18. Fever	Keirewa sesen	Emona
19. Brown Ear Tick infestation	Kobore Lmancher	Ileleba emadang
20. Jaundice	Adarlenu	Lonyang
21. Recumbency	Meinyototo	Eureni
22. Parasite in the liver	Lkurui lemonyua	Ngirtan

Cattle Clinical signs English Name	Samburu Name	Turkana name
23. Respiratory distress/ difficulty in breathing	Keirrum	Akiyang'a
24. Nasal discharge	Koruko Lchama (Mucus consistency) Koruko Nkumeshin (Watery consistency)	Ngithurma
25. Swollen udder	Kejei nyawa	Edeke aket
26. Mastitis milk	Kenyokuo kule	Akile naang'akot
27. PM- Lung lesions	Kerep lbusbus lmarain	Lotai

Goat Diseases	Samburu Name	Turkana Name
1. Contagious Caprine Pleuropneumonia (CCPP)	Lkipei	Loukoi
2. Peste des Petits Ruminants (PPR)	Ngoritit /Serr /Nkiriata /Lodua	Lonoo
3. Sheep and Goat Pox (SGP)	Nariri /Tune	Tune
4. Coenurosis	Sirko	Nkerep
5. Coccidiosis	Nkiriata Elodo	Eremorit Ng'aokot
6. Helminthiasis	Ntumuai	Ngiritan
7. Contagious ecthyma (Orf)	Abituro	Ng'borouk
8. Mange	Iputo	Echudano
9. Enterotoxaemia	Sirr / Nadomanyita	Lobus
10. Foot rot	Ng'ojini	Lukulup

Sheep Diseases	Samburu Name	Turkana Name
1. Coenurosis	Sirko	Sirko/ Nkerep
2. Helminthiasis	Ntumuai	Ngiritan
3. Foot and Mouth Disease (FMD)	Lkulup	Lokulup
4. Enterotoxaemia	Lbus/ Nadomanyita	Lobus
5. Sheep and Goat Pox (SGP)	Tune/ Nariri	Tune
6. Pneumonia	Lkipei / Nkijepe	Lotai
7. Mange	Iputo	Echudanu
8. Anaplasma	Ndis	Lony'ang
9. Peste des Petits Ruminants (PPR)	Kinyoot/ Nkiriata/ Lodua	Lokiyoo / Lonoo
10. Babesiosis	Nkula	Lonyang'

Goat and Sheep clinical signs English Name	Samburu Name	Turkana name
1. Sudden death	Keebusori/ Ibus / ikiee	Toton
2. Diarrhoea	Nkiriata	Aremor Ngachin Imorikina Kangaakot- blood tinged
3. Anorexia/off-feed	Medaa	Nyedakaah
4. Bloating	Mberiri	Akitiebukin/ Aurenem
5. Intestinal haemorrhage	Nadoo manyita	Loreng' Malteny
6. Enlarged liver and Gall bladder enlargement	Nesapuku lodua- enlarged gall bladder	Ebut emany- enlarged liver Kilelebun ekulam- enlarged gall bladder
7. Emaciation	Saso	Erogo
8. Nasal discharge	Lchama	Thuruma/ Esurem-sheep Ethuruma / Esurem- Goat
9. Lacrimation	Keishir ngonyek / ikiyo ee nkonyek	Lokiyo / Akiyo
10. Salivation	Samplal	Ngakajeela / Lokajeela
11. Sub-mandibular oedema (Bottle jaw)	Njian ee nkweshi	Lobolbolio
12. Respiratory distress/ difficulty in breathing	Kepsing'a	Egolit Arukum-
13. Abortion	Aiburu	Akiyech
14. Mouth lesions	Ng'oldonyok ee nkutuk	Ngajemei Naakutuk-sheep Ngajemei Aakutuk-goat
15. Foot lesions	Ng'oldonyok ee nkejek	Ngajemei Angakejen
16. Pox lesions	Lpepedo	Abuth
17. Pox lesions in the intestines	Riririu ee manyit	Ngajemei Aakook
18. Coughing	Alua	Awala / Akiwaa
19. Lung adhesions to the rib cage due to fibrinous pneumonia	Kerap lbusbus marain	Atapakin Euko Namaran- Sheep Euko etapakin namaran- Goat
20. Anaemia	Meeta lodoo	Lokwakonyen
21. Presence of worms in the faeces	Ntumuai	Ngiritan Anachin
22. Rough hair coat and scaly skin	Kei julus	Echudanu- rough hair coat Ekurara- scaly skin
23. Red urine	Nkula	Ngakul Naarengak
24. Jaundice	Ndis	Akiring' alinyang'
25. Nervous signs- circling and incoordination	Kemanaa- circling Kewushush- incoordination	Akirim- circling Akitertero- incoordination
26. Larvae in the brain	Ibulbuli	Ekurut Loeyei Lodam
27. Constipation/hard stool	Kogol moidio	Egogong' Ngachin

Goat and Sheep clinical signs English Name	Samburu Name	Turkana name
28. Excessive bleating due to pain or discomfort	Ibua / mion	Alakakin Nagkiyo nakaalak
29. Swelling of the mouth	Injina ee nkutuk	Abwere Aakutuk
30. Lameness	Ngojini	Akingwal
31. Alopecia	Keidoso	Emaam Ngajul
32. Heavy tick infestation	Lmanchir	Ileleba Ngimadang'
33. Post mortem fluid in chest cavity	Kitii lpile lgoo	Ngapethpeth angakipi alouko

Chicken diseases	Samburu name	Turkana name
1. New Castle Disease	Lchama / Nkonkoro	Lonoo
2. Infectious Coryza	Moyen Enkonyek	Eboot Akou
3. Fowl pox	Nariri enkokon	Ng'ajemai
4. Ectoparasites	-	-
5. Fleas	Losusu	Losusu
6. Lice	Lashe	Elachet
7. Mites	Mpaipayan	Losaa
8. Scaly feet/Scaly leg mite	Nkasimai	Ngooeye
9. Ticks	Lmancher e nkokon	Emadang
10. Fowl Cholera	Nkiriata	Eriomorit
11. Chronic Respiratory disease	Nkurungata e nkokon	Ipiyorit Akienga
12. Helminthiasis	Ntumwa	Ngirtan
13. Infectious bronchitis	Kebirua	Egolit arukum
14. Gumboro	Disease not common no name	Etunati

Chicken clinical signs English Name	Samburu name	Turkana Name
1. Depression	Keiguuta	Aburun
2. Loss of appetite	Mokuru ndaata	Nyimuji
3. Nasal discharge, coughing, sneezing	Lchama / Ikirobi	Akaritho
4. Feeling cold	Nkijepe	Akatorot
5. Ruffled feathers	Kejuluus	Epuriarit
6. Drooping wings	Ailiang nkaipupo	itoweikinit
7. Diarrhoea	Nkiriata	Eriomorit
8. Twisted neck	Konkoro	Edeke eutune
9. Nervous signs -Circling	Kemanaa	Irimi

Chicken clinical signs English Name	Samburu name	Turkana Name
10. Pox lesions on the head and unfeathered skin	Nariri	Ng'ajemei
11. Sudden death	Lbus	Ang'edar /Itular
12. Weakness of joints	Lgila	Akichodo
13. Premature egg laying-soft shell	Kebirua	Ngakaekuwam / Akiyechun
14. Abscess	Ntubu	Eyang'abulon
15. Difficulty breathing	Lchama	Egulo ngumeth
16. Wounds	Ng'oldonyot	Ajemei
17. Worms in faeces	Ntumuai	Ngiritan anachin
18. Bloody diarrhoea	Nkiriata elodo	Aremor angaakot

Reference

1. Mapping Priority Livestock Diseases in Samburu County Report (2021). A report submitted to ACTED supported by Funds from USAID Bureau of Humanitarian Assistance (BHA).

Chapter 2:

Cattle Diseases

2.1 | Lumpy Skin Disease (LSD)- Nariri Eenkishu (Samburu), Tune (Turkana)

Definition

- Vector-borne viral disease of domestic cattle characterised by sudden appearance of skin nodules.

Distribution (Epidemiology)

- Occurrence common during the heavy rainy season when there is presence of large number of biting insects.
- Endemic in Samburu.
- Occurs in Southern and Eastern Africa, Middle East, Asia, and Eastern Europe.
- No occurrence record in North and South America.

Causative agent (Aetiology)

- Lumpy skin disease virus (LSDV) from the family Poxviridae and genus Capripoxvirus (CaPV) is a large double stranded DNA virus.
- Stable virus with little genetic variability (mutations)

Transmission and Pathophysiology

Short distance transmission (equivalent to how far insects can fly (usually < 50 km)

- 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.
- 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°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, involving the epidermis, dermis, subcutaneous tissue and sometimes underlying muscle.

- Nodules also affect the nasal, oral, ocular and genital mucosa.
- Increased nasal and oropharyngeal secretions 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).

Post mortem lesions

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

Morbidity and Mortality rate

- The world animal health organisation (OIE) estimates the global morbidity rate to be between 10 and 20% and mortality rate to be 1 to 5%.
- In Samburu county group discussions with livestock keepers estimated an apparent morbidity rate of 17%, mortality rate of 5% and case fatality rate of 29%.

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.
- In Samburu county, LSD occurrence is common during the short rains season locally known as Lgergerua (April to May) in the Samburu community and Ngirupei (November and December) in the Turkana community.

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.
- Laboratory tests to be conducted include - Polymerase Chain Reaction (PCR) for LSDV antigen genome identification and Enzyme-Linked Immunosorbent Assay (ELISA) for serological analysis of antibodies.
- Serology does not differentiate genus or species level of LSDV, SPPV and GTPV.

Differential Diagnosis (DDx)
Diseases 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 hind legs or neck muscles q48 hours PRD IM
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. LUMPIVAX™- a freeze-dried, live attenuated vaccine that is available 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, 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

1. Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. OIE (World Organisation for Animal Health) ISBN 978-92-9044-804-4. https://rr-asia.oie.int/wp-content/uploads/2020/04/oie-atlas-of-transboundary-animal-disease_ahs-chapter.pdf
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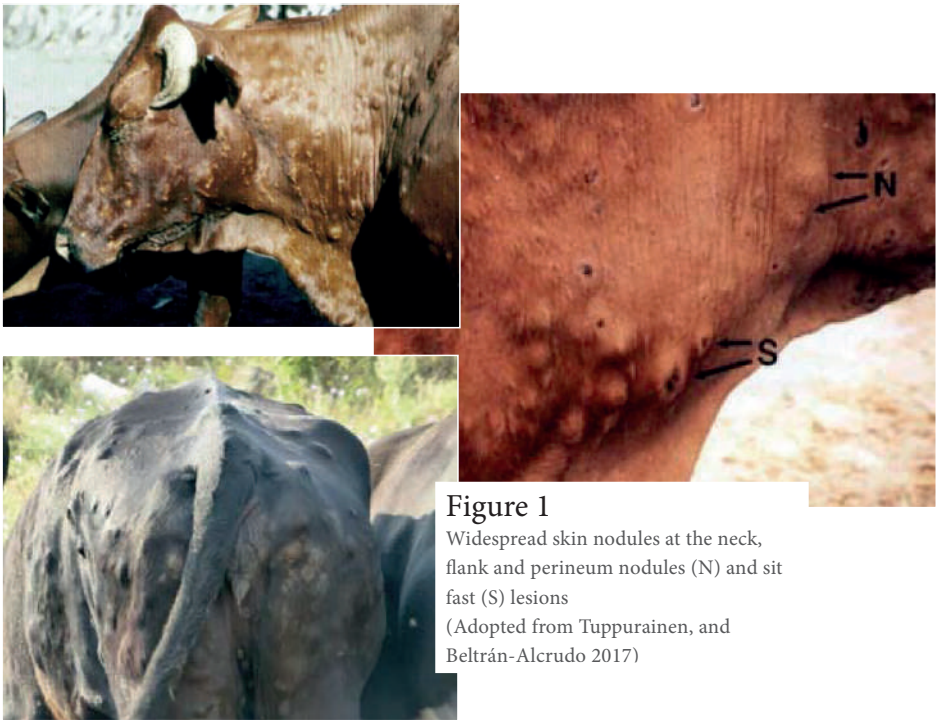

Figure 1

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

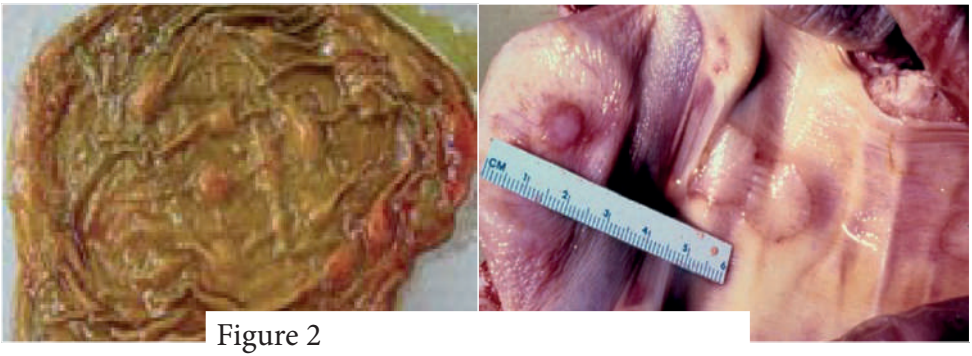

Figure 2

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)- Lkulup (Samburu), Lokulup (Turkana)

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

- Endemic in Samburu County
- Occurs in Africa, Middle East, Asia, and Eastern Europe.
- No occurrence recorded in Central and North America, Australia, New Zealand, Western Europe and Indonesia.

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.
- 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.”

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.

Morbidity and Mortality Rates

- The world animal health organisation (OIE) estimate the global morbidity rate in unvaccinated herds to be 100%, mortality rate in adult cows is between 1 to 5% but higher in calves (20%).
- In Samburu county, FMD has an estimated apparent morbidity rate of 45%, mortality rate of 8% and case fatality rate of 18%.

Seasonality of occurrence

- Outbreaks at any season due to introduction of sick animals, presence of naïve (not immune) animals. In pastoralist settings shared pasture and water are a key driver of FMD outbreaks.
- In Samburu county, FMD occurrence is common during the long rains season locally known as Ltumuren (October to December) in the Samburu community and Akiporo (March and June) in the Turkana community

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

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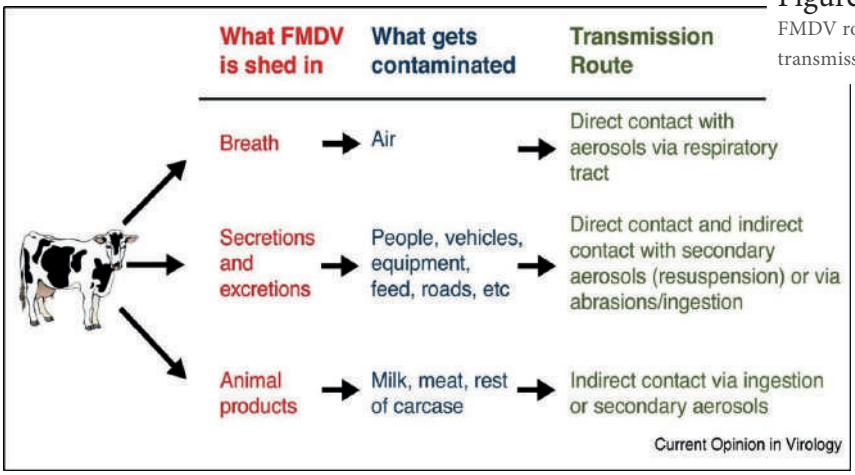

Figure 3
FMDV route of transmission

Picture source:
https://www.sciencedirect.com/science/article/pii/S1879625717300962

Figure 4
Profuse salivation (saliva hanging in long ropy strings up to the ground)

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

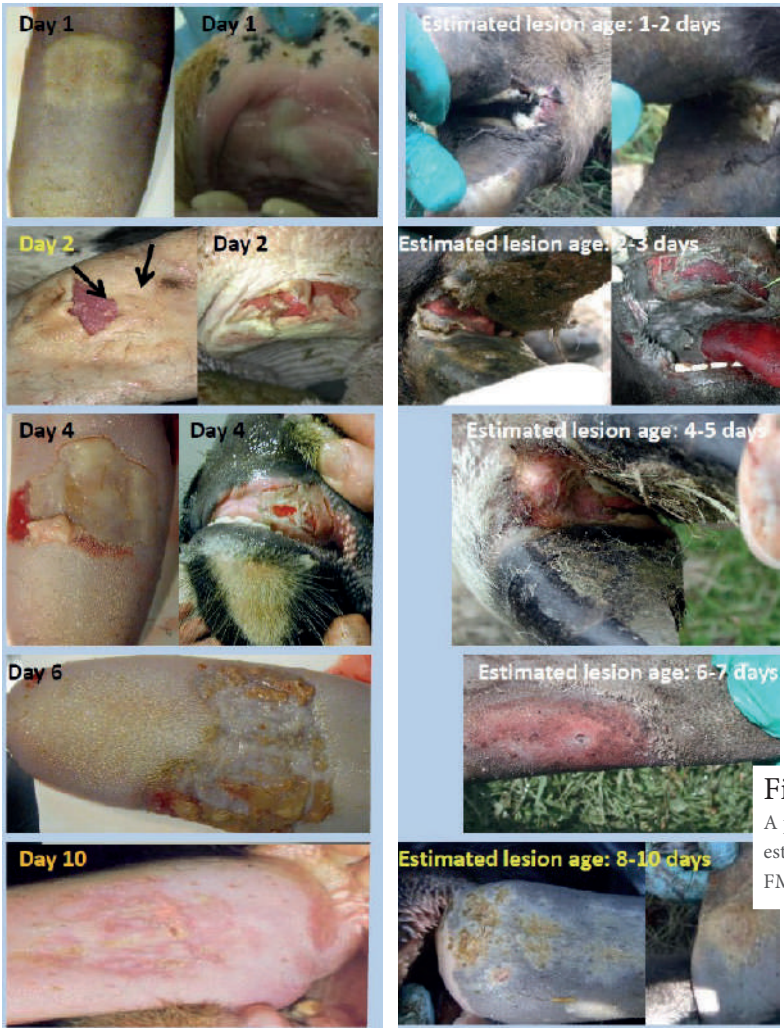

Figure 5
A pictorial guide to estimate the age of FMD lesions

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)- Lipis (Samburu and Turkana)

Other name(s): 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.
- It is estimated that annually, ECF causes the death of 100,000 cattle of all ages in Kenya. This makes ECF the most important disease of cattle in the country.

Distribution (Epidemiology)

- ECF occurs in 13 countries in Sub-Saharan Africa.
- Endemic in Samburu county.

Causative agent/ Aetiology

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

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.
- Eggs of the brown ear tick are not infected with Theileria parasites, so the larvae which hatch from them cannot transmit the disease. Only nymphs and adults are infective.
- About 2-10% of ticks in an ECF endemic area are infected with Theileria parasites. This means that the presence of brown ear ticks on a susceptible animal does not necessarily mean that the animal will develop ECF. However, only 1 tick is needed to transmit the disease to a susceptible animal.
- Animals that recover are immune to subsequent challenge with the same strains but may be susceptible to heterologous strains. However, most recovered or immunized animals remain carriers.
- 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 fatal disease like adults.
- Exotic breeds (Bos Taurus) are more susceptible when compared to indigenous Bos indicus breeds.

Clinical signs

- ECF incubation period is 8 to 12 days.
- Fever occurs and continues throughout the course of infection and may be as high as 41 to 42 °C.
- Marked enlargement of all palpable and superficial lymph nodes, particularly, the ones in front of the shoulder (pre-scapulars) and below the ears (parotids) and the on the flanks (pre-crurals).
- Lacrimation, increased nasal discharge and coughing.
- 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.
- Haemorrhages are common on the serosa and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Anaemia is not a major diagnostic sign (as it is in Babesiosis) because there is minimal division of the parasites in RBCs and thus no massive destruction of them.
- The mucosa of the digestive tract has lesions that resemble "cigarette burn" these are ulcers in the rumen. In addition, the colon, may show "zebra striping" due to destruction of the cells of the gut wall.
- Surfaces of the kidneys may show white spots (lymphoid infarcts), which extend down into the kidney tissue.

Morbidity and Mortality Rates

- The fatality rate for untreated ECF is 100% in cattle from non–endemic areas especially exotic breeds (Bos Taurus).
- In endemic areas, the morbidity rate is high at 100% even amongst indigenous cattle breeds.

However, the mortality for untreated ECF in indigenous breeds is lower and is estimated to range from 20% to 50%.

- Cross breed cattle show an intermediate degree of susceptibility.
- In Samburu county, ECF has an estimated apparent morbidity rate of 11%, mortality rate of 7% and case fatality rate of 64%.

Seasonality of occurrence

- Occurrence of disease is dependent on availability of an infected tick, susceptible animals and effectiveness of tick control strategies. Disease incidence can therefore occur all year round.
- In Samburu county, ECF is common during the short rainy season locally known as Lgergerua (April to May) in Samburu community and during the long rainy season, Akiporo (March and June) in the Turkana community.

Diagnosis

- Presumptive diagnosis is based on clinical signs.
- 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.

Differential Diagnosis (DDx)

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

Treatment

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

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

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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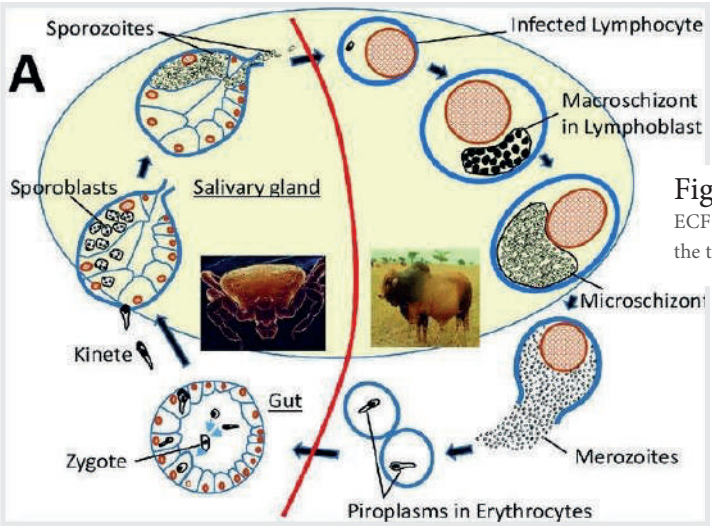

Figure 6
ECF parasite life cycle in the tick and cattle

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

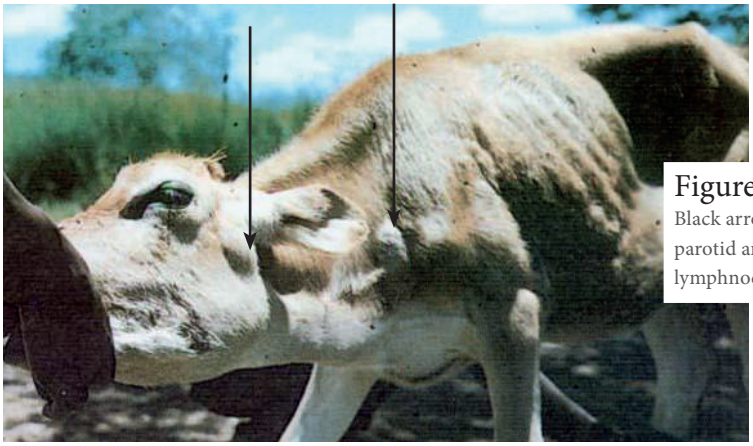

Figure 7

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

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

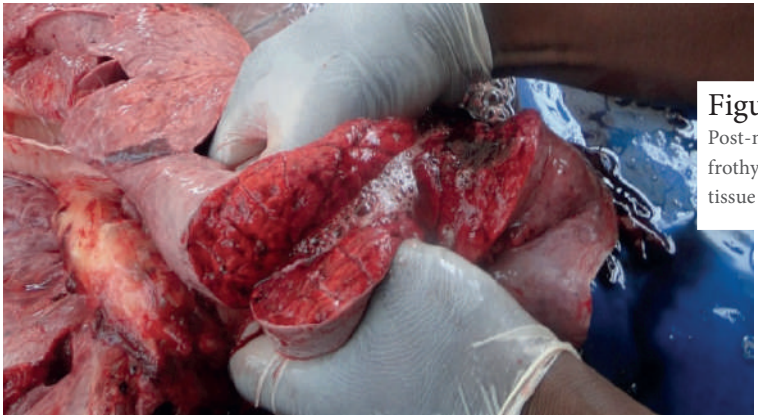

Figure 8

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

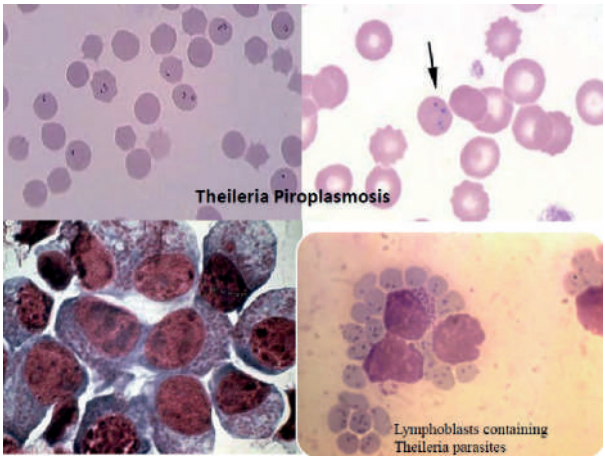

Figure 9

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- Ndis (Samburu) , Lonyang (Turkana)

Other name(s): 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.
- The Anaplasma bacteria infects red blood cells of cattle, sheep, goats, buffalo, and some wild ruminants.
- Anaplasmosis is the second most important tick borne disease in Kenya after ECF.

Distribution (Epidemiology)

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

Causative agent/ Aetiology

- Anaplasma bacteria is from the order Rickettsiales, family Anaplasmataceae, genus Anaplasma.
- 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.
- The Anaplasma genus also includes species that were previously known as Ehrlichia like A. bovis (formerly E bovis), and A. platys (formerly E platys). These can invade erythrocytes of other mammalian hosts and therefore can be potentially zoonotic.

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.
- Transplacental transmission has been reported and is usually associated with acute infection of the dam in the second or third trimester of gestation.
- There is a strong correlation between age of cattle and severity of disease. Calves are more resistant to disease (although not infection) than older cattle. This resistance is not due to colostrum antibody from immune dams but it is from the fact that calves born in endemic areas become infected with A. marginale early in life and gain carrier status without manifesting any signs. This situation is called enzootic stability.

