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M.VET

ANIMAL HEALTH AND PRODUCTION FOR

DEFINITIONS TC "A: DEFINITIONS" \f C \l "1"

(i) **ENZOOTIC DISEASES:** Refers to diseases present (endemic) among animals in a particular region, country or locality, for example, FMD, Newcastle disease and Anaplasmosis in Tanzania.

(ii) **SPORADIC DISEASES**: refers to a disease occurring in single cases here and there.

(iii) **PANZOOTIC DISEASE**: Means a disease that affects all animals in an area.

(iv) **EPIZOOTIC DISEASE**: Is a term applied to a disease that appear infrequently affecting a large number of animals in a large area of land at the same time and spreads with great rapidity e.g. FMD and Rinderpest.

(v) **EPIDEMIOLOGY**: This is a systemic characterization and explanation of patterns of disease, and the use if this information in the resolution of health problems.

(vi) **INFECTIOUS DISEASE**: The disease that is caused by living organisms, often microorganism, that infects animals and are transmissible from one infected animal to another. The Microorganisms are bacteria, Viruses, Protozoa, Mycoplasm etc

(vii) **NON-INFECTIOUS DISEASE**: Diseases that are not transmissible from one animal to another, they are caused by a number of factors, such as poisons, abnormal growth of the body (cancer or tumor), inadequate or excessive nutrition and secretion.

(viii) **MIXED DISEASE**: Refers to disease caused by more than one agent in the same animal.

(ix) **QUARANTINE:** Implies governmental regulations for the prevention of the spread of infectious disease by which an animal or animal products, which have come from infected countries or areas, are detained at the frontiers or ports of entrance, or at other official centers, for a period of isolation, before being allowed to mix with stock of the country.
OR is the physical separation of sick animals from healthy ones, or the placing of restraints on the movements of exposed or infected animals or its items that may have contaminated.

(x) **NOTIFIABLE DISEASES**: Are those which, when they break out upon farm premises, must be notified to the police or government department concern. For example: Anthrax, CBPP, Rabies, African Swine Fever etc.

BOVINE DISEASES TC "BOVINE DISEASES" \f C \l "1"

A: BACTERIAL DISEASES TC "B: BACTERIAL DISEASES" \f C \l "1"

1. MASTITIS TC "1. MASTITIS" \f C \l "2"

The term Mastitis refers to inflammation of the parenchymal tissue of the udder (mammary gland) regardless of the cause.
Mastitis as a disease is characterized by physical, chemical and pathological changes in the glandular tissues of the mammary gland and milk.

ETIOLOGY:
Many infective agents have been implicated as causes of Mastitis, and these are:
Bacteria:

Streptococcus spp
Staphylococcus spp
Corenebacterium (actinomyces) pyogenes
Klebsiella spp

Enterobacter aerogenes
Mycobacterium spp
Bacillus cereus
Pasteurella spp
Mycoplasma bovis
Nocardia spp
Escherichia coli
Leptospira spp

Fungus
Trichosporon spp
Aspergillus spp

Yeasts
Candida spp
Cryptococcus neoformans

Algae
Prototheca trispora
Prototheca zopfii

ROUTE OF INFECTION
The microorganism gain entry into the animal' s body through:

(i) Teat canal
(ii) Udder injury: trauma existing on the skin that allow organism from environment to enter to the parenchyma.
(iii) Haematogenous route: from blood vessels to the parenchyma (e.g. M. bovis)

FACTORS THAT FACILITATE THE DISEASE TO OCCUR (PREDISPONSING FACTORS)
House hygiene: when animals lay down in dirty houses/ beddings
Udder hygiene: dirty udder predisposes the animal to get mastitis
Milker' s hand or machine: dirty hands or machine facilitate disease occurrence.
Efficiency of milking personnel and/ or machines.

Milking hygiene: before and after milking, clean the udder using warm water, then dry by piece of cloth or towel.
Udder and teat injury:
Occurrence of other diseases e.g. FMD
Susceptibility of animals: This is related to:

- Stage of lactation (2months more susceptible than other period)
- Age of cow (older cows are more susceptible than young cows)
- Level of inherited resistance (related to teat shape and anatomy of the teat canal)
- Immunological status of animal (Leukocytic status of each mammary gland prior infection)

NB: The development of disease depends on:
Bulk of infection in the environment including infected quarters
Efficiency of milking personnel and/or milking machines
Susceptibility of the cow.

PATHOGENESIS

The mechanisms by which Mastitis pathogens produce the lesions of the disease are complex. However, can be explained in three stages:

Invasion: where the organisms pass from the exterior of the teat to the milk inside the teat canal.

Infection: when the organisms multiply rapidly and invade the mammary gland.

Inflammation: the stage at which clinical Mastitis appears or a greatly increased leukocytes count is apparently in the milk.

CLINICAL SIGNS

The clinical findings in Mastitis include:
Abnormalities in secretion (Milk):

- Discoloration, Clots, Flakes, Pus etc

(ii) Abnormalities of size, consistency and temperature of mammary gland:

- Fibrosis (firmer feel that other parts)

- Inflammatory swelling
- Atrophy of mammary tissues

(iii) Systemic reaction:
- This comprises of toxemia, fever, general depression, and anorexia may or may not be present.

The clinical forms of mastitis are usually classified according their severity:

i) **Peracute**: Severe inflammation of the quarter(s) with a marked systemic reaction
ii) **Acute**: severe inflammation without a marked systemic reaction
iii) **Subacute**; mild inflammation with persistent abnormality of the milk.
iv) **Chronic**: recurrent attacks of inflammation with little changes in milk.
v) **Subclinical Mastitis**: When there is evidence of inflammation (high somatic cell count in the milk) without any visible abnormality of the milk or under.

DIAGNOSIS

Diagnosis of Mastitis depends on clinical signs and laboratory examination of milk. The methods used include:

(i) Strip cup: Shiny, black plate
(ii) California Mastitis Test (CMT)
(iii) Cell Count in Milk:
- Direct Microscopic Somatic Cell Count
- Electronic Somatic Cell Count
- Bulk Milk Cell Count
- Individual Cow Cell Count

TREATMENT

The treatment of Mastitis can be highly effective in removing infection from the quarter and returning the milk to normal composition. This treatment can be through:

(i) **Parenteral (Systemic) treatment**: For all cases with marked systemic reaction, to control or prevent the development of a systemic or bacteremia, to assist in the treatment of the infection of the infection in the gland, and when the gland is badly swollen and intramammary antibiotic is unlikely to diffuse properly.

(ii) **Udder (Intramammary) infusions**: Strict hygiene is necessary during treatment to avoid the introduction of bacteria, yeast and fungi into the treated quarter. Completely empty the quarter before infusion of the drug. After an intramammary infusion avoid emptying the gland, strictly obey the manufacturers instructions.

(iii) **Direct Intraparenchymal injection**: This is done for grossly swollen udder.

(iv) **Drying off chronically affected quarter:** this is by using 3% silver nitrate (30 – 60mls) or 20 mls of 5% copper Sulphate.

(v) **Supportive Therapy:** This include, Fluid therapy (Lactated Ringer' s solution, Lactate, Acetate etc), Antihistamine in extensive udder damage e.g. Glucocorticoids in treatment of Toxemia, Non- Steroidal – Anti – Inflammatory Drug (NSAID), application of cold to reduce absorption of toxins.

CONTROL

(i) **Dry period treatment**: This is done at the end of the last milking before the cow is turned out. It is a good prophylaxis especially in *Staphylococcus aureus*.

(ii) **Teat dipping or spraying**: This is done after milking. Teat dipping solutions includes: Odophor solution (1% Chl), Hypochlorite solution (4% Chl), Chlorhexidine (Hibitane).

(iii) **Udder washing and teat stripping**: Stripping of a few squirts milk out before premilking wash will reduce refluxing milk from the teat cup into the udder.

(iv) **Other control measures**: Creating awareness to farmers, improve house hygiene, improve milking machine, improve beddings (wet sawdust, shaving and straw predispose cows to Coliform mastitis), infected cows should be milked last and milk given to pigs or dogs, and regular treatment of sores on udder.

(v) **Nutrition and Inheritance**:

- It is commonly believed that the incidence of mastitis increases when cattle go on to lush pastures or are fed diet high in protein.
- Genetic variations in resistance to Mastitis have been proven with regard to *Str. Agalactiae* Mastitis. One of the inherited characteristics, which may affect susceptibility to Mastitis, is teat shape, the characteristic is inherited and cows with cylindrical teats become affected more commonly than cows with funnel shaped teats.

2. ANTHRAX TC "2. ANTHRAX" \f C \l "2"

This is an acute, febrile disease of all warm – blooded animals, including man. The disease is characterized by septicemia and sudden death with the exudation of TARRY BLOOD from the body orifices of the cadaver.

ETIOLOGY

The causative agent is *Bacillus anthracis,* a gram positive, non-motile spore forming bacterium.

EPEDEMIOLOGY

i. Anthrax outbreaks are associated with **NEUTRAL or ALKALINE** soil.
ii. Outbreaks tend to occur in association with marked climatic or ecological changes, such as heavy rainfall, flooding or draught.
iii. Outbreaks originate from a soil – borne infection with different severity within species; the disease is common in:

Cattle and Sheep, Less frequently in Goats, Horses and Man, Swine, Dogs and Cats are relatively resistant.

iv. **Transmission**: The spread of the organism within the area is through: Streams (Water), Insects (Biting flies), Dogs and other Carnivores, Wild birds, and fecal contamination from infected animals.

v. Introduction of infection into a new area is usually through: Ingestion of contaminated animals' products such as bone meal, fertilizers, hides, hair and wool, and contaminated concentrates or forages.

In man Anthrax occurs in three forms:

As localized cutaneous lesions (Malignant carbuncles) from contact of broken skin with infected bloody tissues.

Highly fatal inflammation of the mediastinal lymph nodes – through spore inhalation when handling contaminated wool or hair.

Intestinal Anthrax – from consumption of undercooked meat.

PATHOGENESIS

Upon ingestion/ inhalation/ through skin the bacteria get entry into the animal' s body.

The bacteria are taken by motile phagocytes to the local lymph nodes

In the lymph nodes, multiplication of bacteria occurs

Then the bacteria are carried through the lymphatic vessels to the blood stream

From blood stream, septicemia occurs resulting into massive invasion of all body tissues

Then production of lethal toxins occurs in tissues

Toxins cause edema and tissue damage that lead to death of the particular animal

CLINICAL SIGNS

The incubation period after field infection varies from 1 - 2 weeks

Cattle and Sheep/Goat:

Peracute form:

Sudden death (without premonitory signs); After death, discharge of blood from the nostrils, mouth, anus and vulva occurs.

II. **Acute form (lasting for 48hrs)**

- ❖ Severe depression and listlessness
- ❖ Fever (approaching $42^{O}C$)
- ❖ Deep and rapid respiration
- ❖ Congestion and sometimes haemorrhagic mucosae
- ❖ Anorexia and ruminal stasis
- ❖ Abortion in pregnant cows
- ❖ In milking cows: reduced milk, blood stained or deep yellow in color milk.
- ❖ Diarrhoea and dysentery

III: **Chronic Form:**

Localized, subcutaneous, edematous swelling on the ventral neck, thorax and shoulder.

2. **Pigs:** may be peracute or acute

Acute; swelling of the throat or face, bloodstained froth, and petechial haemorrhages in th2

3. **Horses**: Anthrax in horse is always acute:
   - Ingestion leads to septicaemia, enteritis and colic
   - Insect bites leads to edematous swelling

POSTMORTEM FINDINGS: In all cases of suspected Anthrax the carcass should not be opened.

Absence of rigor mortis

All natural orifices usually exudates a dark, tarry blood which do not clot

Putrefaction and bloating rapid (gaseous decomposition quickly)

When carcass opened: presence of haemorrhages throughout the body tissues, acute spleenomegally (acute swelling of spleen, dark red or black, soft, semifluid spleen), presence of bloodstained serous fluid in body cavities, and severe enteritis (small intestines). Liver, kidney and lymph nodes are usually congested and enlarged.

DIAGNOSIS

a. History of sudden death
b. Clinical findings
c. Microscopic examination of smears (blood; small amounts of blood collected aseptically from a superficial vessel such as ear vein or edema fluid).Blood is stained wit Giemsa or Methylene blue.
d. Experimental animal inoculation (eg guinea-pig or mouse)

DIFFERENTIAL DIAGNOSIS

Lightning strike:- history of electrical storms, presence of thunderstorm, rain and lightings

Peracute blackleg:- Restricted to young animals and crepitating swelling is present.

Acute Leptospirosis:- presence of haemoglobinuria

Peracute lead poisoning:- obvious nervous signs

Acute bloat:- gaseous distension of abdomen, no fever, and history of feeding lush psture

TREATMENT: Early treatment is essential to save diseased animal.

(i) Antibiotics: Penicillin, Oxytetracycline, Streptomycin
(ii) Antianthrax Serum (Intravenous injection)

CONTROL:

Anthrax can be controlled largely by annual vaccination of all grazing animals in the endemic areas and implementing control measures during outbreaks.

(i) Destruction of cadaver and discharges:

This is by burning or burring the cadaver together with bedding and soil contaminated by discharges.

Burial should be at least 2 meters deep with an ample supply of quicklime added.

Disinfections of premises, hides, bones, meal, fertilizer, wool and hair

Quarantine: Segregation of all suspected cases and in-contact animals for at least 2 weeks after disappearance of the disease

Vaccination of Survivors: In enzootic areas annual revaccination of all stock is necessary. Vaccination should be done annually.

3. PASTEURELLOSIS TC "3. PASTEURELLOSIS" \f C \l "2"

Pasteurellosis is caused by pasteurellae that occur in many animal diseases and, although in some instances they act as primary causes, mainly appear as secondary agents.

The most common diseases in animals caused by Pasteurellae are:

a. Pneumonic Pasteurellosis of Cattle (Shipping fever pneumonia or Transit Fever)

b. Septicaemic Pasteurellosis of cattle (Haemorrhagic Septicaemia (HS) or Barbone)

A: PNEUMONIC PASTEURELLOSIS

This a peracute Pasteurellosis caused by Pasteurella species characterized by sudden onset, dullness tachypnoea (increased rate of breathing), anorexia and pyrexia.

ETIOLOGY

Pasteurella haemolytica

Biotype A serotype 1: causing about 75% of all cases
Biotype A serotype 2: causing about 10% of all cases

EPIDEMIOLOGY

Pasteurellae are not primary agent of the disease, they cause the disease in already dearranged/infected lungs and most after Parainfluenza or I BR Virus infection

PREDISPOSING FACTORS

(Stress due to:

- Weaning of calves
- Mixings groups of animals
- Changes of diet
- Exposure to inclement weather (chilling/ cold weather)
- Transport.
- Poor ventilation (over crowding)

NB. The incidence is high in new purchased animals and weaned animal especially .in cold weather.

TRANSMISSION

By inhalation of infected droplets coughed up or exhaled by infected animal or recovered carriers in which the infection persists in the upper respiratory tract.

CLINICAL SIGNS

The disease usually develop in the cattle within 1-14 days after they have been stressed

1) Sudden death in calves may be the first sign of outbreak, coughing which elicited by walking
2) Anorexia
3) Mucopurulent nasal discharge (a crusty nose)
4) Ocular discharge and conjunctivitis
5) Fever 40 - 41˚c
6) Tachypnoea (increase breathing rate)

POST MORTEM LESIONS

1) Catarrhal bronchitis and bronchitis pneumonia
2) Serofibrinous pleurisy / pleuritis
3) Fibrinous pericarditis

DIFERENTIAL DIAGNOSIS

1) CBPP: deep cough, elbow out and grunting respiratory
2) Lung worm pneumonia: coughing, loud breath sounds crackles, not responding to antibiotic
3) Infection bovine rhinotracheitis: rhinits and loud coughing

TREATMENT

Oxytetracycline
Trimethoprime - Sulphamethoxazole
Penicillin
Erythromycin
Anti - inflammatory agent (e.g. corticosteroids)

CONTROL

By avoiding stress to the animals

B: HAEMORRHAGIC SEPTICAEMIA (HS)

This is an acute disease, principally of cattle, water buffaloes and camels, and to much smaller extent pigs and horses.

AETIOLOGY: The disease is caused by two Serotypes of *Pasteurella multocida: P. multocida* type B and *P. multocida* type E.

EPIDEMIOLOGY

The disease occurs with worst epidemics during rainy season, although may occur throughout the year, and is most common in river valleys and cold areas (Occurs in outbreak during period of environmental stress).

Disease outbreak results from (predisposing factors)

a) Exhaustion e.g. long traveling
b) Chilling e.g. change of environment
c) Malnutrition
d) Change of diet
e) Vaccination against other diseases

Spread occurs by the ingestion of contaminated foodstuffs either by coughs or nasal discharges contaminating water and pasture.

Infection originates from clinically normal carrier or clinically sick animas or possibly tick and biting in\sects (saliva of affected animals contains large number of Pasteurella, and carrier animals harbor in tonsils and nasopharyngeal mucosa).

CLINICAL SIGNS

The disease manifest itself as peracute or acute:

1. **Peracute form**: Sudden death without premonitory signs
2. **Acute form:** Death occurs in about 24 hrs

(i) Sudden onset of fever (41- 42^{o}C)
(ii) Profuse salivation
(iii) Severe depression
(iv) Submucosal petechiation

(v) Localization may occur in subcutaneous tissue, resulting in the development of hot, painful swelling about the throat, dewlap, brisket or perineum.
(vi) Severe dyspnoea may occur if the respiration is obstructed
(vii) In later stages of an outbreak, some affected animals develop signs of pulmonary or alimentary involvement
(viii) Recovery is rare especially in Buffaloes

POST MORTEM FINDINGS

a) Blood-tinged fluid is often found in the pericardial sac and in the thoracic and abdominal cavities
b) Petechial haemorrhages are prominent in the pharyngeal and cervical lymph nodes and serosae
c) Haemorrhagic gastroenteritis
d) Edematous lung and lymph nodes

DIAGNOSIS

History of the animal (epidemiology)
Clinical signs and PM lesions
Laboratory confirmation: samples are saliva (affected animal) and blood from the heart and portion of spleen from dead animal

DIFFERENTIAL DIAGNOSIS

Anthrax (Tarry black discharge)
Blackleg (Crepitation on muscle and edematous swelling)
Acute Leptosipirosis (haemoglobinuria)

TREATMENT

Sulphonamides injection
Oxytetracycline injection

CONTROL

- Annual vaccination (immunity after vaccination lasts for at least 12 months)

4: BRUCELLOSIS TC "4: BRUCELLOSIS" \f C \l "2" (SYNONYMUS: Bang' s Disease OR Malta Fever)

This is the contagious disease primarily affecting cattle and characterized by abortion, orchitis and infection of the accessory sex glands in male animals.

OETIOLOGY

Five main species of organism cause disease in animals and human being:

a) *Brucella abortus* Affects cattle, horses, and in man cause undulant fever & horses
b) *Br. Suis* Affects swine, horses and Man
c) *Br. Melitensis* Affects Goats, and cause Mediterranean fever in man
d) *Br. Ovis* Affect sheep
e) *Br. Canis* Affect dogs

TRANSMISSION

- Organisms are shed in milk and uterine discharges if mammary glands are affected respectively.
- The organisms are transmitted through ingestion, inhalation, penetration of the intact skin and conjunctiva, and contamination of the udder during milking.
- Bulls get the infection from infected cows but do not usually transmit infection from infected to non-infected cows mechanically. However, semen from AI transmits the infection.
- Man get the infection through drinking of infected milk, handling of meat / udder / uterus (occupational disease)

- The spread of the disease from one herd to another and from one area to another is almost always due to movement of infected animals from an infected herd into a non-infected susceptible herd.

CLINICAL SIGNS

The clinical findings are dependant upon the immune status of the herd:

(i) Abortion (STORM): Occurs at late stage of pregnancy (65% abort, of these, 65% abort only once, 23 twice and the least three times). In subsequent pregnancies the fetus is usually carried to full term although second or even third abortions may occur in the same cow.
(ii) Retention of placenta, metritis and reduced milk yield.
(iii) In sows, there is irregular estrus, small litters and abortion
(iv) In male animals: Orchitis and epididymitis (One or both scrotal sacs may be affected with acute, painful swelling to twice normal size)
(v) In young animals: Arthritis (swelling of knees) may occur

POSTMORTEM FINDINGS

(i) Edematous placenta (placentitis)
(ii) Necrosis of the cotyledons
(iii) Pneumonia in aborted fetuses (some)
(iv) Cloudy and frequently containing flakes of pus in the amniotic fluid

DIAGNOSIS

(i) History: age of fetuses
(ii) Clinical signs
(iii) Serological tests: CFT, ELISA (Enzyme-Linked Immunosorbet Assay)

DIFFERENTIAL DIAGNOSIS

- ❖ Trichomoniasis (Low MB: 5-30%; mainly infertility, early abortion: 2-4months, pyometra and return to heat after 4-5 months)
- ❖ Vibriosis (low MB:5-20%, abortion 5-6months, and pyometra)

TREATMENT
Not recommended

CONTROL

a) Vaccination:

- ❖ Using *Br. abortus* strain 19 to females calves at age starting 4 – 8 months giving lasting immunity
- ❖ *Br. abortus* 45/20- vaccine is recommended to cows.

b) Eradication on an area basis by test and slaughter policy

c) Hygienic measures:

Isolation or disposal of infected animals

Disposal of aborted fetuses, placenta and uterine contents

Disinfection of contaminated areas

- **5: INFECTIOUS KERATITIS OF CATTLE (SYNONYMUS: PINK EYE, BLIGHT)** TC "*5: INFECTIOUS KERATITIS OF CATTLE (SYNONYMUS: PINK EYE, BLIGHT)*" \f C \l "2"

This is the disease affecting eye in cattle. It is not fatal, but its total economic impact is enormous (huge). In cattle, young animals are most susceptible.

ETIOLOGY
Moraxella (Haemophilus) bovis

TRANSIMISSION

Face flies (*Musca autumnalis)* are responsible for spread of the disease.

The conjunctiva is the probable portal of entry of infection.

PREDISPONSING FACTORS

Season of the year: The occurrence is at maximum during wet season.

Presence of vector: Presence of flies (*Musca spp*)

Dust: Abundant dust normally is associated with the disease due traumatizing effects of dusts

Long grass: Long grasses traumatize the conjunctiva, and thus portal of entry for infection

CLINICAL SIGNS

One or both eyes may be affected

i) Injection (congestion of the cornel vessels
ii) Oedema of the conjunctiva
iii) Copious watery lacrimation
iv) Blepharospasms (frequent blinking of eyes)
v) Photophobia (fearing of light)
vi) Opacity (white to deep red yellow)
vii) Occasionally, animal may become completely blind and those on pasture may die of starvation

DIAGNOSIS

- History
- Clinical signs
- Laboratory confirmation: swabs from conjunctiva sac

DIFFERENTIAL DIAGNOSIS

a) Traumatic injury (conjunctivitis): Foreign matter or evidence of physical injury in the eye.
b) Infectious Bovine Rhinotracheitis (Rhinitis, cough)
c) Rinderpest (Diarrhoea, dysentery and change in temperature)

d) Bovine Viral Diarrhoea /Mucosal Disease (Diarrhoea)

e) Photosensitivity keratitis (Lesions in other parts especially white areas)

f) Thelaziasis (presence of worm or spots)

TREATMENT

蝂* □□衩爀椀愀猀縀過âÂ蝂齕縀槊涡椀搌 匀愀渀猀 鶴匀伀逮Ä̤SÂ＄殀9璉鬖甀搌椀搀挽
ye ointment solution containing Antibiotics

蝂* □□衩爀椀愀猀縀過âÂ蝂齕縀槊涡椀搌 匀愀渀猀 鶴匀伀逮Ä̤SÂ＄殀9璉鬖甀搌椀搀挽
ubconjunctival injection of Antibiotics (2 – 5 mls)

(Chloramphenical, Oxytetracycline, Penicillin-streptomycin mixture and other common
Antibiotics)

CONTROL

Control of some predisposing factors such as dust, fly and long grasses.

6: DISEASES CAUSED BY MYCOBACTERIA SPP TC "**(6) DISEASES CAUSED BY MYCOBACTERIA SPP**" \f C \l "2"

(A) TUBERCULOSIS

Is the chronic contagious disease of animals and man characterized by formation of tubercles particularly in some species of animals

ETIOLOGY.

Mycobacterium spp.

M. bovis Cattle, goat, Pigs, Sheep, Horse & Man

M. avium Birds & Pigs

M. tuberculosis Man and rare in animals.

TRANSMISSION

1. **By air**: Inhalation of contaminated dust particles and droplets leads to lesions in lungs

2. **Ingestion**: Ingestion of contaminated food, raw meat, milk blood leads to alimentary involvement,

CLINICAL SIGNS.

1. **Cattle** (Clinical signs depend on parts of the body affected)
   - Chronic/ persistent cough due to bronchopneumonia
   - Loss of condition **with** pale mucous membrane and staring coat and Low grade fever (39.5 – 40^{0}C)
   - Generalized enlargement of lymph nodes
   - Mastitis; marked indurations and hypertrophy of udder, and amber like fluid in milk.

2. **Pigs**
   - Tuberculous lesions in cervical lymph nodes which may rupture to the exterior
   - Digestive dysfunction leading to emaciation

3. **Sheep and Goat**
   - Bronchopneumonia, which is manifested by cough and terminal respiratory embarrassment.
   - Intestinal ulceration with diarrhea
   - Emaciation.

POST MORTEM PICTURE

Enlarged superficial lymph nodes, containing cheesy foci that may turn into caseous (in acute cases thorax, head and intestines are involved).

DIFFERENTIAL DIAGNOSIS

- ❖ Lung abscess (sudden onset of coughing and auscultation reveals the area)
- ❖ Traumatic pericarditis (pain, death:2wks, gross edema)
- ❖ Chronic CBPP (History and Pm picture)

DIAGNOSIS

Tuberculin test

Intradermal injection of 0.05ml of tuberculin (*M. bovis* culture) Into the skin of the neck.

Calipers are used to measure the reaction; positive reaction constitutes a diffuse swelling at the injection site after 48 -72 hrs.

Serological tests

- − Complementary fixation test
- − Fluorescent antibody test
- − Direct bacteria agglutination
- − Haemogglutination test

Clinical signs: coughing, weight loss etc

TREATMENT

- − Not recommended because of reinfection (zoonosis), cost, and ineffectiveness that may complicate control of the disease in the herd.
- − Isoniazid is effective in treatment.
- − Combination of streptomycin, Para-aminosaliaglic and other acids (as in man)

CONTROL

Test and slaughter policy
Hygienic measures to prevent the spread of infection
Vaccination: BCG vaccines (in human). It is not recommended in animals.
Avoid overcrowding.
Pasteurization of milk for human consumption.

PUBLIC HEALTH: Man is equally susceptible to infection with *M. tuberculosis* and *M. bovis.* Man becomes infected by aerosol route or by drinking unpasteurized tuberculous milk. Meat inspection and condemnation of affected organs or part of the carcass are important weapons for public health. The whole carcass should be condemned if milliary tuberculosis is seen.

B: PARATUBERCULOSIS (Synonyms: JOHNE' S DISEASE)
This is specific, infection enteritis of cattle, sheep and goats characterized by persistent and progressive emaciation in all species affected and by chronic diarrhea, weight loss, debilitation and death with thickening and corrugation of the wall of intestine.

ETIOLOGY:
Mycobacterium paratuberculosis (Johnei); Three strains: - Cattle, Sheep and Goat.

TRANSMISSION.

- Under field conditions, the disease is transmitted principally by ingestion of feed and water contaminated by the feces of infected animals, which are excreting the organism.

- Infection occurs in animals at a very early age, usually under 30 days of age and clinical disease does not occur until 2 – 5 yrs of age. (NB Animals of over 40days are seldom affected).

CLINICAL SIGNS

In cattle, clinical signs do not appear before 2 years of age and are commonest in the 2 - 6 years age group.

