

UNITED REPUBLIC OF TANZANIA



MINISTRY OF LIVESTOCK AND FISHERIES

**DISEASE FACT SHEET FOR TRANSBOUNDARY
ANIMAL DISEASES (TADs)**

FOR FIELD VETERINARY STAFF IN TANZANIA

January 2019

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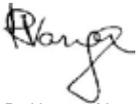
ACKNOWLEDGEMENT

This Animal Diseases Fact Sheet is a revision of the original document developed in 2009 by the Ministry of Livestock and Fisheries Development (MALF) and Food and Agriculture Organization of the United Nations (FAO), to facilitate recognition and reporting of priority animal diseases in order to enhance timely response.

The current version takes into account existence of Guidelines for Surveillance of Prioritized Zoonotic Diseases for Human and Animal Health of the United Republic of Tanzania, 2018.

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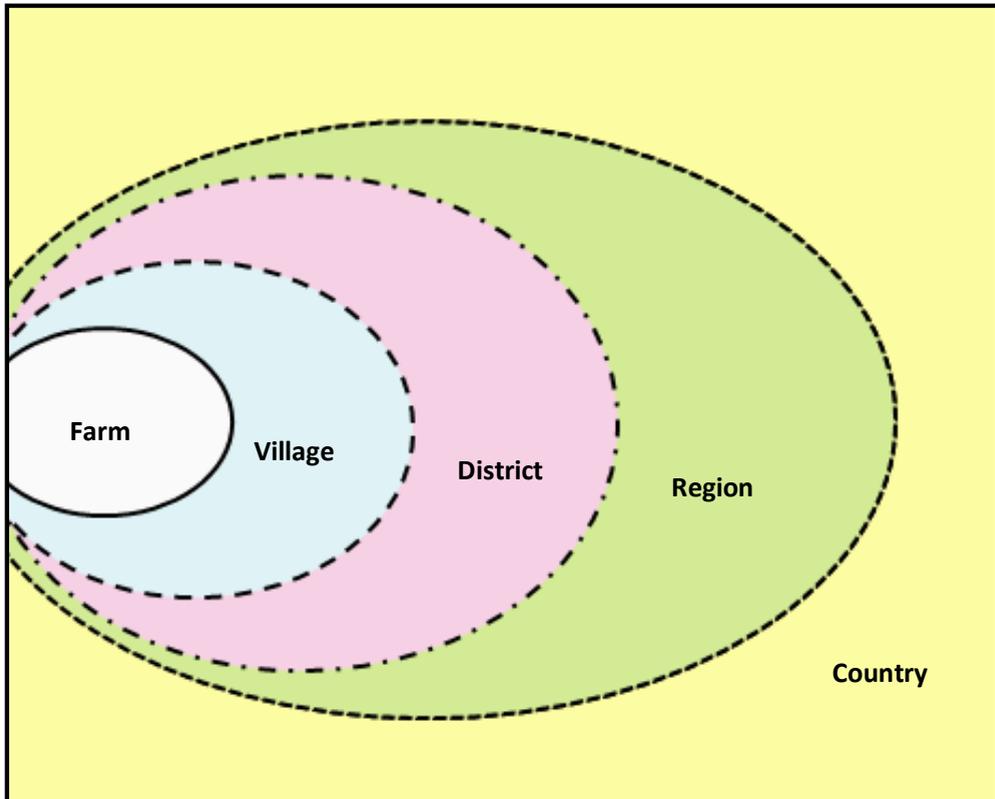
Dr Hezron Nonga
Director of Veterinary Services

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BIOSECURITY IN THE CONTROL OF ANIMAL DISEASES



ACTIONS TO TAKE:

- Educate personnel and livestock farmers on epidemic aspects of the disease
- Ensure use of proper personal protection equipment (PPE)
- Ensure safe collection, handling and shipment of appropriate samples
- Educate on biosecurity measures:
 - ✓ Bio-exclusion (segregation)
 - ✓ Bio-containment (cleaning and disinfection)

1. BIOSECURITY IN THE CONTROL OF ANIMAL DISEASES

DEFINITION

Biosecurity can be defined as security from transmission of infectious diseases, parasites and pests. Biosecurity also means creation of an environment where infectious diseases are contained and prevented from spreading. It encompasses protection of the health of living things, the environment and the economy against biological harm from infectious diseases, parasites and pests.