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

Affected group

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

Clinical signs

- Steadily increasing temperatures but does not exceed 41°C like in ECF.
- Anaemia, weakness and respiratory distress particularly after exercise.
- Jaundice and marked loss of body condition.
- Urine is often brown due to the presence of bilirubin.
- Constipation, faeces are dark and firm and sometimes covered with mucus.
- Severely affected animals may die.
- 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.
- Anaplasmosis frequently occurs at the same time with ECF. The reason is that ECF causes a depression of the animal's immune responses. This then allows an Anaplasma infection which was already present in the carrier state in the animal to flare up as clinical Anaplasmosis.
- Animals with mixed ECF and Anaplasma infection are very sick with poor prognosis. Research at KALRO Muguga in Kenya indicates that even low Anaplasma parasitaemia (2%) can cause severe symptoms if 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.
- 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.

Morbidity and Mortality Rates Estimates

- A recent molecular study in Kenya, estimated the prevalence of Anaplasma in dairy cows in Nairobi county and its environs to be; A. platys (44.8%), A. marginale (31%) and A. bovis (13.8%). Another study found the seroprevalence of Babesiosis, Anaplasmosis and ECF to be 0.42%, 4.64% and 4.92% and 1.45%, 32.11% and 39.05% in the rural and peri-urban areas of Western Kenya respectively.
- Samburu county has an estimated apparent morbidity rate of 19%, mortality rate of 7 % and case fatality rate of 37%.

Seasonality of occurrence

- The presence of active and infected tick vectors determines season of occurrence. reports indicate it can occur in all seasons.
- In Samburu county, Anaplasmosis occurrence is common during the short rainy season locally known as Lgergerua (April-May) in the Samburu community and long rainy season, Akiporo (March and June) in the Turkana community.

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 the tip of either the ear or the tail of the sick animal (peripheral blood smear).
- 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

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 parasites.

Prevention/control

- Vaccine is not available in Kenya.
- Vaccine is available in South Africa, Australia, Israel, and South America using a live vaccine derived from Anaplasma centrale (originating from South Africa). There is a killed vaccine in USA used as an experimental vaccine. It requires two doses (28 days apart) and booster doses every 1 to 2 years depending on herd history
- Attempts to use live or attenuated A. marginale have results in fatal disease.
- Sustained and stringent tick control using acaricides is the most common strategy

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

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)

Further Reading

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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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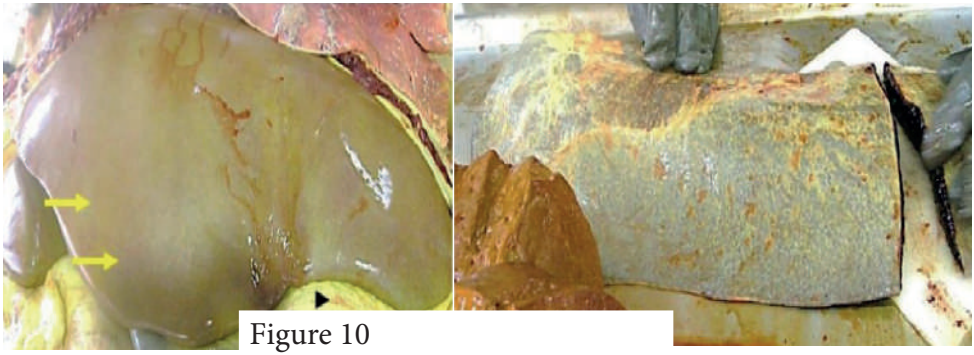


Figure 10

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

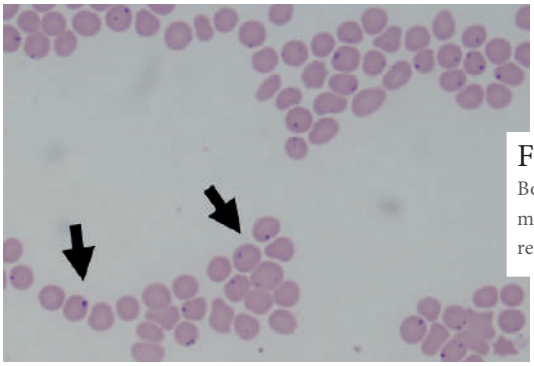


Figure 11

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 | Pneumonia -Lkipei Loonkishu (Samburu) Loukoi (Turkana)

Other name(s): Bronchial pneumonia, Fibrinous pleuropneumonia. Shipping fever and Bovine

Respiratory Disease (BRD) complex. BRD is defined as a "disease complex" for two reasons:

- It is usually caused by a variety of pathogens, both viral and bacterial, that interact with one another to produce serious disease, and
- Behaviour of these pathogens follows a sequential process that, step by step, results in sick animals.

Aetiology:

- Multi-factorial cause with a variety of physical and physiological stressors combining to predispose cattle to pneumonia.
- Bacterial pathogens cause the acute syndrome by invading the bovine respiratory tract that has been compromised by viral infections. Preceding and contributing to the viral infection is stress like low plane of nutrition, change in ambient temperature and humidity. All of which reduce energy reserves.
- Several species of bacteria have been isolated, but the most commonly found species are Mannheimia (M.) haemolytica serotype S1(formerly Pasteurella (P.) haemolytica), P. multocida, Histophilus (H.) somni (formerly Haemophilus) and Mycoplasma bovis (that may be associated with polyarthritis in calves)
- The most important are M haemolytica serotype S1 and P. multocida
- Viruses such as Infectious Bovine Rhinotracheitis (IBR), Bovine Viral Diarrhoea Virus (BVDV), Bovine Respiratory Syncytial Virus (BRSV) and Parainfluenza Virus 3 (PI3) are the primary invading agents that predispose to secondary bacterial infections.
- The bacteria are normal inhabitants of the nasopharynx of cattle.
- If the pneumonia becomes chronic there is development of pulmonary abscessation due to Trueperella pyogenes (formerly Arcanobacterium pyogenes).

Transmission and Pathophysiology

- Under normal conditions, the bacteria remain confined to the upper respiratory tract, in particular the tonsillar crypts, and is difficult to culture from healthy cattle. After stress or viral infection, the replication rate increases rapidly, as does the likelihood of culturing the bacterium.
- The increased bacterial growth rate in the upper respiratory tract, is followed by inhalation of the bacteria and colonization of the lungs.

Clinical Findings

- Clinical signs of bacterial pneumonia are often preceded by signs of viral infection of the respiratory tract. With the onset of bacterial pneumonia, clinical signs increase in severity.
- Auscultation of the cranioventral lung field reveals increased bronchial sounds, crackles, and wheezes.
- Fever (40°–41°C)- earliest signs of the BRD complex - temperatures should be taken in the morning hours of dull animals that are not feeding when taken to pasture (afternoon

temperatures may be affected by high ambient temperatures). Affected animals hang their heads, look lethargic and often stand away from other cattle.

- Serous eye and nasal discharge- earliest indicators of BRD, discharge is watery, sticky and clear. Serous discharge usually starts from the nose, then moves to the eyes as the disease progresses. The eye and nasal discharge then becomes purulent that is an indicator of more advanced BRD, discharge is thick, cloudy and pus-filled. The cloudy appearance is caused by white blood cells that have localised in the respiratory tract to attack the infection. In some acute BRD cases, blood may appear in the nasal discharge due to irritation in the respiratory tract. The protective mucosal lining is broken down and enters the respiratory system, where it is blown out.
- Stiff gait- due to an increased systemic endotoxin load that results in muscle and joint soreness.
- Crusty muzzle- caused by animal licking the muzzle less due to endotoxin overload and mild dehydration as it is not taking in water.
- Salivation- animal's overall feeling of malaise cause it to drool and gape more than usual.
- Mild diarrhoea- endotoxins in the animal's system cause displacement of body fluids, dumping more fluid into the bowel and disrupting normal absorption of food, causing loose stools.
- Rapid, shallow breathing. More blood is distributed to the infected portion of the lungs, causing occlusion of airflow. The animal has to breathe harder to get good air exchange, because parts of its lungs are not working properly. Early morning, when environmental influences are less, is the best time to evaluate breathing. Increased respiration when the environmental temperature is high may be caused more by the external environment than disease.
- Soft coughing. In early BRD cases, the lungs and airways are generally painful, so the animal will try to clear the airway with mild, tentative coughing. Loud, prominent coughing or "honking" indicates far more chronic, advanced cases, at which point treatment is difficult. Moist cough and a rapid, shallow respiratory rate is a good indicator of early BRD.
- In severe cases, pleurisy develop, characterized by an irregular breathing pattern and grunting on expiration.

Post-mortem lesions

- M. haemolytica causes- haemorrhagic fibrinonecrotic pneumonia. The pneumonia has a bronchopneumonic pattern. Grossly, there are extensive reddish black to grayish brown cranioventral regions of consolidation with gelatinous thickening of interlobular septa and fibrinous pleuritis. There are extensive thromboses, foci of lung necrosis, and limited evidence of bronchitis and bronchiolitis.
- P. multocida is associated with only small amounts of fibrin exudation, some thromboses, limited lung necrosis, and suppurative bronchitis and bronchiolitis.
- H. somni infection of the lungs results in purulent bronchopneumonia that may be followed by septicaemia and infection of multiple organs. H somni is associated with extensive fibrinous pleuritic.
- Pulmonary abscessation can occur as the pneumonia becomes chronic. Abscesses develop in 3 weeks but do not become encapsulated until 4 weeks. T. pyogenes is frequently cultured from these abscesses.

Morbidity and Mortality rates

- Morbidity and mortality rates vary as the disease is depends on complex interaction of animal susceptibility and environmental

factors. Studies have determined morbidity rates of about 20% and mortality rate of between 5 to 10% if treatment is not instituted early.

- Samburu county, has an estimated apparent morbidity rate of 2%, mortality rate of 1% and case fatality rate of 50%.

Seasonal occurrence

- Bovine pneumonia can occur at any season but is most common during periods of stress that results in animal consuming less feed or during the changing of seasons that results in low ambient temperatures.
- In Samburu county, occurrence is common during the long rainy season locally known as Ltumuren (October to December) in the Samburu community and the long dry season locally known as Akaamu nakoyen (July to October) in the Turkana community

Diagnosis

- Post-mortem gross necropsy findings and bacterial culture. Because the bacteria involved are normal inhabitants of the upper respiratory tract, the specificity of culture can be increased by collecting ante mortem specimens from the lower respiratory tract by tracheal swab, trans tracheal wash, or Broncho alveolar lavage.
- Lung specimens can be collected for culture at necropsy. If possible, specimens for culture should be collected from animals that have not been treated with antibiotics to permit determination of antimicrobial sensitivity patterns.

Treatment

- Early recognition by trained personnel skilled at detecting the early clinical signs of disease followed by treatment with antibiotics is essential for successful therapy.

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM hind legs or neck muscles q48 hours PRD IM
Amoxicillin -150 Long acting	Broad spectrum bactericidal antibiotic	1 ml per 10 kg BWT IM repeatable if necessary after 48 hours.
Use either Oxytetracycline or Amoxycillin		
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

Further Reading

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

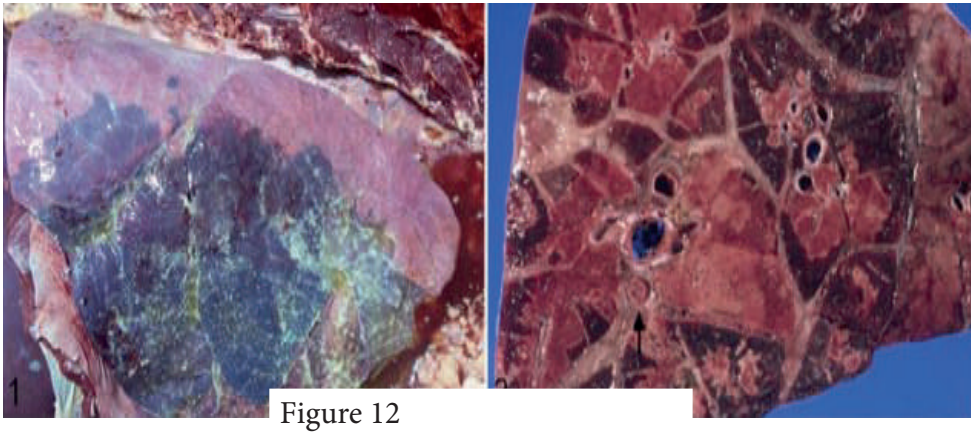


Figure 12

Fibrinous bronchopneumonia lung lesions due to Mannheimia haemolytica serotype S1.

Picture 1: The ventral regions of cranial, middle, and caudal lobes are markedly dark purple, consolidated, regionally collapsed, and sharply demarcated. Thick mats of fibrin strands are adhered to the visceral pleura, and the interlobular septa are expanded by oedema.

Picture 2. Cut section of lung; bovine showing necrotizing bronchopneumonia. The lobular pattern is accentuated by severe oedema of interlobular septa and necrosis, centered on airways imparting a characteristic marble pattern. Necrotic foci are irregular, discrete, sharply delineated from the remaining parenchyma, and bordered by a pale white necrotic rim. Note that a thrombus (arrow) completely occludes the lumen of a blood vessel.

Sourced from: http://vet.sagepub.com/content/48/2/338

2.6 | Fascioliasis- Lkurui Lemonyua (Samburu), Ngirtan (Turkana)

Other name(s): Common liver fluke

Definition

- Common parasitic liver disease of both cattle and sheep caused by Fasciola hepatica

Distribution (Epidemiology)

- Global distribution
- Fasciola hepatica and has a broad host range, including humans.

Aetiology

- Fasciola hepatica leaf shaped parasite from the helminth class Trematode.
- The parasite has as a broad host range, including people.

Pathophysiology

- Eggs are passed in the faeces where miracidia develop. Hatching only occurs in water, and miracidia are short-lived (approximately 3 hours). Miracidia infect pond snail (Galba truncatula, previously known as Lymnaea truncatula).
- Once in the snail miracidia undergoe asexual development and multiplication through the stages of sporocysts, rediae, daughter rediae, and cercariae. After 6–7 weeks or longer if temperatures are low), cercariae emerge from snails,
- The cercariae encyst on aquatic vegetation, and become metacercariae. Metacercariae remain viable for many months unless they become desiccated.
- After ingestion by the host, usually with herbage, the metacercariae excyst in the duodenum, penetrate the intestinal wall, and enter the peritoneal cavity, where they migrate to the liver. The time required for this transit can vary and results in delayed development rates, which affects the efficacy of some treatments because many are effective against flukes only later in their development. The young flukes penetrate the liver capsule and tunnel through the parenchyma for 6–8 weeks, growing and destroying tissue. They then enter small bile ducts and migrate to the larger ducts and, occasionally, the gallbladder, where they mature and begin to produce eggs.
- The prepatent period is usually 2–3 months. The minimal period for the completion of one entire life cycle is 17 weeks. Adult flukes may live in the bile ducts of sheep for years but in cattle they are eliminated from the liver within 5 to 6 months.

Clinical signs

- Fascioliasis symptoms in cattle is determined by the number of metacercariae ingested. Acute disease occurs 2 to 6 weeks after the ingestion of large numbers of metacercariae (usually >2,000) over a short period. Acute disease manifests as distended, painful abdomen; anaemia; and sudden death.
- In subacute disease occurs when cattle ingest 500 to 1,500 metacercariae are ingested over longer periods of time. The cattle survive for longer 7 to 10 weeks even in cases of significant hepatic damage, but deaths occur due to haemorrhage and anaemia.
- Chronic fascioliasis occurs as a result of ingesting moderate numbers 200 to 500) of metacercariae over longer periods of time. Signs include progressive emaciation, anaemia,

unthriftiness, submandibular oedema (bottle jaw), and reduced milk production.

- It should be noted that heavily infected cattle may show no clinical signs other than progressive emaciation despite access to good pasture. The immunity to other pathogens may also be reduced.
- In cattle, a partial acquired resistance develops beginning 5–6 months after infection.

Post mortem lesions

- Severity depends on the number of metacercariae ingested, the phase of development in the liver, and the species of host involved.
- During the first phase, immature, wandering flukes destroy liver tissue and cause haemorrhage. The main lesions seen are enlarged liver that is friable with fibrinous deposits on the capsule. Migratory tracts can be seen, and the surface has an uneven appearance.
- In the second phase when flukes enter the bile ducts, ingest blood and damage the mucosa with their cuticular spines. The lesions are consistent with chronic liver damage such as cirrhosis where the damaged bile ducts become enlarged, or even cystic, and have thickened, fibrosed walls. In cattle but not sheep, the duct walls become greatly thickened and often calcified. Aberrant migrations occur more commonly in cattle, and encapsulated flukes may be found in the lungs. Mixed infections with Fasciola gigantica can also be seen in cattle.
- Tissue destruction by wandering flukes may create a microenvironment favourable for activation of Clostridial spores and animal can succumb to infection.

Morbidity and Mortality Rates

- Morbidity rates are between 40 and 60% in most areas where animals have access to contaminated pasture
- Mortality rates in cattle is generally low between 0 and 13%. Most cases of infection are diagnosed at slaughter for most animals. However, in weaned or yearling calves it may be high at 30% due to ingestion of large numbers of mertcercariae cysts over a short period of time.
- In Samburu county, fascioliasis has an estimated apparent morbidity rate of 4%, mortality rate of 0% and case fatality rate of 0%.

Age group affected and Seasonality of occurrence

- Affects all ages of cattle that have access to swampy pasture with intermediate snail host. However, beef breeds have higher incidence.
- Can occur throughout the year if animals are grazed in the swampy areas. But most common in the long dry season due to availability of pasture.
- In Samburu county, occurrence is common during the long dry season locally known as Lameioodo (June to September) in the Samburu community and Akaamu nakoyen (July to October) in the Turkana community.

Diagnosis

- Composite faecal egg counts where dung samples are collected from 10 animals and a single (composite) egg count performed at the laboratory for faecal egg count.
- Faecal microscopic identification of the oval, operculated, golden brown eggs that must be distinguished from those of paramphistomes (rumen flukes), which are larger and clear. Eggs of F hepatica cannot be demonstrated in faeces

during acute fasciolosis. In subacute or chronic disease in cattle, the number varies from day to day, and repeated faecal sedimentation may be required.

- Diagnosis can be aided by an ELISA (commercially available in Europe) that enables detection at 2 to 3 weeks after infection and well before the patent period using blood serum or milk.
- Blood serum analysis of plasma concentrations of γ-glutamyltransferase, which are increased with bile duct damage, are also helpful during the late maturation period when flukes are in the bile ducts.
- At necropsy, the nature of the liver damage is diagnostic. Adult flukes are readily seen in the bile ducts, and immature stages may be squeezed or teased from the cut surface.

Differential Diagnosis (DDx)

- Helminthiasis
- Paratuberculosis
- Pyelonephritis,
- Chronic mastitis,
- Chronic suppurative pneumonia.

Treatment

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

* 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

- Animals should always drink from safe, uncontaminated water sources. If this is not possible, measures should be taken to control the snail with copper sulphate, but this is short-term and not very effective.
- Animals should be dosed in November – after the start of the rains and again at the end of the rains.
- Examination of the livers at slaughter houses will give an indication of the degree of infestation and thus used to advise livestock keepers when to deworm animals.

Zoonotic potential

- Yes, important parasitic zoonosis
- WHO estimated in 1995 that approximately half of the 2.39 million infected individuals globally were living in three Latin American countries (Bolivia, Ecuador and Peru).
  The Andean highlands represent the most endemic areas of the world, with high levels of prevalence of infection among indigenous communities.

Notifiable disease

No

Further Reading

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

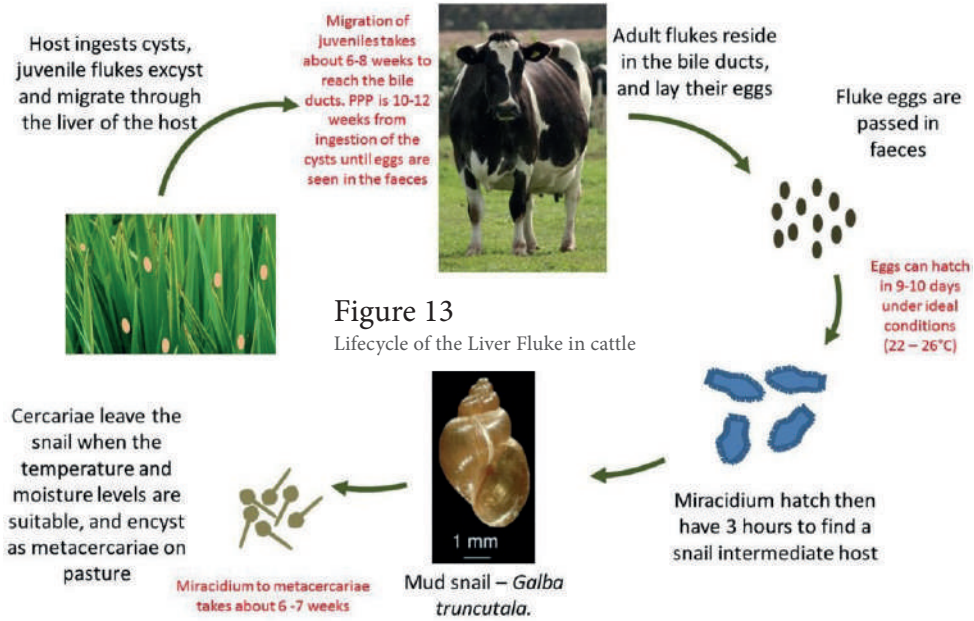
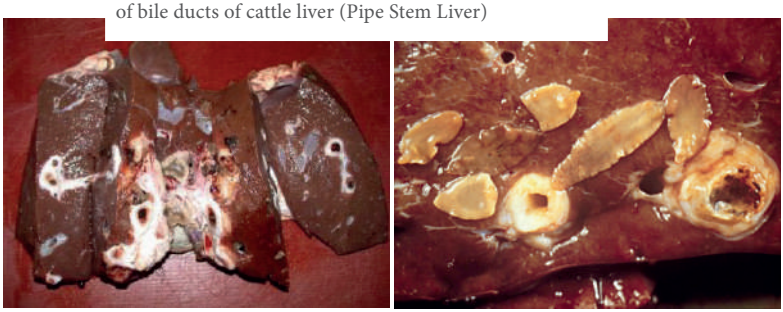


Figure 13
Lifecycle of the Liver Fluke in cattle

Sourced from: https://www.farmhealthonline.com/US/disease-management/cattle-diseases/liver-fluke/

Figure 14

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



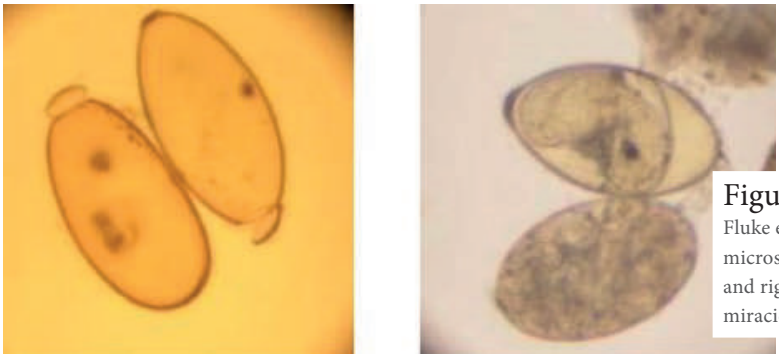


Figure 15

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

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

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

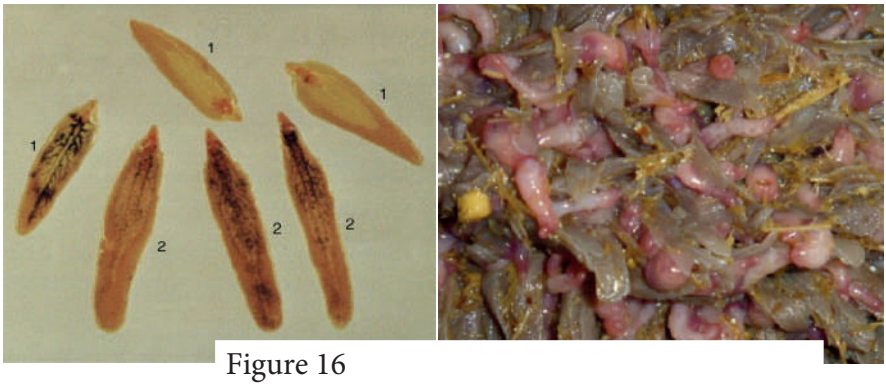


Figure 16

(1) Adult Fasciola hepatica (2) Adult Fasciola gigantica

Left Picture

Adult paramphistomes on the surface of the rumen

Sourced from: https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/liver-fluke

2.7 | Black Quarter (BQ)- Nenkeju (Samburu), Lokwas/ Ngwaat (Turkana)

Other name(s): 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)

- Global distribution

Causative agent/ Aetiology

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

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.

Morbidity and Mortality Rates

- A study in Ethiopia estimated a morbidity and mortality rate local zebu cattle population of 17.9 % and 3.6 % respectively.
- In Samburu county, apparent morbidity and mortality rate is 6% with 100% case fatality rate.

Seasonality of occurrence

- Throughout the year
- In Samburu county, amongst the Samburu community the disease is common during the short dry season known as Lamei dorop (January to March) while in the Turkana community it is common during the long dry season Akaame nakoyen (July to October),

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

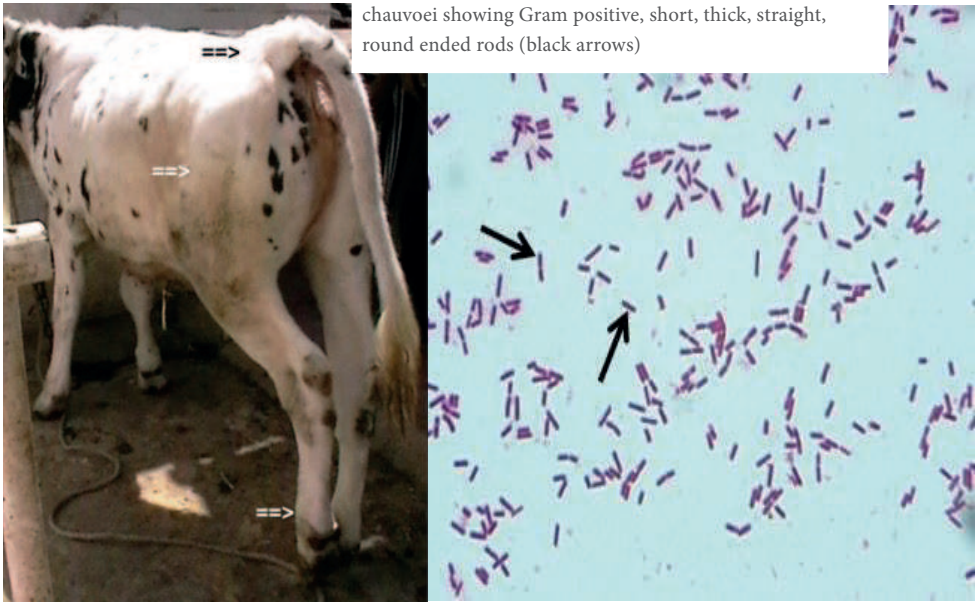
- None, but due to toxaemia the carcass meat is susceptible to rapid decomposition and can be a source of food poisoning to humans if consumed.