1. Emaciation
2. Submandibular edema which disappears as diarrhea develop
3. Fall in milk production.
4. Normal temperature and the animal eats well throughout but thirst is excessive
5. Diarrhoea
   - Continuous or intermittent, Pipe brow-like diarrhea
   - Feces are homogenous and without offensive odor.
   - There is no blood, epithelial debris and Mucus in feces
6. In Sheep and Goats
   - Emaciation and shedding of wool (Sheep)
   - Depression and dyspnoea are evident in goat

PM LESION

- Hypertophy of the lower jejunum, ileum & ceacum, with formation of thick transverse folds
- Enlargement of mesenteric and ileocecal lymph nodes

DIAGNOSIS

1. Characteristic features of clinical Johne' s disease: Chronic diarrhea which do not respond to treatment, Progressive weight loss and emaciation.
2. Serological test (Johnnie' s Test)

3. Fecal culture and biopsy of intestines (but requires 12 – 16 wks incubation period)

DIFFERENTIAL DIAGNOSIS

1. Salmonellosis, Coccidiosis and parasitism (Acute and in Young animals)
2. Secondary copper deficiency (chronic Molybdenum poisoning)
   - Affect large number of animals and respond well to treatment.

TREATMENT

No treatment - cull the animal

Streptomycin - causes only a transient improvement in clinical signs

CONTROL

1. Test and slaughter policy (Johnne' s test)
2. Separate calves from adult animals immediately after birth
3. Vaccination:
   - Vaccination in calves less than 1 month of age

   NB: Control measures are difficult because of carrier animals and lack of effective test for early diagnosis, and long incubation period

7: CONTAGIOUS BOVINE PEUROPNEUMONIA (CBPP) TC "**7: CONTAGIOUS BOVINE PEUROPNEUMONIA (CBPP)**" \f C \l "2"

This is an acute, sub acute or chronic disease of cattle characterized by pneumonia, serofibrinous, pleurisy and edema of interlobular septae of lungs.

ETILOGY: *Mycoplasma mycoides* Var. *Mycoides* (small colony type)

NB: The strain may also affect goats and sheep, but infection does not spread between the two species

TRANSMISSION

Under natural conditions, a number of animals in a group do not become infected, either because of natural immunity or because they are not exposed to a sufficiently large infective dose.

The principal method of spread of this disease is by inhalation of infective droplets from active or carrier case of the disease (Outbreak tends to be more extensive in housed animals and those on transit by train or on foot).

Carrier animals also called, as "**Lunge**r" appears clinically health but has localized infection in the lung in a form of "**Sequestrum**" with a size of pear to an orange. The capsule of the sequestrum under adverse conditions break and the organism escape through the bronchi to the air. Thus the infection spread within the land slowly.

Transplacental infection also occurs.

Outbreaks tend to be more extensive in housed animals and those on transit by train or on foot.

FACTORS FACILITATING THE OUTBREAK OF DISEASE:

Stress due to starvation

Exhaustion (long distance trekking/traveling)

Intercurrent disease

NB: these cause the sequestrum to break down and connect the animal to an active case.

An essential part of the pathogenesis of the disease is THROMBOSIS in the Pulmonary vessels leading to development of Pneumonic lesions.

CLINICAL SIGNS

1) A cute form

a. High fever (pyrexia) up to 40^{o}C; in acute disease the rise of temperature is soon followed by signs of general illness characterized by ***Fall in milk production, dull coat, debility etc***
b. Anorexia and cessation of rumination (Dejected appetite)
c. High respiration.
d. Severe depression, the animal stands apart or lags behind a traveling group.
e. Coughing, first dry later moist.
f. Difficult breathing with extended head
g. Affected animals being disinclined to move, standing with the elbows turned out, the back arched and head extended and lowered.
h. Muco-purulent nasal discharge
i. Edema of lower parts of chest (throat and dewlap)

NB: animal dies within 2 -3 wks after onset of signs. Death results from anoxia and presumably from toxemia.

2) Sub acute form

(a) Cough induced after running; most animals recover but retain the infection in form of sequestrum.
(b) In young calves severe arthritis and synovitis may occur.

POST MORTEM LESIONS

Thickening and inflammation of the pleura often with heavy deposits of fibrin.

The thoracic cavity may contain up to 10 liters of clear yellow or turbid fluid mixed with fibrin flakes.

Affected lobules show various stages of gray and red hepatization (marbled lung)

Interlobular Septa are greatly extended with serofibrinous exudates.

In recovered animals presence of foci and necrotic tissue surrounded by fibrous capsule ‘‘sequestrum’

DIAGNOSIS

1. History of contact with infected animals
2. Clinical signs
3. Pm Changes
4. Complement fixation test
5. Cultural examination

DIFFERENTIAL DIAGNOSIS

Pneumonic Pasteurellosis (Ocular discharge, coughing and conjunctivitis)

Parasitic Pneumonia in Calves (coughing, loud breathing sounds and crackles)

Tuberculosis (enlargement of lymph nodes, loss of condition and chronic coughing)

TREATMENT

Not recommended because of risk of development of carrier state.

Tylosin tartrate is the drug of choice

Others are: Erythromycin, Spiramycin, Sulfadimidine, Organic arsenicals etc

CONTROL

1. Infected animals should be removed from the herd as soon as possible (If possible slaughtered).
2. Serological test to remove the carriers and sub clinical cases.
3. Strict quarantine - last 12 wks after removal of clinical case consistent surveillance (observation) so that clinical cases may be observed
4. Vaccination of all cattle twice in the first year, then annually.

8: FOOTROT TC "8: FOOTROT" \f C \l "2"

(Synonymous: Interdigital necrobacillosis, Foul in the foot)

This is an infectious diseases of cattle characterized by inflammation of the sensitive tissue of the feet and severe lameness.

CAUSES:

1. *Fusobacterium necrophorum*
2. Others*:* *Bacteriodes melaninogenicus*
   Bacteriodes nodosus (Causes Stable footrot disease in cattle and infectious footrot of sheep)

PREDISPOSING FACTORS:

i) Wet humid weather or when condition are wet underfoot
ii) Stony ground/ sharp grave /pasture with course ground.
iii)Breed susceptibility-
- *Bos indicus* (zebu) cattle are much more resistant to infectious Footrot than *Bos taurus* breeds

iv) Soil pH: low pH favors multiplication/survival of bacteria than high pH.

TRANSMISSION

- Discharges from the feet of infected animals are the probable source of infection.
- Infection gains entrance through abrasions to the skin on the lower part of the foot

CLINICAL SIGNS

a. Severe foot lameness appears suddenly usually in one limb only.
b. Moderate systemic reaction with a fever of 39 - 40^{o}C
c. The animal puts little weights on the leg although the limb is carried only when severe joints involvement occurs.
d. Temporary reduction in milk production in cows and bulls show temporary infertility.
e. Swelling of the coronet and spreading of the claws are obvious.

f. Typical lesions occurs in the skin at the top of the interdigital cleft and takes the form of a fissure with swollen, protruding edges. Pus is never present in large amounts but the edges of the fissures are covered with necrotic materials with characterized odor (purulent foul smell)

DIAGNOSIS

Clinical signs: Characteristic site, nature and smell of the lesion, the pattern of the disease ,and the season and climate are usually sufficient to indicate the presence of true footrot.

DIFFERENTIAL DIAGNOSIS

a. Traumatic injury to the bones and joints
b. Puncture by foreign bodies.
c. Bruising of the heels
d. Gross overgrowth of the hoof.
e. Laminitis; may cause lameness but no local foot lesions.
f. Excessive sole wear; animals maintained for longer period on concrete yard. The sole of the foot is worn flat and the walls are separated at the lateral edges.

TREATMENT

1. Parenteral administration of antibiotics or antibacterial drugs; Examples are: Penicillin, Oxytetracycline, Sodium Sulfadimidine; 150-200mg/kg BW IV or IP for 3 days.
2. Local Treatment:
   - Restrain the animal properly by holding the foot up by hand.
   - The foot is scrubbed, all necrotic tissues curetted / removed away and a local dressing applied under a pad or bandage.
   - Any suitable antibacterial ointment/ solution may be applied and secured with a bandage which may be left on for several days.
   - Local treatment may not necessary in the early stages of the disease if the animal can be prevented from gaining access to wt, muddy areas.

- In case where the lesion has remained superficial but has become chronic with separation of the horn from the coronary band, the horn should be trimmed back and the area cleaned and painted daily with 5% Copper Sulphate solution.

CONTROL

1. Prevention of injury in foot will reduce the incidence
2. Provision of footbath containing a 5 -10% solution of Formaldehyde or Copper sulphate.
3. Feed additives e.g. Chlortetracycline, organic iodides etc.

9: DISEASE CAUSED BY CLOSTRIDIUM SPP. TC "9: DISEASE CAUSED BY CLOSTRIDIUM SPP." \f C \l "2"

The clostridia are the major important in farm animals as primary causes of diseases. They rarely act as secondary invaders except where gangrene is already present.
They are all potent producers of exotoxins (Toxins which diffuse readily from the bodies of bacteria during their lifetime) upon which their pathogenicity depends
The toxins of the different organisms vary in their effects and the manner in which they gain entry to the circulation:
They may be ingested preformed in feed as in **Botulism**
They may be absorbed from the gut after abnormal proliferation of the causative organisms in the alimentary tract as in **Enterotoxaemia**.
May be elaborated in a more proper infection of the tissue as in **Blackleg**.

Others develop as local infections with elaboration of the toxins in minor lesions such as **Tetanus, Black disease, Braxy and Bacillary Haemoglobinuria**.
Clostridia are commonly present in soils rich in humus and in the intestinal contents of normal animals.
Endotoxins; toxins retained within the bodies of bacteria until the latter dies or disintegrate.

A: TETANUS (Synonymus: LOCK jaw)

Tetanus is highly fatal non-contagious infection disease characterized by muscle stiffness, prolapses of the third eyelid, convulsions and death due to respiratory failure. The susceptibility to disease ranges: Horses most susceptible and cattle less susceptible. Mortality rate is 80% in young animals but recovery rate is high in adult

ETIOLOGY

Clostridium tetani; Spore forming organism.
The organisms are commonly present in the feces of animals (e.g. Horses) and in soils contaminated by these feces.
The disease occurs in sporadic pattern.

TRANSMISSION

The organism get entry into the animal through many ways. However, spores may lie dormant in the tissues for some time and produce clinical illness only when tissue conditions favors their proliferation. The site of entry are:

i) Puncture wounds (deep) of the hooves – contaminated wounds
ii) Introduction into the genital tract during parturition (cattle)
iii) Wounds in the mouth (cattle)
iv) Castration
v) Shearing (Sheep)
vi) Docking (Sheep)
vii) Vaccination

PATHOGENESIS

Spores from soil or feces contaminating wounds when meet with reduced O_2 tension (under anaerobic condition) germinate and produce toxins **Tetanispasms** which is a neurotoxin affecting the peripheral nerves, traveling in nerve tract up to spinal cord (Ascending paralysis)

> Tetanus localized at site of introduction → proliferates and produce toxins (neurotoxins) under favorable condition (anaerobic condition) → Blood → peripheral nerves →CNS → clinical signs.

CLINICAL SIGNS

The incubation period is 1 - 3 weeks to months even year. The clinical signs are similar in all species:

Muscle stiffness accompanied by muscle tremor and lameness (1^{st} sign)

Prolapse of third eye lid, Spasms of the nostrils and tail.

Difficult mastication due to stiffness of masseter muscles

Constipation, Urine retention; due to partly inability to assume the normal position of urination, Bloat (may/may not) in young animals.

Temperature and pulse rate are normal, convulsion and death

NB: The course of disease varies: Horse & cattle (5-10 days) while sheep (3-4 days)

PM LESIONS

There in no gross or histological findings which are diagnostic to Tetanus.

DIAGNOSIS

1. History - accidental injury or surgery
2. Clinical signs - muscular spasms, prolapse of third eyelid
3. Laboratory examination.
    - Isolation of *Cl. tetani* from wounds or tissues
    - Inoculation of *Cl tetani* into laboratory mice

DIFFERENTIAL DIAGNOSIS

Muscular spasms, prolapse of the 3rd eyelid and recent history of accidental injury or surgery are characteristic findings. However, in its early stages, Tetanus may be confused with:

1. Hypocalcemia tetany: confined to lactating animal and respond to treatment with Calcium salts.
2. Acute Laminitis: no tetany or prolapse of 3rd eyelid.
3. Cerebrospinal meningitis: causes rigidity of necks and hyperesthesia to touch but has depression and immobility rather than excitement and hypersensitivity to sound and movement.
4. Enzootic Muscular dystrophy: there is stiffness without tetany.
5. Polioencephalomacia (edema of brain): no prolapse of 3rd eyelid, there is muzzle twitching, opisthotonos (marked arched spinal column in the concavity facing upward), blindness, inability to stand etc
6. Berenil Poisoning: poisoning leads to tremors, jerky eye movement, unsteady gait, opisthotonos and death.

TREATMENT

Response is poor in horse & sheep but better in cattle

The main principles in the treatment of tetanus are to eliminate the causative bacteria, neutralize residual toxin, relax muscle tetany to avoid asphyxia and maintain the relaxation. Drugs commonly used are:

i) Penicillin - Parenteral and locally at the wound (After antitoxins)
ii) Antitoxin
iii) Muscle relaxant e.g. Acepromazine, Magnesium sulphate injection and/or chloral hydrate

CONTROL

a. Vaccination (Tetanus toxoids) given annually in enzootic areas all susceptible animals

Toxoids: are Alum-precipitated, formalin treated toxin. One injection gives immunity in 10 – 14 days lasting for a year and revaccination in 12 months gives solid immunity for life.

b. Improve hygiene during surgical operations (avoid wound contamination)

B: BOTULISM

This is the rapidly fatal, motor paralysis disease most common in birds, particularly the domestic chicken and wild waterfowl, cattle, sheep and horses. However, Pig, dog and cats are resistant

ETIOLOGY

Clostridium butulinum, a spore forming anaerobe that proliferates only in decaying animal or plant materials.

Cl. butulinum proliferates in decomposing animal matter and sometimes in plant materials producing fatal toxins. Eight types of toxic are available: A, B, C, D. E etc.

TRANSMISSION

- The spread of infection from place to place is through carriage by birds and blowflies.
- The animal gets the infection through ingestion of toxins (Botulism is intoxication and not an infection).
- Dead rodents in hay or ensilage pit provides the source of toxins, dried poultry wastes, decaying grass at the base of old stock etc
- The disease occurs in a number of animals at one time and is fatal.

FACTORS THAT FACILITATE THE ANIMAL TO GET INFECTION

1. PICA syndrome; a phosphorus - deficient syndrome
2. Drinking of river/ lake water contaminated by carcasses of ducks or water flows died of botulism

3. Feeding of spoiled vegetables and potatoes contaminated by *Cl. butulinum*.
4. Spoiled silage: especially when forage is very succulent or is wet by rain when it is made

PATHOGENESIS

- The toxins of *Cl butulinum* are neurotoxins and produce functional paralysis without the development of histological lesion.
- When toxins are absorbed in blood, inhibit acetylcholine release at neuromuscular junction leading to paralysis of skeletal muscles.

CLINACAL SIGNS.

Signs usually appear 3 -17 days after the animal gain access to the toxic material

a. Peracute cases

Sudden death: However a few fail to take water or food for a day beforehand may be shown

b. Acute cases

i) Progressive muscular paralysis affecting particularly the limb muscles and the muscles of jaw and throat

NB: muscle weakness and paralysis commence in the hindquarter and progress on the forequarter the head and the neck

ii) Sternal recumbence

iii) Neck turned into the flank on the ground

iv) Protrusion of tongue

v) Salivation

NB: This variation in signs is often a characteristic of an outbreak

c. Sub acute for chronic cases

i) Sluggish walking due to partial paralysis, incordination, ataxia, inability to rise or lift the head

ii) Sternal recumbence

DIFFERENTIAL DIAGNOSIS

- ❖ Parturient paresis (Occurs in association with recent or near calving, and respond to calcium therapy)
- ❖ Paralytic rabies (History of bite from rabid animal, abnormal behavior e.g. picking up foreign bodies etc)

DIAGNOSIS

1. History of Pica
2. Clinical signs
3. Laboratory examination: Feeding of suspected material to susceptible animals.

TREATMENT

1. Specific or polyvalent antitoxin serum
2. Purgatives to remove the toxic from the alimentary tract
3. Central nervous system stimulants
4. vaccination of remainder animals in the group.

CONTROL

Correction of dietary deficiencies by supplementation with phosphorus or protein
Hygiene disposal of carcasses
Vaccination with toxoid in enzootic areas

C: BLACKLEG (synonymous: Black quarter)

Is an acute febrile infectious disease affecting cattle and buffalo characterized by emphysematous swelling of Muscles, usually in the heavy muscles, severe toxemia and a high mortality. Normally is a disease of cattle and occasionally sheep.

ETIOLOGY *Clostridium chauvoei (Cl. Chauvoei)*

EPIDEMIOLOGY

The bacteria causing the disease reside mainly in the soil. The organism probably gets into the animal body by ingestion.
Most cases occur in cattle from 6 months to two year old, but thrifty calves as young as 6 weeks and cattle as old as 10 – 12 years may be affected.
The disease is more common just after the start of short rains.

PATHOGENESIS

True blackleg develops when spores, which are not lodged in normal tissue, proliferate and produce toxins leading to severe necrotizing myositis locally, and a systemic toxemia, which is usually fatal.

CLINICAL SIGNS

Frequently affect heavy muscles of the hindquarters. If the animal is observed before death, there is a marked:

i) Swelling: Pain, hot, edematous, on palpation produces a crepitating sound, then develops to other parts. The skin on swollen part is discoloured and soon become dry and cracked
ii) Lameness
iii) Depression and high temperature (41^0C)
iv) Complete anorexia
v) Ruminal stasis

NB: Although the lesions are usually confined to the upper part of one limb, occasional cases are seen where lesions are present at the diaphragm, Psoas muscle, the brisket and udder.

POSTMORTEM LESIONS

Cattle found dead of blackleg are often in a characteristic position: lying on the side with the affected hind limb stuck out stiffly.
Bloating and putrefaction occurs quickly
Bloodstained froth exudes from the nostril and anus
Clotting of blood occurs rapidly.

On incision of the swelling/affected part reveal; dark, discolored, swollen tissue with a rancid odor, Serosanguinous fluid containing bubbles of gas
All body cavities contain excess fluid usually blood stained and fibrins.

DIAGNOSIS

Clinical signs
Pm lesions
Laboratory results: *Cl. chauvoei* can be isolated from the lesion (Myositis).

DIFFERENTIAL DIAGNOSIS

Anthrax: characteristic splenic lesion and lack of ligor mortis
Bacillary Haemoglobinuria: liver infarcts, haemoglobinuria
Lightning strikes
Lead Poisoning: muscle tremor, blindness, excitement, diarrhoea etc.

TREATMENT

Heavy doses of penicillin (large dose of 10000 nits/kg bw)

CONTROL

1. Annual vaccination
2. Proper disposal of carcasses of animals dying of blackleg by burning or deep burial to limit soil contamination.

D: BACILLARY HAEMOGLOBINURIA

This is the infectious disease of cattle and sheep characterized by a high fever, haemoglobinuria, and jaundice and by the presence of necrotic infarcts in the liver.

ETIOLOGY *Clostridium haemolyticum*

TRANSMISSION

The disease has shown high incidence/prevalence in
Soil with low lime
Swampy and poorly drainage areas
Liver flukes areas (Fasciola hepatica): Liver flukes cause damage to the liver, and the bacteria take advantages to the necrotic areas

The disease spread from infected to non - infected areas by:
Flooding
Natural drainage
Contaminated hay from infected areas
Carrier animals
Dogs or other carnivores carrying bones or meat

The animal gets infection through ingestion of contaminants of feces or decomposing cadavers

PATHOGENESIS

When the bacteria enter the alimentary canal, invade the wall and transported through blood to the liver and lodge there until damage to the parenchyma of the liver and the resulting hypoxia create conditions suitable for bacteria proliferation. After liver damage (e.g. by fasciolasis - Fasciola hepatica) anaerobic condition suitable for bacteria is created that lead to bacteria to produce toxins; Hemolysin and a Necrotizing agent.

Hemolysin: causes acute haemolytic anemia (severe hemolysis)

Necrotizing agent: causes endothelial damage, and extravasations of blood into the tissues, and plasma in serous cavities.

CLINICAL SIGNS

The disease occurs within 7 - 10 days after animal move to swamp areas, and the disease is characterized by:

i. Sudden death
ii. More often the disease is of short duration, characteristized by:

- Sudden complete cessation of rumination, feeding, lactation and defecation
- Abdominal pain (shown by disinclination to move and an arched back posture)
- Grunting may be evident on walking
- Fever (35.5 - 41^{0}C) at early stage, then subnormal at death
- Edema of the brisket
- urine is dark red (Haemoglobinuria)
- Jaundice and anemia
- Shallow and rapid respiration and dyspnoea that is followed with death.
- Feces are dark brown, there may be diarrhea with more mucus and blood.

NB: The course of illness is 12 hours to 4 days

POSTMORTEM FINDINGS

Wide spread petechial haemorrhages in subcutaneous tissues
Anemic infarcts in liver (pale areas surrounded by a zone of hyperemia and has the general appearance of local necrosis)
Red urine in kidney and bladder
Petechiation is evident throughout the kidney

DIAGNOSIS

Clinical signs
PM lesions
History

DIFFERENTIAL DIAGNOSIS

Acute Leptospirosis: petechiation
Plant poisoning: no fever
Babesiosis: no history of liver flukes.
Anaplasmosis: temperature fluctutation, incomplete anorexia, slightly/no haemoglobinuria, and jaundice.

Enzootic haematuria: pyelonephritis, cystitis, presence of red blood cells in urine.

TREATMENT
Antibiotics: OTC or Penicillin
Blood transfusion and fluid therapy

CONTROL
Control liver flukes
Proper disposal of carcasses
Vaccination

E: OTHER DISEASES CAUSED BY CLOSTRIDIUM

i) MALIGNANT EDEMA (Gas gangrene)

This is an acute inflammation at the site of infection and a profound systemic toxaemia.

CAUSES: *CL. septicum, Cl. chauvoei, Cl. perfringens, Cl. sordellii and Cl. novyi*

EPIDEMIOLOGY
All ages and species of animals are affected
Wound is the portal of entry and a dirty environment, which permits contamination of wounds with soil, is the common predisposing factor.

CLINICAL SIGNS
The disease is characterized by soft, doughy swelling with marked local erytherma accompanied by severe pain on palpation

TREATMENT
Penicillin is the drug of choice; local surgical incision and irrigation with Hydrogen Peroxide fastens the healing

ii) ENTEROTOXEMIA

This occurs in young animals, and is caused by *Cl. perfrigens* A.
The disease is characterized by severe depression, collapse, pallor mucosa, jaundice, haemoglobinuria and dyspnoea.

iii) INFECTIOUS NECROTIC HEPATITIS (Black Disease)

This is the disease of sheep, cattle and sometimes pigs
The disease is caused by TOXINS of *Cl. novyi* elaborated in damaged liver tissues. Under field conditions is associated with liver flukes (Fascioliasis)
The disease is characterized by sudden death; outstanding cases show sudden severe depression, reluctant to move, coldness of the skin, ruminal stasis and periorbital edema

iv) BRAXY (Bradsot)

This is the disease of sheep characterized by abomasitis, toxemia and high mortality rate.

10: ACTINOMYCOSIS (synonymous: Lumpy jaw) TC "10: ACTINOMYCOSIS (synonymous: Lumpy jaw)" \f C \l "2"

This is the disease of cattle characterized by osteomyelitis of the bones of the head, particularly the Mandibles and Maxillae. Rarely affects the soft tissues, particularly the Alimentary canal.

ETIOLOGY: *Actinomyces bovis*

EPIDEMIOLOGY

This is a sporadic disease and is worldwide. The disease is common in cattle and inheritance susceptibility exists, occasionally in horses and pigs. Its importance is through poor response to treatment

TRANSMISSION

Act. bovis is a common inhabitant of the bovine mouth and the infection get entry through wounds to the buccal mucosa caused: rough feeding (sharp pieces of feed), foreign material in feed, dental alveoli (teething), teeth eruption in young cattle, gastrointestinal tract infection from laceration by sharp foreign bodies

CLINICAL SIGNS

a. Painless swelling (bony swelling) on the mandible or maxilla, usually at the level of molar teeth
b. The swelling is very hard and immovable
c. The swelling in later stage become painful to touch, may break through the skin and discharge through the skin on one or more openings
d. The pus discharged is honey-like fluid, sticky containing minute, hard, yellow-white granules
e. Dyspnoea may occur if there is excessive swelling of the maxilla
f. The animal eventually become emaciated
g. When soft tissues are involved (the esophageal glove with spread to the lower esophagus and the anterior wall of the

reticulum) digestion disturbances may occur. The impaired digestion leads to passage of undigested food, chronic bloat and PICA syndrome (Allotriphagia).

h. Occasionally lesions may occur in the brain, lungs, omentum, heart, kidney, testes and mammary gland (this is by hematogenous route)

DIGNOSIS

Clinical signs: Abscess of bony tissues is a characteristic feature.

Laboratory examination: Needle puncture reveals the presence of pus, which may be fetid or caseous depending on the duration of the abscess. Microscope reveals yellow and rosette like colonies G +ve bacteria (Sulfur granules)

DIFFERENTIAL DIAGNOSIS

Abscesses of the cheek muscles and throat region. However, this is movable and localized in soft tissues)

Accumulation of foreign bodies between cheek and teeth. However, enlargement occurs suddenly.

Chronic peritonitis: due to indigestion caused by visceral actinomycosis.

TREATMENT

Sulphonamides, Penicillin, Streptomycin and Broad-spectrum Antibiotics

Iodides given orally or intravenous administered

CONTROL

i) Quick treatment of the affected animals. This prevents the contamination of pasture and feeding troughs

ii) Isolation or disposal of animals with discharging lesions

iii) Proper premises hygiene.

11: ACTINOBACILLOSIS (Synonymous: Wooden tongue)

This is the specific infectious disease of cattle and sheep characterized by inflammation of the tongue, less commonly the pharyngeal lymph nodes and esophageal glove.

ETIOLOGY

Actinobacillus lignieresii (gram -ve bacteria, sulfur granules former)

EPIDEMIOLOGY

Worldwide and sporadic occurrence on particular farm.

TRANSMISSION

Infected discharges are the source of the infection

Transmission is affected by the ingestion of contaminated pasture or feed

Injury to the buccal cavity/ mucosa permits easy entry of the infection (as in Actinomycosis)

PATHOGENESIS

Local infection → acute inflammation → granulomatous lesion with necrosis and suppuration → discharge of pus to exterior.

CLINICAL SIGNS

There is sudden onset of the disease with the animal stopping from eating within 48 hours

- Excessive salivation and gentle chewing of the tongue as if has foreign body is the mouth
- The tongue is swollen and hard, particularly at the base, while at the tip appearing to be normal
- Pain revealed on manipulation of the tongue
- Nodules and ulcers are present on sides and anterior edges of the dorsum of the tongue.
- Lymphadenitis: Submandibular and parotid lymph nodes
- Later on when the acute inflammation is replaced by fibrous tissue, the tongue become shrunken and immobile (difficult prehension)

Severe loss of weight and death through starvation
Rare cases: cutaneous Actinobacillosis occurs in cattle in the mouth, head, flanks, thigh appearing as ulcers discharging yellow pus or nodules.

DIAGNOSIS
Clinical signs: wooden tongue is characteristics

DIFFERENTIAL DIAGNOSIS
i) Rabies and Foreign bodies: due to salivation, chewing and anorexia
ii) TB (For cutaneous form of disease); use Tuberculin test to differentiate

TREATMENT
i. Iodides: Potassium or Sodium Iodide
ii. Sulphonamides
iii. Antibiotics: Penicillin, streptomycin & Broad Spectrum Antibiotics

CONTROL
i) Quick treatment to prevent spread of infection
ii) Institute sanitary measures: dinsinfectantion of premises, burn contaminated feeds etc

12: LEPTOSPIROSIS TC "**12: LEPTOSPIROSIS**" \f C \l "2"

This is a zoonotic disease of farm animals characterized by fever, haemolytic anemia, Jaundice, haemoglobinuria, abortion, mastitis in cattle and interstitial nephritis

ETIOLOGY

Leptospira interrogans

NB. Is a spiral organism (bacteria) seen under dark field illumination.