IMPORTANCE OF BIOSECURITY

Failure in biosecurity can result in disease, human/animal deaths, decreased production and great economic loss. Economic loss is consequential to the infectious disease, parasite/pest infestation as well as to the cost of controlling/eradicating the introduced/prevalent infectious disease or parasite/pest infestation.

ASPECTS OF BIOSECURITY

Biosecurity involves:

Segregation

This involves keeping potentially infected animals and materials away from susceptible animals and humans. It entails-

- Creation of barriers (physical and/or temporal or procedural)
- Control of movement through barriers by:-
 - Enforcing changing of footwear and clothing for all people passing through the barrier
 - Restricting passage of vehicles, animals and humans

Cleaning

Contamination with disease causing agents on physical objects occurs via adherent faecal materials, respiratory and other excretions as well as blood and organ parts and therefore removal of these by cleaning will remove most of the disease-causing agents

- Small objects can be cleaned adequately with soap, water and brush
- High pressure water is needed for large objects such as vehicles as well as premises (recommended pressure is 110-130 bar)

Disinfection

- Disinfection is a "final polishing step" in biosecurity
- Many disinfectants can destroy disease-causing agents under ideal conditions but not in dirt
- Always use a recommended disinfectant

EMPHASIS IN BIOSECURITY

You should always aim at :

- Bio exclusion : Keeping disease-causing agent out of uninfected farm or premise
- Bio containment: Keeping disease-causing agent from spreading
- Prevention: Carrying out disease control measures to protect humans and animals from getting infected



CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP)



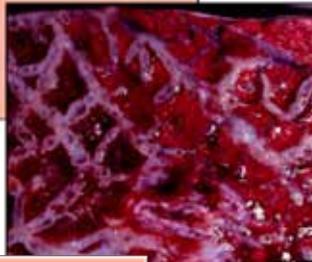
Unthrifty animal



Coughing with extended neck



Swollen joints sign of arthritis



Marbled lung



"Extended neck"

ACTION TO TAKE:

- Examine the whole herd thoroughly
- Fill in the outbreak report form

SAMPLES

- (i) Collect blood in EDTA or Heparin and whole blood decanted and obtain serum;
- (ii) Lung specimen for histology
- Isolate sick animals from the herd and prevent movement
- Submit the duly filled form and samples to the DVO
- Seek further advice from your supervisor

2. CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP)

Kiswahili name: *Homa ya Mapafu ya Ng'ombe*

CAUSATIVE AGENT

Mycoplasma mycoides var. *mycoides*

TRANSMISSION

- Aerosol and droplet infection from infected cattle
- Recovered cattle ("lungers") act as carriers and shedders, especially under stress

EPIDEMIOLOGY

- Species Affected: Cattle
- Morbidity: 90% in susceptible animals
- Mortality: 10-50%
- Distribution: Disease is endemic in Tanzania

CLINICAL SIGNS

- Incubation: Acute 10-14 days; chronic 3-6 months
- Fever, depression; inappetence, loss of weight
- Coughing on exercise; shallow rapid respiration, grunting and gurgling
- Extended neck, lowered head and open mouth
- Arched back and outward rotated elbow
- Arthritis in young animals

LESSIONS

- Fibrinous pleuritis; Straw-coloured fluid in thorax
- Lobar pneumonia with red hepatization
- Marbled appearance of lung lobules due to thickening of interlobular pulmonary oedema
- Enlarged mediastinal lymph nodes
- Walled-off sequestra formation in chronic cases
- Haemorrhage in heart
- Arthritis and tenosynovitis

SAMPLES TO TAKE

- Thoracic fluid; Diseased lung and regional lymph nodes on ice during transportation
- Additional samples in 10% formalin for histopathology; Serum for CFT

DIFFERENTIAL DIAGNOSIS

- Rinderpest; FMD; ; IBR; Hemorrhagic septicaemia; Shipping fever (Pneumonic pasteurellosis)
- ECF; Tuberculosis; Bacterial or viral broncho-pneumonia; Lungworms
- Foreign body reticulum pericarditis; Ephemeral fever; Abscess ; Farcy
- Chlamydial infections; Actinobacillosis; Echinococcal cysts

ACTIONS TO TAKE

Follow the Standard Operation Procedures for an emergency outbreak investigation.