Notifiable disease

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

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

2.8 | Listeriosis – Mporoto (Samburu), Mporroto (Turkana)

Other name(s): Circling Disease

Definition:

- Listeriosis is a sporadic bacterial infection that affects a wide range of animals, including humans and birds.
- The most common form of the disease in adult cattle is encephalitis specifically rhombencephalitis which refers to inflammatory diseases affecting the hindbrain (brainstem and cerebellum).
- The disease is essentially a localized asymmetric infection of the brain stem that develops when L monocytogenes ascends the trigeminal nerve. Clinical signs vary according to the function of damaged neurons but often are unilateral and include depression (ascending reticular activating system), ipsilateral weakness (long tracts), trigeminal and facial nerve paralysis, and circling (vestibulocochlear nucleus), excitement, and recumbency.

Distribution, aetiology/causative agent

- Listeriosis occurs worldwide, more frequently in temperate and colder climates.
- Main source of infection is carrier animals that harbour the bacteria in their intestines.
- The genus Listeria is composed of gram-positive saprophytic bacteria widely distributed in the environment. including soil, water, sewage, decaying vegetation, feed, and human and animal faeces.
- There are 17 species identified. Listeria monocytogenes is the species usually associated with animal and human disease. Other species L. ivanovii or L. innocua have also been linked to ruminant and human disease.
- Contaminated dairy products are one of most common foods involved in human listeriosis outbreaks. Milk pasteurisation has reduced the risk, even though cases continue resulting from post processing contamination and consumption of raw milk products.
- Among ruminants, bovine species has the highest prevalence of carrier animals that shed bacteria asymptomatically.
- The main source of L. monocytogenes infection in ruminants is poorly fermented silage but there are reports of listeriosis in grazing ruminants, In Samburu infections are associated with cattle grazing decaying grass that is being inhabited by wild rats and pigs. Focus Group discussion indicated that livestock keepers believe that the urine of the two wild animals contaminate the grass and cause the disease.

Seasonal occurrence

- In Samburu county incidences are common during the months of June to October the long dry season locally known as Lameioodo in Samburu language and Akaamu nakoyen in Turkana language.

Clinical signs

- Initially, affected animals are anorectic, depressed, and disoriented. They may propel themselves into corners, lean against stationary objects, or circle toward the affected side. Facial paralysis with a drooping ear, deviated muzzle, flaccid lip, and lowered eyelid often develops on the affected side, as well as lack of a menace response and profuse, almost continuous, salivation; food material often becomes

impacted in the cheek because of paralysis of the masticatory muscles. Terminally affected animals fall and, unable to rise, lie on the same side; involuntary running movements are common.

- Ruminants are particularly susceptible to infection, with rhombencephalitis being the most common clinical presentation. The most common clinical manifestation of listeriosis is a localized ascending asymmetric infection of the brain stem of ruminants by Listeria monocytogenes. The resultant meningoencephalitis damages the origins of cranial nerves V, VII, and VIII in the brain stem, resulting in unilateral facial paresis or paralysis, head tilt, loss of sensation, depression, and recumbency.
- Other signs in cattle include abortion, neonatal septicaemia, keratoconjunctivitis/uveitis and enteritis.
- The uterus of all domestic animals, especially ruminants, is susceptible to infection with L monocytogenes at all stages of pregnancy, which can result in placentitis, foetal infection and death, abortion, stillbirths, neonatal deaths, metritis, and possibly viable carriers. The metritis has little or no effect on subsequent reproduction; however, Listeria may be shed for 1 month or longer via the vagina and milk.

Morbidity and mortality rate

- In most cases infection of the herd may approach 100% but morbidity rate, that is animals that show clinical signs are low estimated to be 2 to 10%. Mortality rate is estimated to also be low at between 20 and 30% if treatment is done early.
- In Samburu county the apparent morbidity rate is 7% with a mortality rate of 5% and a case fatality rate of 71%.

Post-mortem lesions

- Gross lesions in adult cattle are few except for congestion of meninges.
- In calves (age <3 weeks), have focal hepatic necrosis and marked haemorrhagic gastroenteritis.
- Aborted foetuses have marked autolysis, clear to blood-tinged fluid in serous cavities, and numerous small necrotic foci in the liver. Necrotic foci may be found in other viscera such as the lung and spleen.

Diagnosis

- Gram-stained smears of abomasal contents in calves and foetus reveal numerous gram-positive, pleomorphic coccobacilli.
- In adults, diagnosis is based on clinical signs of asymmetric brain-stem dysfunction with depression that is confirmed by means of bacterial culture or immunofluorescence assay.

Differential Diagnosis

- Rabies
- Ketosis
- Bovine spongiform encephalopathy
- Polioencephalomalacia (Thiamine deficiency)
- Lead poisoning
- Brain abscesses
- Coenurosis- present with circling, contralateral blindness, and proprioceptive deficits; however, they show no cranial nerve deficits.
- Vestibular disease is common in growing ruminants; these animals typically show ipsilateral spontaneous nystagmus or strabismus, and they remain bright and alert without trigeminal nerve dysfunction.

Treatment

- Due to the urgent need to have the drug at the site of infection use short acting preparations

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting Most effective for acute cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM q24 hours for 3 to 5 days.
Penstrep Procaine penicillin G200000 IU. Dihydrostreptomycin sulphate200 mg	Broad spectrum Bacteriocidal antibiotic	1 ml per 20 kg BWT IM only for 3 to 5 days.
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

Zoonotic risk

- Yes
- Samples from suspected clinical cases of listeriosis carries the risk of zoonotic infection and should be handled with caution. Aborted foetuses and necropsy of septicaemic animals present the greatest hazard.
- Humans have developed fatal meningitis, sepsis, and papular exanthema on the arms after handling aborted material.
- Pregnant animals and women should be protected from infection because of danger to the foetus, with possible abortion, stillbirth, and infection of neonates.
- Although listeriosis in humans is rare (upper estimate of 12 cases per million populations per year), mortality can reach 50%. Most human cases involve older patients, pregnant women, or immunocompromised individuals. In ruminants with meningoencephalitis, L monocytogenes is usually confined to the brain and presents little risk of transmission, unless the brain is handled during necropsy.
- L monocytogenes can be isolated from milk of mastitic, aborting, and apparently healthy cows. Excretion in milk is usually intermittent but may persist for many months. Infected milk is a hazard, because the organism may survive certain forms of pasteurization. Listeria also isolated from the milk, as well as cheeses, from sheep and goats.

Notifiable disease

- No

Prevention and control

- There is a vaccine available but due to sporadic nature of disease occurrence it is not commercially available in Kenya.
- Immediately remove the herd from the area with contaminated grass or withdraw contaminated silage.

Further reading

1. Carolina Matto, Gustavo Varela, Maria Ines Mota, Ruben Gieneechini and Rodolfo Rvero (2017). Rhombencephalitis caused by Listeria monocytogenes in a pastured bull.
Journal of Veterinary Diagnostic Investigation, Volume: 29 issue: 2, page(s): 228-231.
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2. MSD Veterinary Manual https://www.msdvetmanual.com/

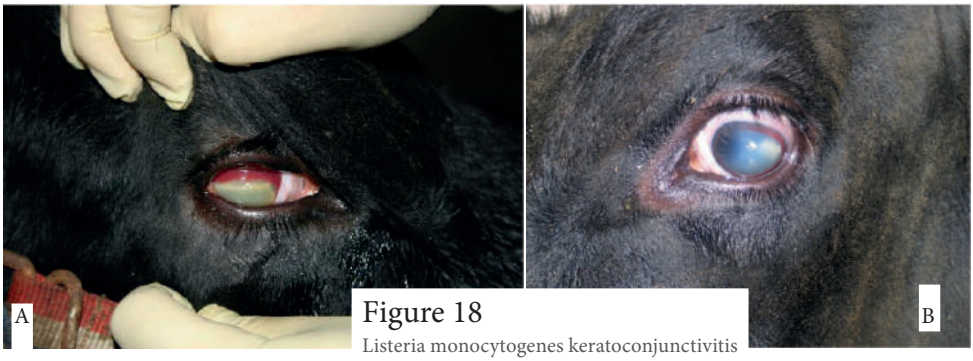


Figure 18
Listeria monocytogenes keratoconjunctivitis and uveitis in dairy cattle

A - anterior uveitis, yellowish anterior chamber fluid, opaque cornea, epiphora and hyperaemic conjunctiva.

B - bluish corneal opacity and white focuses on the cornea, floccules of material attached to the endothelium of the cornea, anterior uveitis, and hyperaemic conjunctivae

Sourced from: https://www.semanticscholar.org/paper/LISTERIA-MONOCYTOGENES-KERATOCONJUNCTIVITIS-AND-IN-Stari%C4%8D-Krizanec/a36210dd2954c9562718a632c5909f710b6b9d5a

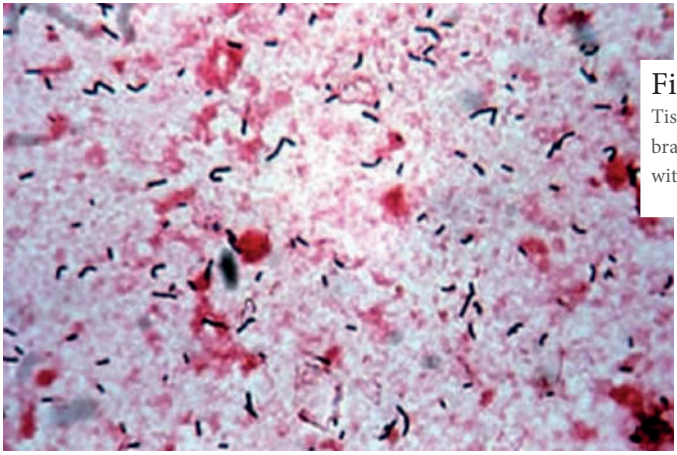


Figure 19
Tissue smear of post-mortem brain stem samples of an animal with Listeriosis

Note the presence of large numbers of the V-shaped gram-positive bacterium Listeria monocytogenes (Gram stain, high power)

Sourced from: https://www.msdvetmanual.com/generalized-conditions/listeriosis/listeriosis-in-animals

2.9 | Bovine Babesiosis (BB)- Nkula (Samburu) Akul Narrengak (Turkana)

Other name(s): 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)

- BB is found in areas where its arthropod vector is distributed, especially tropical and subtropical climates. Babesia bovis and B. bigemina are widely distributed in Africa, Asia, Australia, and Central and South America. Babesia divergens is economically important in some parts of Europe and northern Africa.

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

- 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),
- 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.

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

- High fever (41°C) persists throughout, and is accompanied 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%

Post mortem lesions

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

Morbidity and Mortality Rates

- Morbidity and mortality vary greatly and are influenced by several factors, such as prevailing treatments employed in an area, previous exposure to a species/strain of parasite, age, cattle breed, and vaccination status.
- In endemic areas, cattle become infected at a young age and develop a long-term immunity. However, outbreaks can occur in these endemic areas if exposure to ticks by young animals is interrupted or immuno-naïve cattle are introduced. The introduction of Babesia infected ticks into previously tick-free areas may also lead to outbreaks of disease.
- In Samburu county the apparent morbidity rate is 16%, mortality rate 8% and case fatality rate of 50%.

Seasonality of occurrence

- Throughout the year, depends on tick vector presence and availability of susceptible host.
- In Samburu county, the disease is common amongst the Samburu community herds during the short rainy season called Lgergerua (April to May) while in the Turkana community herds during the long dry season, Akaamu nakoyen (July to October).

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

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

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

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 splenectomized 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 20

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

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

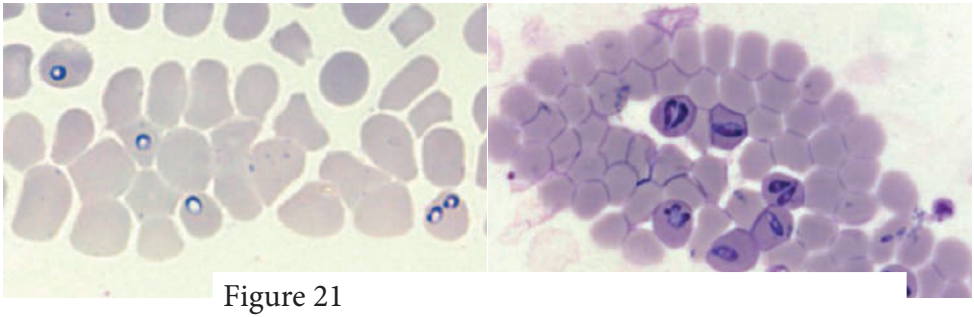


Figure 21

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

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

2.10 | Trypanosomiasis – Saar (Samburu) /Ekwakoit/Lokipi (Turkana)

Other name(s): 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.

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 Samburu community herds, trypanosomiasis in cattle is common during the short dry season (Lamei dorop- January to March) and long dry season (Lameioodo- June to September). In Turkana community cattle herds, the disease is most common during the long dry season, Akaamu nakoyen (July to October).

Morbidity and Mortality rates

- Study estimate a morbidity rate of between 30 and 50% and mortality rate of 6 to 30%.
- In Samburu county an apparent morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25% 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

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

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

Prevention/control

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

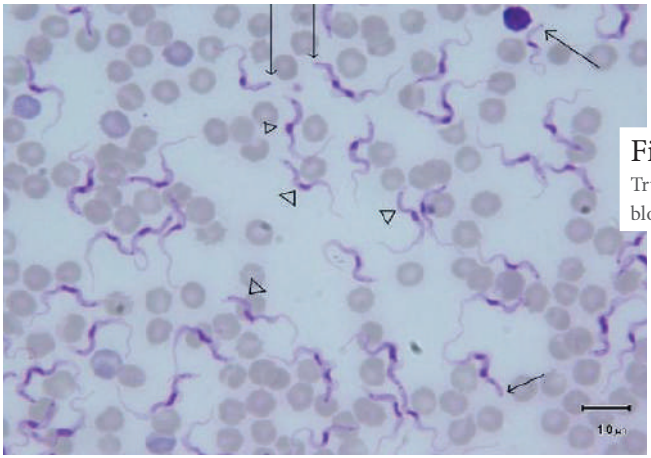


Figure 22
Trypanosome evansi in cattle blood smear

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

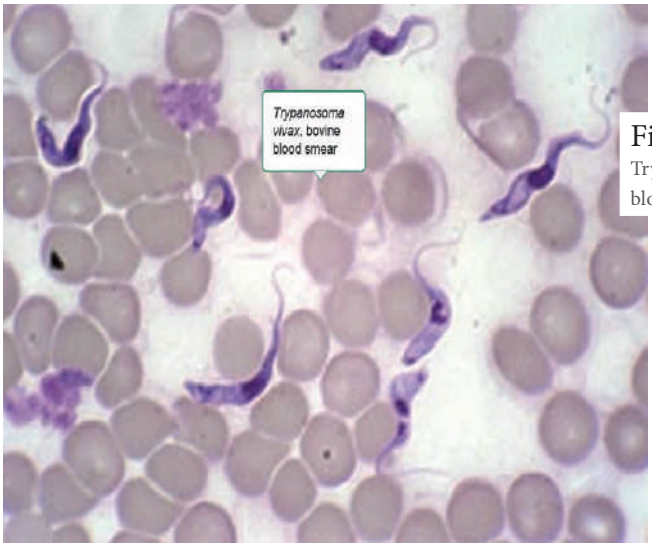


Figure 23
Trypanosome Vivax in bovine blood smear

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

Chapter 3:

Camel Diseases

3.1 | Haemorrhagic Septicaemia (HS) – Nalngaringari (Samburu) Longaruei/ Lokou (Turkana)

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)

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

Causative agent/ Aetiology

- HS is caused by two gram-negative coccobacillus bacteria namely Pasteurella multocida and Mannheimia haemolytica. The two bacteria have several serotypes that cause 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

Morbidity and Mortality Rates

Morbidity is low, but mortality can be as high as 80%. Morbidity depends on immunity and environmental conditions, including both weather and husbandry; morbidity is higher when animals are herded closely, in poor condition, or exposed to wet conditions.

Mortality is nearly 100% unless the animal is treated very early in the disease

In Samburu county, the apparent morbidity rate is 43%, mortality rate 23% and case fatality rate is 53%.

Seasonality of occurrence

Rainy season or cold dry season when animals are in nutritional stress

In Samburu county HS is most common amongst Samburu community camel herds during the short rainy season, Lgergerua (April-May) while in Turkana community herds during the Akiporo log rains season (March-June).

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.

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

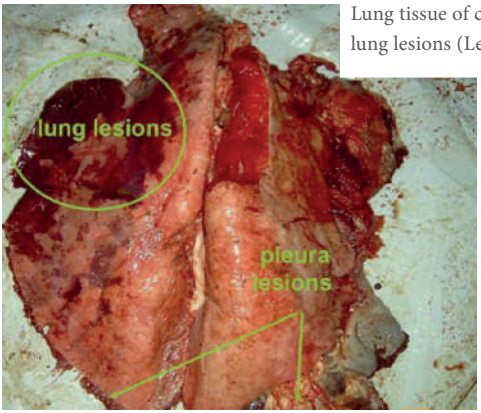
1. Gluecks, I.V., Bethe, A., Younan, M. et al. Molecular study on Pasteurella multocida and Mannheimia granulomatis from Kenyan Camels (Camelus dromedarius). BMC Vet Res 13, 265 (2017). https://doi.org/10.1186/s12917-017-1189-y
2. MSD Veterinary Manual - https://www.msdvetmanual.com/
3. Yaeger, Michael;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

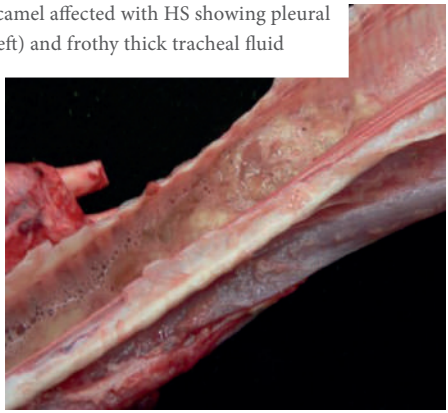
Figure 24

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 25

Lung tissue of camel affected with HS showing pleural lung lesions (Left) and frothy thick tracheal fluid





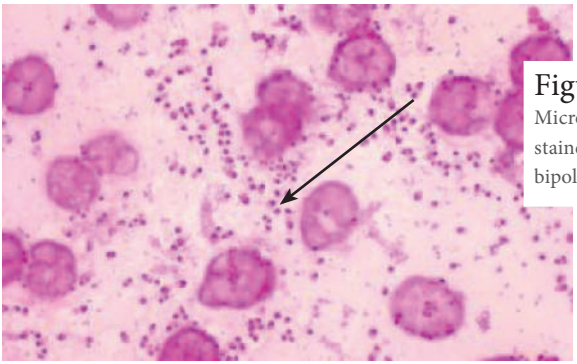


Figure 26

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 Flu- Lchama (Samburu), Lowola (Turkana)

Definition

- Starts as an upper respiratory tract infection mostly due to a virus and in some cases especially in young normal flora bacteria invade and can result in pneumonia

Distribution (Epidemiology)

- Disease occurs in all camel rearing regions of the world
- In Samburu county camel flu is most common during the Lgergerua (April to May) short rainy season amongst Samburu community herds. In Turkana herds the disease is more common during the long rain season, Akiporo that occurs in the months of March to June.
- The apparent morbidity rate in Samburu county is 58% with a 27% mortality rate and 47% case fatality rate.

Causative agent/ Aetiology

- The causative virus and bacteria can be found in the upper respiratory tract of normal camels. Predisposing circumstances, cause the normal flora to invade respiratory tissues and cause serious disease.
- A recent metagenomic sequencing analysis on nasopharyngeal swab samples from 108 MERSCoV-positive dromedary camels in UAE identified 13 genera of viruses from 10 viral families including Coronaviridae, Nairoviridae, Paramyxoviridae, Parvoviridae, Polyomaviridae, Papillomaviridae, Astroviridae, Picornaviridae, Poxviridae, and Genomoviridae.
- A similar study in Sudan that analysed lung tissue identified parainfluenza virus 3 (PIV3), influenza viruses A, B and D, adenovirus, respiratory syncytial virus (RSV), and infectious bovine rhinotracheitis (IBR).
- The most common pathogenic bacteria are Staphylococcus aureus, Corynebacterium pyogenes, Streptococcus pyogenes, Escherichia coli, Klebsiella pneumonia, Pseudomonas aeruginosa, Trueperella pyogenes (formerly Arcanobacterium pyogenes), Mannheimia haemolytica and Pasteurella multocida.

Mode of transmission and pathophysiology and affected group

- 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 viral or 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°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.
- ELISA or molecular testing for viruses

Differential Diagnosis (DDx)

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.

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

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.
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Recent outbreak of Acute Respiratory Disease in Kenya

(Account and Picture source from Emmanuel Lesiantam – Samburu County)

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.

Further reading

1. Li Y, Khalafalla AI, Paden CR, Yusof MF,Eltahir YM, Al Hammadi ZM, et al. (2017) Identification of diverse viruses in upper respiratory samples in dromedary camels from United Arab Emirates. PLoS ONE 12(9): e0184718. https://doiorg/10.1371/journal.pone.0184718
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4. 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

Figure 27

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 28

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

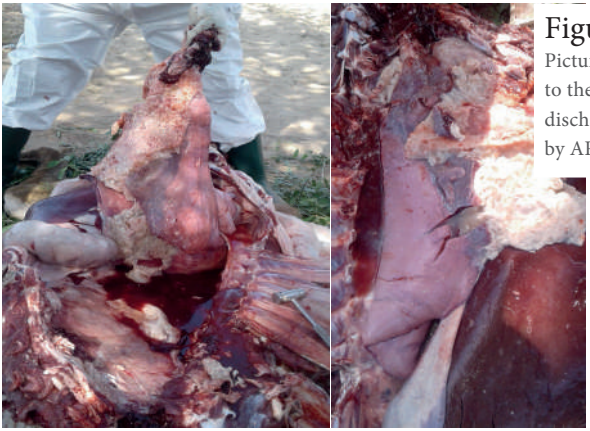

Figure 29

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 – Saar (Samburu) Ekwa Koit/ Saar/Lokipi (Turkana)

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)

- Most camels live outside Africa's tsetse belt; the disease is transmitted by other haematophagus flies like Tabanus, Stomoxys, Haematopota, Chrysops and Lyperosia. Due to the wide geographic range of the flies the disease is endemic in Africa, Asia, South and central America.

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.

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.

Morbidity and Mortality Rates

- Morbidity rates in camel rearing areas varies from 20–70%. Case fatality rate (CFR) in untreated camels can be as high as 100%. Surra in cattle and buffalo tends to be chronic with a much lower CFR.
- Animals subjected to stress, like malnutrition, pregnancy, and physical labour, are more susceptible to disease causing mortality of 3%.
- In Samburu county the apparent morbidity rate is 7% with no mortality rates reported,

Seasonality of occurrence

- In some countries incidence of surra increases significantly during the rainy season when biting fly populations is high
- Studies have reported varying seasonality of occurrence with some reporting outbreaks during the end of the rainy season while others reporting higher incidences during the dry season.
- In Samburu county Trypanosomiasis is most common during the January to March short dry season (Lamei dorop) in Samburu herds. While in Turkana herds it is common during

the Akaamu nakoyen long dry season (July to October).

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 filed 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

- Camels other tsetse-transmitted trypanosomosis,
- Anthrax,
- Chronic parasitism- helminthiasis

Treatment

- Two drugs are recommended for the treatment of evansi infections in dromedaries: suramin and quinapyramine, as sulphate. Suramin marketed as Trypamidium Samorin® and quinapyramine marketed as Triquin® are both 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; Suramin 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 6-12 weeks depending on the dosage and the infection challenge.
- Quinapyramine is easier use as it subcutaneously administered. Quinapyramine methylsulphate is used as a curative

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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

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 nor likely in the near future 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.