EPIDEMIOLOGY

- The disease is found worldwide (cosmopolitan)
- All domestic animals are susceptible, however young animals are most susceptible (Septicaemic development, passive transfer of antibodies to newborn occurs via the colostrums and the antibodies persist for 2-4 months)
- The disease is associated with high rainfall.

TRANSMISSION:

- For most Leptospiral serotypes, Rodents are the important hosts shedding the bacteria through urine
- The source of infection is usually an infected animal which contaminates pastures, drinking water and feed by
  i) Infected urine (especially of pigs e.g. piglet may carry Leptospira for up to 2 months)
  ii) Aborted fetuses
  iii) Infected uterine discharges and genital tract of bulls
  iv) Semen of an infected bull may carry Leptosporae and transmission from such a bull to heifers is by coitus and AI.
    - Water is the major mean of spreading the disease
    - Entrance of the organism into the body occurs most probably through cutaneous or mucosal abrasions.

PATHOGENESIS

- Entry is via abrasions on mucosal membrane or skin; from there the microorganism enters into the blood. Within 4 -7 days replication occurs and then migrate to brain, kidney, uterus, placenta and liver where they produce haemolysin toxin that cause haemolytic crisis (intravascular haemolytic) and capillary damage

CLINICAL SIGN

a. Acute form

- Common in calves up to a month old are most susceptible.
- The disease is manifested by septicaemia, with:
  i) Dullness (Depression) and Anorexia
  ii) Pyrexia - 41.5^{0}C
  iii) Petechiation in mucosal membranes
  iv) Haemoglobinuria
  v) Jaundice, anemia and Dyspnoea
  vi) Severe lameness due to synovitis occasionally occur.
  vii) Death
- Adult animals
  i. Abortion and
  ii. Agalactia (Absence of milk in udder following parturition). Milk flow almost ceases, and the secretion is red-coloured or contains blood clots, and the udder is limp and soft.

b. Subacute form

- Signs are milder as compared to acute
- Abortion may occur 3-4 weeks after infection
- One of the characteristic sign is the fall in the milk yield and the appearance of bloodstained or yellow - orange, thick milk all four quarters without apparent physical change in the udder.

NB: Mastitis outbreak occurs to about 50%.

c. Chronic form

Severe "storms" of abortion occurring most commonly in groups of cattle, which are at the same stage of pregnancy when exposed to infection (last third of pregnancy i.e. 7-9 month) without other signs of illness.

Occasionally, Leptospiral meningitis in cattle have been observed and X2 by incoordination, excessive salivation, conjunctivitis and muscular rigidity.

POSTMORTEM FINDINGS:

Enlarged liver (Hepatomegaly), interstitial nephritis and autolyzed aborted fetus.

Acute cases: anemia, Jaundice, haemoglobinuria and submucosal haemorrhages

DIAGNSIS

Epidemiology
Clinical signs
PM findings
Laboratory isolation of the organism

DIFFERENTIAL DIAGNOSIS

1. Babesiosis: All age groups, history and microscopic examination, acute fever, jaundice, rarely abortion.
2. Bacillary haemoglobinuria: Edema of brisket, abdominal pain.
3. Mastitis: Swelling of udder (not in Leptospira)
4. Brucellosis: Retention of placenta, Metritis and Pneumonic calves

TREATMENT

The primary aim of treatment is to control the infection before irreparable damage to the liver and kidney occurs. Thus, treatment should be done as soon as possible after signs appear:

Streptomycin
Tetracycline

CONTROL

1. Eliminate the carrier animals after serological test
2. Control of wild animals (rodents and other wild animals)
3. Maintain hygiene - limiting the spread

4. Vaccination

Zoonosis (occupational hazard) to butcher, farmers and veterinarians. Human get the infection by contamination with infected urine or uterine contents

13: CAMPYLOBACTERIOSIS TC "13: CAMPYLOBACTERIOSIS" \f C \l "2"

Several species of the genus Campylobacter are known to cause disease in farm animals. The most common disease caused by Campylobacter is Vibriosis.

(a) VIBRIOSIS (synonymus: Campylobacteriosis)

This is a venereal disease of cattle spread through coitus from chronically infected bulls and characterized by infertility.

ETIOLOGY: *Compylobacter fetus var. venerealis*

Transmitted by infected bulls
Compybacter are zoonotic causing diarrhea and dysentery in main

SYMPTOMS:

1. Abortion: The pathogens cause uterine reaction which impair embryo nourishment leading to early embryonic death (about 4 - 6 months)
2. Endometritis
3. Increased excretion of mucous leading to vulva discharges
4. There is regular or irregular estrous cycle (Infertility; irregular moderately prolonged diestrus)
5. Bulls: Do not show specific clinical signs, only become carrier unless treated

6. Fetus: Pus in peritoneum; thickened placenta due to inflammation, semi-opaque, petechiae, localized avascularity and edema.

DIAGNOSIS

□□蒴葰 History

□□蒴葰 Clinical signs

□□蒴葰 Examination of vaginal mucosa by using vaginal spectrum (to see endometritis)

□□蒴葰 Laboratory examination: Culture and sensitivity test of material from prepucial area.

TREATMENT

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treptomycin infusion-intrauterine or in prepuce, penis (wash)

CONTROL

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revent natural service (Avoid use of infected bulls)

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se of Artificial insemination

(b) Other Diseases Caused By Campylobacter Spp

- *C. Jejuni:* causes diarrhea in calves and human being
- *C. Col:* cause diarrhea and colic in Piglets
- *C.fetus var. jejuni:* causes winter dysentery in cattle
- *C.fetus var. fetus:* causes sporadic abortion in cattle and enzootic abortion in sheep bacteremia in man

14: COLIBACILLOSIS TC "**14: COLIBACILLOSIS**" \f C \l "2"

This is a disease (most common) of newborn farm animals characterized by diarrhea, septicemia and rapid death

NB: The disease is most common in animals under 3 days of age, but it may occur as early as 12-18 hours after birth and occasionally occurs in calves up to several days of age when there is a mixed infection with viral enteropathogens

ETIOLOGY: *Escherichia coli* (pathogenic *E. coli*): A normal inhabitant of the posterior part of intestine of healthy animals

PREDISPOSING FACTORS

1. Low immunity: Newborn farm animals are born with low immunity (agammaglobulinemic) and must ingest colostrum and absorb colostral immunoglobulins within few hours (24 hrs in calves and 48hrs in piglets) after birth to obtain protection against septicemia and enteric Colibacillosis.
2. Overcrowding: When newborn animals are mixed with older calves
3. Poor hygiene: Dirty houses barns contribute much to the occurrence of the disease
4. Environmental factors: Cold, wet, windy weather have a significant effects on calf mortality due to septicemia and enteric Colibacillosis
5. Nutrition: Vitamin A and Iron deficiency in piglets predispose the newborn piglets to get the infection

TRANSMISION

a. Ingestion of contaminated feed, water etc
b. Intrauterine infection especially in foals
c. Navel and nasopharyngeal infection

SOURCE OF INFECTION

- In calves: contaminated bedding, dirty calf pens, diarrheic calves milk, from cow with mastitis (coliform), skin of the perineum and udder of the cow
- In swine - feces of infected sows and uterus.

SYMPTOMS

There are 2 forms of disease

1. Coli-septicaemia form of colibacillosis (septicaemic)

This occurs in newborn animals, which are deprived of colostrum, and occurs on the 4th day of life. (Most common in calves during the first 4 days of age)

This is acute, the course varying from 24 to 96 hrs. There are no diagnostic clinical signs

- The affected animals are depressed, weak, complete anorexia
- Temperature may be high at initial stage and then fall down at late stage (subnormal) leading to moribund and weakness.
- Diarrhoea and dysentery are uncommon
- If the animal survives the septicemia state localization may appear in 1week. And this lead to arthritis, meningitis, panophthalmitis (Pus in eyes) and less commonly, pneumonia

2. Enterotoxic colibacillosis (Enteric form)

This is the most common form of colibacillosis in newborn calves primarily from 3 to 5 days of age (may occur in calves of 1 day old)

The affected newborn animals collapse and die within 2 - 6 hrs after the onset of signs.

Outstanding clinical signs include coma, collapse and death (shock like syndrome) in severe cases.

Also diarrhea with profuse, watery to pasty, faeces, usually pale yellow to white in colour, and occasionally streaked with blood flecks and very foul smelling and dehydration.

Diarrhoea/defecation is frequent, effortless, and the tail and buttocks are soiled.
The temperature is usually normal in the initial stages but becomes subnormal as the disease worsen.
Abdomen may be slightly distended in early stages when the intestines are fluid-filled then diarrhea occurs.

DIAGNOSIS

Epidemiology: Illness from birth to 2 wks of age; and Clinical signs

TREATMENT

1. Control dehydration and acidosis by using fluids and electrolytes e.g. solutions containing sodium chloride, HCO_3 ions or plasma or combination e.g. Ringers lactate.
2. Use of antibiotics (protectants) e.g. chloramphenical Nitrofurans, Neomycin, Sulfonamides etc.

CONTROL

- Newborn animals must be given colostrums from their dams.
- Proper housing hygienic measures to be reinstituted quickly e.g. cleaning and disinfections.
- Avoid overcrowding and animals of the same age should be kept together, older animals to be separated from young animals
- Avoid contamination of food, water and pasture
- Vaccination ($K99^+$) of dams 2 -4weeks before calving or furrowing is important to stimulate production of antibodies
- Blood transfusion to each newborn animal will prevent the disease.

SUMMARY

SEPTICAEMIC COLIBACILLOSIS: Occurs in newborn animals, which are agammoglobulinemic because they have not ingested sufficient colostrums early enough and absorbed sufficient immunologic – highly susceptible.

ENTERIC COLIBACILLOSIS: Occurs in colostrums-fed animals and is caused by the localization and proliferation of enteropathogenic E.coli, which produce enterotoxins and cause various degree of diarrhea, acidosis and dehydration.

B: BOVINE VIRAL DISEASES TC "B: BOVINE VIRAL DISEASES" \f C \l "1"

1: FOOT AND MOUTH DISEASE (FMD) TC "1: FOOT AND MOUTH DISEASE (FMD)" \f C \l "2"

This is extremely contagious, acute, benign (mild fever) disease of all cloven-footed animals characterized by fever, formation of vesicles outside the mucosal membrane in oral cavity, udder and interdigital spaces. The disease has highest morbidity and mortality rates in young animals.
The disease is not a killing one (MR; young 20% adult 2%) animals are severely affected during the disease and the period of convalescence is prolonged that production (meat and milk) is seriously impaired.

CAUSES
FMD is caused by RNA virus (*Ophthovirus*) under family of Piconarviridae.
There are 7 immunologically distinct strains of virus causing the disease, and these are A, O, C, SAT-1, SAT-2, SAT-3 and ASIA-1.
The most common strain is 'O' and least common is 'C' . There is no cross immunity between strains and substrains.

EPIDEMIOLOGY
Animal affected are Cattle, Buffaloes, Sheep, Goats and Pigs (Not HORSES)
Transmission:
The animal gets the infection through either inhalation or ingestion.
The disease is spread from herd to herd or animal to animal either directly or indirectly.

- **Direct Spread:** By infected animals or possibly infected humans. The viruses are retained in the retropharyngeal region (in the carrier animal) or salivary gland where secretion in the environment is at maximum (in air 100km: wind born, dust, humidity and not rainfall)
- **Indirect Spread:** By transportation of the virus on inanimate objects, particularly uncooked and unprocessed meat, milk, contaminated water, hay, straw and other animal by products.

PATHOGENESIS

Irrespective of the portal of entry, once the infection gains access to the blood stream, the virus shows a predilection for the epithelium of the mouth and the feet, to the less extent; the teat. In these organs the virus causes irritation (persistent local irritation) leading to the formation of vesicles. The incubation period of the disease is 1 – 21 days (usually 3 – 8 days)

- ❖ In cattle the immunity which develops after natural infection varies (1-4yrs); pigs 30 weeks.

CLINICAL SIGNS

- The onset of FMD is heralded by a precipitate fall in milk production (for lactating cows) and high temperature (40 – 41°C).
- Severe dejection (depression) and anorexia
- Acute painful stomatitis
- Salivation: The saliva hangs in a long ropey string and the animal chewing carefully.
- Appearance of vesicles and bullae (1 – 2 cm in diameter) on the buccal mucosa, dental pad and the tongue. The vesicles rupture within 24 hours allowing flow of straw-coloured fluid,

and leaving a raw painful surface which heals in about a week.

- Lesions on the teat and particularly on the interdigital space cause the animal unable to walk (lameness).
- Pregnant animals abort
- Calves (young animals) are rather susceptible than adult and heavy mortality may occur during an outbreak without typical lesion being present; only MYOCARDITIS causing hemorrhages in the heart.

- A SEQUEL TO FMD IN CATTLE: due to probably endocrine damage, a chronic syndrome of dyspnea, anemia, overgrowth of hair and lack of heat tolerant (panting) may develop.
- The MOBIDITY is 100% while MORTALITY is 50%.
- Sheep and Goats only show mild lesion and so to pigs.
- Animals recovered remain carrier with virus localizing in the nasopharyngeal region: Sheep and Cattle (9 months as carrier), Goat 1 month (as Carrier). Under stress these animals release the virus

DIFFERENTIAL DIAGNOSIS

i. Mucosal Disease: (Lesion in buccal cavity, conjunctivitis, diarrhea & dysentery.)
ii. Rinderpest: (Lacrimation, discrete lesions (not vesicles), diarrhea & dysentery.)
iii. Malignant catarrhal fever: (Necrotic lesions, nasal discharge, CNS signs & diarrhea)
iv. Vesicular stomatitis: (Affect also horses, confirmed in laboratory)

v. Swine vesicular disease: (In swine Very short, mild with animals recovering quickly)

vi. Vesicular Exanthema: (In swine: Very short, mild with animals recovering quickly)

DIAGNOSIS

- ❖ Epidemiology: Host, morbidity and mortality
- ❖ Clinical signs: vesicular type
- ❖ Serological test: Complementary fixation test, Serum Neutralizing test and ELISA
- ❖ Animal inoculation: Intradermal injection of fresh vesicular fluid into the plantar pads of the guinea pigs. Vesicles appear on pads in 1 – 7 days and secondary vesicles in the mouth 1 – 2 days later.

TREATMENT

- Treatment with mild disinfectants to inflamed areas for prevention of secondary infection is recommended.
- Symptomatic treatment: e.g. use of Antibiotics to lower the high temperature etc

CONTROL

Quarantine: Restrict movement of animals and by products in and out of the area.

Slaughtering policy: Slaughter all affected animals and compensate the farmers (not applicable in developing countries).

Vaccination: vaccine types depend on the serotype

Vaccinate all animals around the outbreak (ring or barrier vaccination)

Annual vaccination in endemic areas: vaccination can be done once, twice or three times per year depending on the challenge.

NB: Immunity lasts for 4 – 6 months and sometimes up to 1 year.

Treat animal products: Example, pasteurelization of milk.

2: RINDERPEST (SYNONYMUS: CATTLE PLAQUE, SOTOKA) TC "**2: RINDERPEST (SYNONYMUS: CATTLE PLAQUE, SOTOKA)**" \f C \l "2"

This is an acute, highly contagious disease characterized by high fever and focal, erosive lesions confined largely to the mucosa of the alimentary tract. The disease affects ruminants (cattle, goats, buffaloes, giraffes & wildebeest) and swine.

AETIOLOGY

RNA virus: *Morbillivirus* (family Paramyxoviridae)

The virus seems to have some antigenic relationship to the virus of Canine Distemper (Canines), Newcastle disease (Poultry), Measles (Man) and Peste des Petits Ruminants (Sheep & Goats).

EPIDEMIOLOGY

The disease occurs worldwide with exception of North America.

In outbreaks which occur in highly susceptible population, morbidity rate can be up to 100% and Mortality rate of 50% (25 – 90%).

Wild ruminants are common source of infection and are very great hindrance to an eradication programs. Among of these are Buffaloes, Bushbucks, Waterbucks, and warthogs.

Among the races of cattle, the zebus are most resistant.

Close contact between infected and non-infected animals is usually necessary for spread of disease to occur.

Entry of infection occurs principally by inhalation.

CLINICAL SIGNS

The incubation period ranges from 6 – 9 days

The first sign is high fever (40.5^{o}C – 41.5^{o}C) without localizing signs.

Then follows: Anorexia, a fall in milk production, and a harsh, staring coat.

There is inflammation of the buccal and nasal mucosa, conjunctivitis, which is followed by hyperemia of vagina mucosa and swelling of vulva.

First, there is serous lacrimation, which late on become profuse, purulent and normally accompanied by blepharospasm

Profuse salivation, which may be blood-stained and late on purulent as mouth lesions develop.

There will be serous nasal discharge, which late on become purulent

Discrete necrotic lesions inside the lower lips, adjacent gum, cheek mucosa, tongue, nasal, vulva and vaginal mucosae (the lesions are grayish in colour, slightly raised and obviously necrotic. Necrotic material sloughs leaving a raw, red area with sharp edges)

There will be skin lesions affecting the perineum, scrotum, flanks, the inner aspects of the thighs and the neck occur in some cases.

Severe diarrhea and sometimes dysentery with tenesmus appear as lesions develop in abomasums and intestines.

After a period of illness lasting from 3 – 5 days, there is a sudden fall in temperature accompanied by dyspnoea, cough, severe dehydration and sometimes abdominal pain. Death occurs within 24 hours after a fall in temperature.

◊ In enzootic areas where resistant to infection is high, both subacute and skin form occur which is characterized by mild temperature, catarrhal inflammation, small pustules on neck etc.

POSTMORTEM CHANGES

Small, discrete, necrotic areas in the mucosa of mouth, pharynx, the first third of esophagus, nasal cavity, abomasums and small intestines.

Congestion, swelling and erosion of vulval and vaginal mucosae may be present

Haemorrhages at the iliocaecal junction; the haemorrhages are transverse way called ZEBRA STRIPS

Heart may be haemorrhagic, lungs and urinary bladder edematous.

DIFFERENTIAL DIAGNOSIS

FMD: *(Vesicle formation)*

Malignant catarrhal fever: (Eye lesion and CNS changes)

Mucosal disease: (Sporadic occurrence)

Blue tongue: (Sheep and goats only)

DIAGNOSIS

i. Epidemiology: large number of animals affected
ii. Clinical signs
iii. Serological tests: CFT, ELISA, AGD (Agar Gel Diffusion)

TREATMENT

Not recommended because of danger of disseminating the disease

CONTROL

Rinderpest is a notifiable disease

Quarantine: This is followed by ring and barrier vaccination

Vaccination: Annual vaccination of all susceptible animals

Slaughter policy: Not feasible in developing countries

Eradication can be contemplated in enzootic areas if wild ruminants can be eliminated as source of infection.

3: EPHEMERAL FEVER (SYN: THREE DAY SICKNESS) TC "3: EPHEMERAL FEVER (SYN: THREE DAY SICKNESS)" \f C \l "2"

This is arthropod – transmitted disease of cattle and buffaloes characterized by transitory fever, lymphadenitis, watery nasal discharge and myositis, which come in and subside within a few days (mostly 3 days).

ETIOLOGY:
The disease is caused by Virus classified as *Rhabdovirus*

EPIDEMIOLOGY
The disease occurs in low-lying areas, dampy areas, near river or at the onset of rain season.
Insects are responsible for transmission, culicoides and Mosquitoes are possible vector of the disease. However, the disease may occur in areas where insects are not found. Thus suggests other means of transmission.

CLINICAL SIGNS
Biphasic fever and shivering
Inappetence (anorexia)
Lacrimation
Watery (serous) nasal discharge
Dyspnoea
Atony of forestomach
Depression
Stiffness and sometimes lameness (due to myositis)
Affected animals may become recumbent from 8 hours to more than a week
Drop in milk production and abortion may occur
NB: Bulls and heavy cattle are the most severely affected animals.

DIAGNOSIS

- ❖ Clinical signs
- ❖ Serological tests
- ❖ Isolation of virus

TREATMENT

a. Complete rest is the most effective treatment
b. Anti inflammatory drug if given early are helpful
c. Antibiotic to control secondary infection
d. Calcium borogluconate can be given to recumbent animals.

4: LUMPY SKIN DISEASE TC "4: LUMPY SKIN" \f C \l "2"

This is highly infectious skin disease of cattle characterized by the sudden appearance of nodules on all parts of skin.

ETIOLOGY

Lumpy skin disease is really two diseases:
The severe form caused by *Neethling poxvirus*
The mild form caused by *Allerton herpesvirus*

EPIDEMIOLOGY

- All ages and types of cattle are susceptible to the causative virus except animals recently recovered from an attack, in which case there is a solid immunity lasting for about three months
- Biting insects have been suspected as vectors, but outbreaks have occurred under conditions in which insects practically are excluded.
- Contact infection through saliva is another mode of transmission.
- Artificial infection can be produced by inoculation of cutaneous nodular suspension or blood during the early

febrile stages, or by feed or water contaminated with saliva from infected animals.

CLINICAL SIGNS

An incubation period of 2 - 4 weeks is common in field outbreak

i. There is initial rise in temperature, sometimes accompanied by lacrimation, nasal discharge, salivation and lameness.
ii. Multiple nodules appear on the skin suddenly about a week late, they are around and firm, varying from 1 - 4 cm in diameter, and are flattened.
iii. Nodules may develop in the mucosa of GIT, respiratory and genital tracts, which late on ulcerate.
iv. Nodules on conjunctiva cause severe lacrimation.
v. Lymphadenitis may be present.
   - The nodules involve the skin and subcutaneous tissue, sometimes coalesce and form large lumpy lesion called "SIT FASTS"
   - Nodules may ulcerate and suppurative materials fill the nodules.

vi. Nodules on leg cause edema, which lead to necrosis and sloughing off of skin
vii. Mastitis may occur if udder is involved

❖ Morbidity rate is 50% while Mortality rate is less than 10%

PM PICTURE

- Necrotic ulcers in the nasal cavity, mouth, pharynx, trachea, bronchi, and stomach.
- Pneumonia may be present.

DIAGNOSIS

i) Clinical signs
ii) Serological testes: Fluorescent Antibiotic tests
iii) Isolation of Virus
iv) Histopathological examination: presence of inclusion bodies in section of nodule.

TREATEMENT

- No specific treatment is available, but prevention of secondary infection is essential: Use of Antibiotics or Sulfonamides is recommended.

CONTROL

- Vaccination of cattle with sheep poxvirus vaccine.
- Quarantine and restriction of movement are useless.

5: MALIGNANT CATARRHAL FEVER (MCF) TC "5: MALIGNANT CATARRHAL FEVER (MCF)" \f C \l "2"

(SYNONYMOUS: NOSE SICKNESS, MALIGNANT CATARRH)

This is an acute, sporadic, highly fatal, infectious disease of cattle characterized by the development of an erosive stomatitis and gastroenteritis, erosions in the upper respiratory tract, keratoconjunctivitis, encephalitis and lymphadenitis.

ETIOLOGY: *Herpesvirus – 1*

EPIDEMIOLOGY

- MCF in Africa appears to be an infectious of Wildebeest, which causes no harm to them, but when the infection spills over into abnormal hosts, specifically cattle, a highly fatal disease occurs.
- In Africa, outbreak in cattle occurs commonly where they have access to wild ruminants, particularly during calving period of wildebeests.
- Sheep (ewe), also act as carriers
- The disease affects cattle and buffaloes, while wildebeest, goats and sheep are carriers by contaminating the environment especially during calving.
- Outbreak occurs in warmer months particularly when the wildebeests are calving and are in contact with cattle.
- Morbidity is 1 - 2% while mortality is 99%.

TRANSMISSION

- The spread of disease from infected animal or carrier to susceptible cattle is through contact (direct contact)
- The virus is present in the nasal and ocular secretions whereby the rate of contaminating the environment is high but the virus does not live long in the normal air.

  NB: In Tanzania, the outbreak occurs during February and June, which is associated with calving season of wildebeests.

CLINICAL SIGNS

The incubation period varies, 9 days to 9 weeks (average 3 weeks)

i) Affected animals are anorexia and extremely dull.
ii) High and persistent temperature (41.5^0C)
iii) Profuse mucopurulent nasal discharge (bilateral)
iv) Ocular discharge with variable degree of edema of the eyelids, blepharospasms and congestion of capillaries
v) Necrotic areas in the nasal cavity and on buccal mucosa, hard palate, gum and gingivae
vi) Salivation (excessive) with saliva hanging and ropey
vii) Necrotic lesions may occur at the skin -horn junction, the teats, vulva and scrotum, and in acute cases the skin may slough off entirely on touching.
viii) Nervous signs: weakness in one leg, incoordination, head pressing (pushing), paralysis and convulsion at final stage
ix) Lymphadenitis
x) Diarrhoea and dysentery
xi) Opacity of the cornea: commencing as a narrow, gray ring at peripheral spreading centripetally.

NB:In peractue cases, the disease runs a short course of 1 - 3 days with similar

Characteristic signs except opacity. For typical cases, the illness lasts for 3 - 7 days and

rarely up to 14 days.

POSTMORTEM LESIONS

i) Erosive lesions in the mouth, nasal cavity, pharynx, alimentary tract, urinary bladder, kidney and eyes
ii) All lymph nodes are swollen, edematous and often haemorrhagic
iii) Petechial haemorrhages as well as congestion in brain and meninges
iv) Dermatitis

DIAGNOSIS

A definitive diagnosis of MCF is very difficult because of absence of suitable virological technique

i. Epidemiology
ii. Clinical signs
iii. Pm lesions
iv. Serological tests: Virus Neutralization tests (Wildebeests), CFT

DIFFERENTIAL DIAGNOSIS

i. Rinderpest: Rapid spread, high mortality rate without opacity
ii. Mucosal Disease: lesions in interdigital space (lameness) pneumonia
iii. FMD: Vesicle formation without opacity

TREATMENT

- No effective treatment
- Prevent secondary infection by antibiotics

CONTROL

i. Keep away cattle and buffaloes from carrier animals
ii. Isolate the affected animals from the herd

iii. No effective vaccine is available to date.

6: MUCOSAL DISEASE TC "6: MUCOSAL DISEASE" \f C \l "2"

This is acute or subacute or chronic disease of cattle characterized by fever, diarrhoea, erosion of gastro – intestinal tract (GIT), abortion and CNS involvements.

ETIOLOGY: Caused by *Pestivirus* (Family Togaviridae)
The same virus causes two distinct diseases: Mucosal disease (fatal disease) and Bovine Viral Diarrhoea (subclinical infection)

EPIDEMIOLOGY
The disease is worldwide spread affecting cattle and Buffaloes. Immature animals (4 – 8 months) are mostly affected by the mucosal disease.

TRANSMISSION

i. Direct transmission (Through contact)
ii. Indirect transmission (Formates; arthropods)
iii. Vertical transmission (from dam to offspring)

CLINICAL SIGNS
The incubation period is 1 – 3 weeks (approximately 10 days)

i. Rise in temperature 42.5^{o}C
ii. Dullness
iii. Inappetence due to lesions in buccal cavity
iv. Nasal discharge; early stage is serous (watery) late on becomes mucopurulent and copious in nature
v. Superficial erosive lesions occur in the nasal cavity, but no vesicles
vi. Halitosis
vii. Salivation (profuse)
viii. Conjunctivitis and lacrimation
ix. Diarrhoea and dysentery but not as in Rinderpest

x. Dehydration and emaciation
xi. Pneumonia (coughing)
xii. Lesions on interdigital space, lameness
xiii. Abortion or give birth to abnormal calves with CNS disorders: Cerebellar hypoplasia, Cerebellar-ocular agenesis, ocular defects etc

Morbidity rate is 25% while Mortality rate is 90%

POSTMORTEM PICTURE

Dehydration
Ulcerative lesions along GIT

DIFFERENTIAL DIAGNOSIS

i. Malignant Catarrhal Fever: Presence of opacity in MCF
ii. FMD: Presence of Vesicles in FMD
iii. Johnes Disease: Diarrhoea without lesion in Johnes disease
iv. Rinderpest: High morbidity and Mortality rates, no coughing and CNS signs.