FOOT AND MOUTH DISEASE (FMD)



Un-ruptured vesicle on the tongue



Erosion on the tongue



Erosion of hard palate



Lesions on the teats



Drooling saliva

ACTION TO TAKE:

- Examine the whole herd thoroughly
- Fill in the outbreak report form

SAMPLES

- (i) Collect blood for serum extraction
 - (ii) Collect vesicle fluid and store in cool box
- Isolate the herd and prevent movement
 - Submit the duly filled form and samples to the DVO

3. FOOT AND MOUTH DISEASE (FMD)

Kiswahili name: *Ugonjwa wa Miguu na Midomo*

CAUSATIVE AGENT

- Virus of family Picornaviridae, Genus Aphthovirus which has seven immunologically distinct serotypes: A,O,C, Asia1, SAT (Southern African Territories), SAT 1 SAT2 and SAT3. Preserved by refrigeration and freezing ; Inactivated by temperatures > 50o C, pH < 6.0 or > 9.0; NaOH 2%, NaCO3 4%, Citric acid 0.2%

TRANSMISSION

- Inhalation or ingestion of infective material direct from affected animal
- Direct contact with infected animals
- Mechanical fomites (e.g. vehicles, instruments, feed)
- Airborne (up to 60 km overland and 300 km by sea) in temperate countries
- Virus shed in saliva, faeces, urine, milk, semen and breath (for up to four days before clinical signs)

Source of virus is:

- Incubating and clinically affected animals; African buffalo major reservoir of SAT serotypes
- Breath, saliva, faeces and urine; milk and semen (up to four days before clinical signs).
- Meat and by-products with pH above 6.0
- Carriers, especially cattle and water buffalo; convalescent animals and exposed vaccinates (virus in oropharynx for up to 30 months in cattle or longer in buffalo and nine months in sheep)

EPIDEMIOLOGY

Species Affected:

Highly susceptible: Cattle, pigs, sheep, goats, water buffalo; Sheep/goats maintenance hosts due to mild symptoms.

Susceptible: Antelopes, elephants and giraffes. Under experimental conditions: Rats, mice, hedgehogs and armadillos.

Low susceptibility: Camels, llamas, Alpacas, African buffalo

Amplifying hosts: Pigs produce many times more virus particles in respiratory secretions than other hosts

Indicator hosts: Cattle because they generally are the first hosts to show clinical signs

Human infection: 40 human cases so far worldwide with pyrexia, anorexia and vesicular lesions

- Morbidity: Up to 100% in susceptible population
- Mortality: Generally less than 1%; case fatality rate > 60% for young animals of all susceptible species
- Distribution: The disease is endemic in Tanzania

CLINICAL SIGNS

Incubation period: 2-14 days

Clinical signs:

Cattle

- Pyrexia, anorexia, shivering, reduction in milk production for 2-3 days; smacking of lips, grinding of teeth, drooling of saliva; Lameness, stamping or kicking of feet. Recovery generally within 8-15 days
- Complications: mastitis; permanent impairment of milk production, myocarditis, abortions, death of young animals, permanent loss of weight, loss of heat control (“panthers”)

Sheep and goats: Agalactia in milking sheep and goats; Death in young stock

Pigs: High mortality frequently experienced

LESSIONS

- Cattle: vesicles (aphthae) on buccal and nasal mucous membranes, tongue, dental pad, gums, cheek hard and soft

palate; between claws and coronary band ; on mammary glands; after 24 hours, rupture of vesicles leaving erosions; complications: tongue erosions, super-infection of lesions, hoof deformations

- Sheep/goats: Less pronounced lesions; foot lesions may go un-recognised. Lesions in dental pad of sheep
- Pigs may develop severe foot lesions when housed on concrete

SAMPLES TO TAKE

- Tissue from unruptured or recently ruptured vesicle (1g) placed in transport medium at pH 7.2-7.4
- Oesophageal-pharyngeal fluid collected by probing cup (speculum) or swab and frozen below -40o C.

DIFFERENTIAL DIAGNOSIS

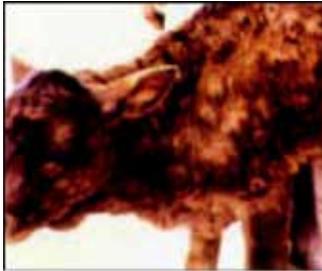
Vesicular stomatitis; Swine vesicular disease; Vesicular exanthema of pigs; Rinderpest; Bovine mucosal disease; IBR; Bovine Mammillitis; Bovine Papular Stomatitis; Bovine Viral Diarrhoea; MCF; PPR; Bluetongue; Foreign bodies and trauma (beware of rabies also!).