Figure 30

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

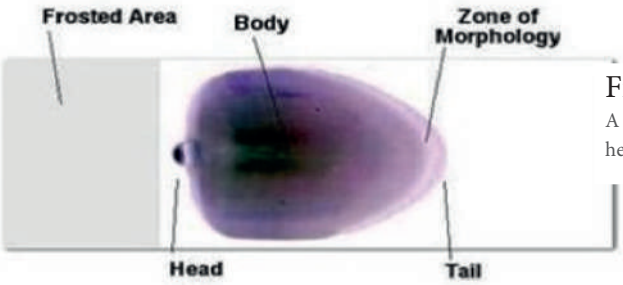


Figure 31

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

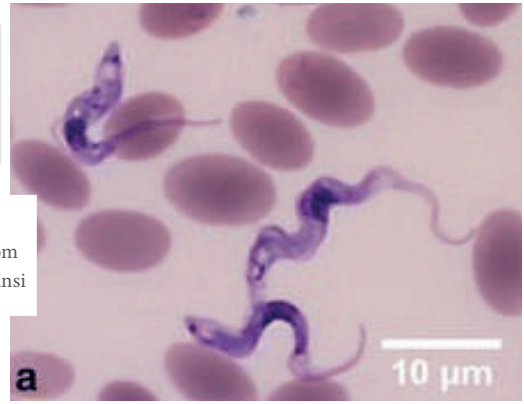
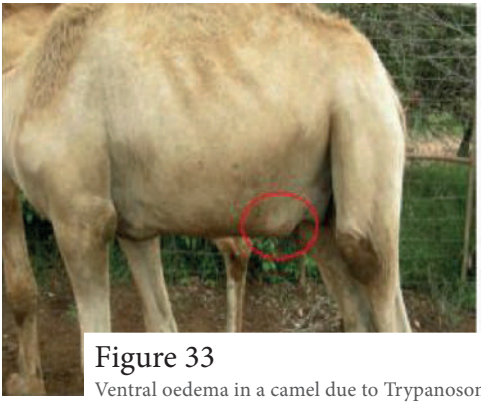


Figure 32
Giemsa-stained blood smears from camel showing Trypanosoma evansi



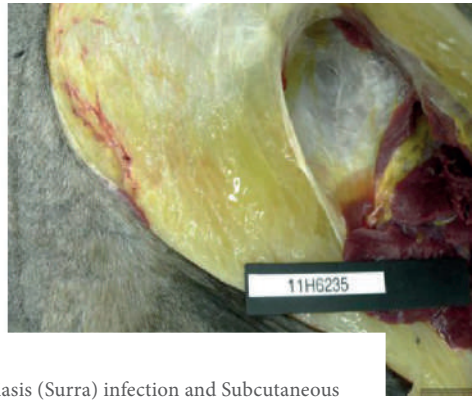
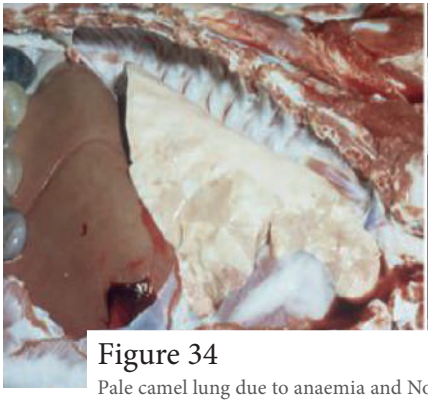


Figure 33
Ventral oedema in a camel due to Trypanosomiasis (Surra) infection and Subcutaneous oedema expansion of the subcutis by clear gelatinous fluid.



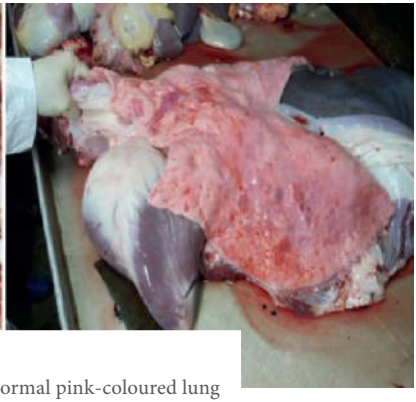


Figure 34
Pale camel lung due to anaemia and Normal pink-coloured lung

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 | Contagious Skin Necrosis- Lmonkoi (Samburu), Akitherit (Turkana)

Aetiology/ causative agent

- Bacteria - no study has ever isolated a single bacterial agent Staphylococcus aureus is the most common isolate.

Transmission and Predisposing factors

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

Occurrence

- CSN (death of living skin tissue) occurs in all regions of the world where camels are raised.
- In Samburu county the disease is endemic and is most common during the months of June to October which is the long dry season locally known as Lameioodo in Samburu language and Akaamu nakoyen in Turkana language.
- The apparent morbidity rate in Samburu county is 9% with a 2% mortality rate and 22% case fatality rate.

Main clinical signs

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

Differential Diagnosis

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

Diagnosis

- Clinical diagnosis of painful skin sores, on which the hair falls out. The sores are usually found on the neck, shoulders, withers, sides and quarters, and rarely on the lower legs.
- Collect sterile swab smear and gram stain if there is a laboratory for bacterial culture and antibiotic sensitivity. Sterile swabs should be

collected from the centre of the ulcer lesion once the necrotic scab is removed.

- The swab should be put in transport media like Stuart's transport medium. Where possible use swabs already pre-packed in media.

Post mortem lesions

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

Treatment and Control

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

Zoonotic potential

- No

Notifiable disease

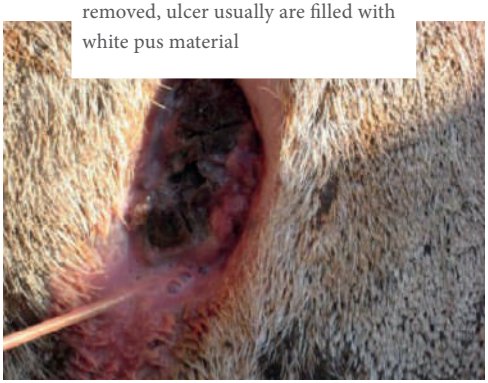
- No

Further Reading

1. 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
2. A field manual of camel diseases: Traditional and modern health care for the dromedary. Compiled by Ilse Köhler-Rollefson, Paul Mundy and Evelyn Mathias. http://vetbooks.ir/a-field-manual-of-camel-diseases-traditional-and-modern-healthcare-for-the-dromedary/
3. Studies on contagious skin necrosis and trypanosomosis in camels. Maha I. Hamed, Mahmoud R. Abd Ellah, Infectious Diseases, Clinical Laboratory Diagnosis, Department of Animal Medicine, Faculty of Veterinary Medicine, Assiut University 71526, Egypt.
https://pdfs.semanticscholar.org/79fa/a0bc28058cf4778b783f3ce20a2a5a37fcf9.pdf

Figure 35

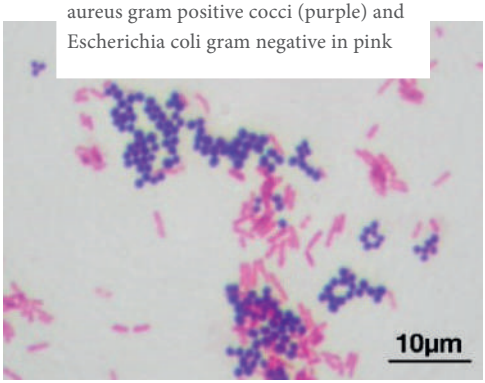
CSN ulcer lesion once crust is removed, ulcer usually are filled with white pus material



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

Figure 36

A Gram stain of mixed Staphylococcus aureus gram positive cocci (purple) and Escherichia coli gram negative in pink



Picture source: (Wikimedia commons)

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.

Transmission and age group affected

- The disease is highly contagious and is more common in the young. It can spread directly from animal to animal, particularly when it is cold and animals huddle together. It can also be spread by fomites (saddles, harnesses) or in areas of common use (bedding, tree trunks, dust baths). Young calves that are in poor body condition are more susceptible.

Morbidity and mortality rate

- In young animals' morbidity rates can be as high as 100% while mortality rate is lower at 30%. In Samburu county, livestock keepers estimated an apparent morbidity rate of 5% with no mortality rate reported.

Seasonal occurrence

- Occurs all year round. In Samburu county, mange is common in all seasons except during the rainfall season in the months of September to December as the animals are in good nutritional state.

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.

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 is a macrocyclic lactone is effective against mange two subcutaneous injections, 2 weeks apart combined with acaricide wash results in clinical resolution of signs.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.
- Prevention- From the prophylactic standpoint, it would be desirable to treat the entire herd but this may be impracticable. Priority should be given then to young camels and breeding males.

Further Reading

1. Richard D. (1987). Camel mange. Rev. sci. tech. Off. int. Epiz., 1987, 6 (2), 475-477.
https://www.oie.int/doc/ged/D8497.PDF
2. Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21.
https://lib.dr.iastate.edu/vdpam_reports/21

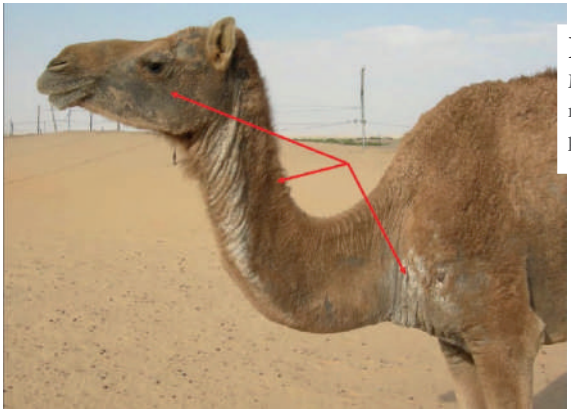


Figure 37
Mange skin lesions in a camel calf
red arrows show alopecia, encrusted plaques and hyperkeratosis of the skin

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 38
Sarcoptic mange Skin scraping showing Sarcoptes scabiei mite

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

3.6 | Caseous Lymphadenitis- Arrar (Samburu), Abuth (Turkana

Definition

- CLA has been known under a variety of names like cheesy gland disease, ulcerative lymphadenitis, actinomycotic infection, pseudoactinomycosis, suppurative lymphadenitis and lymphadenitis.
- Caseous Lymphadenitis (CLA) is one of the most important bacterial infections in livestock that has a wide host range that includes sheep, goat, cattle, camelids and equids.
- It is caused by Corynebacterium (C.) pseudotuberculosis and is characterized by abscessation of one or more superficial lymph nodes and sometimes causes infection of internal organs including mammary gland.

Distribution

- The infection is spread by inhalation, ingestion or directly through wounds. CLA has been reported in from all camel rearing countries including in Australian feral dromedary population.
- The two South American camel species, llama and guanaco, are also affected by this disease in their countries of origin but also in the USA and especially in Europe in which they were introduced as companion animals.
- The virulence of the pathogen is attributed to its exotoxin phospholipase D (PLD) which is produced by all C. pseudotuberculosis strains. Two biotypes exist: ovine/caprine (serotype I or biotype ovis) and equine/bovine (serotype II or biotype bovis) and both have been identified in dromedaries using the nitrate reduction test. Hence, CLA-vaccines for camelids should include both sero types.
- Morbidity of CLA may reach more than 90% in East African countries whereas mortality in Bactrian camels was reported to be 28%. The mortality rate in dromedaries is unknown. Both, young and adult camels are affected by the disease.
- In Samburu county the apparent morbidity rate is 7% with a mortality rate of 1% and a case fatality rate of 14%.
- The disease in Samburu county is most common in Samburu herds during the Lamei dorop short dry season in the months of January to March. In Turkana herds it is more common during the long dry season, Akaamu nakoyen during the months of July to October.

Causative agent and pathogenesis

- C. pseudotuberculosis is a short, irregular ovoid Gram-positive rod almost resembling a coccus. Smears from abscesses show a marked pleomorphism.
- Cultured colonies are small, white and dry and can be surrounded by a narrow zone of haemolysis when sheep or ox blood is used. The plates should be incubated at 37°C for at least 48 hours.
- The facultative, intracellular bacterium multiplies in infected phagocytes and gets disseminated via lymph or blood to secondary sites where it causes abscesses of different size.
- Virulence of C. pseudotuberculosis is attributed to a major exotoxin, phospholipase D (PLD) which increases vascular permeability and also facilitates dissemination of the pathogen into lymph nodes where it inhibits chemotaxis and death of neutrophils as well as inactivation of complement. Additionally, two other toxins, a toxic cell-wall lipid and a haemolysin are excreted by the pathogen. The toxic cell wall lipid is associated with the virulence of the bacterium and the haemolysin causes haemorrhages, increased vascular permeability enhancing bacterial invasion.
- Bacteria has two biotypes known: ovine/caprine and equine/bovine. Recently it has been shown, that strains isolated from dromedaries in the United Arab Emirates and Kenya belong to both serotypes, serotype I (biotype ovis) and serotype

II (biotype bovis) using the nitrate reduction test.

Clinical signs

- Development of superficial abscesses localized mainly in the cephalic, prescapular and prefemoral lymphnodes.
- Pathognomonic in camels are cold, closed painless abscesses up to the size of a lemon or orange in the external lymph nodes.
- In camels, the characteristic laminated (onion ring) pattern seen in goats has not been described. In camelids three different types of pyogranulomatous lesions have been observed: Single abscess, when ripe and opened, the abscess extrudes thick, white-cream like pus; Large central abscess with multiple small abscesses in the peripheral connective tissue capsule and Multiple abscesses and no central abscess.
- A few cases have been seen in dromedaries whereby the abscess breaks through the ribs and the organisms enter the lung producing severe bronchopneumonia with pulmonary caverns

Post-mortem lesions

- Camels rarely die of the disease and most cases are found as incidental findings during slaughter. Most common lesions are pyogranulomatous abscess of the internal organs and lymphnodes.
- Microscopic lesions of the pyogranulomatous C. pseudotuberculosis abscess are composed of: Central necrotic core of pus, several layers of immune cells (macrophages and lymphocytes) and Fibrous capsule which encircles the pyogranulomatous lesion.

Zoonotic potential

- (Rare)

Ten (10) farm and abattoir workers infected with sheep/goat strain that caused chronic lymphomegally that required surgical treatment in (Australia- 1997)

Diagnosis

- Smears from abscesses show C. pseudotuberculosis as a short, irregular ovoid Gram-positive rod almost resembling a coccus.
- Cultured colonies are small, white and dry and can be surrounded by a narrow zone of haemolysis when sheep or ox blood is used. The plates should be incubated at 37°C for at least 48 hours.
- There is no commercial serological test (ELISA), however, a specific polymerase chain reaction targeting C. pseudotuberculosis has been developed.
- Confirmatory diagnosis is through biochemical identification of PCR- unidentified bacteria.

Treatment and Control

- Disinfect and clean stables and pens rigorously, remove the dung, bedding and topsoil from pens, move the herd immediately into a newly erected boma.
- Animal housing should be free from wire and other causes of skin trauma
- External parasites must be controlled
- Purchase of animals should only be allowed from herds with no history of abscessation
- Corynebacterium is sensitive to penicillin, tetracyclines and cephalosporins but the fibrous capsule and the pus in the abscess prevents the medication from reaching the bacteria.
- Antimicrobial treatment is often not rewarding and other treatment methods should be considered. Since erythromycin is able to penetrate tissues. It is proposed a combination of penicillin and erythromycin to treat pseudotuberculosis in camels.
- In cases with multiple abscesses, surgical and antibiotic treatments are recommended Subcutaneous ripe abscesses can be lanced and cleaned out on a daily basis with iodine solution. It is of paramount importance to provide strict aseptic methods, destroying contaminated equipment and disinfection of instruments used.
- There is no available vaccine.

Further Reading

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

Figure 39

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

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

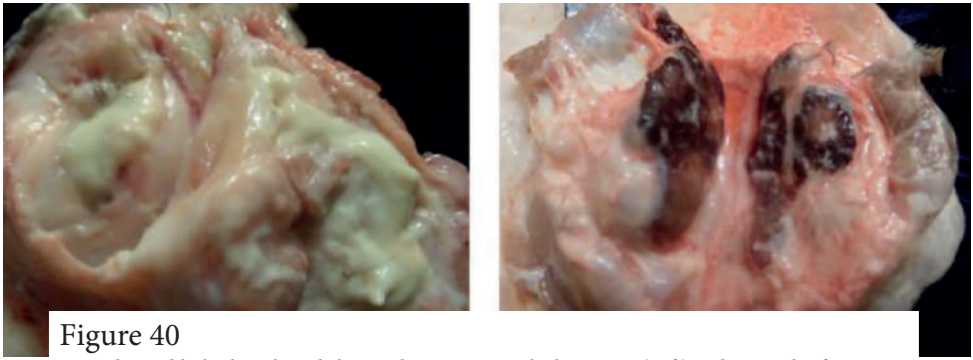


Figure 40

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

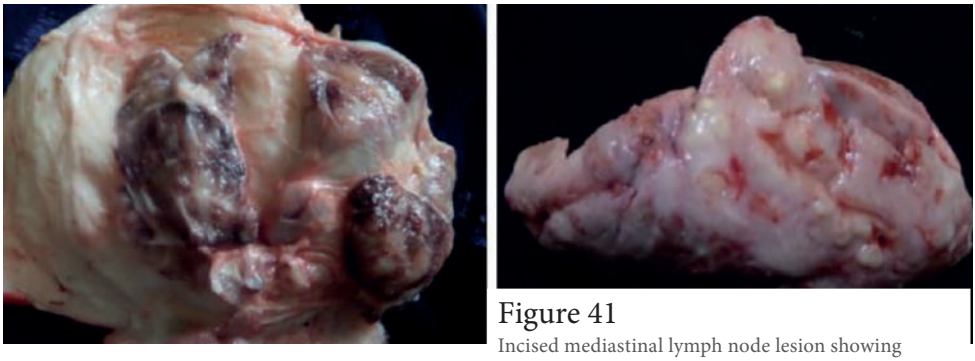


Figure 41

Incised mediastinal lymph node lesion showing enlargement and congestion without abscess formation (Left) and Pulmonary lesion showing multiple small caseous abscesses (Right)

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

3.7 | Contagious Ecthyma (orf)- Abitiro (Samburu), Ng'iboruok (Turkana)

Other name(s): 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.
- Worldwide distribution and is found in all camel keeping countries. The disease is endemic in affected areas with variations in intensity of infection,
- Morbidity and mortality rates occur annually in the rainy season mainly affecting young animals. The age group at risk are those less than one year of age including one month-old camel calves. The occurrence during the rainy season may be due to the optimum condition for the survival and perpetuation of the virus and skin abrasions caused by browsing thorny trees.
- 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.

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.

Morbidity and Mortality rate

- Morbidity rate is about 60-100% while the mortality rate is 5-20%.
- The camel calves die due to an inability to graze and suckle their dam.
- In Samburu county the apparent morbidity rate is 3% with no mortality rate.
- The disease in Samburu county is more common during the short dry season Lamei dorop (January to March) in Samburu herds. In Turkana herds it is more common during the long dry season, Akaamu nakoyen in the months of July to October.

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.
- 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.

Differential diagnosis

- Camel pox
- Mange
- Fungal infections

Treatment

- No treatment since it is viral.
- Local application of antiseptic solution such as povidone iodine or iodine ointment on the affected areas is recommended. Use of Gentian violet spray with oxytetracycline antibiotic also assists in drying lesions and prevents local secondary bacterial infection
- Antibiotic therapy is useful in control of secondary bacterial infection use 20 to 30% long acting oxytetracycline dosage rate 1mL per 10 kilograms.
- Sometimes the disease results in inflammation of the head tissues one can use dexamethasone at a dosage rate of 1mL per 25 kilograms' body weight, intramuscular.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.

Prevention and control:

- Affected animals must be segregated to stop the 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.

Further reading

1. Khalafalla, A.I., El-Sabagh, I.M., Al-Busada, K.A. et al. Phylogenetic analysis of eight sudanese camel contagious ecthyma viruses based on B2L gene sequence. Virol J 12, 124 (2015). https://doi.org/10.1186/s12985-015-0348-7
2. Walid Saber. Contagious Ecthyma (ORF) | Overview of the Disease. Veterinary Medical Encyclopedia http://www.veterpedia.net/infectious-diseases/1403-camels/contagious-ecthyma-orf/overview-of-the-disease.html

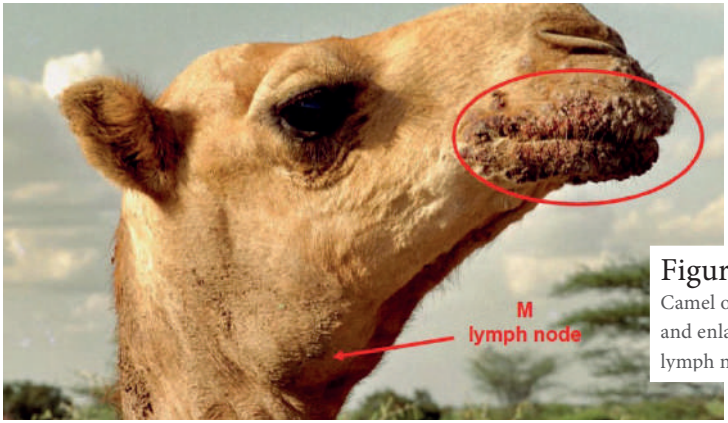


Figure 42
Camel orf oral lesions (red oval) and enlarged sub-mandibular lymph node (red arrow)

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

Chapter 3:

Sheep Diseases

4.1 | Enterotoxaemia (Pulpy Kidney disease)- Lbus/ Nadomanyita (Samburu) Lobus (Turkana)

Aetiolology

- Enterotoxaemia (Pulpy Kidney disease) is a toxemia due to the toxin from Clostridium perfrigens type D. Clostridium perfringens type D is a Gram-positive spore-forming anaerobic (meaning it can grow without oxygen) bacteria that is normally found in the intestines of humans and animals. It is also a common cause of human food poisoning when ingested in sufficient numbers.
- The bacteria under certain conditions it proliferates and produces large quantities of toxins which can be lethal.
- Disease is distributed worldwide.
- In Samburu county the apparent morbidity rate is 28%, mortality rate 15% and case fatality rate of 54%.
- In Samburu county the disease is more prevalent during the long rainy season locally known by the Samburu community as Ltumuren (November and December) and by the Turkana community as Akiporo (March and June).

Predisposing factors

- Ingestion of excessive amounts of feed or milk in lambs and of lush green grass in adults.
- Disease commonly affects animals in good body condition hence the name "overeating disease".
- Factors which result in intestinal stasis or slow the passage of the ingesta through the intestines such as heavy tape worm infestations favour the accumulation of the toxin and occurrence of the disease.

Pathophysiology

- C. perfringens is a natural inhabitant of the intestinal tract. Once the intestinal environment is altered by dietary changes, the organisms rapidly proliferates and produces powerful exotoxins. Alpha toxin, a necrotizing toxin causes vascular leakage, haemolysis and liver damage. Epsilon toxin is also produced and causes renal damage, gastroenteritis and haemorrhages in various organs.

Clinical signs

- Sudden death is the main manifestation of the per acute form of the disease in young lambs although some of the animals may be dull, depressed and anorexia. In acute cases, there is frothy salivation, green or pasty diarrhoea, staggering, recumbency, opisthotonus, colonic convulsions coma and death.
- Colic and bloat may occur. Affected adult sheep often lag behind the rest of the flock and show nervous signs. Champing of the jaws, blindness, salivation, rapid and shallow respiration, atonic rumen and pasty faces may also be evident.

Diagnosis

Samples:

- Faecal sample or intestinal contents for laboratory identification of C. perfringens type D
- Serum sample - presence of hyperglycemia and glucosuria can strongly suggest enterotoxaemia

Necropsy:

- Ulcerated intestinal mucosa
- Watery contents, blood and fibrinous clots in the small and large intestines
- Multi-systemic haemorrhages, particularly of serosal surfaces
- Presence of pericardial effusions

Treatment

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

Control

The most effective way of control is by vaccination.

KEVEVAPI does not have the vaccine source from private sources.

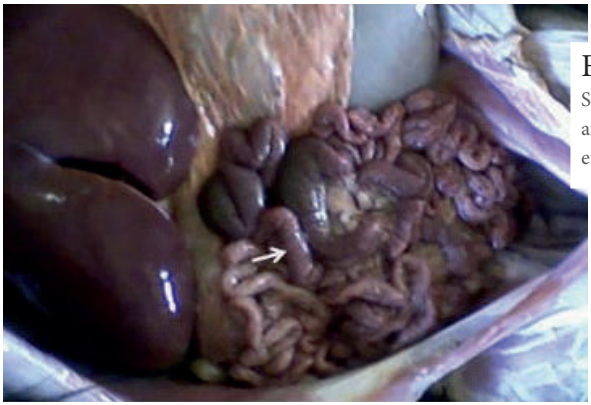


Figure 43

Small intestine distended with gas (white arrow) and congested in a sheep with enterotoxaemia

Picture source:
Hassanein et al., 2017

4.2 | Peste des Petits Ruminants (PPR) Kinyoot/ Nkiriata/ Lodua (Samburu), Lokiyoo/ Lonoo (Turkana)

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 Samburu county and outbreaks are common during the long dry season, Lameioodo (June to October) amongst the Samburu community sheep flocks. The disease in Turkana flocks is common during the long rainy season. Akiporo during the months of March to June.
- The apparent morbidity rate in Samburu county is 25% with a 13% mortality rate and 52% 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

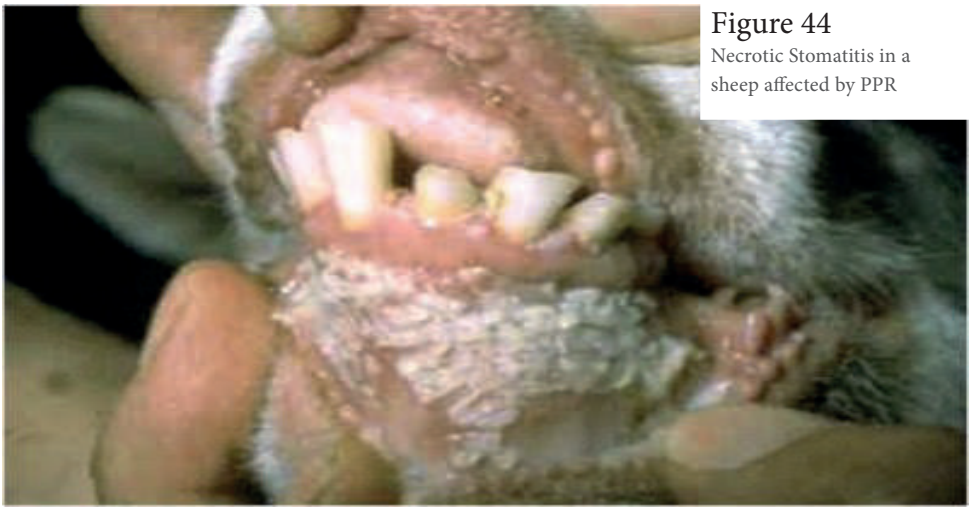


Figure 44
Necrotic Stomatitis in a sheep affected by PPR

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

4.3 | Sheep and goat pox Tune/ Nariri (Samburu), Tune (Turkana)

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 on wool devoid skin including the cheeks, lips and nostrils as well as the tongue and gum.
- In Samburu county, SGP is endemic and common during the long dry season, Lameioodo in the Samburu community most common during the months of June to October. The Turkana community indicated that SGP occurs throughout the year with no seasonal outbreaks.
- The apparent morbidity rate in Samburu county is 14% with a mortality rate of 5% and a case fatality rate of 36%.