DIAGNOSIS

i. Epidemiology: Morbidity, mortality and age factors
ii. Clinical signs
iii. Serological tests: CFT, ELISA, Gel diffusion precipitation (GDP)

TREATMENT

There is no specific treatment; the prognosis for severe cases is unfavorable

CONTROL

i. No proper control measure is recommended
ii. Animals with chronic disease should be culled and destroyed

7: PAPILLOMATOSIS (Synonymous: WARTS) TC "7: PAPILLOMATOSIS (Synonymous: WARTS)" \f C \l "2"

These are benign neoplastic lesions of the skin especially around teats and necks. The neoplasms have a characteristic of crop like (Group) rough on the skin.

ETIOLOGY

Papillomavirus (family Papovaviridae)

EPIDEMIOLOGY

- Warts are very common in young cattle, especially when they are housed, but ordinarily they cause little harm and disappear spontaneously.
- In horses, the lesions are usually small and cause little inconvenience. They usually occur sporadically.
- The lack of susceptibility of adults to natural infection with Warts is thought to be due to immunity acquired by apparent or inapparent infection when young.
- The method of spread is probably by direct contact with infected animal
- Infection gains entry through cutaneous abrasion.
- Transplacental spread also occurs (particularly in horses)
- Morbidity and Mortality rates are very low.

> In cattle, warts occur on almost any part of the body, when a number of animals in a group are affected, it is common to find them all affected in the same part of the body.
> It is known that distinct viruses cause some of these topographically specific warts, so that immunity to one of them does not necessarily confer immunity to others.

CLINICAL SIGNS

The incubation period is 3 – 8 weeks or longer.

i. Warts are solid outgrowth of epidermis occurring on the skin, especially in teats, ventral of abdomen, neck, vulva and penis.
ii. The lesions (warts) may be Sessile or pedunculated, varying in size from 1 cm upwards, they are dry, horny, cauliflower-like appearance in characteristics.

DIAGNOSIS

i. Epidemiology: Location, species affected and age of animals
ii. Clinical signs
iii. Tissue biopsy

TREATMENT

i. Cutting the outgrowths followed by application of tincture of iodine
ii. NB: when cut few, others will regress spontaneously fast
iii. Take part of lesion, clean, grind, filter and inject intramuscularly 1 ml to the animal (autovaccine), the animal will recover spontaneously.

CONTROL

- No specific control measure because the condition is unpredictable.

DISEASES CAUSED BY RICKETISIA TC "DISEASES CAUSED BY RICKETISIA" \f C \l "1"

1. ANAPLASMOSIS TC "1. ANAPLASMOSIS" \f C \l "2"

Anaplasmosis of cattle, sheep and goats is caused by infection with *Anaplasma spp*. In cattle is characterized by severe debility, emaciation, anaemia, and Jaundice. The disease is usually subclinical in Sheep and goats.

Etiology

The rickettsia, *Anaplasma marginale* and *Anaplasma centrale* are the causative agent in cattle and wild ruminants, an *A. ovis* in sheep and goats

Epidemiology.

The morbidity rate is usually high in the outbreak. The mortality rate varies widely depending on susceptibility, and may be 50% or more in introduced cattle.

Young animals are relatively resistant to the disease; the resistance to infection is due to passive immunization as a result of the passage of antibodies from the dam to the calf in the colostrums.
Zebu-type cattle are relatively resistant to infection than *Bos taurus* breeds probably because of their relative resistance to heavy tick infestation
The source of infection is always the blood of an infected animal
Spread from animal to animal occur chiefly by insect Vectors *Boophilus microplus*, *Rhipicephalus Sanguineus*, *Boophilus annulatus*, *Dermocentor* ticks, *Argas* ticks, and biting flies of *Tabanis spp*.
Bovine anaplasmosis may also be spread mechanically by infected hypodermic needles, and castrating, spaying and dehorning instruments, by blood transfusions and embryo transplant
Intrauterine infection also occurs particularly in ewes.
Recovered animals remain carriers for life.

Clinical findings
In cattle the incubation period varies being 3-4 wks or longer after tick-born infection. In most cases the diseases is subacute, especially in young animals

1) **Peracute form (particularly in adult dairy cows)**
   - Sudden onset of high fever, Anaemia, Jaundice Severe dyspnoea and death after 24hrs.

2) **Acute form**
   High temperature (40.5^{0}C). This may remain elevated or fluctuate with irregular periods of normal and fever up to 2 wk
   Slightly or severe anorexia and ruminal stasis
   Pallor and Jaundice mucous membrane
   Emaciation and impaired fertility
   Constipation, pass out pelleted faeces covered with moucous.

Rapid drop in milk production

3) **Sheep and goats**

- Listlessness, anorexia, weakness, ruminal stasis, respiratory distress, pallor of the mucous membrane, increase heart and respiratory rate constipation, edema of submandibular region and ventral side of the neck, and abortion may occur

Pm Findings:

1. Emaciation, Pallor of mucous membrane and thin watery blood
2. Yellowish of mucous membrane (jaundice)
3. The liver is enlarged and deep orange in colour (mottled mahogany colour)
4. Spleen is enlarged with soft pulp
5. Severe constipation (omasal contents are dry and caked)
6. Bile is thick, brownish green and the gallbladder is distended.

DIAGNOSIS

1. History – Outbreak associated with presence of vector and other means of spread
2. Clinical signs
3. Laboratory examination
   - Peripheral blood smear stained with GIEMSA, presence of small dot-like organisms at the periphery of RBC
   - In subacute cases 10% of RBCs are parasitised
   - In paracute cases more than 50%
4. Pm picture

Differential Diagnosis

- Babesiosis – haemoglobinuria
- Trypanosomosis
- Bacillary haemoglobinuria
- Water toxicity in calves (no fever and presence of haemoglobinuria)
- Chronic copper poisoning: (CNS signs).

Treatment

1. Oxytetracline injection
   10% for 3days
   20% – once (in subacute cases)

2. Imidocarb
   3mg/kg Bw given once, for complete sterility 2 injections are required at 5mg/kgBru

3. Supportive treatment
   - Multivitamin injection
   - Purgative – Epsom salt, liquid paraffin.

CONTROL

- Tick control – use of acaricides.

2. HEARTWATER (Synonyms *Cowdriosis*) TC "**2. HEARTWATER (Synonyms *Cowdriosis*)**" \f C \l "2"

This is a tick-borne disease of wild and domestic cattle, sheep and goats characterized by fever, nervous signs, edema of the body cavities and diarrhea. Goats are more susceptible than Cattle.

Etiology

Rickettsial organism: *Cowdria ruminantium* (an obligate intracellular parasite)

Epidemiology

- This is the disease of ruminants (cattle, sheep and goats). Wildebeest, antelopes and rodents acts as carrier.
- Exotic breeds of cattle & goats are more susceptible than local cattle.
- The disease is limited in its occurrence to Africa, south of Sahara where mortality rate is (case mortality rate) is 50-100%, that is, peracute -100% in acute cases 50 - 90%, in mild cases most animals recover
- The disease is transmitted by ticks, *Amblyomma* genus especially *Amblyomma variegatum*
- Young animals have innate resistance

Pathogeneses

The infection is introduced by the bite of an infected tick, multiplies in the local lymph nodes and then invades the endothelial cells of the blood vessels in all organs where they cause vascular damage leading to hydropericardium

One of the endpoints of vascular damage in goats is renal ischema and a resulting nephrosis

Clinical Signs

Incubation period is 1 - 3 wks

a**) Peracute cases**:

Show only high fever, lacrimation, collapse and death with terminal convulsion (within 12hrs)

b) **Acute Form**: Have a course of 6 days

High temperature 41-43^{0}C which remains high throughout the course of illness

Nervous Syndrome, which is characterized by high stepping gait, exaggerated blinking of eyes, chewing movements, circular movements, ataxia, apparent blindness, recumbence, convulsions and death.

In less severe cases the principle sign may be diarrhoea

Necropsy findings

Standard lesions are ascites, hydrothorax, and hydropericardium

There may be subserosal haemorrhages in most cavities

Splenomegaly and lymphadenopathy

Histopathological findings include perivascular infiltration in most organs and encephalitis with ricketisia visible in the brain tissue.

Diagnosis

Clinical signs: in enzootic areas fever of unknown origin

Laboratory examination: identification of ricketsia in a strained, squash preparation of tissue taken from endothelial tissues at postmortem. OR take sample from large blood vessels (e.g. Jugular vein) using special needle (Sarah needle) and crush in glasses, dry, fix and stain with Giemsa to confirm the presence of Rickettsia

Pm picture

Serological tests: ELISA

NB: The clinical and pathological findings are not specific, thus differentiation from other diseases characterized by fever septicemia and nervous signs may be difficult

Treatment

Use of antibiotics: Tetracycline

Control

Control of ticks: Regular dipping of cattle at 3-7 days interval
Early treatment of infected animals: recovered animals remain immune; they remain immune for long period if parasites are in the endothelial tissues.

DISEASES CAUSED BY PROTOZOA TC "DISEASES CAUSED BY PROTOZOA" \f C \l "1"

1. TRYPANOSOMIASES TC "1. TRYPANOSOMIASES" \f C \l "2"

These are group of protozoan diseases manifested by a wide spectrum of features depending on the species of the parasites and host involved. The prominent features are intermittent fever, Anemia emaciation, reduced productivity and high mortality

Terminologies

A: Nagana:

The tsetse transmitted trypanosomiasis group of disease caused by the salivarian trypanosomes (*Trypanosome congolense T. vivax, T simiae, T suis T. brucei, and T uniform*) and are characterized by an acute or chronic course, fever, anemia, emaciation and a heavy mortality rate. Transmitted principally by *glossina* spp; the tsetse flies

B: Sura

This is biting flies (not Glossina) transmitted protozoan disease affecting camels and horses, and caused by *T. evansi.* Biting flies can be Tabanid and stomoxys
Clinically: manifested by central nervous system involvement

C: Dourine

This is a contagious trypanosomiasis of horse caused by *T. equiperdum* and transmitted by coitus. It is characterized by

inflammation of the external genitalia, cutaneous lesions and paralysis.

D: Sleeping Sickness
Tsetse fly transmitted infection of man caused by *T. gambiense* and *T. rhodensiese*

E: Chagga' s Diseases
This is a Zoonotic disease affecting pigs and dogs caused by *T. cruziae*.

BOVINE TRYPANOSOMIASIS (NAGANA)

This is most important disease-affecting cattle in Africa. Is a problem of about 68% of the African continent characterized by intermittent fever, parasitaemia, anaemia, loss of condition reduced productivity and frequently high mortality.

Etiology
The disease is caused by Trypanosomes: *T. vivax, T. Congolense, T. brucei.* They are all members of the salivarian group of trypanosomes and are transmitted via the mouthparts of their vector flies hence the name salivarian trypanosomes.

Epidemiology

- The disease is transmitted by tsetse flies, glossina spp (*G. fusca, G.Morsitans* in savannah area, and *G. palpalis*)
- All breeds of cattle are affected. But some breeds of cattle indigenous to Africa, such as N' Dama are resistant to the infection.
- Wild animals act as carriers e.g. warthogs, bush pigs, buffaloes etc

- Mechanical transmission may occur by other biting flies.
- Carnivores (Lions, Hyenas, dogs and cats) can be infected with *T. brucei* through feeding on a carcass of infected animal.

Pathogenesis

Flies are infected when feeding on infected host. The ingested Trypanosomes undergo development in the tsetse fly producing infective METACYCLIC trypanosomes. Once infected, the fly will transmit Trypanosomes for the rest of its life.

Clinical signs

There are no pathognomonic signs and a clinical examination is of little help in pinpointing a diagnosis.

The incubation period is 5 – 10 days

Fever which is likely to be intermittent and to last for a long period (until parasites become scanty in blood)

Affected animals are dull, anorexic, have an ocular discharge and lose condition (progressive)

The lymph nodes are swollen.

Anaemia (pale mucous membrane and watery blood)

Occasional cases have diarrhoea and some have edema of the throat and underline

The animal become very emaciated, weak and die within 1 – 6 months

Many animal will have mixed infections and these are usually severe cases: Anaemia and sometimes sudden death

Some animals will show cerebral form of diseases, which is manifested by ataxia (loss of power of governing movement), circling and paralysis

Harsh coat and abortion may occur.

Many sick cattle lose the tail switch.

Diagnosis

Nagana is difficult to diagnose without laboratory services because it resembles any other chronic disease and has no

characteristic clinical signs or postmortem lesions. Thus diagnosis will base on

a. Epidemiology – presence of Glossina spp
b. Laboratory examination
    - Demonstration of the parasite in a blood smear. This is easiest if the animal is in the early stage of the disease and is febrile. At other time the number of parasites in the peripheral blood may be very small. Other special methods recommended are
    - Buffy coat method of concentrating and viewing the parasites; Haematocrit centrifuge method of concentrating the parasite into the buffy layer
c. Serological tests: CFT
d. Experimental animals: Guenia pigs

POST MORTEM PICTURE

- Petechiation on serosal membranes
- Lymph nodes and spleen are swollen
- Anaemia.

Differential Diagnosis

Anaplasmosis (high fever, jaundice and anaemia)
Paratuberculosis (no temperature reaction, good appetite and chronic diarrhea)
Round worm infection (Severe in calves, no temperature reaction and reduced appetite)
Traumatic reticulitis (chronic disease with diarrhea and loss of condition, abdominal pain, rumenotomy reveals the presence of nail)

Treatment

There are a wide range of Trypanocidal drugs available
Diminazene aceturate (Berenil ®) – 3.5mg/kg Bw intramuscularly for curative purposes

Homidium bromide and **Ethidium chloride** 1 – 2 mg/kg Bw intramuscularly. Effective in cattle and horses (curative)
Isometamidiun chloride (SAMORIN®) 0.25 – 1mg/kg Bw intramuscularly. Used as curative and prophylactic against *T. congolense, T. vivax.*
Quina pyramine dimethylsalfate (Antrycide®). Used as curative in *T. congolense and T. vivax* at 2.2 – 4.4 mg/kg Bw subcutaneous.
Suramin (Antripol®) Used against *T. brucei* in horses, camel and dogs as curative and prophylactic at 7 – 10 mg/kg Bw intravenously.

Control
Control of the tsetse flies requires a strict regimen of regular spraying of animals, spraying of foliage likely to harbor the fly, destruction of bush and fly trapping. These can be done by

1) Eradication of Glossina through:
   - Vegetation clearing. Desert formation
   - Use of insecticide of pyretroid group e.g. Grenade, Decatix, Ectomin, Dominex etc.
   - Hand catching of tsetse fly
2) Removal of wild animal (reservoirs)
3) Control of animal movements
   - Restriction of movements to free areas OR
   - Movement of animal when tsetse flies are not active (during the night)
4) Early diagnosis, chemotherapy and chemoprophylaxix
   - Applicable at present but limited to expertise, expense and development of resistance
5) Breeding of "sterile male technique" and breed tolerant (Trypanotolerant) cattle

2. THEILERIOSIS TC "2. THEILERIOSIS" \f C \l "2"

This is the group of infection caused by protozoan parasites of genus Theileria transmitted by ticks (hard ticks - ixodid ticks). The

disease affects domesticated and wild animals. Among Theileriosis includes:

i. East coast fever (ECF).
ii. Malignant Bovine Theileriosis: Caused by *Theileria lawrencei*
iii. Mediterranean Coast Fever: Caused by *T. annulata*. Characterized by pallor mucous membrane due to anamea, haemoglobinuria and jaundice. Transmitted by Hyalomma ticks
iv. Begin Bovine Theileriosis: Caused by *T.mutans* and manifested by fever, anorexia, and anemia. Transmitted by Amblyomma
v. Malignant Theileriosis of sheep and goats: caused by *T. hirci* and *T.ovis* . Occurs in sheep and goats and a syndrome similar to ECF
vi. Corridor disease: caused by *T. bovis*

EAST COAST FEVER

This is an acute disease of cattle and buffaloes characterized by fever, lymph node enlargements, weakness, emaciation and a high death rate.

Etiology

The protozoan parasite *Theileria Parva:* There are antigentically distinct strains of parasites showing great deal of variation in virulence.

Epidemiology

- This disease is transmitted by ticks, *Rhipicephalus appendiculatus* being the principal vector and sometimes Hyalomma (Brown ear ticks)
- Cattle and buffalo are the usual hosts with zebu cattle being more resistant than exotic breeds.
- The distribution of diseases is completely dependant on the distribution of the vector tick, and in tick-infected areas all animals become infected.

Pathogenesis

Infective particles, usually nucleated sporozoites of *T. parva*, are injected by the vector tick in its saliva when it is feeding. The particles pass to the local lymph nodes, develop into macroschizonts and are disseminated through the lymphoid system. In the lymphoblastoid cells they develop to merozoites, which migrate to erythrocytes and become piroplasms. The dominating pathological lesions is the damage caused to the lymphoid organs, with erythrocyte damage as a contributing factor in some cases

Clinical Signs

The commonest form of disease is acute form.
The incubation period is 1-3 wks depending on the virulence of the strain and the size of the infecting dose
The mortality rate is 5-50% in calves, =95% in adult.
Morbidity rate is low except if there is bad management
Calves in endemic areas do not show any sign except enlargement of lymph nodes (superficial)

a. The first clinical sign is dullness and staring hair coat
b. Inappetance and swollen external lymph nodes (parotids, prescapular and precural/ prefemoral lymph nodes)
c. Rise in temperature (41.5-42.0^{0}c) which remain high for long period of time
d. Drop in milk production
e. Profuse lacrimation and bilateral serious nasal discharge
f. Difficult breathing (dyspnoea) due to lung edema which causes death
g. In severe cases there is diarrhea, increased salivation, with dysentery but usually only late in the course of the disease
h. Petechiation on mucous membrane of buccal cavity, conjunctiva, vulva, nostrils but no Anemia in ECF
i. Emaciation weakness and recumbence lead to death after 7-10 days
j. Terminally there may be a frothy nasal discharge

k. Occasional cases of brain involvement occur characterized by circling, hence circling disease or **cerebral Theileriosis**: There are localizing nervous signs and convulsions, tremor, profuse salvation and head pressing

Pm Picture

In acute form

Froth from nostrils, trachea and lung, hydrothorax, hydropericardium

Lymph nodes enlarged, edematous and Haemorrhagic

Enlargement of liver and spleen.

Kidney cortexes is congested and have white spots on the surface.

Haemorrhages in caecum.

Ulcers (cigarette burns) in the Abomasal mucosa.

Diagnosis

Epidemiology: Presence of ticks (lack of tick control) and only cattle and buffalos are affected

Clinical signs

Lymph node smears; Koch' s Blue Bodies (KBBs) – Schizonts.

Blood smear: piroplasms

Pm-picture-froth & edema of lungs

Treatment

Buparvaquone (Butalex®): 2.5 mg\kg 1m at 48hrs internal (2doses)

Parvaquone (Parvexon/clexon®): 10mg1kg 1m at 48hrs interval (2 doses)

Halofuginone lactate: Two dosesx1.2mg/kg BW), but recrudescence (reccurrence) is common

Furosemides (Lasix®): Facilitate to remove fluid from body cavity and lung edema

Tetracycline: When given at early stage in local cattle

Anti-inflammatory drugs e.g. Glucocorticoids

Multivitamins

Control

Control vector: control against ticks by dipping using Acaricides

Restrict movement of livestock

Pasture spelling: Resting or leave pasture for a long time >6months

Use of dead -end host: Put donkey in the pasture which are not susceptible

Burning of pasture

Vaccination:

Vaccine is available against ECF and once an animal is immunized it is protected for 2 – 3 years.

Immunization consist of a simultaneous injection of the vaccine and a long acting Oxytetracline (OTC 30%)

Constant challenge is necessary to boost the immunity (natural booster vaccination)

Animals that can not be immunized are:

Younger than 4 weeks of age

Sick animals

Animals in poor condition

Pregnant animals in the last 3 months of pregnancy

Animals which were dewormed with Levamisole products in the last month.

3. BABESIOSIS (syn: Redwater, Red urine, Texas fever) TC "**3. BABESIOSIS (syn: Redwater, Red urine, Texas fever)**" \f C \l "2"

This is a protozoan tick borne disease of cattle, sheep, pigs and horses characterized by fever, intravascular hemolysis causing anaemia, haemoglobinuria and haemoglobinemia

Etiology

Protozoa Babesia causes the disease.

Cattle: *B. bigemina, B. major and B. divergens*

Water Buffaloes: *B. bovis and B. bigemina*

Sheep and goats: *B. motasi and B. ovis*
Pigs: *B. trantmanni and B. perroncitoi*
Horses*: B. equi, B. caballi*
Dogs: *B. canis, B. Gipson*

- The parasites are pear - shaped in RBCs

Epidemiology

- The distribution of causative agents (disease) is governed by the geographical distribution of the insect vector that transmit them
- The disease is transmitted by *Boophilus* ticks, *Rhipicephalus, Hyaloma* and *ixodes*
- Contaminated needles and surgical instruments can transmit the infection physically
- Strong immunity occurs after natural infection with most Babesia spp; if the infection recurs repeatedly the immunity is permanent. If the infection is not repeatedly the immunity will persist for a further 6 months and the host is susceptible again
- All races of cattle are equally susceptible, but zebu and Afrikander cattle have a higher resistance to Babesia than exotic breeds
- The greatest infection rate is in animals in the 6 - 12 months age group and infection is uncommon in animals over 5yrs of age.

Clinical signs

In field infection the incubation period is 2 - 3 wks subclinical infection occur fairly commonly in calves.

i) Dullness, Anorexia, and ruminal stasis
ii) Pyrexia 41.5^{o}C and staring coat

iii) Arched back due to abdominal pain
iv) Tachycardia, tachypnoea and increased heart beat sounds
v) Hemolytic crisis leads to anaemia and pallor mucous membranes
vi) Constipation and tenesmus whereby in late stages a profuse diarrhea that is characterized by pipe-stem may occur.
vii) Haemoglobinemia and Jaundice masking the anemia.
viii) Haemoglobinuria
ix) Pipe stem diarrhoea
x) CNS aberrations characterized by aggressiveness, staggering gait, posterior paralysis, convulsion and comas
xi) Pregnant animals often abort and lactating animals show drop in milk production

- Death occurs within 2 -3 weeks
- Mortality rate is 90% for severe cases

NB A Subacute syndrome also occurs, especially in calves, in which the fever is mild and haemogloinuria is absent.

PM Picture

- In acute cases, Jaundice is marked, spleen is enlarged, swollen and of soft, pulpy consistency
- The liver is grossly enlarged and dark brown in colour
- Gallbladder is distended with thick, granular bile
- Kidneys are enlarged and dark
- Bladder contains red brown urine
- Ecchymotic haemorrhages are seen in epicardium and endocardium.
- Blood-stained fluid in pericardial sac

- Intravascular clotting (Characteristic feature)

Diagnosis

- Epidemiology
- Clinical signs: Fever associated with haemoglobinuria and jaundice
- Laboratory examination: Blood from peripheral circular (Pear shaped parasites)
- Pm picture
- Serological tests: CFT, FAT, ELISA

Differential Diagnosis

Anaplasmosis: No haemoglobinuria
Leptospirosis: Shorter course and more severe in young animals than adult
Bacillary haemoglobÿÿuria: Liver flukes (epÿÿÿÿiology)

Treatment

- Imidocarb (1mizo1®): 1mg/kg BW 1m. Single injection
- Diminazene aceturate (Berenil®)

Control

Control ticks: Dipping etc
Other measures as in other tick borne diseases

4. TRICHOMONIASIS TC "4. TRICHOMONIASIS" \f C \l "2"

Trichomoniasis is a venereal disease of cattle characterized by sterility, early abortion and pyometra

Etiology

The causative agent of the disease is *Trichomonas fetus*

- This is a flagellated protozoa with a pyriform or fusiform Shape.
- The organism is rapidly killed by drying, by the presence of antiseptics, and by adverse conditions or environment

Transmission

- The disease is spread through natural service by breeding/mating cow with infected bulls.
- *Trichomonas fetus* is found only in the genital tract, vagina, cervix, uterus, and contents of the uterus of the cow and on penis, prepuce and possibly a slight distance into the urethra of the bull
- Cows usually develop a temporary immunity whereas when a bull becomes infected, the infection is permanent and only rarely does spontaneous recovery occurs.

Symptoms

(1) Vulvovaginitis and **cervicitis**

- This is observed by 4 to 9 days of service. Characterized by slight edema of the vulva and perivulvular tissues. There will be severe vaginitis (rough, rasp like corrugated mucosa).

(2) Infertility: The most common symptom of Trichomoniasis.
The infertility is caused by the death of fetus, usually 2 months after conception.
This is characterized by the necessity for many services per conception (repeated breeding and delayed calving) and by the

frequent occurrence of a prolonged time between estrual periods after breeding (interestrual periods)

NB: *T. fetus* produces a mild inflammation of the endometrium, cervical and vaginal mucous membrane, and attack the developing ovum, embryo and fetus. But do not interfere fertilization

(3) Abortion

- Abortion due to *T fetus* usually occurs between 3 and 4 months of pregnancy. However, most fetuses aborted before 90 days are not observed. The corpus luteum and cervical seal persist sop that there is no discharge of pus. When cervical seal opens a slight odourless discharge is observed.
- Those aborted from the third to fifth month of gestation are usually slightly macerated and are expelled with the fetal membranes around the fetus along with a variable amount of dark, reddish brown exudates.
- Retained placenta is very seldom observed following a Trichomonad abortion.

(4) Pyometra

- When death of the developing embryo occurs and abortion or maceration and absorption do not occur, pyometra develops
- Since estrus does not occur with pyometra, The condition may go unnoticed for 8 to 9 months unless a vaginal discharge is observed or a diagnosis made on rectal examination
- The pus in Trichomonad pyometra has a characteristic thin, yellow – gray, watery, flocculent consistency often containing yellow flakes of pus and shreds of fetal membrane and tissues. In rare cases fetal bones may be present.

Diagnosis

1) History: History of the introduction of a new bull or non pregnant into the herd.
2) Clinical signs
    - Gradual increase in problem of infertility characterized by failure of conception after frequent services
    - Prolonged period after service before the next estrual period
    - Cows apparently conceiving but coming into estrus several months later.
    - Occurrence of occasional early abortions and pyometra
3) Laboratory examination

In bulls: Flushing of preputial sheath with 5 – 10 cm of physiological saline solution and examine the organism under the microscope

In cow: By examination of the fetal membrane, fetal fluids and exudates in the uterus and from pyometra fluid

Differential Diagnosis

1. Vibriosis: (Abortion after 5^{th} months of gestation: endometritis and aborted fetuses containing pus in peritoneum)
2. Brucellosis: (Abortion after 5^{th} months of gestation: retained placenta, orchitis in male animals)
3. Leptospirosis: (Other signs in young animals: In subacute infection; abortion is accompanied by fall milk production, blood stained milk without physical change in the udder)

Treatment

1. Cows

Douching or flushing of the vaginal and possibly the uterus with 2 – 3 mm of Lugol' s iodine solution in 100 mm of water. This can be done once or at several intervals of a week or 10 days apart

2. Bull treatment

a. Local treatment of prepuce and penis with either Bovoflavin ointment, Acriflavin ointment or hydrogen peroxide. Also Sodium iodide can be used locally or by intravenous administration. However, repeated intervals are required, thus time consuming and expensive
b. Presently the treatment of choice is the oral administration of Dimetridasole at 50mg/kg Bw Also Metronidazole (drug used in human being against *T.vaginalis*) can be used intravenously at 10 – 15 mg/kg Bw.

Control

1) Use of artificial insemination instead of natural service
2) Early diagnosis and treatment of diseased cows
3) Prevent the introduction of Trichomoniasis into a herd by knowledge of the reproductive efficiency in the herd of origin of any stock to purchase; thorough examination of newly purchased suspect bulls by taking preputial samples for culture at least 3 to 6 times at weekly intervals.