ACTIONS TO TAKE

Follow the Standard Operation Procedures for an emergency outbreak investigation.



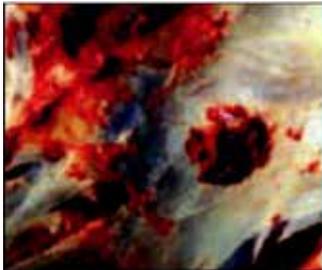
LUMPY SKIN DISEASE (LSD)



Skin nodules on calf



Lesions in the trachea



Lesions on the muscle



Lesions in the lung

ACTIONS TO TAKE:

- Examine the whole herd thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Collect blood in EDTA or Heparin and Serum (whole blood, decant and obtain serum)
 - (ii) Skin biopsy, Lung lesion and lymph node on ice and 10% buffered formalin
- Isolate the herd and prevent movement
 - Submit the duly filled form and samples to the DVO
 - Seek further advice from your supervisor

4. LUMPY SKIN DISEASE (LSD)

Kiswahili name: *Mapele Ngozi*

CAUSATIVE AGENT

Lumpy skin disease (LSD) is caused by a virus in the family Poxviridae, genus Capripoxvirus. It is closely related antigenically to sheep and goat pox virus.

TRANSMISSION

Transmission of the LSD virus is primarily by biting insects, particularly mosquitoes (e.g. *Culex mirificens* and *Aedes natronus*) and flies (e.g. *Stomoxys calcitrans* and *Biomyia fasciata*).

Direct contact is also a minor source of infections (LSD virus can be present in cutaneous lesions, saliva, nasal discharge, milk, semen, muscles, spleen, and lymph nodes).

EPIDEMIOLOGY

Species Affected: Lumpy skin disease is primarily a disease of cattle

Morbidity: 5-50%

Mortality: Low in most cases (1–3%), but depending on the presence of insect vectors and host susceptibility can be as high as 20–85%.

Distribution: The disease is endemic in Tanzania.

CLINICAL SIGNS

Incubation period: 4-14 days

Clinical signs

- Fluctuating fever (4-14 days); hyper-salivation and excessive nasal discharge
- Various-sized cutaneous nodules (diameter 5-50 mm) first seen on perineum 4-10 days after onset of fever, then throughout body (gastrointestinal, respiratory and genital tracts); a few to hundreds in number; may show scab formation and may become erosions.
- Festering sores
- Swelling of superficial lymph nodes ; swelling of limbs and lameness
- Diarrhoea ; extreme emaciation
- Infertility (bulls and cows) and abortion
- Mastitis with permanent damage to tissue of udder

LESSIONS

- Thrombosis of skin vessels followed by cutaneous infarction and sloughing particularly on legs, leading to festering sores; joint and tendon inflammation from secondary infection
- Ulcerative lesions in mucosa of respiratory and digestive tract
- Reddish, haemorrhagic to whitish lesions in lungs
- Nodules and interlobular oedema in lungs
- Permanent damage to skin

SAMPLES TO TAKE

- Nodules and scab for histological and ultrastructural examination
- Lung; lymph nodes

DIFFERENTIAL DIAGNOSIS

- Allergies; Urticaria; Dematophilosis (Streptothricosis); Bovine ephemeral fever; Cattle grubs; Vesicular disease; Photosensitization; Besnoitiosis; Sweating sickness of calves; Pseudolumpy skin disease (Herpes mamillitis)

ACTIONS TO TAKE

Follow Standard Operation Procedures for an emergency outbreak investigation.



PESTE DES PETITS RUMINANTS (PPR)



Mucopurulent nasal discharge



Mucopurulent ocular discharge



Necrosis of tongue epithelium



Necrosis of tongue epithelium



Erosion on the gums and dental pads



Intestinal haemorrhages ("Zebra striping")

ACTIONS TO TAKE:

- Examine the whole flock thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Collect blood in EDTA or Heparin and Serum (whole blood, decant and obtain serum)
 - (ii) Gum debris in PBS, tears (swabs), lymph node, spleen, lungs
- Isolate sick animals from the herd and prevent movement
 - Submit the duly filled form and samples to the DVO
 - Seek further advice from your supervisor

5. PESTE DES PETITS RUMINANTS (PPR)

Kiswahili name: *'Sotoka ya mbuzi na kondoo'*

CAUSATIVE AGENT

A paramyxovirus in genus Morbillivirus antigenically very similar to rinderpest virus.