Clinical signs

- Characteristic raised skin lesions on the skin, lesions more widespread in the skin of the groin, axilla and perineum.
- Fever of 40°C 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 45
Lung lesions in sheep and goat pox

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

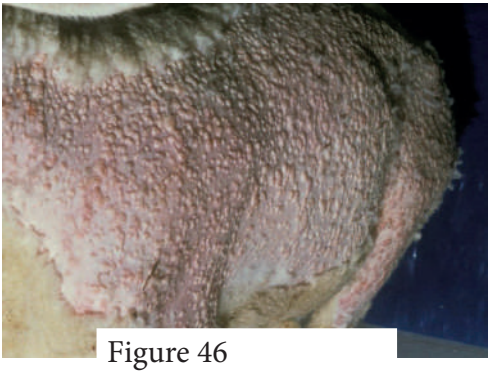


Figure 46
sheep pox skin lesions

Picture source:
Hurisa et al., 2018

4.4 | Foot and mouth disease (FMD) Lkulup (Samburu) Lokulup (Turkana)

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 Samburu county the apparent morbidity rate is 16%, mortality rate 8% and case fatality rate 50%.
- In Samburu, FMD is common amongst Samburu 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 47
FMD foot lesions in the coronary band and interdigital space.

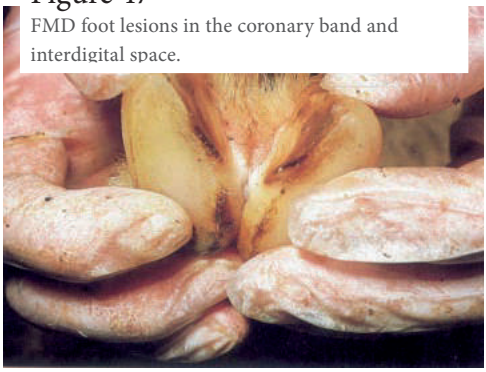


Figure 48
FMD Gum lesions

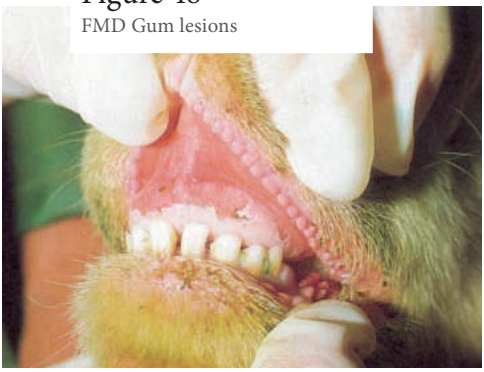
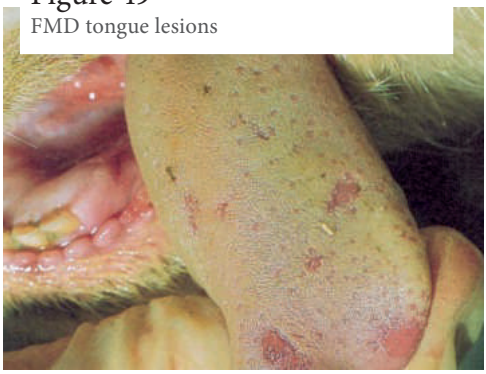


Figure 49
FMD tongue lesions



Picture source:
Business Queensland

4.5 | Helminthiasis Ntumuai (Samburu) , Ngiritan (Turkana)

- Helminths in sheep can be divided into nematodes (round worms), trematodes (flukes) and cestodes (tape worms).
- Nematodes include: Haemonchus contortus, Nematodirus species, Bunostomum species, Ostertagia circumcinta, Trichostrongylus species
- Cestodes include: Monenzia expanza is the most common cestode of sheep
- Trematodes include: Fasciola hepatica (liver fluke) is the most common trematode of sheep
- 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.
- In Samburu county, helminthiasis is common during the long wet season known as Ltumuren (November to December) in Samburu community and Akiporo (March to June) in Turkana community.
- In Samburu county the apparent morbidity is 32% with a 17% mortality and 53% case fatality rate.

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 haemorrhage of the intestinal mucosa, diarrhoea and dehydration. The key features of hemonchosis are sub-mandibular oedema and anaemia.

Liver fluke disease
Pathophysiology

- Liver fluke eggs are passed in the bile and faeces and hatch in 2–3 weeks to form the free-swimming miracidia. The miracidia penetrate the body of an intermediate host (usually freshwater snails) and develop through sporocysts and rediae stages, finally forming cercariae. The cercariae leave the intermediate host, swim to grassy vegetation, and become cyst-like metacercariae, which may remain in a dormant stage on the grass for 6 months or longer until ingested by a ruminant. The ingested metacercariae penetrate the small-intestinal wall, migrate through the abdominal cavity to the liver where they locate in a bile

duct, mature and remain for up to 4 years.

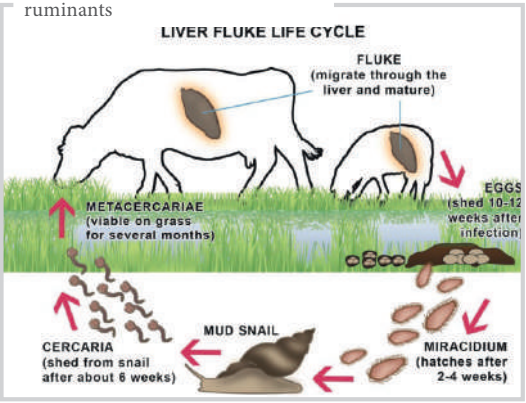
- Acute liver fluke disease is related to the damage caused by the migration of immature flukes which leads to liver inflammation, haemorrhage, necrosis, and fibrosis.
- Chronic disease may result from fluke-induced physical damage to the bile ducts and cholangiohepatitis. Blood loss into the bile may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

Clinical signs

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

Diagnosis	Treatment & 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 50
Lifecycle of liver fluke in ruminants



Picture source:
Farmhealthonline.com

Figure 51
Bottle jaw associated with haemonchosis in sheep.

Picture source:
National Animal Disease Information Service (NADIS)

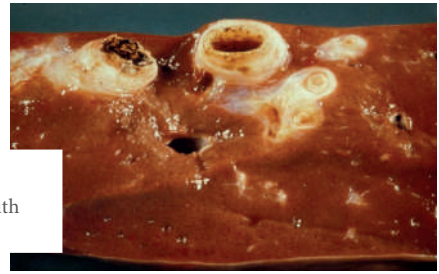


Figure 52
Fibrotic sheep liver with liver fluke tracts

4.6 | Babesiosis Nkula (Samburu) Lonyang' (Turkana)

Aetiology

- Babesiosis is caused by Babesia spp; Babesia ovis and B. motasi. They are transmitted vertically by ticks of the genera Rhipicephalus, Dermatocenter, Ixodes and Haemophysalis.
- In Samburu county the apparent morbidity rate is 15%, mortality rate 5% and case fatality rate of 33%.
- In Samburu county the disease is common during the long dry season locally known as Lameioodo (June to October) in Samburu community and Akaamu (July to October) in the Turkana community.

Predisposing factors

- The occurrence of babesiosis depends on the distribution of transmitting ticks. It is distributed in different countries including Iran, Kenya, Rwanda, Bolivia and Uganda.

Pathophysiology

- The infective stage of Babesia organisms is the sporozoite and it is produced in the salivary gland of the tick vector. It is injected into the host, the sheep, by feeding ticks. The parasite attacks erythrocytes causing haemolysis due to intracellular reproduction, leading to haemoglobin release in circulation.

Clinical signs

- The main clinical signs are haemoglobinuria, anaemia and moderate jaundice.

Diagnosis	Treatment	Control
Laboratory sample: Thin blood smears stained with Giemsa for microscopic examination of Babesia trophozoites.	Treatment is based on diminazene aceturate (Berenil®/ Veriben®) at 3.5mg/ Kg body weight intramuscularly in a single injection; or imidocarb diproprionate (Imizole®, Imochem®)at 6.6mg/ Kg body weight intramuscularly or subcutaneously in two treatments two weeks apart	Transmission and spread is usually controlled by use of acaricides to eliminate ticks and by exercising good pasture management.

Figure 53
Coffee coloured urine due to haemoglobinuria in babesiosis

Picture source:
National Dairy Development Board, India

4.7 | Pneumonia Lkipei/ Nkijepe (Samburu) Lotai (Turkana)

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 Samburu county, pneumonia cases are common during the long dry season. locally known as Lameioodo (June to October) in Samburu community and Akaamu (July to October) in the Turkana community.
- The apparent morbidity rate in Samburu county is 31% with a 16% mortality rate and 52% 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.

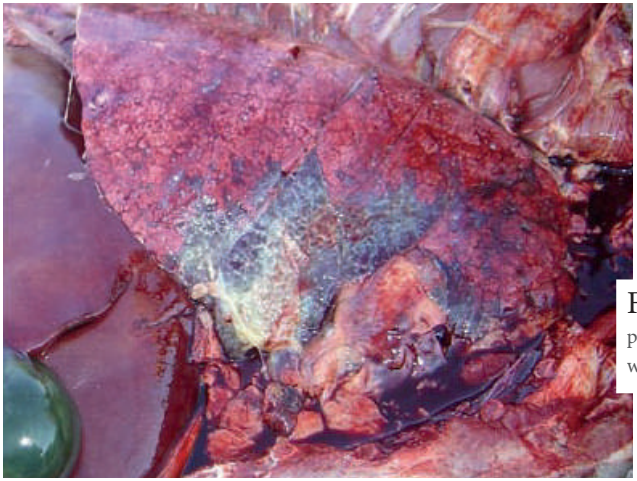


Figure 54
purple-red lesions on lung associated with Pasteurella pneumonia in sheep

Picture source: 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.

4.8 | Coenurosis- Sirko (Samburu),Sirko/ Nkerep (Turkana)

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 grazed by sheep by dog faeces can result in larval invasion of the central nervous system and clinical disease. The life cycle is completed when the carnivorous definitive host ingests infested sheep's brain.
- In Samburu county the apparent morbidity rate is 14% with a 8% mortality rate and 57% case fatality rate.
- In Samburu county the disease is common during the long dry season locally known as Lameioodo (June to October) in Samburu community and Akaamu (July to October) in the Turkana community.

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 sheep.	Treatment with medication is not usually successful, farmers resort into culling affected animals	Control is done by breakage of the sheep-dog cycle by proper disposal of carcasses and deworming of dogs with praziquantel

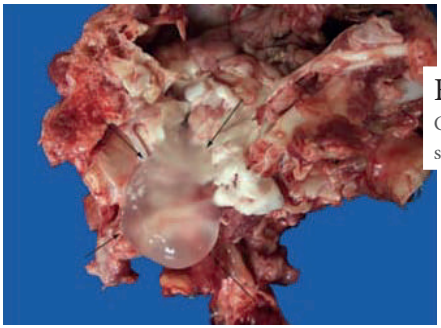


Figure 55
Coenurus cerebralis larval cyst in sheep brain

Picture source:
Batista et al., 2010

4.9 | Mange – Lputo- Samburu, Echudanu- Turkana

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 Samburu county the apparent morbidity rate is 13% with a 3% mortality rate and 23% case fatality rate.
- In Samburu county mite infestations are common during the long dry season locally known as Lameioodo (June to October) in Samburu community and Akaamu (July to October) in the Turkana community.

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 56

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

4.9 | Anaplasmosis Ndis (Samburu) Lonyang' (Turkana)

Aetiology

- A. ovis is a tick transmitted disease. Transplacental transmission has been reported and is usually associated with acute infection of the dam in the second or third trimester of gestation.
- Anaplasmosis may also be spread through the use of contaminated needles or other surgical instruments (Iatrogenic transmission)
- Infected animals remain carriers for life.
- In Samburu county the apparent morbidity rate is 18% with a 7% mortality rate and 39% case fatality rate.
- Disease has a seasonal occurrence in Samburu county, most cases are seen are common during the long dry season locally known as Lameioodo (June to October) in Samburu community and Akaamu (July to October) in the Turkana community.

Pathophysiology

- Anaplasma infect erythrocytes and each organism replicates by binary fission to form up to eight individual organisms within a simple vacuole.
- The parasites leave the erythrocyte in a non-haemolytic mechanism that is not understood to infect new erythrocytes. During the acute phase, the number of infected erythrocytes doubles every 24–48 hours. The removal of the infected erythrocytes by the haemopoietin system mainly the spleen results in anaemia after 30–40 days after infection.

Clinical signs

- Progressive anaemia and jaundice, and constipation.
- Urine may be brown but, in contrast to babesiosis, haemoglobinuria does not occur.
- There may be a transient fever with the body temperature rarely exceeding 41oC.
- Pregnant animals may abort.

Diagnosis	Treatment	Control
Laboratory sample: Whole blood - The presence of typical inclusion bodies in erythrocytes of sheep and goats with anaemia is sufficient to establish diagnosis Necropsy: On post-mortem, the carcass is generally jaundiced, the spleen is enlarged and soft, the liver is mottled and jaundiced and the gall bladder is distended with thick brown or green bile	Tetracycline antibiotics and imidocarb are currently used for treatment. Prompt administration of tetracycline drugs in the early stages of acute disease usually ensures survival. A commonly used treatment consists of a single IM injection of long-acting oxytetracycline at 15-30mg/Kg body weight.	The key to controlling anaplasmosis is vector control by acaricide application every two weeks. The carrier state may be eliminated by administration of a long-acting oxytetracycline preparation (15-30 mg/kg, IM, at least two injections with a 1-wk interval)

Figure 57
Jaundiced liver with distended gall bladder as seen in anaplasmosis in sheep

Picture source:
Government of Western Australia, Department of Primary Industries and Regional Development.

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Chapter 3:

Goat diseases

5.1 | Peste des Petits Ruminants (PPR) Ngorotit /Serr/Nkiriata/Lodua (Samburu), Lonoo (Turkana)

- 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.
- Samburu county apparent morbidity rate is 29% with a mortality rate of 8% and case fatality rate of 28%.
- In Samburu county PPR incidences in goats is common during the short rainy season called Lgergerua (April and May) in Samburu community. In Turkana community PPR is common during the long rain season (Akiporo) that occurs in the months of March and June.

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.

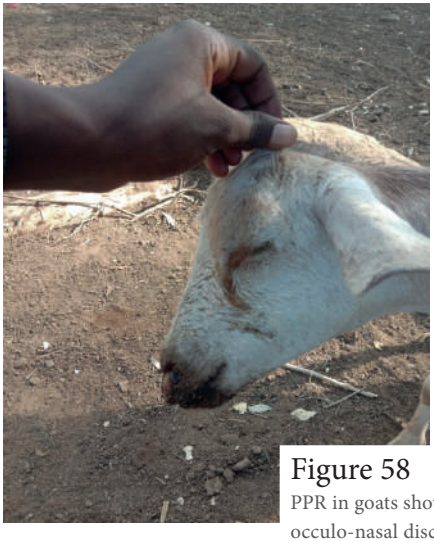
- For antibody detection, competitive ELISA and virus neutralization are the OIE-recommended tests
- The specimens required are lymph nodes, tonsil, spleen, and whole lung for antigen or nucleic acid detection, and serum for antibody detection.

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



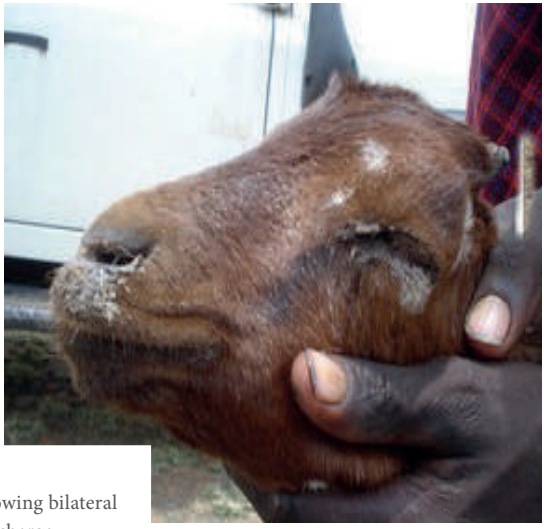


Figure 58
PPR in goats showing bilateral occulo-nasal discharge

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

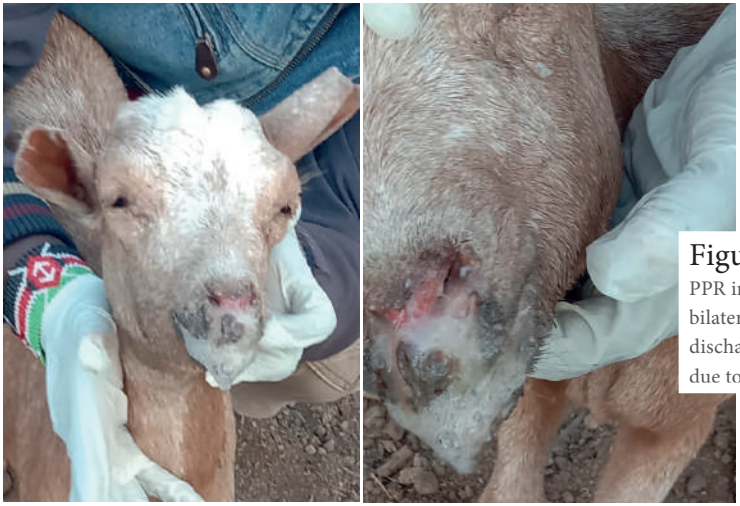


Figure 59
PPR in a goat kid showing bilateral mucopurulent nasal discharge and hypersalivation due to oral lesions

Picture source:
Emmanuel Lesiantam – Samburu County

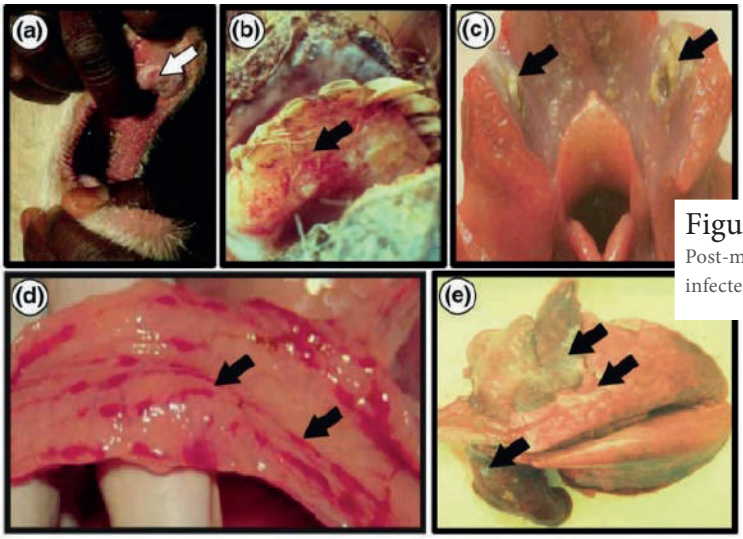


Figure 60
Post-mortem lesions in goats infected with PPR

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

5.2 | Helminthiasis Ntumuai (Samburu), Ngiritan (Turkana)

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 Samburu county the apparent morbidity rate is 22% with a 7% mortality and 32% case fatality.
- Most helminth infestations in the Samburu community herds occur during the short rain season, Lgergerua (April to May). Turkana communities' goat herds have increased helminth infestation during the long rain season, Akiporo (March to June).

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

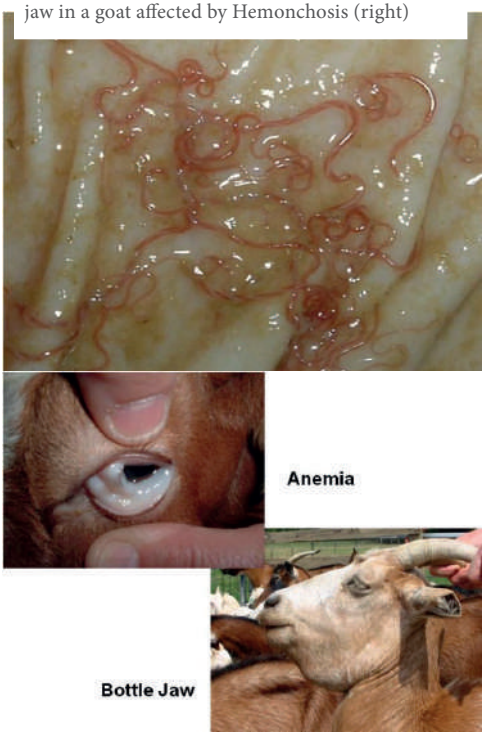
- Lungworms- Benzimidazoles (fenbendazole, oxfendazole and albendazole), 5mg/kg body weight, orally.
- Macrocyclic lactones (ivermectin, doramectin, eprinomectin and moxidectin), 0.4 mg/kg orally.
- Levamisoles and repeat two weeks later
- Multivitamin supplementation

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 61

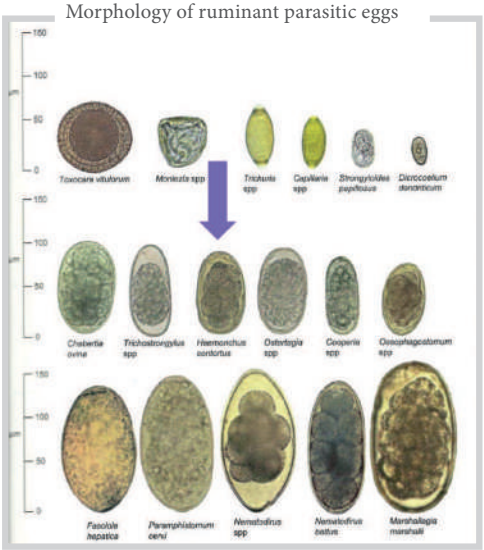
Haemonchus contortus adult worms in the abomasal lumen in a goat (left) anaemia and bottle jaw in a goat affected by Hemonchosis (right)



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

Figure 62

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

5.3 | Sheep and Goat Pox (SGP) Nariri/ Tune (Samburu), Tune (Turkana)

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 and mucosa including the cheeks, lips and nostrils as well as the tongue and gum.
- In Samburu county, SGP is endemic and common during the long dry season, Lameioodo in the Samburu community most common during the months of June to October. The Turkana community indicated that SGP occurs throughout the year with no seasonal outbreaks.
- The apparent SGP morbidity rate in goats in Samburu county is 30% with a mortality rate of 20% 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 63
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

5.4 | Foot rot Ng'ojini (Samburu), Lukulup (Turkana)

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 Samburu county the apparent morbidity rate is 8% with no mortality rate reported. Foot rot is most common during the long rainy season locally known as Ltumeren (October to December) in Samburu community and Akiporo (March to June) in Turkana community.

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.

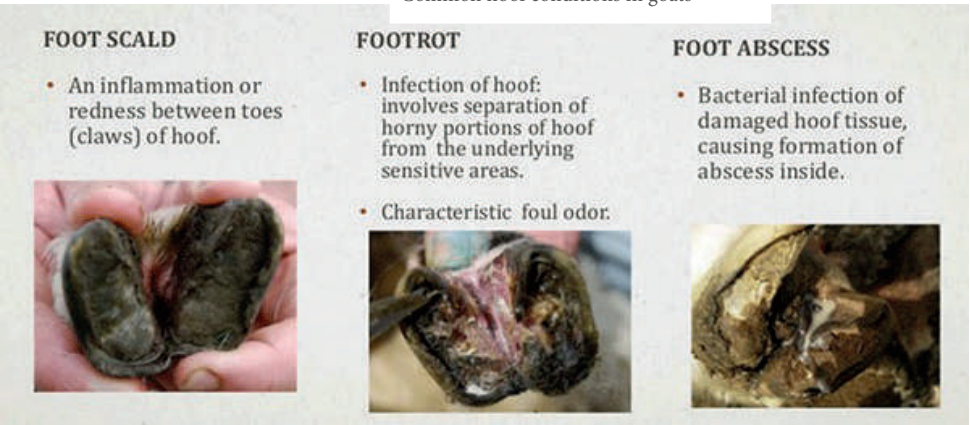
Control

- 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



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)

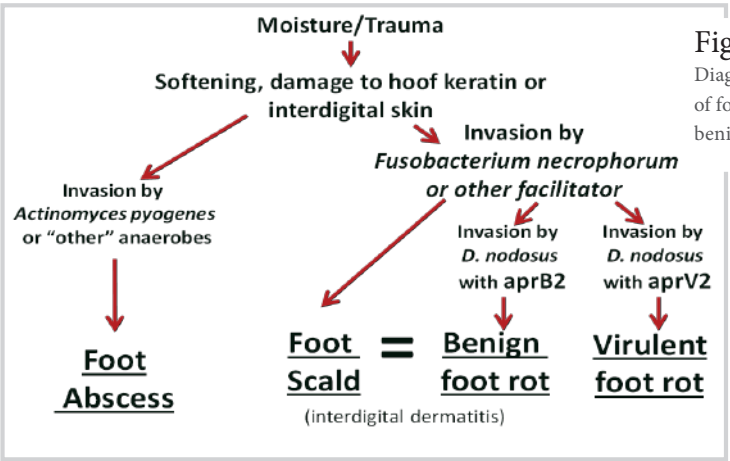


Figure 65
Diagram depicting differences of foot abscess, foot scald and benign or virulent foot rot

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

5.5 | Coccidiosis Nkiriata Elodo (Samburu), Ngerep (Turkana)

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 Samburu county the apparent morbidity rate is 10% with a mortality rate of 8% and a case fatality rate of 80%. The cases amongst Samburu community herds are common during the long rainy season, Ltumuren (October to December) and in the Turkana community during the long dry season Akaamu nakoyen (July to October).

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.
- I f 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 66
Eimeria species unsporulated oocysts in goat kid faecal sample

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

Figure 67
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/

5.6 | Contagious ecthyma (orf)- Abituro (Samburu), Ng'borouk (Turkana)

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 Samburu county the apparent morbidity rate is 7%, mortality rate 2% and case fatality rate of 29%.
- Orf in Samburu county is common during the long rainy season locally known by the Samburu community as Ltumuren (October-December) and Akiporo (March to June) by the Turkana community.

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.