DISEASES CAUSED BY FUNGI TC "DISEASES CAUSED BY FUNGI" \f C \l "1"

Fungal infection in domestic animals is of two types
Systemic mycoses
Dermatomycoses

SYSTEMIC MYCOSES TC "SYSTEMIC MYCOSES" \f C \l "2"

Systemic mycoses in animals are usually sporadic infection which occur by chance and cause non - specific syndrome because of variation in the organs in which they localize

Their epidemiology is based on the failure of the disease to be transmitted from one host to another, all infections being derived from sources in the environment

The causative fungi exist as saprophytes in organic matter and the portal of entry is by inhalation of dust containing fungal element, with primary focus developing in a lung

Some of common diseases caused by systemic mycoses are

i. **Abortion**: Rhizopus, Absidia, Mucor, Aspergillus
ii. **Mastitis**: Algae (protothecaspp) yeast (Cryptococcus neoformans0
iii. **Chronic pneumonia**: Coccidioides immitis, Aspergillus spp, Histoplasma spp, Rhinosporidia spp and candida albicans
iv. **Gastro intestinal tract diseases**: Rhizopus, Absidia, Mucor, Aspergillus, Histopasma, Candida albicans.

(b) DERMATOMYCOSES TC "(b) DERMATOMYCOSES" \f C \l "2"

- The most common disease of skin in domestic animals caused by fungi is RINGWORM.
- Ringworm of the skin is caused by the invasion of the Keratinized epithelial cells and hair fibers by dermatophytes

Etiology

The causative agents are fungi, which grow on the hair or skin or both. The common fungi are

Trichophyton spp

Miscrosporum spp

Trichophyton spp produce spores in long chains

Microsporum spp produce a Mosaic pattern of spores

Both are capable of growth on hair.

Epidemiology

Ringworm occurs in animals in all countries but more commonly where animals are housed in close proximity to each other for long periods

High humidity being conducive to multiplication of the fungi

Animal susceptibility is determined largely by immunological status so that young animals are most susceptible

Transmission between species occur readily and in rural areas 80% of human ringworms may derive form animals

Warm humid atmosphere and a slightly alkaline PH of the skin facilitate the growth of fungi

Transmission

- Direct contact with infected animals is a common method of spread of ringworm and licking with the tongue undoubtedly will spread of the fungus
- Indirect contact with any in animate objects, particularly bedding, harness, groaning kits, is probably more important

Pathogenesis

Ringworm fungi attack chiefly keratinized tissues, particularly the stratum corneum and hair fibers resulting in autolysis of the fiber structure, breaking of the hair and alopecia.

Exudation from invaded epithelia layers, epithelia debris and fungal hyphae produce the dry crusts characteristic of the disease.

Clinical findings

- The typical lesion is a heavy, gray – white crust raised perceptibly above the skin.
- The lesions are roughly circular an about 3cm in diameter, but in severe cases the lesions may coalesce to form large lesions.
- In early stages the surface below the crust is moist, in oldest lesions the scab becomes detached and alopecia may be the only obvious abnormalities.
- Lesion are most commonly found on the neck head and perineum but a general distribution over the entire body may occur particularly in calves
- Itching does not occur and secondary infection in unusual.

NB In all instances it is very important to remember that ringworm spreads from the center outwards.

Diagnosis

1. Epidemiology: Close proximity of animal, humidity calves mostly affected
2. Clinical signs: Crust/scab growing outwards without itching/irritation

Laboratory examination.

- Presence of fungal mycelia and spores
- Skin scrapings should be made after defatting the skin with either or alcohol, scraping are warmed gently in a 20% solution of either potassium or sodium hydroxide. Spores or mycelia are examined direct on the microscope or after culture, Spores are the diagnostic feature and appear as round or polyhedral, highly retractile bodies in chains (Trichophyton spp or Mosaics (Microsporum spp) in hair

follicles, epithelial scales, and in or on the surface of hair fibers.

Treatment

Local or systemic treatments used, the later when lesions are widespread

Local treatment

The crusts should be removed by scraping or brushing with soft wire brush and the medicament brushed or rubbed vigorously. Suitable topical applications include weak solution of Iodine, Whitfield' s ointment, 10% ammoniated mercury ointment, Solutions of quaternary ammonium compounds (1 in 2–to 1 in 1000), Ointment containing propionic and undercylenic acids and their esters, Hexetidine etc

NB The above topical treatments are probably of grater value in the early stages of

an outbreak when the lesions are small and few in numbers

Systemic treatment

I. Intravenous injection of sodium iodide (1g/14kg Bw) as a 10% solution.
   - More than one injection is often required and should be accompanied by topical application of fungistatic agents
II. Oral administration of Griseofulvin 5 – 7.5 mg/kg Bw daily for 7 days

Control

1. Isolation and treatment of infected animals
2. Hygienic improvements
   - House cleaning with commercial detergents
   - Avoidance of contaminated inanimate objects.

4. Supplementation of the diet, particularly with Vitamin A to young housed animals.
5. Vaccination of all animals in the group is recommended.

B: PORCINE DISEASES TC "**B: PORCINE DISEASES**" \f C \l "1"
ERYSIPELAS TC "**ERYSIPELAS**" \f C \l "2"
Erysipelas of pigs is the major disease of animals caused by *Erysipelothrix rhusiopathiae* but there are several other minor conditions occurring in other domesticated animals

A: ERYSIPELAS IN PIGS
Erysipelas in pigs appear as acute, septicaemic form aften accompanied by **diamond – shaped skin lesion,** and a chronic form manifested by a non-suppurative arthritis and a vegetative endocarditis.

i. Etiology
Erysipelethrix rhusiopathiae (insidiosa)
There are at least 22 serotypes, which are immunologically distinct.

Epidemiology
Pigs of all ages are susceptible although adult pigs are mostly susceptible than piglets. Piglets are resistant due to maternal antibodies providing active immunity without visible disease.
Source of infection

- **Contaminated soil, feed and water** (by feces of affected or carrier pigs)
- **Infected animals of other species**; birds and fish (fish meals)

Rodents and birds are means of spread of microorganisms from one herd to another.

Entrance of the organism is through ingestion or skin abrasions

Morbidity and mortality rates in swine vary considerably from place to place largely due to variation in virulence of the particular strain of the organism involved.

iii. Clinical findings (Incubation period 1 – 7 days)

Peracute form

Sudden death or collapse without premonitory signs.

Sometimes high fever and cyanosis (purplish-red in skin) may occur.

Acute form

- Initially there is high fever (42^{o}C) though the animal may be active and continue eating, then later on complete anorexia, thirst and occasional vomiting may occur.
- Marked red to purple discoloration of the skin in ventral surfaces of abdomen (**Diamond shaped)** In recovered pigs, the skin lesion disappears in about 10 days without residual effects.
- In outbreak, one or two may be found dead or severely affected pigs may show marked red to purple discoloration of the skin. Others may show: reluctance to rise, incoordination while walking, and conjunctivitis with ocular discharge may be present.
- Acutely affected pregnant sows may abort, and suckling sows may show agalactia
- After a course of 2 – 4 days the pig recovers or dies with diarrhea, dyspnoea and cyanosis evident terminally.

NB

- The classical diamond – shaped lesion is red urticarial plaque of about 2.5 – 5cm square or a more diffuse edematous eruption with the same appearance. The lesions are most common on the belly, inside the thighs, on the throat, neck and the ears and appear usually about 24 hours after the initial signs of illness

Chronic form

- **Arthritis** – A non-suppurative proliferative arthritis involving intervertebral joints, and limb joints : elbow, hip, hock, stifle and knee joints causing lameness and stiffness. The joints are obviously enlarged and are usually hot and painful
- There may be **alopecia, sloughing off the tail and tips of the ear, and dermatitis** in form of hyperkeratosis of the skin of back, shoulders and legs.
- **Endocarditis-** this is characterized by sudden death without previous illness, especially at times of exertion such as mating or movements between pens. Also progressive emaciation and inability to perform exercise may be noticed. With forced exercise dyspnoea and cyanosis occurs.

Iv: Necropsy Findings

In acute form

- Swollen and hemorrhagic mesenteric lymph nodes, congestion of lungs and liver, and infarcts in the spleen and kidney
- Small peteachial to ecchymotic haemorrhages throughout the body but best commonly under the kidney capsule, pleura and peritoneum.

Chronic form:

- Non – suppurative proliferative arthritis is characteristic
- Skin lesion – Diamond skin
- Endorcardial lesion – crumbly vegetations in the valves, often sufficient large to apparently block the valvular orifice.

V: Diagnosis

Epidemiology, Clinical signs – Diamond skin lesion, and Pm picture

Vi: Treatment

Penicillin and antierysipelas serum: Administrated together by dissolving the penicillin in the serum

Penicillin alone is adequate when the strain has only slight virulence

Chronic cases – cull the animal (do not respond well to either treatment because of the structural damage which occur at the joints and endocardial valves).

Vii: Control

- Avoid introduction of animals in the herd, which have succumbed to disease.
- General hygiene combined with distocking the place
- Slaughter all incontact animals
- Inject penicillin and serum to incontact animals

B: ERYSPELAS IN OTHER ANIMALS

i: Erysipelas in cattle.

Infection with *Erysipelothrix Rhusiopathiae* has been associated with arthritis in calves.

There is **non-suppurative arthritis** with ulceration of articular cartilages leading to lameness, recumbency and severe loss of condition.

ii: Erysipelas in Sheep

Erysipelas in sheep is associated with **arthritis** or **laminitis** and rarely as **endocarditis**

There is post dipping laminitis:

Dips that become grossly contaminated with organic mater are likely to cause the disease. Up to 90% of the flock may be affected

- Severe lameness begins 2- 4 days after exposure usually in one leg, sometimes in all legs. However, there is no underrunning of the hoof as in foot rot. No abscessation as in foot abscess and most cases recover spontaneously in 10 – 14 days but penicillin should facilitate recovery.

2. SWINE DYSENTERY

This is a contagious disease of pigs characterized clinically and pathologically by mucohaemorrhagic colitis.

i: Etiology

Treponema hyodysenteriae, a large strongly beta hemolytic spirochete

ii: Epidemiology

- Reported worldwide, causing heavy mortality in growing pigs (7 – 16 wks old) occasionally in piglets and adults
- The **source of infection is infected pigs**, Pigs that have recovered from clinical disease with or without treatment (carriers shade the organisms and infect in – contact animals for 50 – 90 days)
- **Infection is by ingestion** and is enhanced by conditions leading to oral – fecal cycling
- The **spread within a group is slow** taking up to 7 – 14 days and the disease may spread to involve adjacent or other pens of pigs over a 2 – 3 wk – period. However, **morbidity** within a group of pigs may approach 100%, and if the

disease is not treated the case fatality rate may rise up to 50%

iii: Pathogenesis

- Ingestion of microorganism leads to multiplication and invasion of colonic crypts. This is followed by progressive erosion of superficial epithelium, excessive mucus production, edema, hemorrhages of the lamina propria and pseudomembrane production.
- The erosive colitis is the cause of the diarrhea, dysentery and excessive quantities of mucus in the faeces
- Death usually results from chronic dehydration and bacterial toxaemia.

NB: Swine dysentery is a colonic malabsorption syndrome.

iv: Clinical findings

Most commonly the disease initially affects only few pigs within the group but spread over a period of 2 weeks to involve majority. The affected pigs show moderate fever, slight depression and some reduction in appetite.

Faeces are characteristic:

They are only partially formed, usually of porridge like consistency, passed without apparent conscious efforts and splatter on contact with the pen floor.

Affected pigs commonly defecate anywhere and on anything in the pen.

The faeces are light grey to black and on close inspection much mucus is present and flecks of blood and epithelial casts may be seen.

NB: Blood in feces generally occurs 2 -3 days after the initial onset of diarrhea.

- Affected pigs become **progressively dehydrated** and their abdomens appear gaunt and sunken
- **Death usually** occurs some days to weeks after the initial onset of signs and results primary from dehydration and toxaemia.

- Less common an outbreak may start with the sudden death of one or two pigs with no evidence of premonitory signs or terminal hemorrhagic diarrhea. This occurs more commonly in market age pigs and adults in the herd where the disease has been introduced for the first time.
- A **chronic form** of the disease with **persistent diarrhea and failure to grow** occur in some pigs with irreversible lesions of the colonic mucosa. The disease respond well to treatment but following withdrawal of treatment, the disease may recur within the same group of pigs.

V: Necropsy findings

- Inflammation and necrosis with varying degrees of hemorrhages into the lumen of colon
- The submucusal glands are enlarged and frequently visible through the serosa of the colon as opaque spots.
- There are no abnormalities in the small intestines
- The draining lymph nodes are enlarged and congested.

Vi: Differential Diagnosis

Swine dysentery must be differentiated from other disease causing diarrhea in growing pigs:

i. **Coliform gastroenteritis**: Initial sign are dead or severely sick pigs with fever, skin discoloration, anoxia and profuse watery diarrhoea.
ii. **Salmonellosis:** As in coliform gastroenteritis: Lesions are also present in the small intestine and other organs
iii. **Hog cholera:** High fever, conjunctivitis and dirrhoea, complete anorexia and vomiting)

Together with symptoms as of caliform gastroenteritis and Salmonallosis. This occurs in all ages, while the coliform gastroenteritis has close association with weaners.

NB: In all these diseases the onset and spread within a group is much more sudden and rapid than with Swine dysentery and death occurs earlier

vii: Diagnosis

Clinical signs – slight onset (insidious in onset), incomplete appetite, and faces are soft and mucohaemorrhages

Pm – Necrosis is the colon only.

Laboratory exanimations: Isolation of *T. hyodrosenteriae* from feces of affected pigs

Serological tests: Elisa, Micro-titration agglutination test (MAT) etc

Viii: Treatment

- Treatment for swine dysentery is usually administered to all pigs within the group.
- Treatment by water medication rather than feed medication is preferable in that it is generally easier and faster to institute.
- Feed medication is suitable for prophylaxis
- Pigs with severe hemorrhagic diarrhea and toxaemia may not drink sufficient medicated water and should be treated by parenteral injection.
    1. Organic arsenicals such as **Sodium arsamilate**
    2. Nitroimidazole: **Dimetridazole**
    3. Lincomycin; **Spectinomycin**

    NB: All water treatments should be given for 5 – 7 days feed medication for
    treatment should continue for at least 14 days and preferably 21 days

    4. Tylosin: At 8.8mg/kg BW intramuscularly for 3 - 7 days.

5. Lincomycin: At 11mg/kgBW intramuscularly for 3 – 7 days

Ix: Control

- Effective control of Swine dysentery is dependent on elimination of the source of the organism from the affected pigs, the prevention of reinfection and avoidance of the introduction of carrier animals into herds considered free of disease.
- Early treatment with adequate level of drugs for a sufficient length of time.
- Improvement of house

3. AFRICAN SWINE FEVER TC "**3. AFRICAN SWINE FEVER**" \f C \l "2"

This is a peracute, highly fatal, and contagious disease of pigs characterized clinically and at postmortem picture by viremia and hemorrhages.

i. Etiology

The disease is caused by unclassified virus: The virus is physically, chemically and antigenenically distinct from that causing hog cholera – *pest virus* (Family – **Togaviridae**) (Pigs artificially immunized against hog cholera as still fully susceptible to ASF virus.

ii. Epidemiology

- ❖ Morbidity rate is 100% and case fatality rate is approximately 80%

- African Swine Fever is indigenous to the African continent where **warthogs, bush pigs** and **forest hogs** act as **reservoirs** of the virus.
- **The method of transmission** of the disease from the reservoirs in wild pigs to the domestic pigs is via the **ARGASID TICKS** (*Ornithodoros moubata*)

  The viremic warthog provides a source of infection for the ticks.

  The virus can be maintained in warthog associated argasid ticks for long periods in the absence of fresh source of infection; So Argasid tracks act as reservoir as well as vector of infection (ticks can act as vector for up to 8 years after exposure to the virus)

  Sporadic outbreaks may occur in endemic areas when the virus spreads from infected ticks or warthogs to domestic pigs.

  The tick has main habitat in burrows, which are inhabited by the warthogs.
- In domestic pigs, infection spread via **oral and nasal routes**.

  Also the spread can be by **indirect contact** of infected pens, the **ingestion** of contaminated feeds and water and by feeding uncooked garbage contaminated by infected pigs.
- Transmission via the hog louse (*Haemotopinus suis*) is also common.
- Recovered pigs act as important source of infection remaining persistently infected and a carrier indefinitely.

iii. Pathogenesis

The virus invades through the tonsils and respiratory tract and replicates in the draining lymph nodes prior to the occurrence of a generalized viremia occurring 48 – 72 hrs post-infection.

Viremia persists for 2 – 4 weeks. The viremia leads to inhibition of fibrin formation and thrombocytopenia, which results into development of haemorrhages, serous exudation, infarctions, local edema and enlargement of tissues.

iv. Clinical signs

The incubation period varies from 5 to 15 days

Initially there is high **fever** (40.5^{o}C) appearing abruptly and persists, without other apparent signs for about 4 days

The fever then subsides and the pigs show marked **cyanotic blotching** of the skin.

This is followed by **depression, anorexia, huddling together, disinclination to move, weakness and incoordination.**

Extreme **weakness of the hindquarters with the difficulty in walk** is a characteristic sign

There may be **dragging of hind limbs with coordination remaining only in forelegs**.

Rapid pulse rate, **serous to mucopurrulent nasal and ocular** discharge may occur.

Dyspnoea and coughing are present in some pigs.

Diarrhea, sometimes **dysentery and vomiting** occurs in some outbreaks

Abortion in pregnant sows.

Survivors usually are carriers for life, although the virus is not continuously.

Death usually occurs within a day or two after the appearance of obvious signs of illness, and is often preceded by convulsion.

iv. Postmortem pictures.

- **Petechial hemorrhages** in all serous surfaces and in lymph nodes, epicardium and endocardium.
- Severe submucosal **congestion** and edema in the colon
- **Edema and congestion of the lungs**

- ❖ Thoracic and abdominal cavities are effused with **blood-tinged fluids**

V: Diagnosis:

- ❖ Clinical signs
- ❖ Pm pictures
- ❖ Serological tests: ELISA, RIA, FAT etc
- ❖ Laboratory examination: Isolation of virus from buffy coat – culture of lymph nodes etc.

Vi: Differential Diagnosis:

- ❖ **Hog cholera**: Constipation, conjunctivitis then diarrhea, and fever which persists throughout the course of illness. However, the diseases can be differentiated by laboratory tests, serological tests and geographical distribution.

Vii: Treatment

- ❖ No treatment is available.
- ❖ Once attempt, high possibility of turning to carrier state after recovery (Not recommended to treat).

Viii: Control:

Quarantine of infected premises
Slaughter of all infected and incontact animals and distocking for 3 months.
Suitable hygienic precautions are recommended.
Restock from clean herd.

NB: No vaccine is available.

C: CANINE DISEASES TC "**C: CANINE DISEASES**" \f C \l "1"

1: RABIES (LATIN WORD FOR MADNESS) TC "**1: RABIES (LATIN WORD FOR MADNESS)**" \f C \l "2"

This is an infectious disease of all warm-blooded animals characterized by nervous derangement, often by a change in temperament and progressive paralysis (Nervous derangement = motor irritation) . Rabies is always fatal in human being, and there is danger not only from being bitten by rabid animals, but also from contamination by saliva of wounds, cuts, fingers, eyes etc.

i: Etiology

A *Lyssavirus* (one of the **Rhabdovirus** group)

The virus is readily killed by sunlight and boiling; whereby air drying and disinfections takes longer time.

ii: Transmission

- The main way of transmission of the disease is through **BITE** by rabid animal (Virus present in saliva 5 days before appearance of clinical signs. Thus introduction of virus through broken skin either by bite, or existing fresh wound or mucous membrane).
- Other less common ways are:
  - Ingestion of infected tissue **(in carnivores)** especially when there is lesion on the buccal cavity.
  - Also virus may be present in the milk of affected animals
  - Indirect transmission through abrasions, cut fingers, eyes, wounds by contamination from saliva.
  - Inhalation (**Aerosol virus)**

iii: Source of infection

- The man source of infection is an infected animals. In many countries, dogs and cats are still the most important vector.
- Wild animals provide a reservoir of infection. Among of these wild animals are Foxes (Europe), Jackals & Bats (Asia) Foxes, Bats and coyotes (North America), bats (Central America), vampires & Bats (southern America), Hyena & Wild Carnivores (Africa).

iv: Incubation period

- The time elapsing between infection and onset of symptoms varies greatly with the **location of the bite**, its **severity** and the **quantity of virus** in the saliva.

- In most rapidly developing cases the symptoms may be shown as early as **9th day** after being bitten, at the other extreme cases signs, may appear **several months** after the incident.
- The average incubation period in dogs, sheep and swine are from 15 – 60 days, in horses and cattle 30 – 60 days, in man varies from 18 days - 6 months (In young animals the incubation period is shorter than in adult animals).
- Natural or acquired immunity appear to be a major influence on length of the incubation period. Adult dogs are more resistant to infection, and they have a longer incubation period than younger dogs. Adult animals may have been given vaccine or had a mild exposure in the past.
- Variation in susceptibility between species is present: Foxes, rats and coyotes are extremely susceptible; cattle, rabbits and cats are highly susceptible; Swine, dogs, sheep and goats are moderately susceptible; and birds are not susceptible.
- There is no immunity after natural infection, but immunity can be produced artificially by vaccination.
- Mortality rate (case fatality rate) is 100% once clinical signs appear.

V: Pathogenesis

Following deep introduction of rabies virus by a bite of rabid animal; Initial viral multiplication starts in the striated muscle cells at the site of infection. Then the virus infects the neuromuscular spindle providing an important site of virus entry into the nervous system. This leads to invasion of spinal cord,

then to the brain where ascending wave of neuronal infection and neuronal dysfunction occurs. The immune response during this phase of infection is minimal and explains why neutralizing antibody and inflammation are usually absent at the time of onset of encephalitic signs.

NB: Bite – initial viral multiplication at muscle cells – neuromuscular site – spinal cord – brain

Vi: Clinical signs

In dogs there are three stages of development of typical symptoms.

a) **Prodromal phase**.

The prodromal / dull stage is often not noticed, or, if it is, only scant attention is paid to it.

The habits of the dog change (change of behavior):

Disregards its usual playthings or companions.
Shows a tendency to hide in dark corners.
May appear itchy or irritable as regards its skin.
Frequent urination
Erection in male and sexual desire
Noisy Dogs become quiet and dull, while dogs that are normally of gentle, quiet disposition may become excitable.
Has hallucinations upon awakening

After 2 or 3 days of such behavior the next stage is reached.

b) Excitative phase (stage of excitement).

There is tendency towards violence.
The dog pays no attention to threatening.
It becomes **easily excited** and very uncertain in its behaviors
There is **photophobia**; runs in dark places.
Refuses its ordinary food but eats straw, stones, wood, coal, carpets and pieces of sacks (unusual objects).
May wander for long distance; in its travel **it bites and snaps at objects**, which it encounters, real or imaginary,

animate or inanimate, some rabid dogs bite several people.

If is shut up in a kennel, it persists continually in its efforts to escape. Should it be released or should it escape, it almost invariably runs away from home.

Drooling of saliva (Due to paralysis of laryngeal muscles).

Wanders aimless with open mouth.

There is **protrusion of 3rd eyelids**; the eyes are fixed, expressionless and the pulps are dilated.

Conjunctivae are reddened, and have mucopurulent exudates

Convulsion and incoordination may occur toward the end of stage.

The stage lasts for 1 – 7 days.

(c) Paralytic phase (paralytic stage)

The affected dog is less dangerous because of its inability to bite, but its saliva is effective.

- This is characterized by **paralysis**, especially of the lower and hindquarters.
- The dog begins to **stagger** in its gait, and finally falls.
- The lower **jaw drops**, the **tongue rolls out** of the mouth, and there is **great salivation.**
- **Barking ceases** (due to paralysis of the muscles of throat and larynx).

Vii: Postmortem pictures.

- There are no characteristic macroscopic lesions in the brain or any other organs.
- Traumatic injury and general or local hyperemia are seen.

- The microscopic lesion consists of a very diffuse or severe meningoencephalomyelits.
- The most characteristic alteration is the presence of an intracytoplasmic inclusion body that is known as the **NEGRI BODY**. When the tissue especially the hippocampus is stained with basic fuchsin and methylene blue, the inclusion body appears as a purplish-red body with centrally located blue granules. One or more Negri bodies are found in a single cell (ganglion cells).

Viii: Differential diagnosis.

- **Pseundorabies**: (Due to salivation) disease of swine and other animals. There is extensive pruritis, respiratory distress, diarrhea, vomiting, and no change of behavior.
- **Canine distemper**. (Due to encephalitis-change in temperament): There is diarrhea, vomiting and hyperkeratosis.
- **Oesophageal foreign** body (due to salivation): Evidence of foreign body, sudden occurrence.
- **Toxoplasmosis** (in brain – neurological signs): Intermittent fever and anemia.

Ix: Diagnosis.

- Based on **clinical signs**.
- Fluorescent antibody test on impression smear from the brain.
- **Histopathology**: Intracytoplasmic inclusion bodies (negri bodies) from Hippocampus in 10% formalin stored brain.
- **Animal inoculation**: Use mouse/mice to inoculate saliva/ or brain tissue from the rabid animal. Mice will develop clinical signs after 4-18 days.
- **Isolation of virus.**

NB: organs acting as source of virus:- Brain, Spinal cord, and salivary gland.

X: Treatment.

Not recommended.
Confine the rabid animal for at least 10 days.

Xi: Prevention / control

Vaccination. (Modified live vaccine)

In dogs and cats- 3 months of age and repeat annually.
Mass vaccination of dogs and cats and wild animals.
Strictly control of movement of animals between countries.
Eradicate all stray dogs.
Vaccination of wild animals (**vims - laden baits**)

Xii: Management of animals (Dog and cat) exposed to rabies.

For the case of vaccinated animals, revaccinate it and keep under observation for 90 days.
In case of unvaccinated animals bitten by rabid dog, euthanize/kill it or confine the bitten animal for 6 months.
If still surviving after 6 months, you can vaccinate it and confine it for 1 month, if still health you can release it.

Xiii: Management of Rabies Suspects

- Dogs showing evidence of rabies should be confined in a maximum security cage or compartment with a rock
- A regular patient card should be attached to the cage along with an additional card stating "RABBID ANIMAL. DO NOT HANDLE"
- After death the animal should be removed by attendant wearing heavy rubber gloves
- Head should be cut and transported to the VIC under refrigeration
- The animal should first be placed in leakproof metal container, which then is placed in a large leakproof container and packed with crushed ice for shipment. The container should bear the label " CAUTION – THIS

PACKAGE CONTAINS THE HEAD OF AN ANIMAL SUSPECTED OF HAVING DIED OF RABIES". A history of the case should be enclosed in an envelope attached to the outside container.

RABIES IN OTHER ANIMALS

i. CAT.

The excitement phase is more common than in dogs.

The cat attacks other animals and man with great vigor, and attempt to injure their faces with teeth or claws.

The course of the disease is usually shorter than in the dog.

The cat may be found in a field or garden unable to walk but still able to bite.

Occasionally cats (sometimes dogs) may die from rabies without any observed symptoms.

ii. CATTLE.

- These animals are affected through having been bitten by a rabid dog, cat or fox.
- The stage of excitement is short and the prodromal stage is most evident.
- Affected cattle behave in an unusual manner, they may bellow, salivate from the mouth, rumination and milk production cease, sexual excitement is noticed, and there are a great loss of condition.
- Death may occur from 2-6 days or more after the commencement of the condition.

iii. SHEEP, GOAT AND SWINE.

- Sheep and goats are affected in a manner similar to cattle.
- However, the stage of excitement is shorter than paralytic stage.
- Pigs become excitable, show muscular spasms before paralysis.

iv. HORSES.

- There is facial twitching, biting of woodwork or self-multilation, head tossing, frequent whinnying, abnormal posture, apparent lameness, ataxia and paralysis of hindquarters.
- The horse may continue to eat and drink until shortly before death.