TRANSMISSION

Requires close contact; virus present in ocular, nasal, and oral secretions as well as faeces.

Most infections through aerosols from sneezing and coughing animals; fomite transmission can occur.

Animals may be infectious during incubation period, but there is no known carrier state.

EPIDEMIOLOGY

Species Affected: Primarily goats and sheep; infected cattle and pigs asymptomatic and are not reservoirs.

Morbidity and Mortality:

- Morbidity and mortality up to 100%; also can have high morbidity with mortality closer to 50%
- Goats generally more susceptible than sheep

CLINICAL SIGNS

Incubation period: The incubation period can range from 3 to 10 days, 4–5 days being typical.

Clinical signs: Acutely febrile, highly contagious disease with oral erosions and/or gastrointestinal signs

- Sudden fever lasting 5–8 days before animal either dies or begins to recover; can have subacute and chronic forms with inconsistent signs developing over 10–15 days
- Restlessness and inappetance; necrotic stomatitis common and can be severe
- Serous to mucopurulent nasal discharge; may remain mild or progress to a severe catarrhal exudate that crusts over, blocking nostrils leading to respiratory distress; small necrotic areas on nasal mucosa
- Congested conjunctiva crusting on medial canthus; profuse catarrhal conjunctivitis with matted eyelids
- Profuse diarrhea, severe dehydration; emaciation, dyspnea, hypothermia; death may occur 5–10 days
- Bronchopneumonia with coughing common late in the disease
- Abortion may occur in infected pregnant animals

LESSIONS

- Rinderpest-like; inflammatory and necrotic lesions in oral cavity and throughout gastrointestinal tract.
- Unlike rinderpest, respiratory system affected; small erosions, petechiae in nasal mucosa, turbinates, larynx, trachea. Bronchopneumonia with consolidation, atelectasis; pleuritis and hydrothorax.
- Carcass generally emaciated; Conjunctivitis and erosive stomatitis; necrotic lesions inside surface of lower lip, adjacent gum, commissures, tongue, hard palate, pharynx and upper oesophagus.
- Lymph nodes generally congested, enlarged and edematous; spleen may be enlarged and congested.
- Rumen, reticulum, and omasum rarely have lesions; abomasum with outlined hemorrhagic erosions.
- In small intestine, small streaks of hemorrhages and sometimes erosions in first portion of duodenum and the terminal ileum. Peyer's patches have extensive necrosis and sometimes severe ulceration.
- Most severe lesions in large intestine: congestion at ileo-cecal valve, ceco-colic junction and rectum;
- "Zebra stripes" of congestion are often seen in the posterior part of the colon on the mucosal folds.

SAMPLES TO TAKE

EDTA blood, clotted blood or paired serum samples (2 weeks interval), gum debris, nasal and lachrymal discharge swabs; mesenteric lymph nodes, spleen; lung, tonsils and sections of ileum and large intestine. Samples shipped fresh on ice (not frozen) within 12 hours. Samples to be collected in acute phase of disease (with clinical signs).

DIFFERENTIAL DIAGNOSIS

Rinderpest, Contagious caprine pleuropneumonia, Bluetongue, Pasteurellosis, Contagious ecthyma, FMD, Heartwater, Coccidiosis and Mineral poisoning.

ACTIONS TO TAKE

- Follow the Standard Operation Procedures for an emergency outbreak investigation
- Quarantine affected area
- Destroy exposed or infected animals and burn or bury carcasses
- Apply disinfectants (PPR virus can be killed by most common disinfectants e.g. phenol, or sodium hydroxide 2% for 24 hours) as well as alcohol, ether and detergents

NB: PPR virus can survive for long periods of time in chilled or frozen tissues.