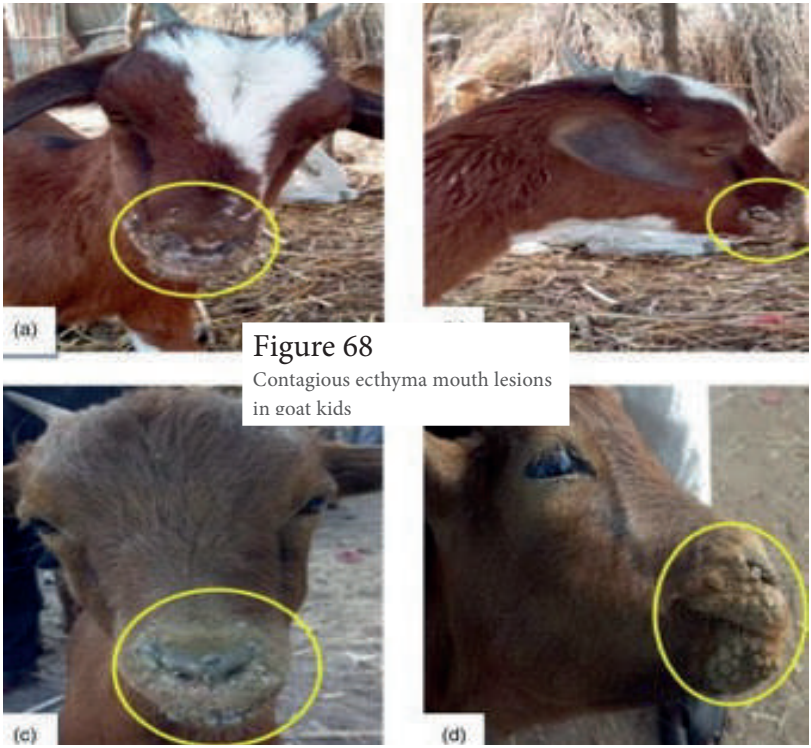


Figure 68
Contagious ecthyma mouth lesions in goat kids

Further Reading

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5.7 | Coenurosis Sirko (Samburu), Nkerep (Turkana)

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 Samburu county the apparent morbidity rate in goats is 13% with a 10% mortality rate and 77% case fatality rate.
- Coenurosis manifestation is more common during the long dry season locally known as Lameioodo (June to September) in Samburu 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

Diagnosis is confirmed on necropsy by the presence of Coenurus cerebralis cysts in the brain of goat.

Treatment

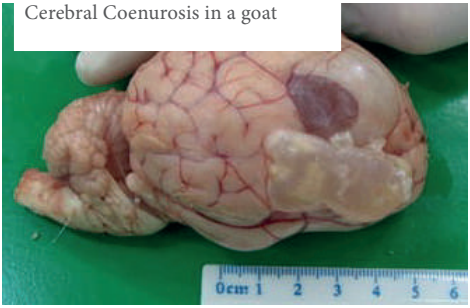
Treatment with medication is not usually successful, farmers resort into culling affected animals

Control

Control is done by breakage of the goat-dog cycle by proper disposal of carcasses and deworming of dogs with praziquantel

Figure 69

Cerebral Coenurosis in a goat



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

5.8 | Enterotoxaemia – Sirr/ Nadomanyita (Samburu), Lobus (Turkana)

Aetiology

- Enterotoxaemia, also known as overeating or pulpy kidney disease, is a condition caused by Clostridium perfringens type D.
- Bacteria are normally found in the soil and as part of the normal microflora in the gastrointestinal tract of healthy goats.
- Under specific conditions, these bacteria can rapidly reproduce in the animal's intestine, producing large quantities of toxins. The epsilon toxin produced by C. perfringens Type D is the most significant toxin in producing the disease.
- Young animals are most susceptible and the main sign is sudden and high mortality rates of lambs and kids.
- Adult animals are also susceptible to enterotoxaemia but they develop immunity due to frequent exposure to low doses of the toxins.
- In Samburu the apparent morbidity rate and mortality rates are both 4% with a case fatality rate of 100%.
- The disease in Samburu county is common during the long rainy season locally known as Ltumuren (October to December) by Samburu community and Akiporo (March to June) by Turkana community.

Pathophysiology

- Excessive consumption of milk or lush grass that results in rapid fermentation.
- When natural immunity is compromised such as when animal is sick or recovering from an illness or is stressed.
- When animals are heavily parasitized with gastrointestinal parasites, including nematodes, cestodes (tapeworms) and coccidia.
- When the ration is rich in carbohydrates (grains) and low in roughage.
- When motility of the gastrointestinal tract is reduced.

Clinical signs

- The peracute form is frequent in young animals. It is characterized by sudden death that occurs approximately 12 hours after the first signs of the disease appear.
- Some kids or lambs may show signs of central nervous disease, such as excitement or convulsions. Sudden death may occur in minutes after showing neurological signs.
- Typical clinical signs include- Loss of appetite, Abdominal discomfort, Profuse and/or watery diarrhoea that may be bloody.

Diagnosis

- Diagnosis is based on clinical signs, history of sudden death and confirmation by necropsy examination.
- Diagnosis can be confirmed by positive identification of enterocolitis, anaerobic culture, and identification of Clostridium perfringens type D from the faeces or intestinal contents from clinical or necropsy specimens of affected animals.
- The presence of hyperglycaemia and glucosuria can strongly suggest enterotoxaemia in live or dead animals.
- A post-mortem examination of the large and small intestines can identify watery contents, blood and fibrinous clots, and small ulcers on the mucosa. The kidneys on gross examination may have a soft pulpy consistency.

Treatment

- Clostridium perfringens C & D antitoxin according to the manufacturer's recommendations (5 mL of C & D antitoxin subcutaneously. Not easily available for livestock keepers in Kenya.
- Antibiotics, especially penicillin based.
- Anti-bloating medication
- Intramuscular thiamine (vitamin B1) to prevent encephalomalacia
- Supportive therapy such as intravenous or subcutaneous fluids and corticosteroids
- Administer rumen ingesta from healthy animals, to encourage repopulation of the microflora in the GI tract

Prevention

- Effective vaccines are commercially available but not readily accessible in Kenya.
- All animals (especially young animals) within the herd should be vaccinated.
- Use vaccines that are labelled for use in sheep and goats and follow the manufacturer's recommendations. Some of the commercially available vaccines against enterotoxaemia are also combined with tetanus toxoid.
- Young animals should be vaccinated at 4 weeks of age and again one month later. All adults including bucks should be vaccinated at least once per year.

5.9 | Mange in goats- Iputo (Samburu) Echudano (Turkana)

- In Samburu county the apparent morbidity rate of goat mange is 8% with a mortality rate of 2% and case fatality rate of 25%.
- Most incidences of goat mange in Samburu are reported during the long dry season when animals are in nutritional stress. The long dry season runs from the months of June to October. The Samburu community refer to this season as Lameioodo while the Turkana community call it Akaamu nakoyen.

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 70
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

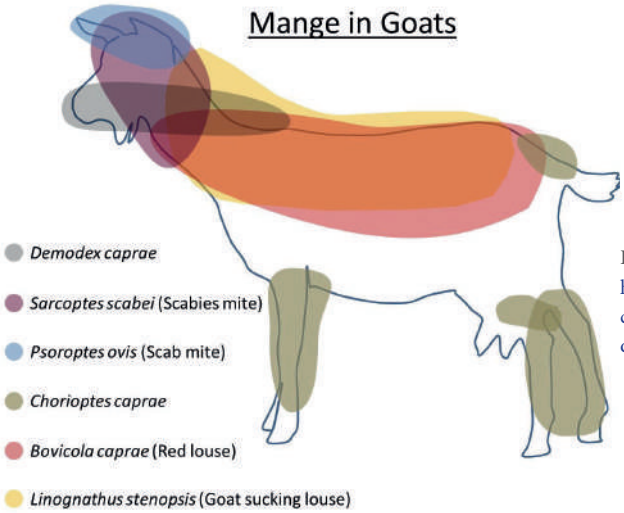


Figure 71
Location of ectoparasite in goats indicates if it is a mite or flea infestation

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

5.10 | Contagious Caprine Pleuropneumonia (CCPP)- Lkipei (Samburu) Loukoi (Turkana)

Causative agent

- CCPP is caused by bacterium Mycoplasma capricolum subspecies capripneumoniae.

Distribution

- Occurs in many countries in Africa, Asia and the Middle East.
- Endemic in Samburu county and in Samburu community goat herds incidences are more common in the months of April and May which is the short rainy season (Lgergerua). in Turkana goat herds disease is more common during the months of March to June which is the long rainy season (Akiporo).

Morbidity and Mortality rates

- In naive herds, the morbidity rate reaches 100% and the mortality rate can be as high as 80%.
- In Samburu county the apparent morbidity rate is 44% with a 16% mortality and 36% case fatality rate.

Transmission

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

Clinical signs

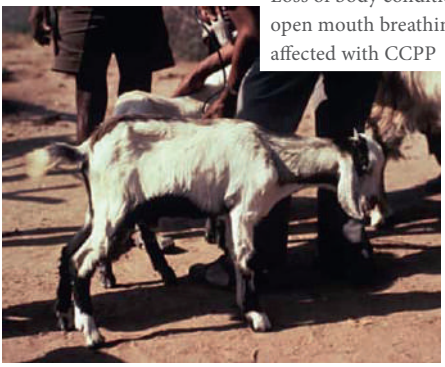
- The incubation period is 6 to 10 days. Peracutely affected goats can die within one to three days with minimal clinical signs. Acute symptoms include fever, lethargy, violent coughing, extended necks, laboured breathing, loss of appetite and abortions.

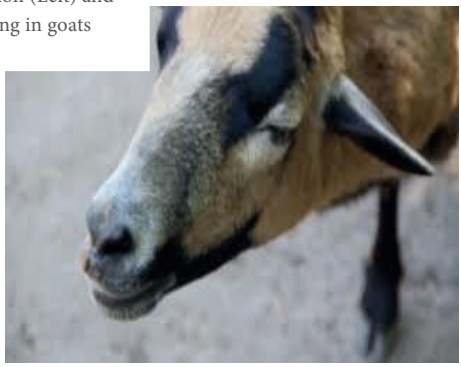
Control

- 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

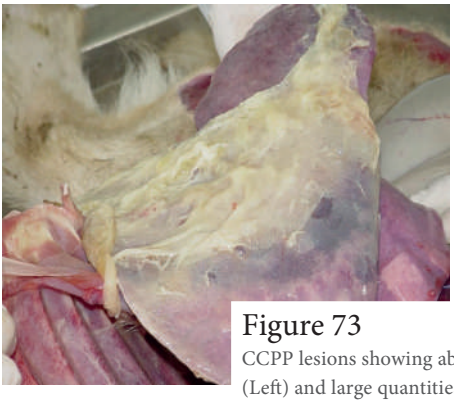


Figure 73

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

Further Reading

1. Merck Veterinary Manual (Online Version)
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Chapter 6:

Chicken Diseases

6.1 | New Castle Disease (ND)– Lchama/ Nkonkoro (Samburu), Lonoo (Turkana)

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 Samburu 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 Samburu the disease has been estimated to have an apparent morbidity rate of 58% with a mortality rate of 48% and case fatality rate of 83%.
- The disease in Samburu county is occurs throughout the year but most outbreaks are seen during the months of June to October during the long dry season locally known as Lameioodo in Samburu language and Akaamu nakoyen in Turkana language.

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 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 Samburu 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)

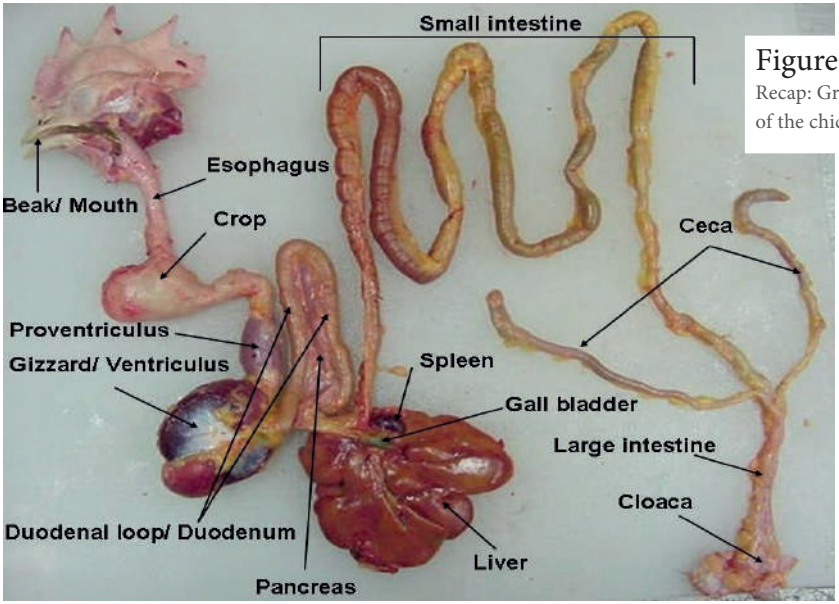


Figure 74
Recap: Gross anatomy of the chicken

Picture source:
Picture book of infectious poultry diseases (2010). Produced by FAO ECTAD Southern Africa. https://www.itpnews.com/uploads/2017/03/color%20atlas%20of%20poultry%20disease%20-%2001.pdf?direct_access_media=1

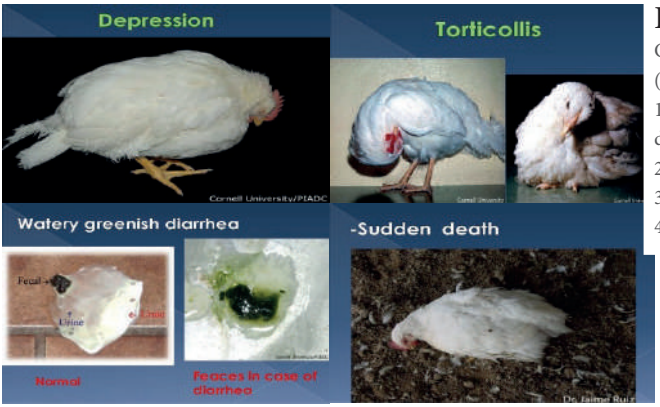


Figure 75

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

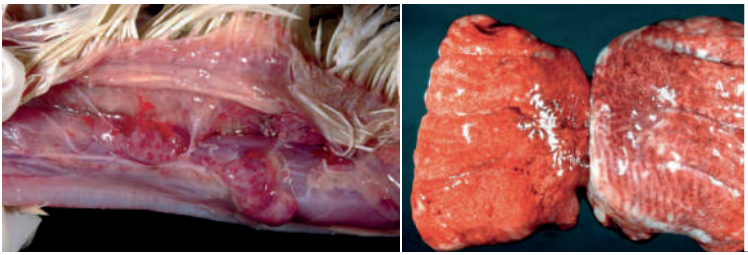


Figure 76

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

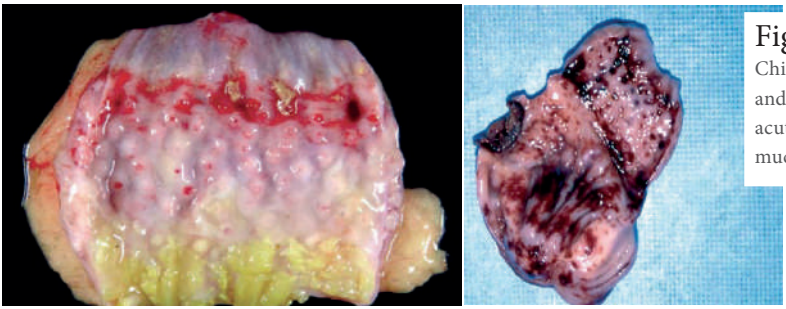


Figure 77

Chicken proventriculus and ventriculus. Severe, acute multifocal mucosal haemorrhages

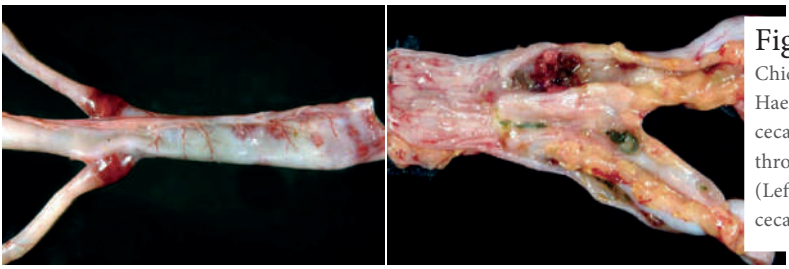


Figure 78

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

6.2 | Coccidiosis – Nkiriata elodo (Samburu), Eremorit ng'aokot (Turkana)

Other Name(s): 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.

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 79

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).

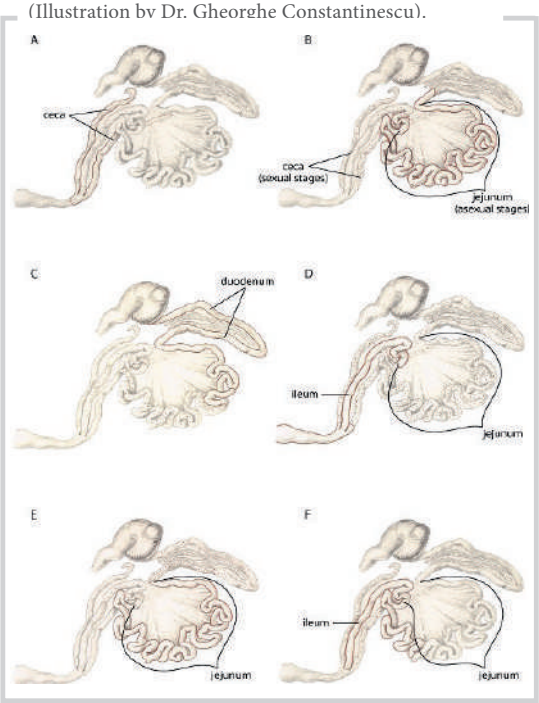
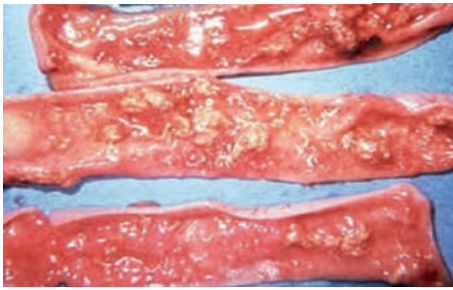


Figure 80

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 – Narirri enkokon (Samburu), Ng'ajemai (Turkana)

Other Name(s): 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 Samburu county the estimated apparent morbidity rate is 14% with a mortality rate of 5% and case fatality rate of 36%.
- The disease in Samburu county is most common during the months of June to October which is the long dry season locally known as Lameioodo in Samburu community and Akaamu nakoyen in Turkana community.

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 81

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

Figure 82

Diphtheric form of fowl pox infection showing yellow cheesy oral lesions

6.4 | Helminthiasis- Ntumwa (Samburu), Ngirtan (Turkana)

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 Samburu county the apparent morbidity rate due to helminths is 6% with a mortality rate of 2% and case fatality rate of 33%.
- Helminths impact in Samburu county are mostly see during the months of June to October which is the long dry season locally known as Lameioodo in Samburu community and Akaamu nakoyen in Turkana community.

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.

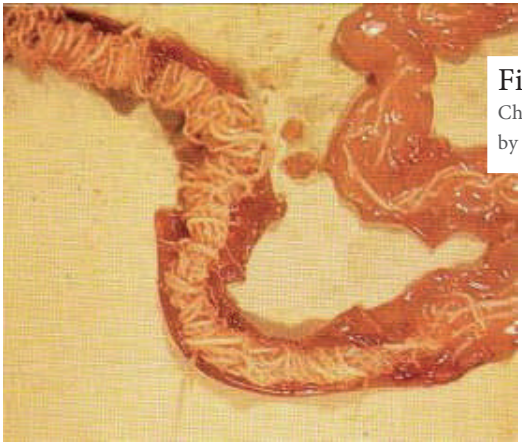


Figure 83
Chicken intestinal tract impacted by Ascarid worms

Picture source:
https://core.ac.uk/download/pdf/211564736.pdf

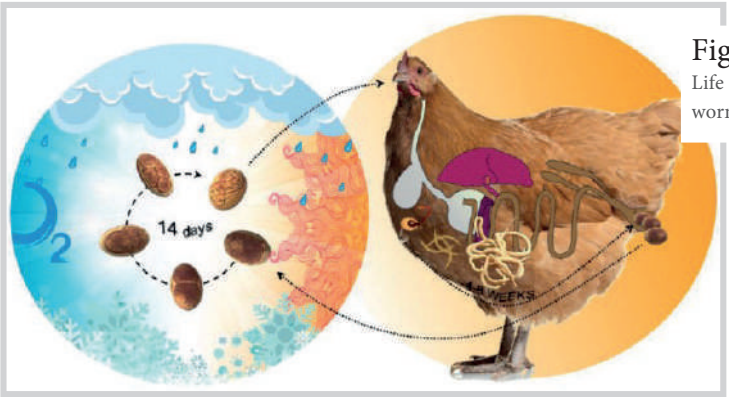


Figure 84
Life cycle of the large round worm of chicken Ascarid galli

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

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

English name	Local name	Causative agent
Fleas	Samburu: Lososu Turkana: Lososu	The main flea pest is the sticktight flea; Echidnophaga gallinacean. Attach on the skin of the head
Lice	Samburu: Lashe Turkana: Elachet	Chewing lice; Order Mallophaga. They feed on feathers, feather debris, skin scales and other dermal scurf
Ticks	Samburu: Lmancher enkokon Turkana: Emadang	Argasid Ticks (soft-shelled ticks) are most common. Argasid ticks are nocturnal feeders and favor the soft un-feathered skin beneath the wings
Body or Scaly leg Mites	Samburu: Nkasimai Turkana: Ngooeye	Ornithonyssus spp remain on chickens permanently; while Dermanyssus mites parasitize chickens nocturnally Knemidocoptes mutans- scaly leg mites

- In Samburu county the apparent morbidity rate due to external parasites in chicken is estimated to be 11% with no morbidity rates reported.
- External parasitism in chicken flocks of Samburu county occurs throughout the year with no seasonal outbreaks reported.

Main clinical signs:
Ectoparasites affect chicken by:

- Transmitting diseases to chicken- ticks transmit spirochetosis.
- Sucking blood causing birds to be weak, less productive and more vulnerable to disease. If too many, cause anaemia and death in young birds.
- Birds spend more time scratching leading to reduced 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:

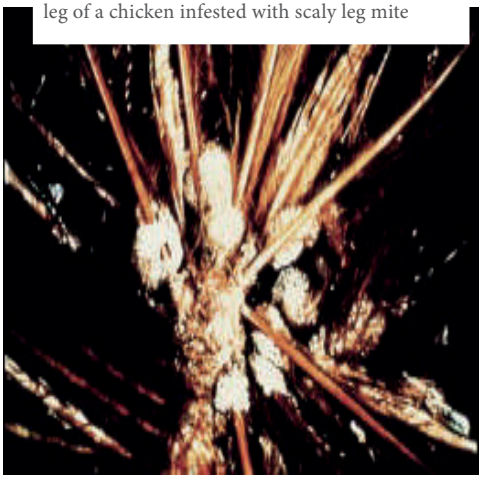
- Ectoparasites may be treated with a carbamate insecticide such as Carbaryl 7.5% insecticide powder – external use (Sevin Poultry dust) applied as a powder to birds at two-week intervals. Cages and housing can be treated with carbamate dust topically. Ensure to apply to base of feathers in case of mite 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 85

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- Moyen Enkonyek (Samburu), Eboot Akou (Turkana)

Other name(s): 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 Samburu county infectious coryza has the apparent highest morbidity rate at 67% with a mortality rate of 35% and case fatality rate of 52%.
- The disease has seasonal prevalence in Samburu county. Amongst the Samburu community disease is prevalent during the short wet season locally known as Lgergerua (April to May). While in the Turkana community the disease is most common during the short dry season Akaamu naurien (January to February).

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 86
Chicken with swollen facial tissue due to infectious corvza

6.7 | Fowl Cholera- Nkiriata (Samburu) Eriomorit (Turkana)

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.
- In Samburu county the apparent morbidity rate sue to fowl cholera is 23% with a 15% mortality rate and 65% case fatality rate.
- There is seasonal prevalence of the disease amongst Samburu flocks. Amongst the Samburu community increased cases are seen during the short dry season Lamei dorop (January to March). While in the Turkana community it occurs during the long rainy season, Akiporo (March to June).

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.

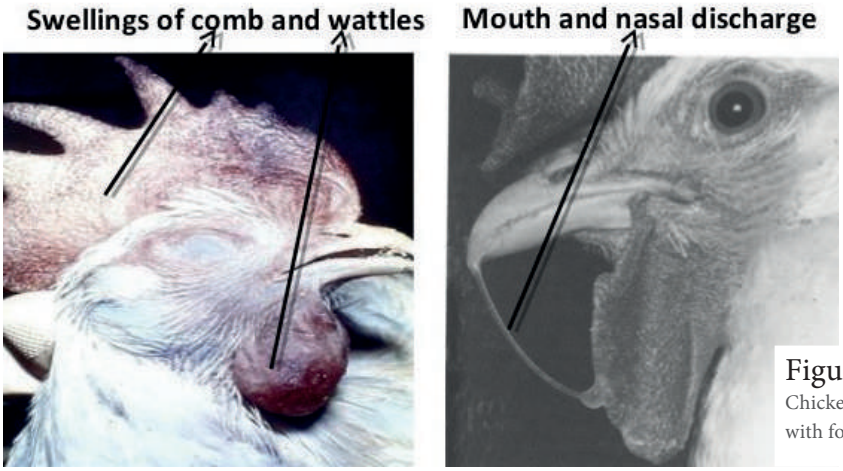


Figure 87
Chicken infected with fowl cholera

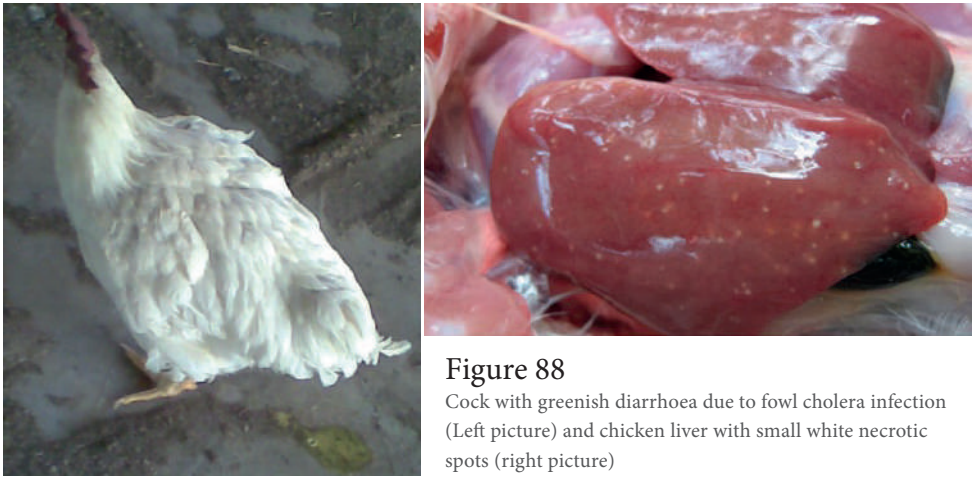


Figure 88
Cock with greenish diarrhoea due to fowl cholera infection (Left picture) and chicken liver with small white necrotic spots (right picture)

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

6.8 | Chronic Respiratory Disease (CRD) Nkurungata enkokon (Samburu), Ipiyorit Akienga (Turkana)

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 Samburu county the apparent morbidity rate is 20% with a mortality rate of 11%and case fatality rate of 55%.
- CRD in Samburu county is common the short wet season (Lgergerua-April to May) in Samburu community or during the long rainy season (Akiporo-March to June) in Turkana community.