2: CANINE DISTEMPER. TC "**2: CANINE DISTEMPER.**" \f C \l "2"

This is highly infectious disease of dogs characterized by pyrexia, Oculo-nasal discharge, gastrointestinal tract involvement (diarrhea), respiratory tract involvement, Neurological involvement and hyperkeratosis of the foot pads and the nose.

i. Etiology.

Paramyxovirus (CDV): Antigenically related to Rinderpest virus, Newcastle disease virus, measles virus, and Peste des petits ruminant virus.

ii. Epidemiology.

Worldwide, morbidity rate 95% and mortality 30-70%(50%).

All ages of animals are susceptible, but young animals between the age of 3 and 12 months are the most susceptible.

All members of canidae and mustelidae are susceptible to canine distemper. These classes contain all those animals that do not have retractable claws (as oppose to the felidae), and include; Dogs, Fox, Ferret, Mink etc.

Transmission: Transmitted through inhalation of aerosols contaminated during coughing or sneezing. The viruses are found in all body secretions.

* Age at the time of exposure is a dominant factor in determining the course and prognosis of distemper. Puppies without immunity usually die in about 2 weeks after exposure.

iii. Clinical signs.

The incubation period of the disease is stated to be from 4 to 21 days, though it may be longer.

The signs vary greatly, depending on what organs are involved (In old dogs 3 months).

- **Transient fever**, depression, and decreased appetite.
- **Conjunctivitis** that varies in severity, the mucous membranes of the eyelids becomes swollen, congested, and purulent discharge appears. This may make the edges of eyes to adhere to one another.
- **Keratitis and opacity** may be present.
- **Nasal discharge**, first serous, and later mucoid and purulent.
- **Sneezing and coughing** may occur, and breathing may be faster than normal.
- **Broncho-pneomonia**; coughing more frequently (bronchitis) harsh, dry and painful.
- The discharge from the nostrils increases in amount and is stained with blood streaks.
- **Gastro-enteritis**, vomiting and diarrhea, loss of appetite. In severe cases blood may be passed, and ulcers may be present in the mouth.
- **Encephalitis**; this give rise to convulsion, tilting of the head, moving in circles, paralysis of hindquarters (that may be accompanied by incontinence of the faeces and urine), change in temperament with tendency to viciousness may occur.

- **Integument system**; there is hyperkeratosis (Thickening and swelling) of the pads of the feet and nose (hard pad disease).
- If distemper occurs in puppies before residue teeth (temporary teeth) are worn-out, enamel **hypoplasia** (yellow or brown colored enamel) or **distemper teeth** develop. The syndrome differs from that usually observed in old dogs; dehydration, inappetence, and hemorrhagic diarrhea. The most constant lesion is HAEMORRHAGIC ENTERITIS.
- The mortality associated with uncomplicated disease in dogs is low, and encephalitis is the usual cause.

iv. Diagnosis.

- Clinical signs.
- Serological test – Elisa.
- Virus isolation.

V: Differential Diagnosis

i) **Infectious canine Hepatitis** (prolonged bleeding time, anaemia, vomiting & diarrhoea)
ii) **Leptospirosis** (Jaundice and haemoglobunuria)

Vi: Post Mortem Picture

- In Puppies: Haemorrhagic enteritis
- In older dogs: Catarrhal or purulent exudates in respiratory tract; in cerebellum and resipiratory tract: perivascular cuffing (inclusion bodies in epithelial cells)

Vii: Treatment.

b) **Good nursing** is highly important.
c) **Supportive therapy**, e.g. Vitamin B complex, fluid therapy, antibiotic and nutritional supplements.
d) In severe neurological signs, give **phenobarbitone and antibiotics**.

NB: The dog may recover after a week or 10 days.

Vi. Control.

- **Vaccination.**
  - Vaccination should be done on 8th - 12th week and a booster dose annually
  - Combined vaccines against Distemper, infections canine hepatitis and parvovirus; Distemper canine parvovirus, and Leptospirosis; are in the market.
- Affected dogs should be isolated and all incontact dogs should be given booster distemper vaccine.

3: PARVOVIRUS DISEASE. TC "**3: PARVOVIRUS DISEASE.**" \f C \l "2"

This is enteritis of an acute onset mostly of puppies.

i. Etiology: *Canine Parvovirus* (DNA virus)

ii. Epidemiology.

- Worldwide affecting mostly puppies than adult dogs (Puppies are most susceptible than adult dogs)
- Ingestion of fecal contaminated materials is the major route of infection.
- Affected dogs (puppies) shed the virus in feces for about 2 week.

iii. Clinical signs.

- Vomiting is the initial sign and can be severe or protracted.
- Anorexia, lethargy and rapid dehydration follow rapidly.
- Diarrhoea is usually seen within 1 – 2 days; the feces are either streaked with blood or dysentery and remain fluid until death or recovery.

Iv. Postmortem picture.

- Enlargement of small intestines with lumen filled with watery – bloody intestinal contents

- Necrosis or dilatation of intestinal crypts
- Collapse of intestinal villous (villi)
- Occasionally inflammation of the heart muscles

v. Diagnosis.

- History: no vaccination records
- Epidemiology: puppies mostly affected
- Clinical signs: vomiting and diarrhea

vi. Differential Diagnosis.

- Canine distemper
- Severe parasitism
- Infectious canine Hepatitis (Fever, keratitis, jaundice, petechiations etc)

vii. Treatment.

- Fluid therapy
- Antiemetic agents: Atropine
- Antibiotics
- Prompt intensive care

viii. Control

- Vaccination

D: EQUINE DISEASES TC "**D: EQUINE DISEASES**" \f C \l "1"

1: STRANGLES *(Distemper)* TC "**1: STRANGLES *(Distemper)***" \f C \l "2"

This is an acute disease of horses characterized by **inflammation of the upper respiratory tract** and abscessasion in adjacent lymph nodes.

Etiology

Streptococcus equi

ii: Epidemiology

- **Horses are the only** species affected.
- The disease occurs in animal **of any age** but particularly in the 1-5 year age group.

- Out breaks of the disease occurs during cold and wet weather.
- The source of infection in strangles is the nasal discharge from the infected animals, which contaminates pasture and feed and water troughs.
- Infection occurs by ingestion or by inhalation of droplets
- The infection is capable of persisting in the pharynx of clinically normal horses for up to 10 months and this provide a means of carrying the infection over long periods when the disease is dormant. Under adverse climatic condition the disease can flap up.
- Morbidity rate: 10 -100% and Mortality rate: 1-2%.

iii. Pathogenesis

Invasion by inhalation or ingestion leads to infection of pharyngeal and nasal mucosa casing acute pharyngitis and rhinitis.

The spread to local lymph nodes (by drainage) results in abscessation and the infection many spread to other organs causing the development of suppurative processes in the kidney, brain, liver, spleen, tendon sheaths and joints.

iv. Clinical findings

Incubation period is 4 – 8 days.

a) Sudden development/onset of the disease with complete anorexia, fever (39.5 – 40.5^{o}C).
b) Nasal discharges: first there is pharyngitis that may be so severe that the animal is unable to swallow, and attempts to swallow food or water are often followed by regurgitation through the nostrils.

c) Coughing: This is soft, moist which causes apparent pain and is easily stimulated by compression of the pharynx. The head may be extended to ease the pain in throat.
d) Swelling of lymph nodes: The affect lymph nodes are hot, swollen and painful. If treatment is not done at about 10 days, rupture may occur leading to discharge of thick, cream – yellow pus soon afterwards. For severe cases, pharyngeal, submaxillary and parotid nodes are involved.
e) Complications of the disease as a result of metastasis and abscess formation in other organs; lungs – development of acute pneumonia, cerebral involvement will lead to purulent meningitis which is characterized by excitation, hyperesthesis, rigidity of the neck and terminal paralysis. Abscession in liver spleen also may occur.
f) Purpura haemorrhagica (Allergic dermatitis).This develops after an attack of strangles has subsided due to the development of sensitivity to streptococcal protein.
g) Edema of the head and on lower parts of the body.

V: Pm Picture:

In the rare fatalities that occur, Pm picture will be: extensive Suppuration in internal organs especially the liver, spleen, lungs, pleura large vessels and the peritoneum.

VI: Diagnosis

1. Clinical signs: purulent nasal discharge and enlargement of lymph nodes.
2. Pm picture
3. Laboratory examination: Nasal swabs and discharges from the abscess can be examined for presence of *Streptococcus. Equi.*

Vii: Differential Diagnosis

- ❖ Equine Viral rhinopneumonitis (no coughing)
- ❖ Equine viral enteritis (no coughing)
- ❖ Equine influenza

NB: In all these, no marked enlargement of lymph nodes.

Viii: Treatment

- Parenteral administration of Sulfonamides
- Penicillin injection (***currently in use***)

Ix: Control

- Isolation of all affected animals
- Thorough cleaning and disinfections of houses, pails, brooms, gloaming brushes and burning of beddings
- Vaccination.

2: GLANDERS TC "**2: GLANDERS**" \f C \l "2"
Glanders is a contagious and high fatal disease of SOLIPEDS, occurring in either acute or chronic form, and characterized by nodules or ulcers in the respiratory tract and on the skin.

i. Etiology
Pseudomonas Mallei

ii. Epidemiology
Solipeds: Horses Mules and Donkeys are the species usually affected.
Horses tend to develop the chronic form, mules and Donkeys the acute form.
Man is susceptible and the infection is usually fatal, cases usually occur in persons working with the organism in the laboratory or in close contact with affected animal.

Animals that are badly fed and kept in a poor environment are more susceptible.
Infected animal or carriers (that have made an apparent recovery from the disease) are the important sources of infection.
Animals get the disease by ingestion or drinking of contaminated feed or water.
The infection spread through folder, utensils, contaminated by nasal discharge or sputum.
Glanders is restricted geographically to Eastern Europe, Asia, North Africa and East Africa.

iii. Pathogenesis
Invasion occurs mostly through the intestinal wall and a Septicemia (acute form) or bacteremia (chronic form) is setup.

iv. Clinical Signs
Incubation period; varies with several months in horses

- **In acute form (mules and donkeys)**
  - High fever, cough and nasal discharges
  - Ulceration on the nasal mucosa
  - Nodules on the skin of the lower limbs or abdomen.
  - Death due to septicemia occurs in few days.

- **Chronic form**

The signs are related to the lesions in the predilection sites:

Pulmonary localization; chronic cough, frequent epistaxis and labored respiration.

Nasal and skin form: formation of nodules, which later ulcerate, serous nasal discharge that later become purulent and blood stained, nodules may discharge pus of the color and consistency of dark honey.

Animals affected with the chronic form are usually ill for some months, frequently showing improvement but eventually either dying or making an apparent recovery.

v. Postmortem Picture

Acute form: multiple petechial hemorrhages throughout the body and a severe catarrhal bronchopneumonia with enlargement of bronchial lymph nodes.

Chronic form: Lungs – military nodules scattered throughout the lung; ulcers on the mucosa of respiratory tract (upper part); Nodules as ulcers on the skin.

vi. Diagnosis

- Clinical signs: lymphangitis and nasal ulcers.
- Pm picture
- serological test: CFT, ELISA, MALLEIN TEST
  - *(Mallein [0.1 ml] is injected intradermally into the lower eyelid. The test is read at 48hrs where positive animals have edema of the eyelid)*
- Laboratory examination.

vii. Treatment

Sodium sulfadiazine: For a period of 20 days

NB: Penstreptomycin does not respond

viii. Control

- Complete quarantine of the affected animals
- Early treatment of all infected animals
- Chronic cases should be destroyed
- Vigorous disinfections program for food and water trough and premises should be instituted to prevent spread while eradication being carried out.

3: EQUINE EHRLICHIOSIS TC "**3: EQUINE EHRLICHIOSIS**" \f C \l "2"

This is equine disease characterized by Jaundice, anemia, respiratory tract involvement and central nervous system signs.

i. Etiology

Ehrlichia equi (Rickettsia)

ii. Transmission

The method of transmission/ spread has not been determined, but transmission by insect, especially ticks is a favored explanation.

iii. Clinical Signs

- ❖ High fever (40 – 42^{0}C)
- ❖ Pallor mucous membrane
- ❖ Jaundice, anorexia and depression
- ❖ Increased respiratory movements
- ❖ Edema of extremities

iv. PM pictures

Petechiation throughout the body and edema of legs and vasculitis

v. Diagnosis

Clinical signs

PM picture

vi. Treatment

Broad spectrum Antibiotics e.g. Tetracycline

4. VESCULAR STOMATITIS TC "**4. VESCULAR STOMATITIS**" \f C \l "2"

Vesicular stomatitis is an infectious disease caused by a Virus and characterized clinically by the development of vesicles on the mouth and feet.

i. Etiology

Vesiculovirus (family Rhabdoviridae)

ii. Epidemiology

Primarily is the disease of horses; but affects also cattle, donkey and pigs. Goats and sheep are resistant.

Young animals are much more resistant to infection than adult animals

Humans are susceptible; infection causes influenza – like disease.

The morbidity rate varies considerably (5 – 10%). However in dairy herds may be as high as 80%. There is no mortality rate.

iii. Transmission

Insects, including biting flies and mosquitoes play a large part in the spread of disease both locally and from infected to clean areas.

The saliva and vesicular fluid from clinically affected animals are highly infective

Transmission by ingestion of contaminated pastures also occurs

iv. Pathogenesis

As in FMD, there is a primary viremia with subsequent localization in the mucous membrane of the mouth and the skin around the mouth and coronet.

v. Clinical signs

Incubation period varies from 2 – 5 days and longer

- Sudden appearance of mild fever and development of vesicles on the dorsum of the tongue, dental pad, lips and the buccal mucosa (sometimes vesicles do not appear)
- The vesicles rupture quickly and the resultant irritation causes profuse ropey salivation and anorexia
- In milking cows there is a marked decrease in milk yield
- Lesions on the feet and udder occur only rarely except in milking cows where teat lesions may be extensive and lead to development of mastitis
- In horses; the lesions are limited to the dorsum of the tongue or lips, the udder of the mare and the prepuce of the males
- Recovery is rapid, affected animals being clinically normal in 3 – 4 days and secondary complications are relatively rare
- In pigs; vesicles develop on or behind the snout or on the feet and lameness is more frequent than in other animals

vi. Diagnosis

- Epidemiology: host involved
- Clinical signs
- Serology: CFT

vii. Differential Diagnosis

- FMD
- Vesicular exanthema in pigs

  VD has low mortality than the two diseases; different species susceptibility; mild form and no mortality.

viii. Treatment

- Mild antiseptic mouthwash so as to comfort the animal and facilitate recovery

ix. Control

- Hygienic and quarantine precautions to control the infection within a herd are sufficient controls. The disease dies out of its own accord.
- Vaccination

5. AFRICAN HORSE SICKNESS TC "**5. AFRICAN HORSE SICKNESS**" \f C \l "2"

This is a highly fatal, infectious disease of horse, mules and donkeys characterized by pulmonary involvement, edema of the head and profuse sweating.

i. Etiology

Viral disease- *Orbivirus (*Family Retroviridae*)*

ii. Epidemiology

AHS is confined to the African continent, though some Outbreaks have happened in Iran, Pakistan, India, Turkey, Cyprus and Spain. The disease continues as an enzootic disease in Africa, recurring annually in most of Southern, equatorial and Eastern Africa, South of Sahara. In enzootic areas it is virtually impossible to raise horses.

The incidence of the disease is often seasonal because of the seasonal variations in the number of vectors.

The disease is spread by the passive transfer of very small quantities of blood by biting insects. **Biting midges & Culicoides** are most probable vectors. Others are **ticks** (Hyalomma), **Mosquitoes** (Aedes, Anopheles & Culex)

NB: Spread does not occur between animals in direct contact unless the vector insect is present.

- Artificial transmission of the disease may occur by intravenous injection of blood.

- Clinically affected equidae (horses & donkeys) are the major sources of infection while Elephants, Zebra and Dogs are natural reservoirs of the Virus.
- Morbidity rate is 100% (However varies with the number of vectors present) while Mortality rate in susceptible horses (90%), Mules (50%) and low in Donkeys.

iii. Pathogenesis

African horses sickness is the disease of **vascular endothelium**, with virus clones affecting endothelium in different organs, resulting in a variety of "forms" of the disease.

iv. Clinical findings.

The incubation period in naturally infections is about 5-7 days.
Three clinical forms of the disease occur, an **acute or pulmonary form**, **a Cardiac or subacute form in susceptible and a mild form known as horse sickness fever.**
An intermittent fever of 40 - 41^{o}C is a characteristic of all forms.

<u>Acute Form</u> (Pulmonary horse sickness)
This is the most common form in susceptible animals.

- Initially there is **Fever** that is followed by **laboured breathing** and **severe coughing.**
- Profuse **nasal discharge** of yellowish serous fluid and froth.
- **Profuse sweating**, **staggery gait and recumbence**.
- The **appetite may be good** until the breathing become so laboured that the animal is unable to eat.
- Death occurs after **4 - 5 days.**

Sub acute form (Cardiac horse sickness).

This is most common in enzootic areas with longer incubation period (up to 3wks).

Slow development of **fever** that persists for long period.

Most common sign is **Edema in the head region**, particularly in the temporal fossa the eyelids, lips and chest.

Bluish oral mucosa.

Petechiation under the tongue.

Restlessness due to mild abdominal pain.

Auscultation of the heart and lungs reveals evidence of **hydropericardium, endocarditis and pulmonary edema**

Paralysis of oesophagus with inability to swallow and regurgitation of food and water through the nose is common.

The mortality rate is not as in acute disease but recovery is prolonged.

A mixed form may occur with pulmonary form following the cardiac form.

C) **Mild Form** (Horse sickness Fever)

- This presents **no diagnostic signs** and may go unrecognized.
- Occurs in areas in which the disease is enzootic and when an existing immunity is partially overcome.
- **Fever (Transient)** 40.5 ^{0}C over a period of 1-3 days but returns to normal about 3 days later.
- **Poor appetite**.
- Slight **conjunctivitis and Moderate dyspnoea**.

v. Necropsy findings

- Gross findings in acute cases include severe **hydrothorax**, pulmonary edema and Moderate **ascites**. Congestion of liver, the pharynx, trachea and bronchi are filled with yellow serous fluid and froth.

- In cases of cardiac form; **marked hydropericardium, endocardial haemorrhages**, **myocardial degeneration and anasarca**, especially of the supra orbital fossa.

vi. Diagnosis

- Epidemiology-in TZ the disease occurs during rainy season.
- Clinical signs
- Pm Picture.

vii. Treatment.

- Supporting therapy; in cardiac form; Good nursing, antibiotic injection, adlib water and Furosamine (Lasix®) to remove lung edema.

viii. Control.

- Vaccination- Annually in enzootic areas.
- Vaccination and quarantine horses at the point of embarkation.

6. EQUINE INFECTIOUS ANAEMIA (Synonymous: Swamp fever) TC "**6. EQUINE INFECTIOUS ANAEMIA (Synonymous: Swamp fever)**" \f C \l "2"

This is contagious disease of horse characterized by jaundice, edema, anemia and CNS involvement.

i. Etiology: *Lentivirus* {Family Retrovirus}

ii. Epidemiology

All breeds and age group of horse are susceptible

Biting flies particularly Tabanidae and mosquitoes can transmit the infection.

Infection can also be transmitted through contaminated surgical instrument, needles and injection of minute quantities of virus.

The morbidity rate varies considerably and may approach 100% where the population of carrier horses and insect vectors are particularly dense

iii. Pathogenesis

The possible scheme of pathogenetic events includes:

- Primary entry and infection of macrophages
- Destruction of macrophages and release of virus and its components
- Production of antibodies to antigenic components
- Formation of antigen – antibody complexes, which induce fever and glomerulitis
- Specific complexes cause hemolytic or phagocytosis by activating the reticuloendothelial system

iv. Clinical signs The incubation period is 2 – 4 weeks

- ❖ There is initial depression, profound weakness, and loss of condition
- ❖ Ataxia is a prominent sign in many cases
- ❖ Intermittent fever, which may rise and fall rapidly, sometimes varying as much as 1^0C within one hour
- ❖ Jaundice
- ❖ Edema of ventral abdomen, prepuce and legs
- ❖ Petechial hemorrhages in the mucosae, especially under the tongue and conjunctiva
- ❖ Anemia
- ❖ There is characteristic increase in heart sounds
- ❖ There is a considerably enlargement of spleen which may be detectable per rectum

- ❖ Pregnant mares may abort
- ❖ Many animals show temporary recovery from this acute stage, others become progressively weak, recumbent and die after a course of 10 – 14 days of illness.

v. Necropsy Findings

- ❖ There may be subcutaneous edema, jaundice and petechial or ecchymotic hemorrhages
- ❖ Enlargements of spleen, liver and local lymph nodes
- ❖ In chronic cases; emaciation and pallor of tissues

vi. Diagnosis

- ❖ Clinical diagnosis
- ❖ Pm picture
- ❖ Serological tests: CFT, ELISA.

vii. Treatment

- ❖ No specific treatment is available.
- ❖ Supportive treatment including blood transfusion, and haematinic drug may facilitate clinical recovery.

viii. Control: Test and slaughter policy, and No vaccine is available

7.EQUINE ENCEPHALOMYELITIS (Syn: Viral encephalomyelitis of horses) TC "**7.EQUINE ENCEPHALOMYELITIS (Syn: Viral encephalomyelitis of horses)**" \f C \l "2"

This is an infectious disease affecting horses and characterized clinically by signs of deranged consciousness, motor irritation and paralysis.

i. Etiology : Three strains of the causative *Alphavirus* (Family Togaviridae) exist: The Eastern (EE), Western (WE) and Venezuelan (VE).

ii. Epidemiology

- ❖ The disease is restricted to the Americas; USA, Canada, Venezuela, Brazil and Argentina.
- ❖ Morbidity varies widely depending upon seasonal conditions and the prevalence of the insect vectors.
- ❖ Man is also susceptible to the disease; the infection in man is mild, influenza like illness in which recovery occurs spontaneously.
- ❖ Spread occurs by insect bites, chiefly from mosquitoes, ticks, blood sucking bugs, and chicken mites and lice.
- ❖ Wild birds act as the reservoir of the infection.
- ❖ Spread may occur occasionally from horse to horse by direct contact or by insect transmission, but the major route of spread appears to be from birds through insects to horses and man.

iii. Clinical signs

The diseases caused by different viruses are clinically indistinguishable.

The incubation period is 1 – 3 weeks

In early stages there is fever, which may be accompanied by anorexia and depression, but the reaction is so mild that it goes unobserved.

CNS involvement:

The early nervous signs include hypersensitivity to sound and touch and apparent blindness, affected animals may walk blindly into objects or walk in circles.

Involvement of muscle movements occurs leading to tremors of shoulder and facial muscles and erection of penis.

The animal stand with the head hung low; appears to be sleepy and may have a half-chewed mouthful of feed hanging from lips.

Paralytic stage:

Inability to hold up head and the tongue may hang out.

Incoordination, particularly in the hindlegs and circling is common.

Defecation and urination are suppressed and the horse is unable to swallow.

Complete paralysis is the terminal stage; the horse goes down, unable to rise and usually dies within 2 – 4 days from the first signs of illness.

iv. Necropsy findings

There are no gross changes.

Histological examination of brain reveals perivascular accumulation of leukocytes and damage to neurons. No inclusion bodies are present

v. Diagnosis

- ❖ Clinical signs
- ❖ Epidemiology
- ❖ PM picture
- ❖ Serological tests: CFT, ELISA.

vi. Treatment: Only supportive treatment should be undertaken

vii. Control

- ❖ Quarantine and Vaccination of all horses

DISEASES OF SMALL RUMINANTS TC "DISEASES OF SHEEP AND GOATS." \f C \l "1"

1. CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP) TC "1. CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)" \f C \l "2"

This is highly contagious disease of Goats characterized by pneumonia, serofibrinous inflammation and pleurisy.

i. Etiology

Mycoplama mycoides var. Mycoides (large colony type) (or Mycoplasma strain F 38)

NB: The disease has many similarities clinically and at necropsy to CBPP But is not transmissible to cattle.

ii. Epidemiology

The disease is readily transmitted by inhalation.
Carriers (recovered animals) and infected animals are the source of infection into the flock
Reports of the disease come mostly from North Africa, East Africa, Spain, Asia, and India.
Morbidity is 100 % and case mortality of 60 - 100 %.
Young animals are relatively resistant compared to adult.

iii. Clinical Signs

Incubation period is 6 – 10 days.

- Coughing (chronic) and nasal discharges (catarrhal).
- Dyspnoea.

- Lagging behind the flock.
- Lying down a lot of time, but the animal can stand and walk.
- Fever (41.5 C)
- At terminal stage there will be mouth breathing, tongue protrusion and frothy salivation.
- Death occurs 2 or more days after appearance of signs.
- Under adverse climatic conditions the disease may occur in a septicaemic form with little clinical or postmortem evidence of pneumonia

iv. Pm Picture

Normally the lesions may be confined to one lung:

- Thickening and inflammation of the pleura often with heavy deposits of fibrin, serous effusion containing shreds of fibrin.
- The affected lobules show various stages of gray and red hepatization (marbled lung)
- Affected areas of lung are commonly covered with a lemon- yellow deposit containing much pleural exudates.
- Pleural cavity contains clear, straw-coloured exudates.
- In less acute cases, soft gelatinous adhesions between the parietal and visceral pleurae may be found.
- The bronchial lymph nodes are swollen and edematous.

  NB: There is no formation of sequestra in the lungs; the interlobular septal tissue is much less involved.

v. Diagnosis

History: The presence of clinically infected animal or carriers.
Clinical Signs
Pm Picture

vi. Treatment

- Tyrosin tertrate (10 mg /kg Bw)
- Tetracycline (O T C) 15 mg /kg Bw)

vii. Control

1. Vaccination: Alluminium hydroxide terpene vaccine

2.OVINE AND CAPRINE CONTAGIOUS OPHTHALMIA TC "**2.OVINE AND CAPRINE CONTAGIOUS OPHTHALMIA**" \f C \l "2"

This is a disease of sheep and goats characterized by conjunctivitis and keratitis.

i. Etiology: *Rickettsia conjunctiva*

* The disease is not transmissible experimentally to cattle, Guinea Pigs or Rabbits.

ii. Transmission

Spread of disease may be:

Indirectly by flies, long grass and dust contaminated by the tears of infected animal.

Directly by means of exhaled droplets or immediate contact.

Predisposing factors:

Warm summer months when conditions are dry and dusty.

Heavy fly population (Musca spp)

Concurrent diseases, poor nutrition and adverse weather (low immunity)

Carrier animals (animals with high immunity) may carry the infection persistently in the eyes for a period of up to 250 days

The source of infection is infected animals and the carrier animals, and recovered animals has immunity that last for 2-3 years

iii. Clinical Signs

a) In Sheep

- ❖ Initially there is lacrimation and blepharospasms
- ❖ Then follows keratitis with cloudiness of the cornea and some increase in vascularity.
- ❖ The watery discharge later becomes purulent.
- ❖ Local ulceration of the cornea may occur causing collapse of the eyeball.
- ❖ Recovery commences in 3- 4 days and is complete at about 10 days. However, in some animals the cloudiness of the cornea may persist for several weeks or even permanently.
- ❖ Both eyes are equally affected in many cases and spread through the flock is rapid

b) In Goat

The disease is milder with little apparent keratitis (ophthalmia) Conjunctivitis is followed by the development of granular lesions on the palpebral conjunctiva.

iv. Diagnosis

- ❖ Epidemiology, Clinical signs
- ❖ Laboratory examination: swabs or scrapings from the affected conjunctiva should be examined for presence of *Rickettsia* in the endothelial cells.

v. Treatment

Chloramphenicol Ointments or Ethidium bromide (0.5% Ointment) or Oxytetracycline Ointment

vi. Control

- ❖ Isolation of affected animals, removal to grassier, least dusty pasture may reduce the rate of spread.