NEWCASTLE DISEASE (ND)



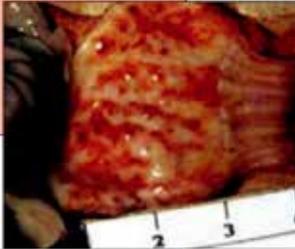
Twisted necks
(torticollis)



Chicken with leg
paralysis



Haemorrhages in
trachea



Haemorrhages in
proventriculus



Haemorrhages in
proventriculus



Cloacal
haemorrhages

ACTION TO TAKE:

- Examine the whole flock thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Live birds: trachea and cloaca Swabs
- (ii) dead birds: Tissue samples including trachea, lung, spleen, cloaca and brain transported on ice
- Isolate flock and prevent movement
- Submit the duly filled form and samples to the DVO
- Seek further advice from your supervisor

6. NEWCASTLE DISEASE (ND)

Kiswahili name: *Mdondo*

CAUSATIVE AGENT

- Newcastle disease viruses are a group of Avian paramyxovirus type 1 (APMV-1) in the genus Rubulavirus, family Paramyxoviridae. There are nine avian paramyxovirus serotypes designated APMV-1 to APMV-9
- Newcastle disease virus strains are grouped into pathotypes based on their clinical signs and virulence
- These pathotypes include:
 - Lentogenic or asymptomatic enteric: causing mild or subclinical respiratory signs
 - Mesogenic causing respiratory and occasional neurologic disease with low mortality
 - Velogenic, the most virulent pathotype with high mortality rates. It is divided into two forms: Neurotropic causing respiratory and neurologic lesions and viscerotropic causing hemorrhagic intestinal lesions

TRANSMISSION

- Direct contact with faeces, respiratory discharges, contamination of the environment including food, water, equipment, and human clothing. Virus is shed during incubation period and for a short time during recovery
- Some psittacine species can shed the virus intermittently for a year or more
- Virus is present in all parts of the carcass of an infected bird
- Viruses can survive for long periods in environment, especially in faeces
- Virus readily destroyed by heat (80°C for 1 minute), soaps, detergents, hypochlorites, alkalis, gluteraldehyde

EPIDEMIOLOGY

Species Affected:

- Many avian species are affected by Newcastle disease viruses
- Of poultry, chickens are the most susceptible; ducks and geese are the least
- Inapparent infections and carrier states can occur in psittacine and some wild bird populations. Mortality: Very high (up to 100% of affected birds)

Distribution: The disease is currently widespread in the country

CLINICAL SIGNS

Incubation period: 2–15 days. In chickens with the velogenic form, an incubation period of 2–6 days is common.

Clinical signs

- Viscerotropic velogenic ND: Sudden decrease in egg production; marked depression, loss of appetite, increased respiration, swollen heads, blue combs, profuse greenish diarrhea, dehydration and collapse; death in 2 days. Surviving birds with twisted necks (torticollis)
- Neurotropic velogenic ND: Severe respiratory and nervous signs including coughing, gasping, head tremors, wing and leg paralysis, twisted necks. Depression, loss of appetite, drop in egg production
- Mesogenic ND: Mainly respiratory signs with coughing but not gasping. Depression, loss of weight, decrease in egg quality and production, for up to 3 weeks. Nervous signs latter in course of infection
- Lentogenic ND: Symptoms mild or absent; mild respiratory signs; drop in egg production

Lesions

- Oedema of interstitial tissue of neck especially near the thoracic inlet and congestion and sometimes haemorrhages on tracheal mucosa
- Petechiae and small ecchymoses and/or foci of necrosis on mucosa of the proventriculus especially around orifices of the mucous glands. Additional lesions: oedema, haemorrhages, necrosis, or ulceration

SAMPLES TAKEN

For virus isolation swab from trachea and cloaca of live birds, or tissue samples from dead birds including trachea, lung, spleen, cloaca and brain. Faeces can also be used for culture. Samples should be transported on ice.

DIFFERENTIAL DIAGNOSIS

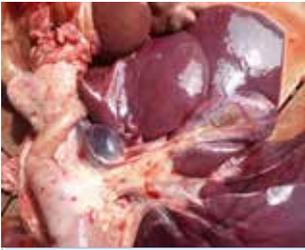
Fowl cholera; Highly pathogenic avian influenza; Infectious laryngotracheitis, Coryza; Fowl pox (diphtheritic form); Psittacosis (chlamydiosis in psittacine birds); Mycoplasmosis; Infectious bronchitis.