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.

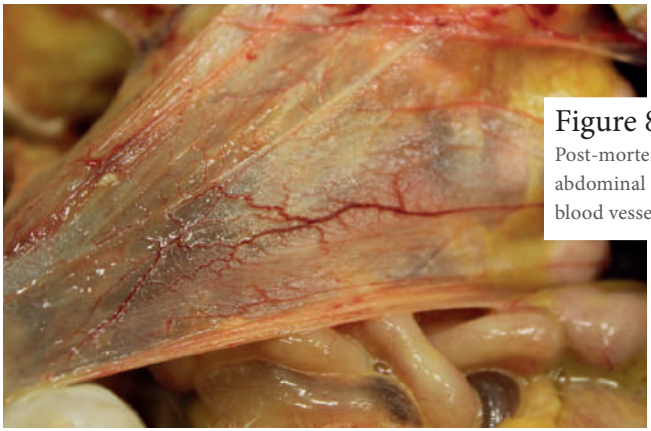


Figure 89
Post-mortem lesions of a bird with CRD with abdominal sac thickened and cloudy with blood vessels visible

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)- Kebirua (Samburu), Egolit Arukum (Turkana)

- 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.
- In Samburu county the apparent IB morbidity rate is 15% with a mortality rate of 5% and case fatality rate of 33%.
- IB prevalence in Samburu county occurs during the months of June to October which is the long dry season locally known as Lameioodo in Samburu community and Akaamu nakoyen in Turkana community.

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

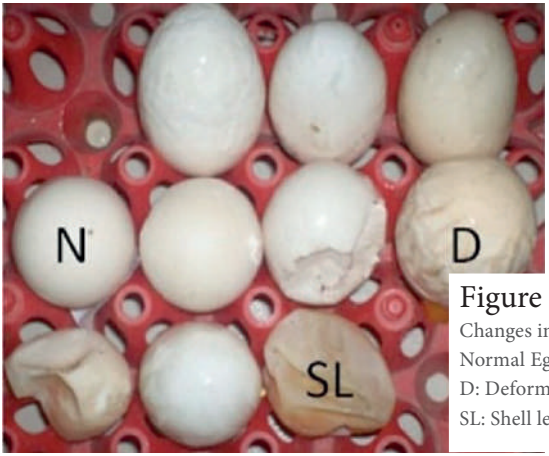
- The trachea, sinuses, and nasal passages contain serous, catarrhal, or caseous exudates, and the air sacs have a foamy exudate initially, progressing to cloudy thickening. If complicated by infection with E coli, there may be caseous airsacculitis, perihepatitis, and pericarditis.
- Young birds will 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 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.



Picture source:
https://www.researchgate.net/profile/Tofazzal-Md-Rakib

Figure 90

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

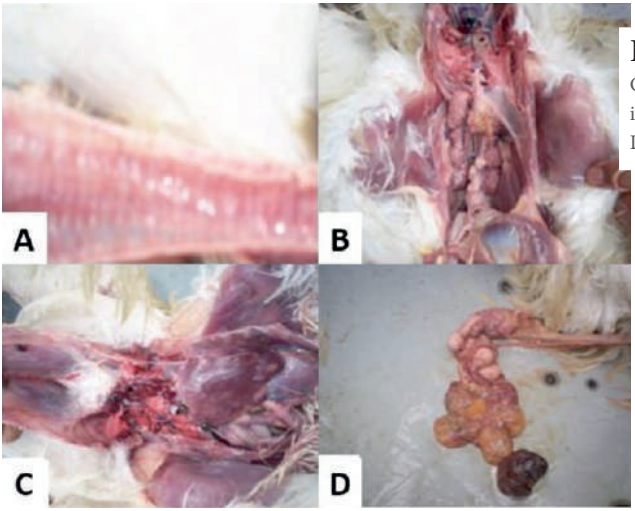


Figure 91

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)

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.
- In Samburu Gumboro disease occurs during the short dry season in the months of January to March, the season is locally known as Lamei dorop in Samburu language and Akaamu Narien in Turkana language.
- The apparent IBD morbidity rate in Samburu is 16% with a 13% mortality rate and case fatality rate of 81%.

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.

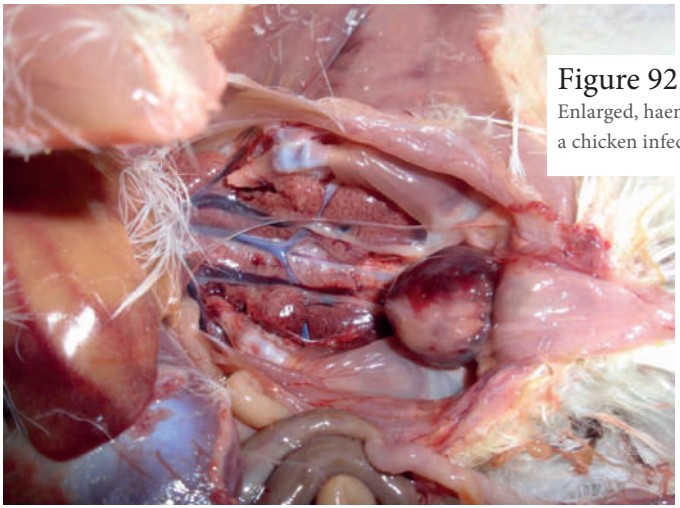


Figure 92
Enlarged, haemorrhagic bursa of Fabricius in a chicken infected with IBD (Black Arrow)

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: Samburu Livestock Vaccination calendar

Key:	Cattle	Goats	Chicken
	Camel	Sheep	

	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

- 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.

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Anthrax

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.

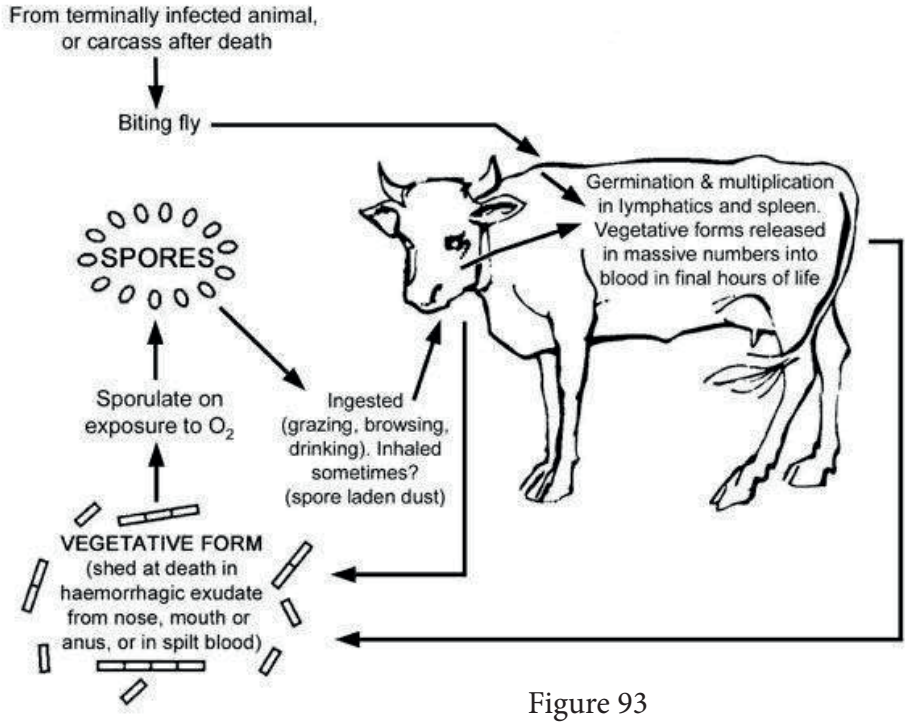


Figure 93
Anthrax Transmission cycle in cattle

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

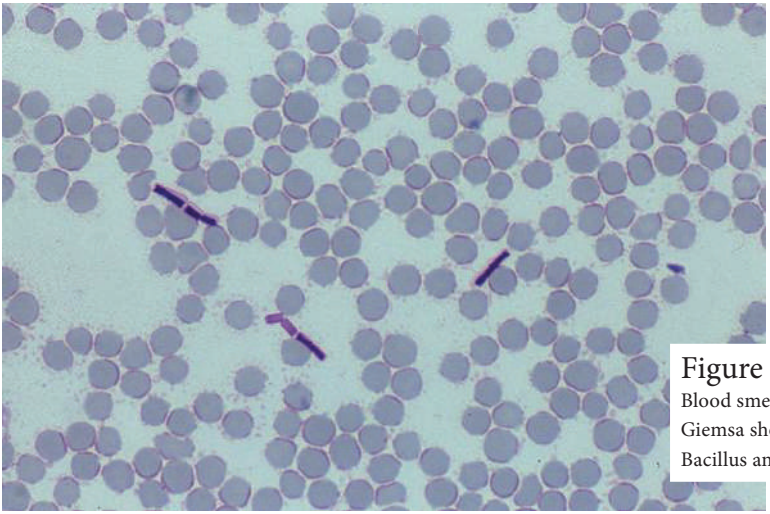


Figure 94
Blood smear stained with Giemsa showing characteristic Bacillus anthracis bacteria

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

Rabies

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.

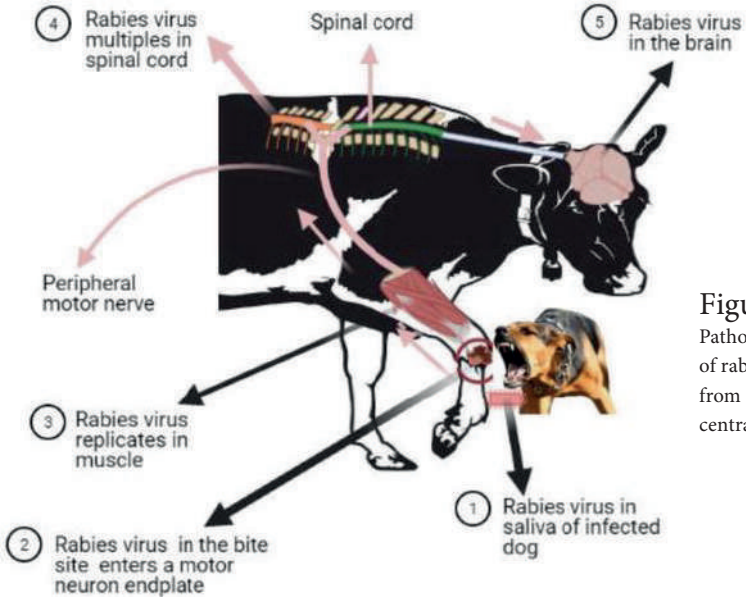
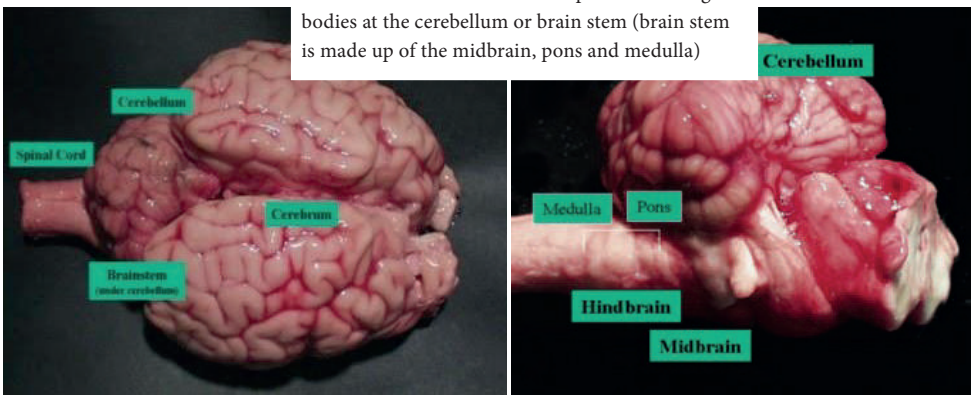


Figure 95
Pathogenesis and spread of rabies virus in animals from the bite site to the central nervous system

Picture source:
https://www.intechopen.com/online-first/77118

Figure 96
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)



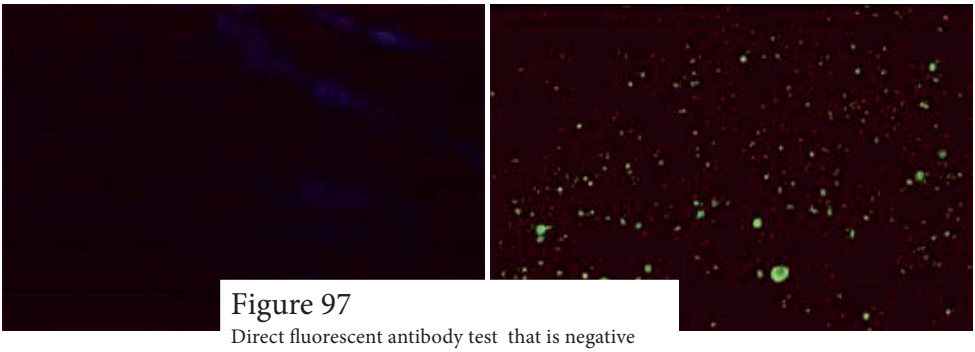


Figure 97
Direct fluorescent antibody test that is negative (Left) and positive (Right) for the rabies virus

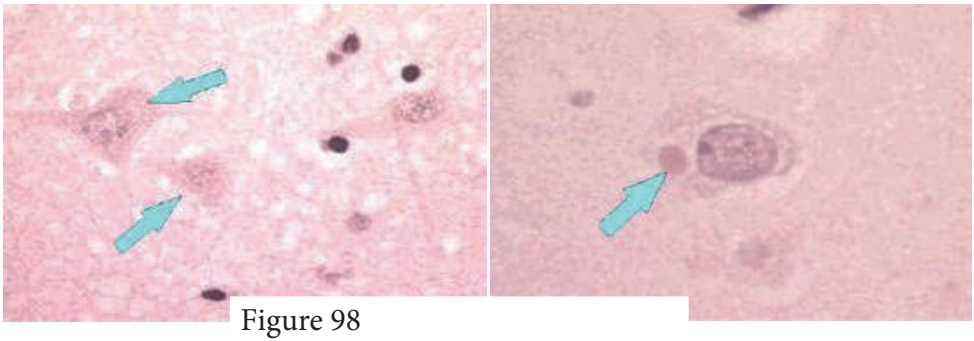


Figure 98
Histology brain tissue with neuron without Negri bodies (right) and with Negri bodies (left)

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

Rift Valley Fever

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

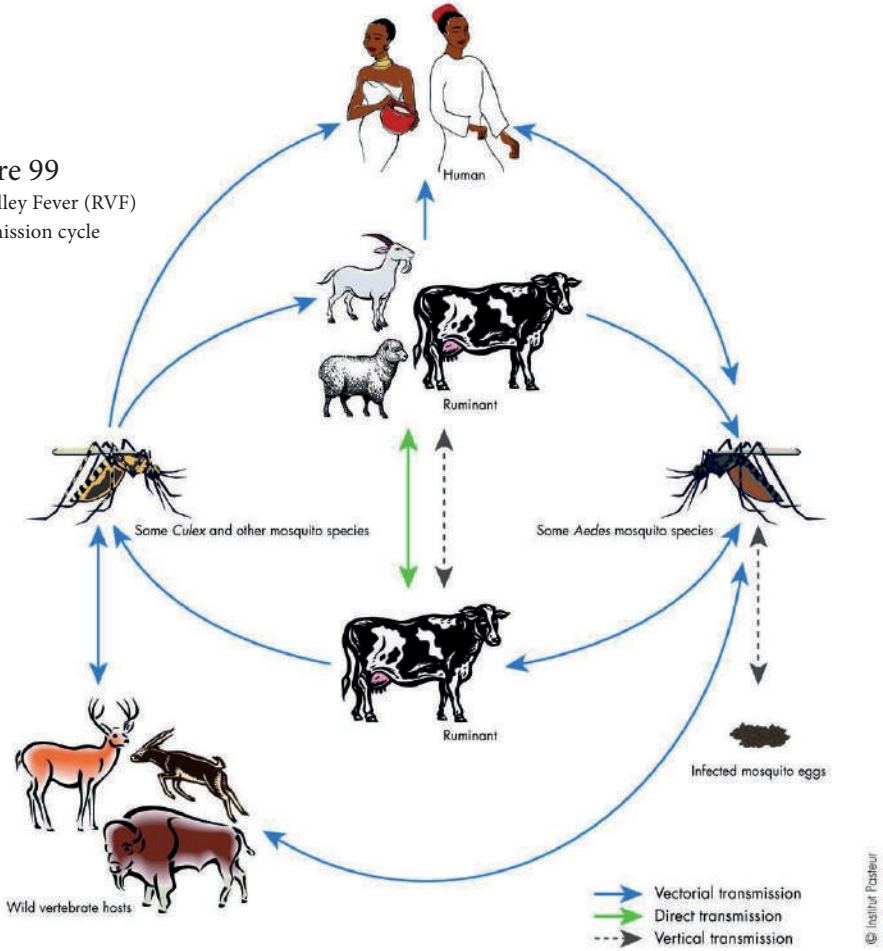
- There is no treatment of sick animals, supportive therapy to animals with clinical signs. Care should be taken not to spread infection through re-use of hypodermic needles or self-infection during treatment of the sick animals.
- Conduct Participatory disease surveillance (PDS) Surveillance during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

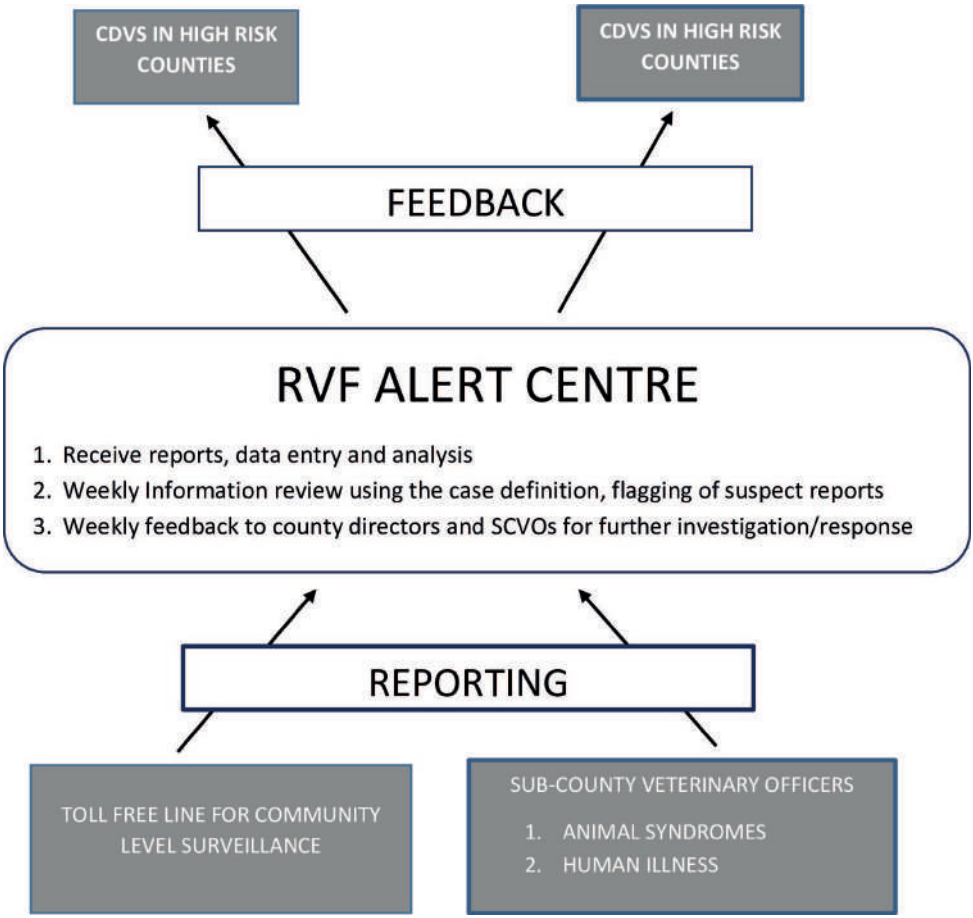
Figure 99
Rift Valley Fever (RVF)
Transmission cycle



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

Figure 100

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 101

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

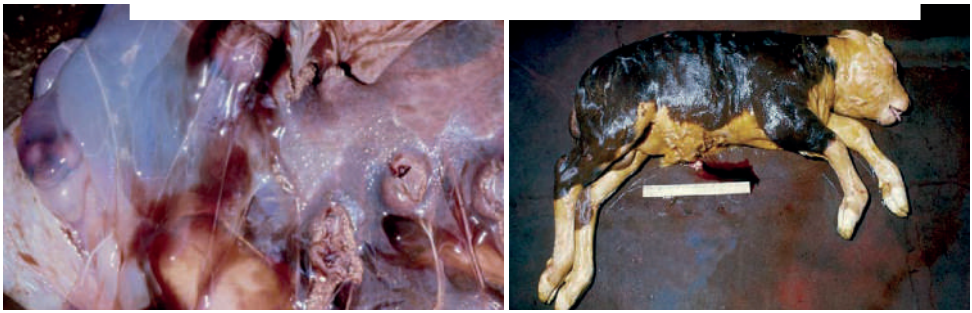
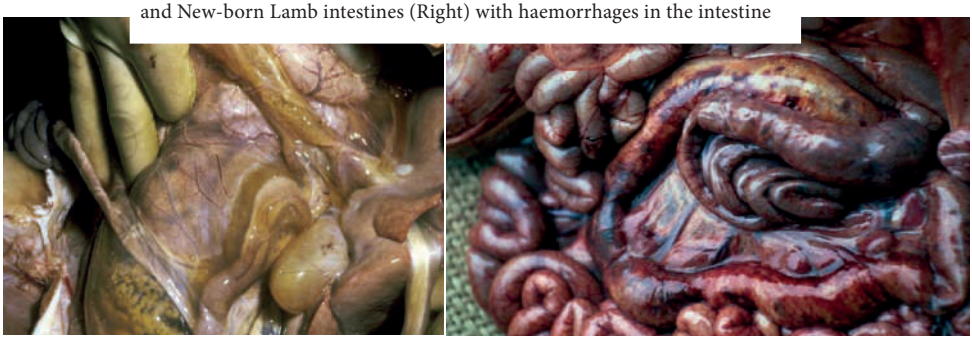


Figure 102

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



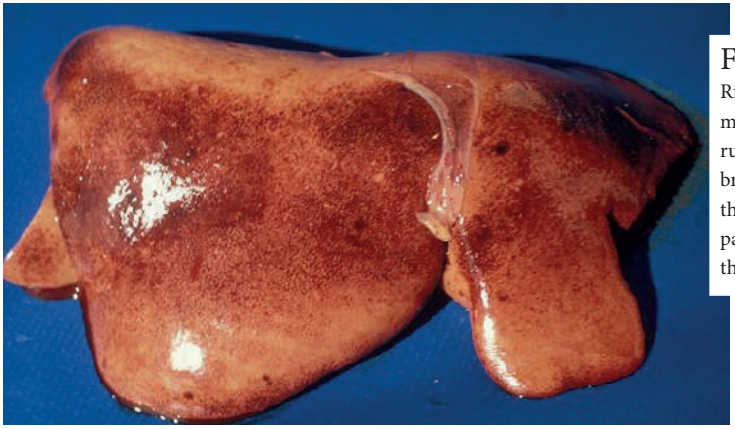


Figure 103

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

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.

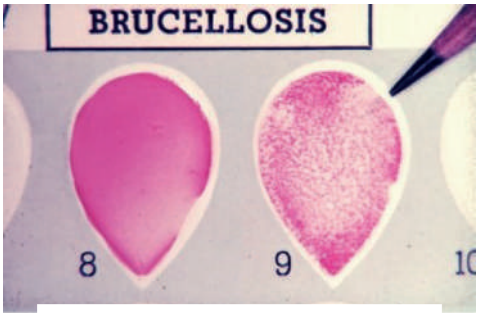


Figure 104
Rose Bengal or card test showing negative (left) and positive (right) reactions

Figure 105
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.