3. ORF: ***(Synonyms: Contagious Ecthyma: Contagious Pustular Dermatitis; Sore Mouth)*** TC "**3. ORF**: ***(Synonyms: Contagious Ecthyma: Contagious Pustular Dermatitis; Sore Mouth)***" \f C \l "2"

This is the highly infectious viral disease of sheep and goats characterized by the development of pustules and scabby lesions on the muzzle and lips.

i. Etiology: *Parapoxvirus* (family paxviridae)

ii. Epidemiology

- ❖ The disease occurs worldwide whenever sheep are raised causing unthriftness and some economic losses.
- ❖ The morbidity rate in naive flocks can be about 90% while mortality rate may range from (15-75%) due to the extension of lesions in the respiratory tract.
- ❖ Outbreaks occur at any time but are most common in wet condition.
- ❖ The disease is commonest in lambs 3-6 months of age although lambs 10 – 12 days of age and adult animals can be severe affected.
- ❖ Recovered animals are solidly immune for 2 – 3 years, but no antibodies appear to be passed in the colostrums and newborns of immune dams are susceptible.
- ❖ The most affected are sheep, goats and cattle.

- ❖ Spread in a flock is very rapid and occur by contact with other affected animals or inanimate objects such as ear-tagging pliers, emasculator etc.
- ❖ The occurrence in dry season may suggest that abrasion of the skin by dry feed may provide a ready portal of entry for infection.
- ❖ The scabs remain highly infective for long periods, but survival of the disease in the flock may be the result of chronic lesions, which survive for long periods on individual animals.
- ❖ In man typical lesions occur at the site of infection, usually an abrasion infected while handling diseased sheep or milking affected cows or by accidental means when vaccination. The lesions are very itchy and respond poorly to local treatment.

Papules: solid elevation of skin
Pustules: small collection of pus
Scabs: firm mass consisting of dry dead inflammatory exudates and epithelial debris.

iii. Clinical Signs

Lesions develop initially as papules and then pustules, stages that are not usually observed.

- ❖ Then thick, tenacious covering a raised area of ulceration, granulation, and inflammation develops.
- ❖ The first lesions develop at the oral mucocutaneous junction, usually at oral commisures.
- ❖ From here the lesions spread on to the muzzle and nostrils, the surrounding haired skin and, to a lesser extend, on to the buccal mucosa.

- Scabs appear as discrete, thick 0. 5 cm in diameter, or be packed close together as a continuous plaque.
- Affected lambs suffer hunger due to resticted suckling and grazing.
- Rarely systemic invasion occur and lesions appear on the coronets and ears, around the anus and on the nasal and buccal cavities.
- There is severe systemic reaction and extension down the alimentary tract that may lead to severe gastroenteritis, and extension down the trachea may be followed by bronchopneumonia
- Recovery is usually after 3 weeks.

Diagnosis

- Epidemiology: high rate of outbreak, dry season
- Clinical signs: Scabs
- Serological tests: CFT, ELISA

Treatment

- There is no specific treatment
- Removal of the scabs and application of ointments to facilitate healing
- The provision of soft, palatable food is recommended.

Control

Isolation of affected animals

Vaccination: vaccination is completely effective for at least 2 years.

4. BLUETONGUE DISEASE (Syn: Catarrhal fever of sheep).

TC "**BLUETONGUE DISEASE (Syn: Catarrhal fever of sheep).**" \f C \l "2"

This is a non - contagious infection of sheep and cattle characterized by Catarrhal stomatitis, congestion and blue discolouration of the buccal and nasal mucosae, rhinitis, enteritis and lameness due to inflammation of the coronary bands and sensitive laminae of the feet.

Etiology: *Reovirus* (genus Orbivirus).

Epidemiology

- The infection is widespread in the Africa continent, Middle East, America, Australia & Europe.
- Under natural conditions the infection is common in Sheep and Cattle, Antelope, camels but not in Goats. However, except **sheep** in other animals the virus produces little clinical illness.
- Among the sheep, sucking lambs (<2 months) are relative resistance to infection.
- British breeds and merinos are more susceptible than native Africa sheep.
- The spread of infection is only though insect bites; sand flies (culicoides), Mosquitoes (Ades), Argasid ticks (Ornithodoros), Sheep ked (Melophagus ovinum).
- Disease outbreak occurs according to the insect population and the disease is more prevalent in wet seasons and in low – lying areas, conditions which favour insect multiplication.

- The morbidity rate varies with the size of the insect population and the immune status of the sheep. For example, when the disease occurs in a flock for the first time the morbidity may reach 50 – 75% and the mortality 20 – 50%.

Pathogenesis

A viraemia occurs in the early stages of the disease and localization of the virus in vascular endothelium follows. Destruction of the vessel walls produces the characteristics epithelial lesions of blue tongue.

Clinical signs

Incubation period is of less than a week (2-4 days)

- First there is severe febrile reaction (40.5-41^{o}c). The fever continues for 5 or 6 days.
- 48hrs after temperature rise, nasal discharge. The nasal discharge is mucopurulent and often blood stained and the saliva is frothy.
- Swelling and edema of the lips, gums, dental pad and tongue occur (often these are cyanotic – Bluetongue).
- Excoriation/ sloughing off the buccal mucosa follows and the saliva becomes blood stained with the mouth having an offensive odor.
- Necrotic ulcers develop, on the lateral aspect of the tongue, which is swollen and purplish-blue in colour.
- Diarrhoea and dysentery may occur.
- Foot lesions; including laminitis and coronitis leading to lameness and recumbence normally occur.

- The appearance of the dark red to purple band in the skin just above the coronet due to coronitis is an important diagnostic sign.
- Death in most fatal cases occurs about 6 days after the appearance of signs.
- In **sheep in enzootic** areas the disease is much less severe and often inapparent. Two syndromes occur:
    - Abortive form in which the pyretic reaction is not followed by local lesions.
    - In subacute form the local lesion are minimal; emaciation, weakness and convalescence are severe.
- **In Cattle**: most infections are inapparent although a few animals may develop a clinical syndrome but not like that seen in sheep. Fever (40 - 41^{o}C), stiffness & laminitis excessive salivation, edema of lips, nasal discharge and fetid breath. There may be sloughing of the hoof and ulcerations in tongue, dental pad and muzzle.

Pm Picture

- Mucosal and skin lesion as above.
- Generalized oedema, hyperemia, and haemorrhages and necrosis of skeletal and cardiac muscles
- Aspiration pneumonia.
- Petechial or ecchymotic haemorrhages at the base of pulmonary artery are a distinctive feature.

Diagnosis

- ❖ Epidemiology – seasonality due to insect vector activities.
- ❖ Clinical signs.
- ❖ Pm picture
- ❖ Serological tests: ELISA.

Differential Diagnosis

- ❖ FMD (vesicles formation)
- ❖ ORF (Scab formation)
- ❖ Nairobi Sheep Disease (no laminitis)

Treatment

- ❖ Local irrigation with mild disinfectant solution.
- ❖ BSA for controlling secondary infection.

Control

- ❖ Quarantine
- ❖ Vaccination.

5. NAIROBI SHEEP DISEASE (Synonym: Kisenyi Sheep Disease). TC "**5.NAIROBI SHEEP DISEASE (Synonym: Kisenyi Sheep Disease).**" \f C \l "2"
This is a tick – borne viral infection of sheep and goats characterized by fever, gastro-enteritis, abortion and high mortality.

Etiology.: Bunyaviridae family.

Epidemiology.

- The disease has been recognized in Burundi, Eastern Congo, Kenya, Rwanda, Tanzania and Uganda (The disease of Eastern & Southern Africa).
- The disease is transmitted by the brown tick (*Rhipicephalus appendiculatus)* all stages of this tick can be infectious. Bont tick (*Amblyomma Variegatum*) and other species of Rhipicephalus occasionally can transmit the disease.
- Goats and sheep are susceptible animals, with sheep and goats reared in the endemic areas having a high level of innate resistance, and reactions in them are characteristically abortive and symptomless.
- Sheep and goats introduced in to the endemic areas react severely and may die (mortality rate – 70%).
- Young ones possess a high innate resistance than adult.
- A natural infection confers a lifelong immunity.

Clinical signs.

The incubation period is 5-6 days.

The acute reactions are characterized by:

Sudden onset of fever and anorexia.
Nasal discharge and dyspnoea
Severe diarrhoea and dysentery sometimes
Collapse and death within 3-4 days of illness
Pregnant ewes abort
Genital organs of ewes are swollen and congested.

Necropsy finding.

The striking pathology lesions are:

- Haemorrhagic gastroenteritis, which is more severe in the caecum and anterior colon.
- Lymphadenopathy and splenitis.
- Petechiation on the pericardium and ecchymoses involving the endocardium.

Diagnosis.
Epidemiology
Clinical signs
Postmortem lesions
Serological tests: enzyme-linked immunoabsorbent test (ELISA), Haemagglutination test etc

Differential Diagnosis
Heart water disease (due to fever & diarrhoea in less acute): But have CNS symptoms.
Blue tongue disease: But have lameness, bluishness of tongue, pads & lips

Treatment

- No treatment.
- Only supportive treatment.

Control
Tick control.
Vaccination of sheep and goats before importation into endemic area.

6. RIFT VALLEY FEVER (Synonyms: Enzootic hepatitis, Hepatitis enzootica). TC "**6. RIFT VALLEY FEVER (Synonyms: Enzootic hepatitis, Hepatitis enzootica).**" \f C \l "2"

This is a viral infection of ruminants and man characterized in young animals by fever, hepatitis, and death in pregnant animals by abortion and in man by an influenza-like disease.

Etiology: The causative agent is *Phlebovirus* (family Bunyaviridae).

Epidemiology

The disease is still confined to the Africa continent.

Sheep, Cattle, Camels, Domestic buffalo, Monkey, Man and Rats are highly susceptible and Goats moderately so, but pigs, Rabbits and Poultry are not.

The disease in domestic ruminants and camels is transmitted by biting flies; Mosquitoes and other blood-sucking insects. Man is infected through contact with affected animals or through handling infected tissues (via abrasions).

The incidence of the disease varies with the size of the vector population and is greatest in warm, most seasons.

The immune gained by an animal after natural attack is lifelong while immunity gained by calves following the ingestion of colostrums lasts for about 5 months.

Pathogenesis

The invasion of virus is followed by acute hepatic insufficiency caused by destructive of liver cells by the rapidly multiplying virus.

Clinical signs.

The incubation period ranges from 12-96 hrs.

- In lambs and calves there is sudden outset of high fever, incoordination that is followed by collapse and sudden death within 36hrs in 95-100% of affected lambs and 70% of young calves.
- In adult sheep and cattle abortion is the outstanding sign but the mortality rate in adult sheep may be as high as 20-30% and 30% in cattle. However, in fatal cases sudden death is preceded by a high fever for 1-2 days.
- Goats show a febrile reaction but few other clinical signs

In man there is an abrupt onset of anorexia, chills, fever, headache and muscular and joint pains. Deaths from the disease are rare, but some patients loose vision due to retinal haemorrhages.

Pm picture

- Extensive hepatic necrosis is the characteristic lesion in Rift valley fever.
- Other non-specific lesions include venous congestion and petechiation in the heart, lymph nodes and alimentary track.
- Macroscopically there is focal or diffuse necrosis of liver.

Diagnosis

Epidemiology.
Clinical signs.
Pm picture.

- Serological test: ELISA, CFT.

Differential Diagnosis

- Bluetongue (Due to abortion in enzootic areas): But there is high mortality rate.
- Ephemeral fever: Muscle stiffness.
- Enterotoxaemia: culture of intestinal contents, epidemiology – adult, young – diarrhea.

NB: But Rift valley Fever has characteristics hepatic lesions.

Treatment: No treatment.

Control

- Vaccination: The annual vaccination of all susceptible animals is recommended.

AVIAN / POULTRY DISEASES TC "**DISEASES OF POULTRY**" \f C \l "1"

1. SALMONELLOSIS TC "**1. SALMONELLOSIS**" \f C \l "2" TC "**1. SALMONELLOSIS**" \f C \l "2"

Salmonellosis is a disease of all animals including human being caused by a number of different species of salmonella and manifested clinically by one of three major syndromes; peracute, septicaemia, and acute or chronic enteritis.

Etiology

- *Salmonella typhimurium* (man & animals)
- *Salmonella paratyphi* (man)
- *Salmonella Pullorum* (poultry)
- *Salmonella gallinarum* (poultry)
- *Salmonella dublin* (cattle)
- *Salmonella anatum* (sheep,horses & goats)

- ❖ *Salmonella arbotus equi* (horses)
- ❖ *Salmonella choleraesuis* (pigs)
- ❖ *Salmonella typhi* (man)
- ❖ *Salmonella newport* (cattle and horses)

(a) FOWL TYPHOID

This form of salmonellosis clinically observed in **growers or adult birds**, although chicks can be affected.

Etiology: *Salmonella gallinarum*

Species affected:

- Chicken (domestic chicken), Turkey, Ducks, Pheasants (Jamii ya kanga), Guinea fowl, Peafowl (Bata mzinga), Grouse (Bata bukuni) and Quails.
- The causal agent is passed out by infected birds in the droppings whereby lateral spread is by ingestion of such material (contaminated food and water).
- *S. gallinarum* persists in faeces for at least a month and in infected carcasses for much longer periods.

Transmission

i. Man, rats, dogs, foxes and wild birds may carry parts of infected carcasses from flock to flock, and infected carcasses may also contaminate ponds and streams.
ii. Recovered birds frequently remain carriers for long periods and are assumed that the movement of such birds could readily be a means by which the disease spread.
iii. Egg transmission is rare but can occur with the opportunity for lateral spread of infection to incontact birds in the hatchery or rearing units.
iv. Attendants, feed dealers, chicken buyers, and visitors who travel from house to house and from farm to farm may carry

infection unless precautions are taken to disinfect foot wear, hands and clothing.

v. Trucks, crates, and feed sacks may also be contaminated and thus spreading the disease.
vi. Wild birds, animals and flies may be important mechanical spreaders, especially if they have been feeding on carcasses of dead birds or offal from packing plants or hatcheries.

Morbidity rate within the flock is high = 80 – 100%.
Mortality rate for untreated flock = 50%.

Clinical signs

Incubation period varies with the immune status of the birds and virulence of the strains: can be as short as 4-6 days in most susceptible flocks.

In growers and mature birds

- In acute outbreak, the first sign is an increase in mortality followed by a drop in food consumption.
- For birds in lay, a drop in egg production is noticed.
- Depression, with affected birds standing still with ruffled feathers and closed eyes & drooping of wings.
- Increased respiratory rate with rapid breathing & gasping.
- The most characteristic clinical signs are a watery to mucoid yellow diarrhoea.
- In birds that do not die within 2 or 3 days of developing these signs a chronic phase follows characterized by; progressive loss of condition and development of intense anaemia which produces shrunken, pale combs and wattles.
- Egg transmission is not a regular feature of the disease but can occur and leads to an increase in dead-in-shell

embryos and small, weak, moribund or dead chick on the hatching trays.
- Sub acute outbreak of the disease may result in a grumbling sporadic mortality over a long period.

In chicks and poults
- Dead chicks on the hatching trays.
- Affected chicks will show non specific and similar to those seen in pullorum disease; weakness, reluctance to move, a tendency to huddle, drop in food consumption, and yellow, pasty dropping which adhere to the feathers around the vent and gasping on lung involvement.

Post-mortem findings

The carcasses of birds dying in the acute phase of the disease have septicaemic and jaundiced appearance, subcutaneous vessels injected (prominent), and the skeletal muscles congested and dark in colour.

A consistent finding is a swollen friable liver, dark-red or almost black in colour and its surface has a distinctive coppery bronze sheen.

The spleen may be enlarged.

Catarrhal enteritis, particularly involving the small intestines.

The intestines typically contain viscous slimy, bile stained material.

The gall bladder is engorged with viscous slime bile.

Treatment

Therapy with a number of antibacterial agents will reduce clinical signs and mortality in a flock affected with fowl typhoid.

- Furazolidone (Nitrofuran) compounds given in water for 5 – 7 days (continuously) or in feed for 10 days are generally considered to be the best treatment routine.
- Other antibacterial agents: Sulfonamides (Triquin®, Mycomas®), streptomycin etc.

Control.

- Proper house hygiene: Use of strong disinfectants to clean the poultry house, foot wash etc.
- Culling of chronically affected birds.
- Early diagnosis and proper treatment of all disease birds.
- Control of movements of birds and other animals including man in poultry houses (Foot wash).
- Proper disposal of dead birds.

(b) PULLORUM DISEASE

Pullorum disease is seen predominantly in chicks less than 3 weeks of age, rarely the condition occurs in adult birds. Species affected are chicken, turkey and pheasants.

Etiology: *Salmonella pullorum*

Transmission.

- The most important mode of transmission is from an infected parent bird via the ovary to the newly hatched chick (Egg transmission).
- The infected birds become adult carriers with *S. pullorum* persisting in the ovaries and being excreted in the ova.
- Also lateral spread from chick to chick in hatcheries and rearing units occur
- Transmission may occur by cannibalism of infected birds, egg eating & entry through wounds.
- Thus pullorum disease is passed from hen to chick by vertical transmission and the there is rapid lateral spread from chick to chick in hatcheries and rearing units.

Clinical signs.

The first indication is usually excessive numbers of dead-in-shell chicks and deaths shortly after hatching (for chick hatched from infected eggs).

Affected birds show variable and non-specific signs; depression with tendency to huddle, respiratory distress, lack of appetite, and white viscous droppings that adhere to the feathers around the vent & drooping of wing.
The mortality varies considerably and in extreme cases can be 100%.
Sub acute form may occasionally be seen in growing birds; and shown by lameness, swollen hock joint and poor growth rates.
Older birds appear listless; have pale and shrunken combs and sub optimal egg production may be the only signs of the disease in this disease age group (older birds).

- Both morbidity & mortality are highly variable and are influenced by age, strain susceptibility, nutrition, flock management and characteristic of exposure. The greatest losses usually occur during the 2nd week after hatching with a rapid decrease during the 3rd & 4th week of age.

Post-mortem picture.
Chicks that die shortly after hatching are likely to have peritonitis with an inflamed, unabsorbed yolk sac. The lungs may be congested, and the liver is dark and swollen with haemorrhages visible on the surface.
In chicks that die after showing signs of disease for 1 or 2 days, the caeca are enlarged and distended with casts of hard dry necrotic material, discrete, small, white necrotic foci are found in the liver, lungs, myocardium and gizzard wall. Also the gall bladder will be engorged with viscous slime bile.
In growers: arthritis on hock joints. The joints are enlarged due to the presence of excess lemon or orange colored gelatinous material around the joints.
In adult birds: Abnormal ovary with ova irregular cystic, misshapen, discolored and pendunculated with prominent thickened stalks.

Diagnosis.
Epidemiology – age

Clinical signs.
Pm picture.
Serological test: Agglutination test.

Treatment.
Antibacterial agents e.g. Furazolidone (Nitrofurans), mycomas, chlortetracycline, Chick formula, Triquine and Colistin.
Sulfonamides: - sulfaquinoxaline, sulfadiazine etc.

Control: Test and removal of the affected parent stock
- Proper house hygiene.

(c) SALMONELLOSIS IN OTHER DOMESTIC ANIMALS.
Infected animals excrete salmonella organisms in faeces and contaminate the environment.
Carrier animals may pass organisms constantly or intermittent in the faeces. The infection normally occurs by ingestion of contaminated material such as pasture, feed or water.

Predisposing factors.

Physiological statue of the animal.
Level of nutrition.
Management factors: Housed animals generally are more susceptible to infection from purchased feeds containing animal byproducts than are pastured animals, e.g. bone meal, fish meal, stored feeds (contaminated by the dropping of rodents), etc.
Intercurrent infection: may render cattle susceptible to salmonella infection.

Clinical signs.
i. Cattle:

The acute disease:
Fever, dullness and loss of appetite.
Loss/ drop in milk production.
Acute diarrhoea – has offensive smell, watery faeces, sometimes blood stained, mucus and necrotic tissues.
Pregnant animals may abort.
Recovery is slow, taking several weeks.
Sub acute form:
Absence or presence of mild fever.
Pregnant animals may abort without clinical signs.
Dullness and loss of appetite.
Brown scours with offensive fluid faeces containing blood and mucus.
Calves.
Normally calves get the septicaemia form of disease that is characterized by:

- Profound depression, high fever, pneumonia, weakness, and may develop arthritis.
- Mortality rate may range from 10 – 20%.

ii. Sheep and Goats.
Sudden death (septicaemia form).
Diarrhoea, dysentery and collapse.
For less acute cases, diarrhoea may be intermittent.
Loss of condition / weight and falling off of fleece.
Pregnant animals may abort.

iii. Pigs.
Young piglets (pigs) up to 4 months are most susceptible:

- Fever and diarrhoea,
- Purple colouration of the skin on ears and abdominal walls.
- Death may occur within 3 – 4 days.
- In sub acute or chronic may be the only clinical signs is diarrhoea.

- Sometimes may show emaciation with pulmonary complications.

Pm picture.

- Swollen, edematous and Haemorrhagic mesenteric lymph nodes.
- Jaundice and pneumonia (calves).
- Severe enteritis.
- Necrosis in liver and kidney.

Diagnosis.

Epidemiology.

- Clinical signs.
- Pm picture
- Laboratory examination: Faecal swabs for culture and isolation of *salmonella spp*.

Treatment.

- Chloramphenicol (injection) – 20mg/kg BW – IV 6 hrs 3 days.
- Nitrofurazones: orally at 20mg/kg BW daily for 3 – 5 days.
- Vigorous disinfections of building (proper house hygiene)
- Proper disposal of infected material should be carried out. (Carcasses should be burnt).

NB: Salmonellosis is a zoonotic disease, care must be observed when dealing with animals or animal products.

2: INFECTIOUS CORYZA (Synonyms: Fowl Coryza) TC "2: INFECTIOUS CORYZA (Synonyms: Fowl Coryza)" \f C \l "2"

Infectious Coryza, as its name implies, is a disease of the upper respiratory tract characterized by seromucoid nasal discharge, conjunctivitis and facial oedema.

Etiology: *Haemophilus paragallinarum* (Haemophilus group of bacteria)

Epidemiology

- The disease occurs principally in chicken, but has also been reported in Pheasants and Turkeys
- All ages of fowls are susceptible but older birds tend to react more severely.
- Birds, which have recovered from field infection, are said to be immune to reinfection for at least a year.
- The main source for disease spread is clinically affected and carrier birds.
- Airborne spread of the disease is the major mean of transmission of the disease.
- The disease occurs worldwide.
- Factors that predispose to more severe and prolonged disease include:
    - Intercurrent infection with other pathogens, such as Fowl pox, Infectious Bronchitis, Infectious Laryngotracheitis, Mycoplasma and Pasteurellosis.
    - Cold and wet conditions.

Clinical signs

The incubation period ranges from 3 – 10 days

The bacteria show tropism for the mucous membranes of nasal passages, infraorbotal sinuses, conjunctiva, and occasionally lower trachea and lungs are affected.

- The first sign in susceptible birds is depression

- Serous nasal discharge
- Conjunctivitis
- Facial edema and swelling of wattles
- Reduction in appetite and production
- Rales
- Birds may have diarrhea with foul smell when complicated with other bacterial infection.

NB: If uncomplicated by other infections, mortality rate is very low and the course of illness
is usually 14–21 days.

Gross Lesions

- Mucoid or catarrhal inflammation of the nasal passages, infraorbital sinuses and conjunctivitis
- Mucoid or catarrhal inflammation of upper trachea and occasionally lungs and air sacs
- Subcutaneous edema of the face and wattles is prominent

Diagnosis

Epidemiology
Clinical signs
Pm pictures (if any)
Laboratory examination: nasal exudates swabs to culture the Heamophilus organism
Serological tests: Plate and tube agglutination test, Agar gel precipitation, and Fluorescent antibody test

Treatment

- A number of drugs reduce the worst effects of the disease, and these include: Sulphathiazole, Streptomycin and Tetracycline given for up to 7 days in drinking water.

Control

- The introduction of new stock should be limited to day-old chicks or old birds, which are known to be free from the infection (so that to prevent the spread).

- Proper housing of all birds
- In non-endemic areas, eradication may be done by removal of survivor flock and the thorough cleaning, disinfection, and resting of building for at least 2 weeks before restocking with clean birds.
- Vaccination: using killed whole culture of organism (BACTERIN) given twice at 3 weeks apart starting at 17th week of age.

3. INFECTIOUS LARYNGOTRACHEITIS (ILT) TC "**3. INFECTIOUS LARYNGOTRACHEITIS (ILT)**" \f C \l "2"
This is an acute disease of chicken characterized by signs of respiratory depression, gasping and expectoration of bloody exudates.

Etiology: α– herpesvirus (alphaherpesvirus); Family Herpesviridae.

Epidemiology

- ❖ The virus seems to be naturally infective only for the fows and occassionally the pheasants.
- ❖ All ages of fowls are susceptible, but young chicks are highly susceptible, heavier breeds are more susceptible than lighter breeds and males are more susceptible than females.
- ❖ Transmission of the disease is by aerosal route; the virus is present mainly in the exudates from the nares, oropharynx, trachea and conjunctiva.
- ❖ The virus enters the body through the upper respiratory tract and conjunctiva. Recovered birds act as carriers excreting the virus intermittently for long period, perhaps for several years.

- ❖ The living diseased bird excretes virus in the greatest amount; very important source of infection.
- ❖ Mechanical transmission also may occur by personnel, animals, birds, contaminated equipment and litter.
- ❖ Predisposing factors to more severe disease are:

Deficiency of vitamin A
Excess ammonia in the atmosphere
Concurrent infections with other pathoges eg NCD, Fowl pox, Infectious bronchitis, Infectious coryza.

Clinical signs

The incubation period ranges from 6 – 12 days.
In individual bird, the infection may give rise to peracute, acute or mild/ asymptomatic disease

i) Peracute form:

Birds may be found **dead without premonitory** signs or show **sudden acute dyspnoea** with severe **coughing** and **expectoration of mucus and blood – stained exudates** followed by death in 1 – 3 days.

ii) Acute form:

- **Nasal discharge**
- **Dyspnoea** with bird breathing with long drawn-out gaps, with a wide-open beak and often moist rales can be heard.
- **Conjunctivitis** with frothy exudates at the anterior canthus of the eyes
- There may **be cyanosis of the faces and wattles**.
- Deaths usually occur in 3 – 4 days.

iii) Mild form:

There may be one or more of the following signs: Moist rales, slight coughing and head shaking, Nasal exudates and conjunctivitis, Decrease in egg production, swelling of infraorbital sinuses.
Most chicken recover in 10 – 14 days.

Post mortem lesions
In peracute form: Haemorrhagic tracheitis and partial or complete respiratory obstruction (blood stained mucus)
In acute form: caseous, diphtheritic exudates, mucus and some haemorrhages in trachea.
In mild form: Not severe tracheitis together with edema and congestion of the epithelium of the conjunctiva and infraorbital sinuses.

Differential Diagnosis

- ❖ Infectious coryza
- ❖ Fowlpox disease (diphtheritic form)

Diagnosis: Epidemiology

- ❖ Clinical signs
- ❖ Pm pictures

Treatment
No drug is effective in treatment of ILT

Control

- ❖ Avoid mixing vaccinated or recovered chicken with susceptible ones
- ❖ Use of proper house hygiene

❖ Vaccination (Eye drop, coarse spray and inclusion in water)

Use of live vaccine there is high possibility of spreading the virus, particularly within 7-10 days of vaccination, and the production of carriers.

4: FOWL POX TC "4: FOWL POX" \f C \l "2"

This is a slow – spreading viral disease characterized by cutaneous lesions and sometimes with diphtheritic form in which lesions appear in the mouth and upper respiratory tract.

Etiology: Fowl poxvirus (Avipox genus and family Poxviridae)

Epidemiology

- Fowl poxvirus does not penetrate intact skin, thus a break on the skin is required for virus to enter the epithelial cells, replicate and cause disease.
- The major factor in the spread of the disease from one chicken to another is by direct contact. Also biting insects such as Mosquitoes (Culex & Aedes) transmit the disease from bird to bird and are responsible for widespread outbreak of the disease. Mosquitoes can remain infective for several weeks and produce consecutive infections.
- Pos may occur in birds of all ages, even as young as few days or week old.
- The disease occurs most commonly in chicken, turkeys, and pigeons especially when they are housed.
- The incubation period of the natural disease varies from 4 – 10 days.