ACTION TO TAKE

Follow Standard Operation Procedures for an emergency outbreak investigation



AFRICAN SWINE FEVER (ASF)



Haemorrhages in omentum/peritoneum and gall bladder



Cyanosis



Enlarged spleen



Enlarged and haemorrhagic kidneys

ACTIONS TO TAKE:

- Examine the whole litter thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Collect blood in EDTA and whole blood decanted to obtain serum;
- (ii) Collect lymph nodes, spleen, tonsils and kidneys

- Detailed epidemiological investigation, with tracing of possible source of infection
- Slaughter of all pigs and proper disposal
- Clean and disinfect thoroughly
- Control of pig movements
- Surveillance of infected and surrounding area
- Submit the duly filled form and samples to the DVO
- Seek further advice from your supervisor

7. AFRICAN SWINE FEVER (ASF)

Kiswahili name: *'Homa ya Nguruwe'*

CAUSATIVE AGENT

African Swine Fever virus of the genus Asfivirus.

TRANSMISSION

Direct contact with infected/dead domestic or wild pigs. Virus present in blood, tissues, secretions and excretions of sick and dead animals. Indirect contact, through ingestion of contaminated material (e.g. food waste, feed, or garbage); contaminated fomites, or biological vectors (soft ticks of the genus *Ornithodoros*);

EPIDEMIOLOGY

Species Affected: Primarily domestic pig; wild pigs (warthogs and bush pigs) are usually asymptotically infected and act as reservoir.

Morbidity and Mortality:

- Morbidity and mortality up to 100% for acute form; also can have high morbidity with mortality 30 – 70 for sub acute form

CLINICAL SIGNS

Incubation period: The incubation period can range from 4–19 days; acute form 3–4 day

Clinical signs:

- Fever (40.5–42°C)
- In white skinned pigs reddening of the skin of tips of ears, tail, distal extremities, ventral aspects of chest and abdomen and may develop a bluish tinge (cyanosis)
- Anorexia, listlessness and incoordination within 24–48 hours before death
- Increased pulse and respiratory rate
- Vomiting, diarrhoea (sometimes bloody) and eye discharges may exist
- Abortion may occur in pregnant sow
- Death within 6–13 days, or up to 20 days
- Survivors are virus carriers for life

Note: Less intense signs for subacute and chronic form of the disease

LESSIONS

- Pronounced haemorrhages in the gastrohepatic and renal lymph nodes
- Petechial haemorrhages of the renal cortex, also in medulla and pelvis of kidneys
- Congestive splenomegaly
- Oedematous areas of cyanosis in hairless parts
- Cutaneous ecchymoses on the legs and abdomen
- Excess of pleural, pericardial and/or peritoneal fluid
- Petechiae in the mucous membranes of the larynx and bladder, and on visceral surfaces of organs
- Oedema in the mesenteric structures of the colon and adjacent to the gall bladder; also wall of gall bladder

SAMPLES TO TAKE

EDTA blood, serum samples mesenteric lymph nodes, spleen; tonsils and kidney kept at 4°C.

DIFFERENTIAL DIAGNOSIS

- Classical swine fever (CSF or hog cholera)
- Porcine reproductive and respiratory syndrome (PRRS)
- Erysipelas
- Salmonellosis
- Aujeszky's disease
- Pasteurellosis
- other septicaemic conditions



CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)



**Distended neck signs
of labour breathing**



**Thick nasal
discharges**

ACTIONS TO TAKE:

- Examine the whole flock thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Collect blood in EDTA or Heparin and whole blood decanted to obtain serum;
 - (ii) Lung and lymph node
- Isolate sick animals from the flock and prevent movement
 - Submit the duly filled form and samples to the DVO
 - Seek further advice from your supervisor

8. CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)

Kiswahili name: 'Homa ya Mapafu ya Mbuzi'

CAUSATIVE AGENT

Mycoplasma capricolum subsp. *capripneumoniae* (Mccp).

TRANSMISSION

Infected aerosol and droplet. A carrier state is likely but not proven.

EPIDEMIOLOGY

Species Affected:

Goats are the primary hosts, sheep may be affected in outbreaks affecting mixed herds.