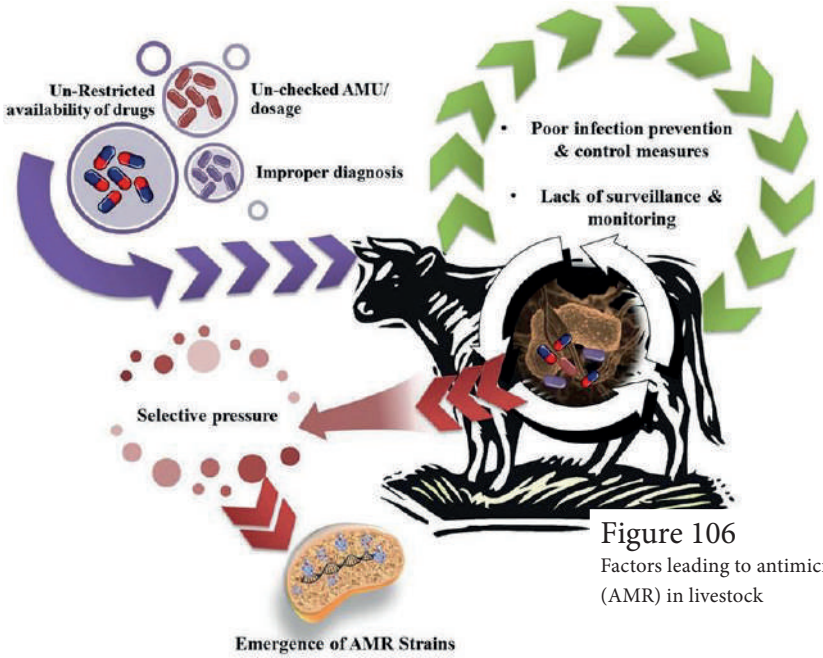


Figure 106
Factors leading to antimicrobial resistance (AMR) in livestock

Figure 107
Conceptual representation of possible movement of antibiotic- resistant bacterial strains/genes between different ecosystems

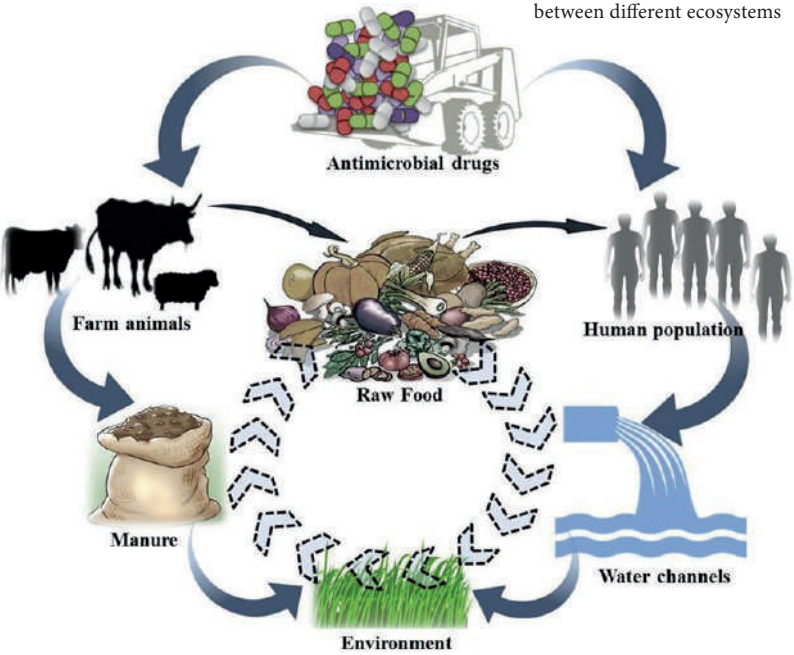


Figure 108

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.) Fluro and other quinolones Glycopeptides Macrolides and ketolides Polymyxins	Aminoglycosides Ansamycins Carbapenems and other penems Glycylcylines Lipopeptides Monobactams Oxazolidinones Penicillins (natural aminopenicillins and antipseudomonal) Phosphonic acid derivatives Tuberculosis and other mycobacterial drugs	Amphenicols Cephalosporins (first & second gen.) Lincosamides Penicillins (anti-staphylococcal) Pseudomonic acids Riminofenazines Steroid antibacterials Streptogramins Sulfonamides Sulfones Tertacyclines	Aminocyclitols Cyclic polypeptides Nitrofurantoin Nitroimidazoles Pleuromutilins	Bambermycins Quinoxalines Ionophores Orthosomycins

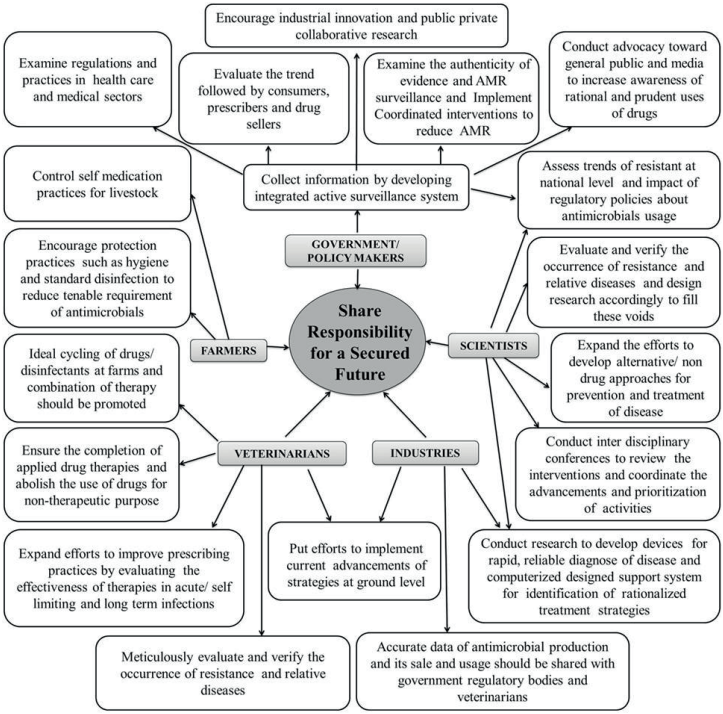


Figure 109

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

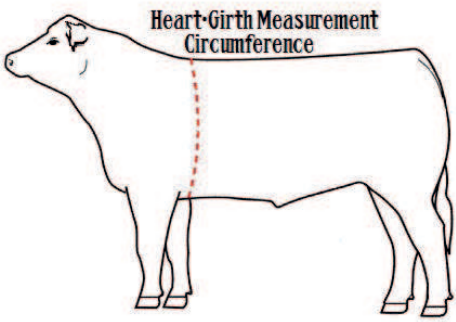
- Livestock owners need to know the weight of their animals in order to determine proper dosage of a medication, or to track how individual animals grow and use their feed. Approved and properly calibrated livestock scales are the most accurate and consistent method for determining body weight. However, 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 110

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 111

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 112

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.

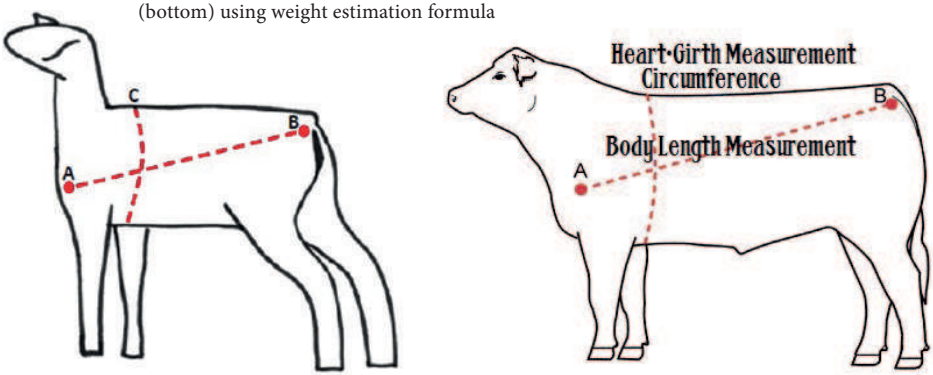
A common tape measure is used to determine the heart girth and body length measurement. These measurements are then used to calculate the animal's weight using the weight estimation formulas described below;

- 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 113

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 Practices for Priority Livestock Diseases as described by livestock keepers

Cattle Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Foot and Mouth Disease (FMD) Samburu- Lkulup Turkana - Lokulup	Topical application of mouth and feet lesions with a paste made from pounded barks and roots of Lkiloriti (Acacia nilotica)	- Wash mouth and foot lesions with salty water (common table salt- NACL) - Smear honey on mouth lesions -Wash mouth and feet lesions with salty water and or Cattle urine (Nkula) - Apply ghee on mouth lesions Hot foment foot lesions by soaking feet into water dissolved with an alkaline salt called Magadi* (NACL, NAHCO3) or dip feet into heated cow dung
Anaplasmosis Samburu-Ndis Turkana- Lonyang	Oral drench of cold decoctions* from; - fruits and roots of Seketet (Myrisine africana) - bark of Ltepes/Ldepe (Acacia tortilis) - bark, stem and twigs of Sumuroi/ Sumuruai*** - whole plant of Lmakutikut (Clerodendrum myricoides Hochst - bark and stem of Lbukoi (Terminalia brownii Fries)	- Oral drench of mixture of raw eggs, changaa** and salty water -Oral drench of donkey faeces - Bleeding animal from jugular vein - Forcing animal to inhale tobacco powder mixed with little amount of kerosene
Babesiosis Samburu-Nkula Turkana- Akul Narrengak	-Oral drench of decoctions* from; -bark of Ltepes/Ldepe (Acacia tortilis)	None
Lumpy Skin disease Samburu-Nariri enkishu Turkana- Tune	None	- topical application on skin lesion used engine oil

* 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 Disease	Ethnoveterinary Plants	Ethoveterinary Practice
East Coast Fever (ECF) Samburu- Lipis Turkana- Lipis	Oral drench decoction of; - Akayei*** - whole plant of Lmakutikut (Clerodendrum myricoides Hochst	- Hot iron burning of swollen lymphnodes
Black quarter Samburu-Nenkeju Turkana- Ngwaat	None- sudden death	- Burning affected muscle with hot iron rod (hot iron branding) - Hot fomentation of affected muscles.
Fascioliasis (liver fluke) Samburu-Lkurui Lemonyua Turkana- Ngirtan	None	- Take animals to graze in natural salt lick areas it will clear the parasite
Pneumonia Samburu- Lkipei Loonkishu Turkana- Loukoi	- oral drench of cold decoction of the herb Emuth***	- Hot iron burning/branding along the ribs
Trypanosomiasis Samburu- Saara Turkana- Saar/Ekwakoit/ Lokipi	None	- Bleeding animal from jugular vein - Take animals to graze in natural salt lick areas it will clear the parasite
Listeriosis Samburu- Mporoto Turkana- Mporroto	- Oral drench of cold decoction of the herb Lokaagogon***	Kill the wild pig (Lguyia), burn the hide and give the flaked hide dirt as an oral drench. -Oral drench of solution of donkey dung

Key

*= Decoction is an extraction procedure where crude plant parts like leaves, bark or roots that have been pounded and crushed and soaked in water or boiled in water with the process lasting for a specific time period.

** = traditional home-brewed spirit that is a potent alcoholic drink made from fermentation and distillation of grains like millet, maize and sorghum.

***= Botanical name not known by FGDs and Validation participants or from literature review.

* 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.

Camel Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Camel flu Samburu-Lchama Turkana- Lowola	- Oral drench of ground seeds of Zanthoxylum usambarense- Loisuk (Samburu) Ethugugu (Turkana) - juice of fruit of Solanum incanum L. (Sodom Apple) Ltulelei (Samburu) put into nostrils - oral drench of milky secretion of Euphorbia candelabrum (Samburu-Mpopong and Turkana Eligoi)	- Hot iron branding at the bridge of the nose - Oral drench of sheep fat (Latanger) mixed with soup made from head and hooves (hide burnt first before make the soup) given as an oral drench.
Haemorrhagic Septicaemia Samburu- Nalngaringari Turkana-Lokou/ Longaruei	- Oral drench of ground seeds of Zanthoxylum usambarense- Loisuk (Samburu) Ethugugu (Turkana)	-Oral drench of sheep fat (Latanger) mixed with soup made from head and hooves (hide burnt first before make the soup) given as an oral drench. - Hot iron burning of swollen lymph nodes
Contagious skin necrosis Samburu-Lmonkoi Turkana-Akitherit	Topical application of milky secretion of Euphorbia candelabrum (Samburu-Mpopong and Turkana Eligoi)	Animals taken to access salty lick areas (Suguta)
Trypanosomiasis Samburu- Saar Turkana- Ekwakoit/ Saar/ Lokipi	None	Bleeding the animal from jugular vein
Contagious ecthyma (Orf) Samburu-Abitiro Turkana- Ng'iborouk	None	-if animal severely affected apply sheep fat (Latanger) or ghee on mouth lesions
Camel mange Samburu-Lpepedo Turkana- Emitina pepedo	None	- Topical application of used engine oil - Take animals to be washed with salty water (Suguta)
Caseous lymphadenitis Samburu- Araar Turkana- Abuth	Topical application of milky secretion of Euphorbia candelabrum (Samburu-Mpopong and Turkana Eligoi)	Take animals to access natural salt licks (Suguta area)

* 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.

Goat Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Contagious Caprine Pleuropneumonia (CCPP) Samburu-Lkipei Turkana- Loukoi	-Oral drench of cold decoction of Emacher* and Emuth*	- Hot rod used to make burn/brand marks on the skin over the lungs (rib area) - Hot foment with warm water the lung/rib area
Peste des Petits Ruminants (PPR) Samburu-Ngoritit/ Serr/ Nkiriata/ Lodua Turkana- Lonoo	-fruits/seeds Myrsine africana L.(Seketet) crushed and disoolved in water and given as an oral drench	Wash mouth lesions with warm salty water
Sheep and Goat Pox (SGP) -Samburu- Nariri/ Tune Turkana – Tune	None	None
Coenurosis - Samburu- Sirko Turkana-Nkerep	- Oral drench of cold decoction of the herb Emacher* - administer a little juice of Solanum incanum- Sodom apple fruit into nostrils Ltulelei (Samburu) Etulelo (Turkana)	None
Coccidiosis - Samburu- Nkiriata Elodo ; Turkana- Eremorit Ng'aokot	None	None
Helminthiasis - Samburu- Ntumuai Turkana- Ngiritan	Fruits/seeds Myrsine africana L.(Seketet) crushed and disoolved in water and given as an oral drench	Oral drench with warm salty water
Contagious ecthyma (Orf) Samburu-Abituro Turkana-Ng'borouk	None	Wash wounds with warm salty water Apply ghee on mouth lesions Hot iron burning/branding on the skin over the bridge of the nose
Mange - Samburu-Iputo Turkana-Echudano	None	Wash animal with sand and water
Enterotoxaemia Samburu-Sirr / Nadomanyita Turkana- Lobus	None	None Occurs mostly in sheep not goats
Foot rot - Samburu- Ng'ojini Turkana-Lukulup	None	Dip hoof affected in salty water

* 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 Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Enterotoxaemia Samburu-Lbus/ Nadomanyita Turkana- Lobus	- chewing tobacco (Nicotiana tabacum) administered through forced inhalation - tobacco mixed with water, administered as an oral drench.	- Bleeding animal from the jugular or tips of the ears -Ear notching -Oral drench of cow milk mixed with water
Peste des Petits Ruminants (PPR) Samburu-Kinyoot/ Nkiriata/ Lodua Turkana- Lokiyoo/ Lonoo	-fruits/seeds Myrsine africana L.(Seketet) crushed and dissolved in water and given as an oral drench	-Hot iron burning/branding of nose bridge
Sheep and Goat Pox (SGP) Samburu- Nariri/ Tune Turkana – Tune	- Oral drench of leaf sap of Aloe secundifora Samburu-Sukuroi, Turkana-Echuchuka	None
Coenurosis Samburu- Sirko Turkana-Sirko / Nkerep	Administer a little juice of Solanum incanum- Sodom apple fruit into nostrils Ltulelei (Samburu) Etulelo (Turkana)	None-once signs start animal dies
Foot and Mouth Disease (FMD) Samburu-Lkulup Turkana- Lokulup	None	- Wash mouth and feet lesions in salty water solution - Hot iron branding of skin over the mouth and hoof area
Helminthiasis Samburu-Ntumuai Turkana- Ngiritan	- Fruits/seeds Myrsine africana L.(Seketet) crushed and dissolved in water and given as an oral drench - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	-Taking animals to drink salty water -Hot iron branding perineum area
Pneumonia Samburu- Lkipei/Nkijepe Turkana- Lotai	- Myrsine Africana L. (Seketet) seeds oral drench - Oral drench of leaf sap of Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	Hot iron branding of skin over ribs - Hot foment chest area
Mange Samburu-Iputo Turkana-Echudano	None	Allow animals to access salty water

* 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 Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Anaplasmosis Samburu- Ndis ; Turkana- Lony'ang	- Myrsine Africana L. (Seketet) seeds oral drench - Terminalia brownie (Lbukoi) oral drench	Administer onion juice through the nostrils
Babesiosis Samburu- Nkula Turkana- Lonyang	- Myrsine Africana L. (Seketet) seeds oral drench - chewing tobacco (Nicotiana tabacum) administered through forced inhalation	None

Chicken Disease	Ethnoveterinary Plants	Ethoveterinary Practice
New Castle Disease (NCD) Samburu-Lchama / Nkonkoro Turkana- Lonoo	- Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka - Onions crushed and given orally - Mwarubaini leaves (neem) given as an oral drench - Myrsine Africana L. (Seketet) seeds oral drench	-Isolate sick birds Warm water/milk given orally - Amoxicillin - capsule (powder) human formulation oral - Paracetamol human formulation (Panadol) crushed and put in drinking water - Tobacco and omo mix wash given orally
Infectious coryza Samburu- Moyen enkonyek Turkana-Eboot Akou	- Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	- Isolate sick birds -Warm water/milk given orally - Tobacco and omo mix wash given orally -Topical application vaseline/ arimis/paraffin/kerosene topical/oil/ methylated spirit/sanitizer
Fowl pox Samburu-Nariri enkokon Turkana-Ng'ajemai	- Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	- Isolate sick birds Vaseline / arimis/ paraffin/kerosene topical/oil/ methylated spirit/sanitizer -Tobacco+ omo mix wash

* 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 Disease	Ethnoveterinary Plants	Ethoveterinary Practice
Ectoparasites Fleas Samburu-Lososu Turkana-Lososu Lice Samburu- Lashe Turkana-Elachet Mites Samburu- Mpaipayan Turkana- Losaa Scaly feet/Scaly leg mite Samburu-Nkasimai Turkana-Ngooeye Ticks Samburu- Lmancher enkokon Turkana-Emadang	- Topical application of Aloe vera sap Samburu-Sukuroi, Turkana-Echuchuka	- Apply used engine oil on lesions - Move chicken to new house and burn the old house - Topical application of Powder of ashes -Wash bird with Tobacco and omo mix - Topical application of Vaseline/ arimis/ paraffin/kerosene topical/oil/ methylated spirit/sanitizer
Fowl Cholera Samburu- Nkiriata Turkana- Eriomorit	-Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	None
Chronic Respiratory Diseases Samburu- Turkana-	- Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka - Myrsine Africana L. (Seketet) seeds oral drench	Isolate sick birds
Helminthiasis Samburu- Ntumwa Turkana- Ngiritan	-Myrsine Africana L. (Seketet) seeds oral drench	None
Infectious bronchitis Samburu- Kebirua Turkana- Egolit arukum	- Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	None
Gumboro Samburu- new disease has no local name Turkana- Etunati	-Ground pepper seeds in the drinking water - Oral drench of leaf sap of Aloe vera secundifora Samburu-Sukuroi, Turkana-Echuchuka	None

Annex:

7: Ethnoveterinary Control Practices for Priority Livestock diseases as described by livestock keepers

Cattle Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Foot and Mouth Disease (FMD) Samburu- Lkulup Turkana - Lokulup	-Avoid outbreak area -Vaccination -Isolate sick herd or animal (avoid sharing the same grazing area or watering area	-Brand marks made with a hot iron at the side of the animal to protect young calves from FMD
Anaplasmosis Samburu-Ndis Turkana- Lonyang NB: Disease not common in Turkana herds	Regular spraying with acaricides	None
Babesiosis Samburu-Nkula Turkana- Akul Narrengak NB: Disease not common in Turkana herds	Regular spraying with acaricides	None
Lumpy Skin disease Samburu-Nariri enkishu Turkana- Tune NB: Disease not common in Turkana herds	-Regular spraying with acaricide - Avoid outbreak areas or herds -Vaccination	None
East Coast Fever (ECF) Samburu- Lipis Turkana- Lipis NB: Disease not common in Turkana herds	Regular spraying with acaricide	Remove one parotid lymph node (rights side preferred) when animals are calves but it is not encouraged as the calves do not grow and add weight like the rest.

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Cattle Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Black quarter Samburu-Nenkeju Turkana- Ngwaat	-Move cattle from boma/area when one animal dies -Avoid grazing animals in area where there has been a previous outbreak -Vaccination	None
Fascioliasis (liver fluke) Samburu-Lkurui Lemonyua Turkana- Ngirtan	-Regular use of anthelmintic-injectable flukicides - Avoid grazing animals in swampy areas	Allow animals to access salty water areas
Pneumonia Samburu- Lkipei Loonkishu Turkana- Loukoi	Isolate sick animal	None
Trypanosomiasis Samburu- Saara Turkana- Saar/Ekwakoit/ Lokipi	Avoid areas with biting flies that transmit Trypanosomiasis	- Graze sheep in front of cattle (sheep smell repels Tsetse flies) - Smoke cattle boma in the evening to chase away biting flies
Listeriosis Samburu- Mporoto Turkana- Mporroto	Burn decaying grass where the wild pigs make their home burrows.	

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Camel Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Camel flu Samburu-Lchama Turkana- Lowola	- No Prevention available (Comes like the wind) - Isolate sick animals/herds and ensure they don't share browse areas or watering points	None
Haemorrhagic Septicaemia Samburu- Nalngaringari Turkana-Lokou/ Longaruei	-Avoid areas or herds that are experiencing an outbreak -Isolate sick animals/herds and ensure they don't share browse areas or watering points	None
Contagious skin necrosis Samburu-Lmonkoi Turkana-Akitherit	-Isolate affected animals –build separate boma for them if necessary	Allow animals access to salty lick areas/water and browse
Trypanosomiasis Samburu- Saar Turkana- Ekwakoit/ Saar/ Lokipi	- Avoid areas known to have biting flies that transmit the disease	Bleeding the animal from jugular vein if they look weak or they are not adding weight
Contagious ecthyma (Orf) Samburu-Abitiro Turkana- Ng'iborouk	None	None
Camel mange Samburu-Lpepedo Turkana- Emitina pepedo	-Regular spraying with acaricides - Regular use of anthelmintics especially when calves are young or juvenile -Separate infected animals including building a separate night boma	None
Caseous lymphadenitis Samburu- Araar Turkana- Abuth	None	None

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Goat Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Contagious Caprine Pleuropneumonia (CCPP) Samburu-Lkipei Turkana- Loukoi	-Isolate sick animals/herds -Avoid mixing animals from different herds -Vaccination -Regular use of oral anthelmintics	None
Peste des Petits Ruminants (PPR) Samburu-Ngoritit/Serr/ Nkiriata/ Lodua Turkana- Lonoo	- Isolate sick animals/herds -Avoid mixing animals from different herds -Vaccination -Regular use of oral anthelmintics	None
Sheep and Goat Pox (SGP) Samburu- Nariri/ Tune Turkana – Tune	- Isolate sick animals/herds -Avoid mixing animals from different herds -Vaccination -Regular spraying with acaricides	None
Coenurosis Samburu- Sirko Turkana-Nkerep	None	None
Coccidiosis Samburu-Nkiriata Elodo Turkana- Eremorit Ng'aokot	- Regular use of anthelmintics - Regular clearing of manure from goat boma	None
Helminthiasis Samburu-Ntumuai Turkana- Ngiritan	-Regular use of anthelmintics -Regular clearing of manure from goat boma	None
Contagious ecthyma (Orf) Samburu-Abituro Turkana-Ng'borouk	- Avoid herds with disease - Avoid mixing of herds	None
Mange Samburu-Iputo Turkana-Echudano	- Regular use of acaricides -Clearing dung from the boma	Allow goats to have access to salt licks
Enterotoxaemia Samburu-Sirr / Nadomanyita Turkana- Lobus	-Milk does before the kids suckle in the evening -Avoid animals eating lush pastures	None
Foot rot Samburu-Ng'ojini Turkana-Lukulup	Clearing dung from boma	-

* 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 Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Enterotoxaemia Samburu-Lbus/ Nadomanyita Turkana- Lobus	-Vaccination -Regular use of anthelmintic	None
Peste des Petits Ruminants (PPR) Samburu-Kinyoot/ Nkiriata/ Lodua Turkana- Lokiyoo/ Lonoo	-Vaccination -Regular use of anthelmintic -Regular spraying with acaricides	None
Sheep and Goat Pox (SGP) Samburu- Nariri/ Tune Turkana – Tune	-Isolate sick animals -Vaccination	
Coenurosis Samburu- Sirko Turkana-Sirko / Nkerep	None	None
Foot and Mouth Disease (FMD) Samburu-Lkulup Turkana- Lokulup	-Vaccination -Avoid herds/areas with outbreak	None
Helminthiasis Samburu-Ntumuai Turkana- Ngiritan	-Regular deworming	Animals should be taken to salt lick areas
Pneumonia Samburu-Lkipei/Nkijepe Turkana- Lotai	- Isolate sick animals -Regular deworming of animals	None
Mange Samburu-Iputo Turkana-Echudano	-Regular use of acaricide spray	None
Anaplasmosis Samburu-Ndis Turkana-Lony'ang	-Regular spraying with acaricides -Regular use of anthelmintics	None
Babesiosis Samburu- Nkula Turkana- Lonyang'	- Regular spraying with acaricides	None

* 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 Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
New Castle Disease (NCD) Samburu-Lchama / Nkonkoro Turkana- Lonoo	- Isolate sick animals - Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Regular use of sevin powder to control ectoparasites -Vaccination -Use of oral antibiotics fortified with multivitamins in drinking water during outbreaks -Prompt disposal of dead birds -Regular cleaning of the chicken house	None
Infectious coryza Samburu- Moyen enkonyek Turkana-Eboot Akou	- Isolate sick animals - Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Regular use of sevin powder to control ectoparasites -Vaccination -Use of oral antibiotics fortified with multivitamins in drinking water during outbreaks	None
Fowl pox Samburu-Nariri enkokon Turkana-Ng'ajemai	- Regular use of acaricides to control ectoparasites - Regular cleaning of the chicken house and use of ash in the chicken house floors	None
Ectoparasites Fleas Samburu-Losusu Turkana-Losusu Lice Samburu- Lashe Turkana-Elachet Mites Samburu- Mpaipayan Turkana- Losaa Scaly feet/Scaly leg mite Samburu-Nkasimai Turkana-Ngooeye Ticks Samburu- Lmancher enkokon Turkana-Emadang	- Regular use of acaricides to control ectoparasites - Regular cleaning of the chicken house and use of ash in the chicken house floors	None

* 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 Disease	Conventional Veterinary Medicine	Ethoveterinary Practice
Fowl Cholera Samburu- Nkiriata Turkana- Eriomorit	- Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Use of oral antibiotics fortified with multivitamins in drinking water during outbreaks -Prompt disposal of dead birds -Regular cleaning of the chicken house	None
Chronic Respiratory Diseases Samburu-Nkurungata enkokon Turkana- Ipiriyorit Akienga	- Isolate sick animals - Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Prompt disposal of dead birds -Regular cleaning of the chicken house	None
Helminthiasis Samburu- Ntumwa Turkana- Ngiritan	-Regular use of oral anthelmintics - Clean the chicken house regularly and use of ash in the chicken house floors	None
Infectious bronchitis Samburu- Kebirua Turkana- Egolit arukum	- Isolate sick animals - Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Prompt disposal of dead birds -Regular cleaning of the chicken house	None
Gumboro Samburu- new disease has no local name Turkana- Etunati	Isolate sick animals - Dissolve ground pepper seeds in drinking water for the rest of the flock -Construct a proper chicken house -Prompt disposal of dead birds	None

* 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.

SAMBURU COUNTY

VETERINARY HANDBOOK

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