Clinical signs

Individual signs of typical pox infection may be in one of the two forms or in a combination: Cutaneous lesions of the head region and featherless areas such as legs, feet and vent; Diphtheritic lesions of the mouth region, and infection of nasal chambers with accompanying coryzalike signs. However, the cutaneous lesion/ form is usually the predominant one in most disease outbreak.

i) Cutaneous form

The cutaneous lesions vary in appearance;

- First there is papules which rapidly progress through vesicle to pustules and finally to crust/ scab.
- After about 2 weeks the scab will drop off and a healed lesion is left which may or may not leave a scar

NB: The lesions occur on unfeathered skin of the head, neck, legs and feet.

ii) Diphtheritic form

- First small white nodules are observed in the upper respiratory and digestive tracts
- These nodules coalesce to form raised yellow plaques on the mucous membrane
- Most lesions are found in the mouth, larynx, trachea, nares, oesophasgus and conjunctiva
- Lesions in the oesophagus and larynx give rise to innapetance, lesions in trachea give rise to difficulty in breathing (dyspnoea), and lesions in nares can give rise to nasal discharge while those on conjunctiva give rise to ocular discharge and rare cases result into blindness
- Morbidity rate is very slow while mortality rate is moderately high in diphtheritic form (50%)

Differential Diagnosis: The diphtheritic form can be confused with:

- ILT = coughing and cyanosis of wattles
- Avitaminosis A (No nodules, hyperkeratosis of mucous membrane of mouth and oesophagus, corneal hyperkeratosis etc)

Diagnosis

- Epidemiology: slow morbidity and mortality, all birds are affected
- Clinical signs: Cutaneous and Diphtheritic forms
- Serological tests: ELISA

Treatment

- There is no satisfactory treatment
- Proper husbandry and supportive treatment should be practiced to alleviate environmental stress

Control

Vaccination (fowl pox vaccine and pegion pox vaccine)
Two main routes are used:

i) Wing web or Thigh route

For wing web method, vaccine is inoculated into the skin of the wing web usually using a bifurcated needle, which has a groove that holds the vaccine fluid. For thigh method, feathers are removed from the thigh and vaccine brushed into the resulting follicles.

Whichever method is used, the skin should be examined 7 10 days later for existence of pox lesions (swelling of skin or a

scab at the site where vaccine was applied for successful vaccination; and no swelling for unsuccessful vaccination)

ii) Via drinking water

This requires two vaccinations of high doses at 5 to 26 days of age is recommended.

Vaccination is indicated in three types of flocks.

a) When the flock was infected the previous year.

All young stock produced on the premise or introduced from other sources should receive fowl pox vaccine.

When several lots of birds are raised each year, each lot should be vaccinated at the appropriate age.

Vaccinated birds should be isolated to prevent possible spread of infection to nonvaccinated birds.

b) If fowl pox was present previous year and pigeon pox vaccine was used, birds should be

revaccinated with fowl pox vaccine, because immunity from pigeon pox vaccine is not of long duration.

c) In areas where pox disease is prevalent, fowl pox vaccine should be used for protection against infection from neighboring flocks

TIME OF VACCINATION

- First dose at 4 weeks old chicks
- Second dose at 1 – 2 months before egg production is expected
- For chicken held for second year of egg production, should be revaccinated.

5: COCCIDIOSS TC "5: COCCIDIOSS" \f C \l "2"

Coccidiosis is contagious enteritis caused by infection with either or both *Eimeria* and *Isospora* spp and occurs in all domestic animals and poultry. A high rate of subclinical infection may occur or there may be diarrhoea and dysentery. In some cases there is anaemia and chronic form of the disease is characterized by inferior growth rate and production.

A: FOWL COCCIDIOSIS

This is a disease of the digestive system/ tract characterized by diarrhoea, often with blood in faeces, lack of activity, weakness, emaciation and high mortality in chicks that looked healthy the previous day.

Aetiology: At least seven species of *Eimeria* have been implicated. And these can be divided into three major groups according to the pathogenicity (severity):

High pathogenicity, dysentery, high mortality and morbidity: *Eimeria brunetti, E. necatrix* and *E. tenella*.

Medium pathogenicity, lesions, but low mortality and morbidity: *E. acervulina,* and *E. maxima*

Low pathogenicity with no lesion: *E. mitis* and *E. praecox*

Epidemiology

All age groups are susceptible to the disease, although chicks of 5 – 7 days and growing birds are highly susceptible. The appearance of clinical signs depends on the life cycle of the parasites; that is, the maximum time required to complete two asexual and one sexual cycle within the host is 5 – 6 days, and bleeding may occur late or day 4, usually reaching a peak on day 5, with cessation about day 7 of the life cycle.

Ingestion of viable sporulated oocysts is the only natural method of transmission. Susceptible chicken get the infection by ingestion of infective oocysts and the disease will start.

Humans are second only to chicken in transport and dissemination of oocysts. Mechanical transmission is commonly affected by manure clinging to shoes or by fomites carried from one pen to another. Hands and clothing may also serve to transmit the single cyst necessary to start infection cycle. Flies, beetles, cockroaches, rodents, pets and wild birds have also been incriminated as mechanical vectors. However, oocysts may also be carried in dust in the atmosphere.

Oocysts may survive as long as 86 wks in shaded soil.

The severity of an attack is directly proportional to the number of organisms ingested by a bird at a time and the species of the organism.

All species of *Eimeria* reported from chickens appear to be widely distributed throughout the world.

The disease is likely to occur only under conditions of high stocking density, which favours the build up of potentially pathogenic population of the parasites. Thus 'COCCIDIOSIS IS SPECIFICALLY IMPORTANT IN INTENSIVE POULTRY OPERATIONS'

The oocysts will remain in the buildings from previous crop of birds and will also be carried in on two legs (human) or possibly by other vertebrate and invertebrate agencies.

In chicken/ animals, there is acquired immunity normally by infection and maintained by continual reinfection. However, this immunity is species specific. Thus, in chicken for example, immunity to *E. maxima* does not confer complete resistance to *E. tenella* and so on.

Clinical signs

The nature of lesion (clinical signs) varies between species of Eimeria which are site specific:

- Diarrhoea, often with blood in faeces is seen.

- Dullness (lack of activity)
- Weakness
- Emaciation
- Decrease in egg production in layers
- Huddling together of the affected birds
- Depressed growth rate or actual weight loss may occur (due to impaired feed conversion)

NB: Early exposure to the GUMBORO agent increases severity of cecal Coccidiosis and may decrease effectiveness of some anticoccidial drugs.

Post-Mortem picture

The entire length of the external surface of the digestive tract from the gizzard to the lower rectum needs to be examined:

- **On the serosal surface**: there may be whitish plaques or petechial or whitish streaks or rounded colonies of oocysts.
- **On opening**:
  - While cutting watch for thickened areas which indicate parasitic invasion of the mucosa or Submucosa
  - Inside: presence of mucus, blood, casts or cores (which may be whitish, red or brown) and cheesy coagulation necrosis
  - Faecal materials may contain blood (e.g. cecal materials indicating *E. tenella* infection).

Differential Diagnosis

- Histomoniasis(mainly in Turkeys: sulphur – coloured droppings, necrotic foci in liver, ulceration of ceca, cyanosis of head – Black head)
- Haemorrhagic syndrome (Blood in faeces, haemorrhages in muscles, internal organs and anaemia)
- Necrotic enteritis (dark faeces +/- blood, congested small intestines, necrotic liver etc)

Diagnosis

- **Clinical signs**: The presence of dysentery, diarrhoea or soft, mucoid faeces, poor growth and drop in egg production. Also sudden increase in daily mortality.
- **Pm examination**: A few sick birds should be sacrificed for this purpose so that fresh material is available. Lesions should be noted from the serosal surface and mucosal surface.
- **Laboratory examination**: examination of wet smears diluted with isotonic saline under coverslip with appropriate lighting will normally be sufficient to detect schizonts, gametes and oocysts.

Treatment

Many anticoccididials are used to treat, the common ones are:

- Sulfa drugs: eg Sulfamethazine, Sulfaquinoxaline (these are either given in feed or water)
- Nitrofurans (Nitrofurazone)
- Amprolium
- Sodium Sulfachloropyrazine monohydrate (Esb_3)

Control

Control of Coccidiosis in poultry relies on:

Good hygiene: This may substantially reduce the number of oocysts contaminating the environment, and most importantly can ensure that litter is kept dry so as not to provide good sporulation condition.

Prevent overflowing waterier and leaking water pipes

Maintain birds on perforated floors.

Use of drugs (prophylactics): Use of coccidiostat, which inhibits but do not kill the parasites (Coccidiocides kill the parasites). Broiler feed almost always contains an anticoccidial agent, which prevent disease and control subclinical Coccidiosis. Some of drugs are:

Salinomycin, 0.004 – 0.0066% (Bio – Cox); nil withdraw period

Amprolium, 0.01 – 0.025% (Amprol); nil withdraw period

Decoquinate, 0 003% (Deccox); nil withdraw period

Sulfadimethoxine, 0.0125% + Ormetroprim 0.0075% (Refenaid); nil withdraw period
Nitrofurazone, 0.0055% (Amifur); 5 days as withdraw period.

B: COCCIDIOSIS IN CATTLE (Synonymous: Red dysentery)
Cause: *Eimeria zürnii*
Clinical Signs
The incubation period ranges from 1 - 8 weeks
Developmental form occurs wholly in the large intestine and caecum where considerable denudation of epithelium occurs, resulting in extensive haemorrhages.

- Persistent diarrhoea which become haemorrhagic
- Emaciation after at least a week
- Fever and digestive disturbance
- Milk diminished or stopped
- Straining during defecation and even eversion of the rectum

NB: Young animals are more likely to succumb the disease with mortality of
2 – 10%;
In adults is self-limiting disease.

C: COCCIDIOSIS IN SHEEP AND GOATS
Causal agents: At least 7 species of *Eimeria* occur in these animals
Clinical signs
Anaemia accompanied by diarrhoea and emaciation
No fever
The lesions are mainly in the small intestines

Treatment
Isolation of sick animals and careful nursing
Use of Sulphamezathine or Sulphaquinoxaline (orally)
Amprolium in feed and water at 10 mg/Kg Bw for 5 days.

6: ASPERGILLOSIS TC "6: ASPERGILLOSIS" \f C \l "2"

This is a disease of mammals and birds produced by the growth of fungus *Aspergillus* in the tissues of the body. Infection probably occurs chiefly through **inhalation** of the fungal spores, which may be abundant in hay or straw under condition of dampness. Entry of the spores into the body may also be by way of the mouth (**ingestion**): in herbivorous animals from contaminated fodder or bedding; in cats and dogs from eating of infected birds or rodents. Once in the animal' s tissues, hyphae grow out from spores, and from the branching filaments more spores are produced causing local necrosis and abscess formation. Numerous organs and tissues can become infected, including the nose and nasal sinuses, the lungs, brain, uterus, and mammary glands.

(a) In cattle and Horses

May cause abortion and pneumonia.

(b) In Dogs and Cats

Aspergillosis is a common cause of chronic nasal discharge from one nostril.

NB: Brain infection may occur in all species, and give rise to symptoms related to Encephalitis,

Fever, excitement, convulsion, paralysis and loss of consciousness.

(c) FOWL ASPERGILLOSIS (Synonymous: Brooder pneumonia; Pneumomycosis)

Aspergillosis in poultry usually is an infection associated with the respiratory system. Other sites of infection, including the visceral organs, liver, eye and brain may also be involved.

Cause: *Aspergillus fumigatus*

Epidemiology

The route of entry is by **inhalation** of large number of spores from mould. Also Aspergillosis can be **egg borne**; *A. fumigatus* can grow inside the egg such that lowered hatchability and high embryonic mortality result.

Outbreak of Aspergillosis is associated with:

- Aerobic environments with a high humidity and relatively warm temperature (> 25^0C)
- Mouldy litter, grain and feed
- Dust (from both inside a poultry house and on ranges)
- Unclean and improperly sanitized hatchery equipment

Young chicks and poults are most susceptible to the infection from *A. fumigatus*.

Predisposing factors to poultry toward Aspergillosis are:

Management factors: inadvertent chilling of young birds, deprivation of feed and/ or water, high ammonia level, and dust in the poultry house.

Primary bacterial or viral infections

Nutritional deficiencies

Acute outbreaks may occur in which there are high morbidity and high mortality, particularly in young birds. In adults, an occasional bird in a flock may become affected while others remaining healthy.

All species of birds may be affected.

Clinical signs

Dyspnoea, gasping and accelerated breathing may occur

Inappetence, emaciation, increased thirst and pyrexia may be present

Serous nasal discharge may be present

Encephalitis; revealed by torticolis and lack of equilibrium

Occasionally infection on eyes may occur that is characterized by formation of yellow cheesy pellet beneath nictitating membrane (mucous membrane of conjunctiva) causing lids to bulge. Also central ulceration of the cornea may occur.
There may be temporary lowered egg production
In chicks mortality may reach 50% especially in confined birds

Post mortem findings
Lesions are found mainly in the respiratory tract but occasionally in the viscera, eyes and brain

- There are **caseous nodules** varying in diameter (10 - 15 mm); these are yellow, of an elastically soft or cartilaginous consistency, and homogenous caseous.
- Older birds typically show **plaque – like caseous lesions** of the air sac walls with grey-brown superficial coloration

Diagnosis
Neither signs nor lesions are diagnostic of this infection.

- Pulmonary nodules cannot be distinguished by gross examination from lesions associated with pullorum disease.
- Diagnosis depends on microscopic detection of the fungus in tissues, followed by isolation and identification by appropriate microbiological methodology

Treatment
At present there is no feasible treatment for Aspergillosis. However, attempts can be done by using: either Nystatin, Amphotericin B, Trichomycin or Griseofulvin
NB: All are given in drinking water

Control
Control is best accomplished by removing the cause by:
Avoidance of mouldy litter or water leakage
Daily cleaning and disinfections of feed and utensils
Spraying the ground around containers with chemical solution e.g. 1:2000 solution of Copper Sulphate.

Careful selection of mash, grain and litter is essential.

7: INFECTIOUS BURSAL DISEASE (GUMBORO) TC "7: INFECTOUS BURSAL DISEASE (GUMBORO)" \f C \l "2"

Gumboro disease is an important viral disease of poultry throughout the world. Clinically it affects young chickens, usually up to 6 weeks of age. The acute form is characterized by sudden onset, short course and extensive destruction of lymphocytes particularly in the Bursa of fabricius (cloaca bursa) and also in other lymphoid tissues.

Causative agent: *Birnavirus*

The virus is highly resistant to physical conditions and chemical agents.

It can survive temperature of 60^0C, stable at pH 2 and resistant to organic solvents; it is susceptible to **formalin** and **iodophors**.

It can remain infective in the environment for at least 4 months.

NB: The hard nature of the virus is one reason for its long persistent survival in poultry houses even when thorough cleaning and disinfection procedures are followed.

Epidemiology

- Gumboro disease is of worldwide distribution, occurring in essentially all major poultry-producing areas of the world.
- Young birds (chicks) are susceptible, with the period of greatest susceptibility lying between 3 – 6 weeks of age. Susceptible chicken young than that of 3 weeks does not exhibit clinical signs but have subclinical infections.
- Natural infections of Turkeys and Ducks have been recorded but no disease has yet been attributed to them within these species.

- The disease is an infectious and highly contagious disease. The affected chicks excretes virus in its droppings for up to 2 weeks after infection.
- There is no evidence that Gumboro is transmitted through the egg or if true carrier state exists in recovered birds. Also there is no direct airborne spread of the virus
- Antibody transmitted from the dam via the egg yolk can protect chicks against early infections with Gumboro for 3 – 5 days of age.
- In fully susceptible flock, the disease appears suddenly and there is high morbidity rate, usually approaching 100%
- Mortality usually begins on the third day (3rd day) post infection and will peak and recede in a period of 5 – 7 days; the mortality rate is high 20 – 60%.

Clinical signs

In acute form:

Whitish, watery diarrhoea usually soiling vent feathers
Depression and anorexia follow within a few days
Some birds will have inflamed vent
There is ruffling of the feathers
Deaths occur within 2 days of the first signs of the disease, reaching a peak in 2 or 3 more days and rapidly decline so that the course of the disease is about 7 or 8 days

In less acute form or subclinical infections:

- The severe clinical signs are absent but there may be a check in growth and production.

Post mortem picture

i) Birds that succumb to the infection are dehydrated
ii) Frequently, haemorrhages are present in the thigh and pectoral muscles
iii) There is increased mucus in the intestines
iv) The liver may be swollen and show peripheral infarcts
v) Occasionally splenomegally occurs

vi) The cloaca bursa is first enlarged, inflamed, oedematous with cream coloured materials and after about 4 – 4 days atrophies. Haemorrhages may be seen in the bursal internal and serosal surfaces and a caseous core is formed within the lumen from sloughed epithelial tissues

vii) Occasionally, haemorrhages are observed in the mucosa at the juncture of the proventriculus and gizzard.

Differential Diagnosis

This is normally in less severe forms, or where the bursa does not show typical lesions

Coccidiosis
Newcastle disease
Haemorrhagic syndromes (for muscular haemorrhages)
Avitaminosis A (for caseous plugs in the bursa)

NB: Confirmation of diagnosis may be made by serological tests

Diagnosis

History/ epidemiology, Clinical signs and Gross lesions

Treatment

- No therapeutic treatment is effective
- Only supportive treatment e.g. Vitamins, & Broad spectrum antibiotics (OTC 20%)

Control

Vaccination: 1st at 7th day, 2nd at 21st or 28th day (3rd or 4th week) of age.

Sanitary precautions to prevent spread of infection by: preventing contact with infected birds and preventing contact with contaminated formats.

8: NEWCASTLE DISEASE TC "8: NEWCASTLE DISEASE" \f C \l "2"

This is a viral disease affecting chicken and characterized by either nervous system, gastrointestinal or respiratory tract involvement

Causative agent

The disease is caused by a group of closely related viruses, which form the avian *paramyxovirus* type 1 serotype (pmv-1). Based on the disease produced in chickens under laboratory conditions NCDVS (PMV-1) have been placed in five pathotypes.

Viscerotropic velogenic NDVs: causing a highly virulent form of disease in which

Haemorrhagic lesions are characteristically present in the intestinal tract.

Neurotropic velogenic NDVs: causing high mortality following respiratory and

nervous signs.

Mesogenic NDVs: causing respiratory and sometimes nervous signs with low

mortality.

Lentogenic respiratory NDVs: causing mild or in apparent respiratory infection.

Asymptomatic enteric NDVs: causing neither apparent enteric infection, clinical signs nor pathology. Detected / isolated in the guts or faeces and by presence of specific antibodies.

Epidemiology

Over 200 species of birds have been reported to be susceptible to natural or experimental infections of NDVs. NDVs strains have been shown to infect all major and minor species of domestic poultry, although some species e.g. ducks and geese, tend to show few signs of disease even when infected with the most virulent strains of NDV for chicken.

Spread

- The mode of transmission from bird to bird is clearly dependent on the organs in which the virus multiplies:
  - Bird showing respiratory disease shed virus in aerosols of mucus, which may be inhaled by susceptible birds.
  - Viruses that are mainly restricted to intestinal replication are transferred by ingestion of contaminated faeces, either directly or in contaminated food or water or by inhalation of small infections particles produced from dried faeces.
- Man seems to play the central role in the spread of NDV, usually by the movement of live birds, fomites, personnel, and poultry product (e.g. dead birds and faeces for fertilizers) from affected premises to susceptible birds.
- Also feral birds and other wildlife contribute to the spread of disease either by infection or by mechanical transfer (e.g. stray racing pigeons)

Clinical signs

The disease produced following infection with NDV may vary considerably with the infecting virus, species of birds, immune status, age and conditions under which they are reared.

i. Sudden death may be produced by infection with highly virulent virus in fully susceptible chickens.
ii. Typical signs are:
    - Appearance of shell – less or soft – shelled eggs, followed by complete cessation of egg laying, may be an early sign in adult fowl.
    - Depression
    - Diarrhoea: greenish Diarrhoea, sometimes bloodstained and profuse.
    - Edema of the tissues around the eyes and throat.
    - Respiratory distress: increased respiration rales when listening at the breast of the disturbed bird.
    - Nervous signs: may appear within a day or two or later, paralysis of legs or wings and torticollis.

The moderately virulent or Mesogenic virus usually cause severe respiratory disease, followed by nervous signs.
The viruses of low virulence may cause no disease or mild respiratory distress for a short time in chicken and turkeys.
Mortality rate may range from 50 – 100% and Morbidity rate of 50 – 100%

Pm picture

Virus causing respiratory disease may induce **inflammation of the trachea**, often with **haemorrhages,** the air sacs may also be inflamed and **appear cloudy and congested**. **Serous or catarrhal exudates** may be present in nasal passages, larynx and trachea. Luÿÿs are usually normal, although theÿÿower anterior portion may **sometimes be pneumonic.**

The virulent viscerotropic virus usually produces hemorrhagic lesions of the gastrointestinal tract, particularly the proventriculus (grandular part of surface of proventricular). However, the lesions vary considerably in size and severity.

Differential diagnosis

b) Infectious bronchitis
c) Infections Laryngotracheitis (respiratory distress, nasal discharge sometimes blood stained)
d) Infections coryza (Swelling of wattles and face, conjunctivitis, serous nasal discharge& cyanosis of face)
e) Marek' s disease (paralysis of one leg/wing, torticolis)
f) Fowl cholera (sudden death, diarrhea with fetid, watery and greenish feces)
g) Poisonings

Diagnosis

b) History
c) Epidemiology

d) Clinical signs
e) Gross lesions; but these lesions are not pathognomonic for any form of Newcastle disease.

Treatment

- There is no specific and feasible treatment.

Control

i. The simplest and most logical measure against ND is to prevent exposure of susceptible birds to virus:
   - Controlled and reduced access of personal and equipment from outside.
   - Avoid introduction of new bird in the flock.

ii. Vaccination: Birds should be vaccinated with live virus at l day old by eye drop or coarse spray and drinking water followed by revaccination at 3 to 4 wks of age.

- ❖ Immunity against ND begins to develop within 1 wks after vaccination; it is well advanced after 2 wks in healthy birds 10 days of age or older when vaccinated.
- ❖ Duration of immunity from living vaccine may vary greatly from flock to flock and among Individuals. It may worn appreciably within 2 – 6 mouths post vaccination, and revaccination is usually recommended within 2 month to 1 year.
- ❖ Vaccination of broilers requires great attention of timing, route and strain of virus. Viable vaccination must be timed so that adverse effects of parental antibodies have waned but it is early enough to reduce chances of losses from infection with virulent field viruses (passive immunity starts to wane 2 – 3 days). It is recommended to vaccinate after they are 1 – 2 weeks old.

9: MAREK' S DISEASE TC "9: MAREK' S DISEASE" \f C \l "2"

This is a lymphomatous disease of the domestic chicken characterized by lymphoproliferative infiltration (mononuclear infiltration) of peripheral nerves, gonads, iris, various visceral organs, muscles and skin (leading to demyelimination of peripheral nerves).

Etiology : *Lyphotropic herpesvirus*

Readily inactivated by common chemical disinfectants.

The virus can remain in the litter for up to 16 weeks at room temperature.

Dried feathers from infected chickens retain infectivity for up to 8 months at room temperature and for at least 3 years at 4^0C

Transmission

Marek' s disease exists in all poultry - producing countries throughout the world.

Natural infection to chicken usually occurs as early as 3rd week of life, but the diseases usually appears between the 2nd and 5th month. Losses often appear after birds reach maturity.

Direct or indirect contract between birds and virus are the means a spread of MD (airborne spread)

Infected birds, carriers and those at incubation period are the source of infection

Incubation may be as short as 3 or 4 weeks or long as several months.

Several lines of chicken with genetic resistance to MD have been recorded (due to deficiency in number of target lymphocytes)

Incidence of MD is quite variable. A few birds that develop signs may recover from the clinical disease, but in general, mortality is nearly equal to morbidity.

Clinical signs

Classical Marek' s disease.

Mortality is variable 10 – 15 %

The signs depend on the peripheral nerves affected.

Involvement of the brachial and sciatic nerves (common) – lead to progressive spastic paralysis of wings and legs (starts with asymmetric paralysis then complete paralysis).

Sciatic nerve (leg) involvement makes birds to have one leg stretched forward and the other back as a result of unilateral paresis/ paralysis of the legs

Brachial nerves involvement leads to paralysis of wings

Vagus nerve involvement leads to paralysis and dilatation of the crop and/ or gasping

When cervical nerves are involved – torticollis

Involvement of Vagus and Intercostals nerves lead to respiratory signs

When iris is involved, blindness occurs

Non-specific signs such as weight loss, paleness, anorexia and Diarrhoea may be observed, especially in birds in which the course of disease is prolonged.

Acute Marek' s disease

High mortality rate of 10 -30% and morbidity rate of 80%

This is characterized by:

Sudden death

Others show depression before death and some paralytic signs similar to those seen in the classical form.

Transient paralysis

This is uncommon, encephalitic form occurring between 5 and 18 weeks of age.

Birds suddenly develop various degree of paresis or paralysis of the legs, wings and neck.

Mortality rate is low

Recovery is within 24 – 48 hrs.

Post mortem picture

Enlargement of one or more peripheral nerves (brachial or sciatic nerves); affected nerves are enlarged, lost cross-striations, gray or yellow in colouration, and sometimes an edematous appearance.

NB: lesions are unilateral; compare the two nerves in case of slight changes.

Diffuse lymphomatous tumour may occur in liver, gonads, spleen, kidneys, lungs, proventriculus and heart

Diagnosis

Clinical signs
Epidemiology
Pm picture

Treatment: None

Control

Management methods, isolation of growing chicken from source of infection
Vaccination: at 18 day or at hatchery, the vaccine give life long immunity

10: FOWL CHOLERA (syn: **cholera, pasteurellosis**) TC "**10: FOWL CHOLERA** (syn: **cholera, pasteurellosis**)" \f C \l "2"

Contagious disease of domestic and wild birds characterized by acute septicaemia with high morbidity and mortality rates, and a chronic localized disease

Aetiology: *Pasteurella muttocida*

Epidemiology

- Species affected are Chicken, turkeys, ducts, canaries and many wild birds
- In poultry, semi‑mature and mature birds are affected.

- Turkeys are more susceptible than chicken; among chickens – heavy breeds are more susceptible
- Distribution: World – wide
- Transmission
  - Ingestion of contaminated feed or water
  - Inhalation
  - Conjunctival mucosa and wounds (rarely)
- Source of infection: carrier birds, clinically infected birds or rodents.

Clinical signs

Depend of the course of the diseases

1. Peracute : Death without premonitory signs (birds in good condition).

2. Acute The course of illness is short

Dead chicken may be the first sign (laying birds dead in the nest).
Anorexia, depression, nasal discharges,
Diarrhea: fetid water or greenish in mucoid
Increased respiratory rates
Ruffled features

3. Chronic

Survivors of acute diseases or affected by less virulent strain.
Signs are associated is localized infection:

- Swelling of joints, foot pad, or tendons heath leading to lameness,
- Swelling of wattles – with pus inside
- Exudative conjunctivitis & pharyngitis
- Accumulation of cheesy exudates in the conjunctiva and infraorbital sinuses

- In turkey torticollis is common

Post mortem picture

- Lesion in peracute and acute cases may be absent: mainly petechial and ecchymotic haemorrhages in tissues and organs
- Chronic cases: inflammation of the joints, wattles and lungs; cheesy exudates in localized lesions.
- In Turkeys; fowl cholera often occurs as a complication of air sacculitis cased by *M gallisepticum*

Diagnosis

- Clinical signs
- Pathological lesions
- Identification of the organism
- Mice inoculation

Treatment: Limited value in septicemic cases
Suphonamides
Tetracycline
Penicillin

Control
Strictly hygiene: exclusion of personnel, birds or rodents
Depopulation and disinfection of premises (removal of reservoirs)
Vaccination