Morbidity and Mortality:

In naive flocks, the morbidity rate may reach 100% and the mortality rate can be as high as 80%

CLINICAL SIGNS

Incubation period: The incubation period can range from 4–19 days; acute form 3–4 day

Clinical signs:

Peracute: affected goats may die within 1–3 days with minimal clinical signs

Acute:

- Fever ((41–43°C), fatigue and anorexia
- Coughing and labored breathing
- Extended neck, lowered head, open mouth front legs wide apart
- Profuse salivation and frothy nasal discharge
- Abortion in pregnant animals
- Death within 7-10 days

Chronic:

- Chronic cough and labored breathing
- nasal discharge and debilitation

LESSIONS

- Fibrinous pleuritis; Straw-coloured fluid in thorax with encapsulation of acute lesions and numerous adhesions to the chest wall.
- Lobar pneumonia with red hepatization
- Marbled appearance of lung lobules due to thickening of interlobular septa
- Enlarged regional (bronchial) lymph nodes

SAMPLES TO TAKE

- Thoracic fluid; Diseased lung and regional lymph nodes on ice during transportation
- Additional samples in 10% formalin for histopathology; Serum for CFT

DIFFERENTIAL DIAGNOSIS

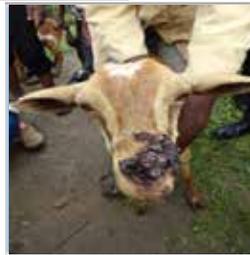
- Peste des petits ruminants, to which sheep are also susceptible;
- Pasteurellosis, which can be differentiated on the basis of distribution of gross lung lesions;
- Contagious agalactia syndrome, also known as Mastitis, arthritis, keratitis, pneumonia and septicaemia syndrome



SHEEP POX AND GOAT POX



Skin Nodules – widely spread



Necrotic papules around the mouth and nose

ACTIONS TO TAKE:

- Examine the whole flock thoroughly
- Fill in the outbreak report form

SAMPLES:

- (i) Collect blood in EDTA or Heparin and whole blood decanted to obtain serum;
 - (ii) Skin biopsies/lesions, lung and other affected tissues
- Isolate sick animals from the flock and prevent movement
 - Submit the duly filled form and samples to the DVO
 - Seek further advice from your supervisor

9. SHEEP POX AND GOAT POX

CAUSATIVE AGENT

Poxviridae, genus *Capripoxvirus*.

TRANSMISSION

Aerosol after close contact with severely affected animals containing ulcerated papules on the mucous membranes. Indirect transmission may occur by contaminated implements, vehicles or products (litter, fodder).

EPIDEMIOLOGY

Species Affected: All breeds of domestic and wild sheep and goats

Morbidity and Mortality:

Morbidity 70–90% and mortality 5–10% in endemic areas

CLINICAL SIGNS

Incubation period: Incubation period is 8–13 days

Clinical signs:

- Fever (above 40°C)
- papules on the mucous membranes of the eyes, eyelids, mouth, anus, and prepuce or vagina
- rhinitis, conjunctivitis and enlargement of all superficial lymph nodes, especially prescapular lymph nodes
- mucopurulent discharge as a result of ulcerating papules on the nose
- laboured breathing due to pressure on the upper respiratory tract from the swollen retropharyngeal lymph nodes draining developing lung lesions.
- papules become necrotic from vascular thrombosis and ischaemic necrosis and form scabs in the next 5–10 days, which persist for up to 6 weeks, leaving small scars
- secondary pneumonia is common

LESSIONS

- Congestion, haemorrhage, oedema, vasculitis and necrosis involving epidermis, dermis and sometimes musculature
- Enlarged lymph nodes draining infected areas
- Pox lesions (papules) on mucous membranes of the eyes, mouth, nose, pharynx, epiglottis, trachea, lungs, on the rumenal and abomasal mucosae, and on the muzzle, nares, in the vulva, prepuce, testicles, udder, and teats

SAMPLES TO TAKE

Full skin thickness biopsies; vesicular fluid if available; scabs; skin scrapings; lymph node aspirates; whole blood collected into heparin or EDTA; paired sera in live animals:

At necropsy: skin lesions; lymph nodes; lung lesions; histology: full set of tissues, especially those with lesions

DIFFERENTIAL DIAGNOSIS

- Contagious ecthyma (contagious pustular dermatitis or orf)
- Insect bites
- Bluetongue
- Peste des petits ruminants
- Photosensitisation
- Dermatophilosis
- Parasitic pneumonia
- Caseous lymphadenitis
- Mange

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Important note

Disease photos and narratives used were partly from the following links

<http://www.oie.int/doc/ged/D13953.PDF>

<http://www.fao.org/docrep/004/x8060e/x8060e00.htm>

<http://www.nadis.org.uk/disease-a-z/pigs/african-swine-fever/>

<http://www.afrivip.org/sites/default/files/CBPP/diagnosis.html>

