Anthrax

Etiology :

Bacillus anthracis ,virulent strain cotain three protein Protective antigen ,lethal antigen , edema factor .

Epidemiology:

1- World wide in distribution .
2- Morbidity rate may be high among all farm animals and thesusceptiblity is highest among ruminant followed by horse. And swin.
3- Source of infection ,sporse from the soil or from fodder grown on infected soil,from contaminated bone meal or protein concentratrat or from infected excreta , blood or other material .
4- Water can be contaminated by infected carcasses .
5- Spread of organism with in the area by Streams,insect,dogs and other carnivores ,wild bird ,by contamination animal product .
6- Infection gains entrance to the body by ingestion ,inhalation or through the skin .
7- Human can infected and cause fatal human illness (human who eat meat from infected animals).

Pathogenesis :

After entry of bacteria move to local lymph node ,proliferation , then bacilli pass via the lymphatic vessels into blood stream .Septicemia ,with massiveinvasion of all body tissue .

-the organism produce alethal toxin caused edema ,tissue damage death resulting from shock and acute renal failure .

Clinical Finding

- incubation period 1-2 weeks in cattle, sheep.

1-Peracute form:

Is most common at the beginning of outbreak,animal found dead without premonitory signs ,the cours 1-2 hr. but fever muscle tremor ,dyspnea and congestion of mucosa can see .

- The animal soon collapse and die after convulsion .
- After death discharge of blood from nostrils ,mouth ,anus and vulva.

2-Acute form :

-course of the disease 48 hr.

- sever depression and listlessness observed first.,high body temperature .
- Respiration is rapid and deep,mucosa congested and hemorrhagic.
- no food is taken and ruminal stasis.
- pregnant cow may abort, reduce milk production and maybe blood stained or deep yellow color.
- diarrhea and dysentery.local edema of tongue and in the throat,sternum,perineum and flank .

Clinical Pathology :

1-in living animal the organism can detected in stained smear of peripheral blood .

2- when there islocal edema the smearfrom edema fluid

3-serological test .

Necropsy Finding :

1- absence of rigor mortis and the carcases undergoes gaseous decomposition
2- All natural orifice usually exude dark tarry blood which dose not clot and putrefaction and bloating are rapid .
3- Present of ecchymotic hemorrhage ,through out the body tissue .
4- Severenteritis and gross enlargement of spleen with softening and liquefaction of its structure are certain indication of the presence of anthrax.

Differential Diagnosis : Sudden death

1- Electrical storms , careful examinationof environment
2- Peracute black leg ,it restricted to young animal ,crepitation swelling .
3- Acute leptospirosis ,occur sporadically and Hburia .
4- Bacliiary hemoglobinuria ,Hburia , infarcts in liver .
5- Peracute lead poisoning ,

6- Hypomagnesemia tetany .
7- Acute bloat ,gaseous distension and exudation of blood from the orifice

Treatment :

Rx - seves ill animal are unlikely to recover.

1- penicillin 10000 unit bw. Twice daily
2- streptomycin 8-10 gm/day two dose im for cattle
3- oxytetracyclin 5mg /kgbw./day at vaccination

In cattle and sheep

-procainepenicillin and streptomycin in larg dose at 12 hr.intervals and antiserum for at least 5 days .

Control :

1-cntrol meat and milk producing animals in infected herd.

2-when outbreak occur placing the farm in quarantine ,destruction of discharges and cadavers and vaccination of survivors.

3-infected carcase should not opened but immediately burned or buried with bedding 2meter deepand the incontact animals must segregated until cases ceas for 2 weeks ,and the animal give hyperimmun serum and used disinfection 5%lysol incontactnwith spores for at least 2 days ,formalin or hydroxid 5-10%

4-vaccination :

- living attenuated strain of organism.
- avirulent spore vaccine one or two dose use in sheep.

In enzootic area annual revaccination of all stock ,milk from vaccinated cow is usually discarded for 72 hr. after injection .

- Stern vaccine ,the organism not appear in milk nor can isolated from blood for 10 and 7 days after vaccination .

Black Leg

Etiology :

Clostridium chauvoei

Epidemiology:

1- diseae in number of animals in soace , the disea enzootic .
2- Morbidity rate 100%.
3- Source of infection ,is soil-borne infection .
4- Disease occur by ingestion of contaminated feed .
5- Contamination of the soil and pasture may occur from infected feces or decomposition of carcasses of animal dying from disease.
6- In sheep the disease wound infection (skin) at shearing and docking and navel at birth causelocal lesion and after vaccination against entertoxemia .
7- In cattle the disease occur between the 6 month and 2 years

Pathogenesis :

Toxin formed by the organism produce sever necrotizing myositis locally in skeletal muscls .

-systemic toxemia which fatal.

- in cattle and sheep atypical outbreak of sudden death occur in which the lethal lesion is a clostridial cardiac myositis.

Clinical Finding :

1-sever lameness with swelling of the upper part of the affected leg ., The animal depressed ,complete anorexia and ruminal stasis .

-high temperature and pulse rate 100-120/min.

-in early stage the swelling is hot,painful ,but soon becom cold and painless and edema and emphysema..

- The skin is discolored and soon become dry and cracked, the lesion present in other location such the base of tongie,the heart, muscle ,diaphragm and brisket and udder .

-the animal die quietly 12-36 hr.after appearance of signs .

-many animal die with out signs having observed.

In sheep :

- Stiff gait and disinclined to move due to sever lameness in one or several limbs.
- Subcutaneous edema is not common and gaseous crepitation can not felt befor death .
- When infection occur through wound of skin ,vulva or vaginathere is an extensive local lesion .
- Sheep and cattle with cardiac myositis caused by Cl. Chauvoei are found dead .

Clinical Pathology :

1-Needle puncture or swab fromwound material for cuture.

2- serum level of lactic dehydrogenase ,SGPT,SGOT increase .

Necropsy Finding :

1- Cattle death from disease are characteristic position: lying on the side with the affected hind limb stuck out stiffly.
2- Bloating and putrefaction occur quickly and blood stained froth exudes from the nostrils and anus .
3- Incision of the affected muscle mass reveal the presence of dark discolored swollen tissue with rancid odor .

Differential Diagnosis :

1-Anthrax, splenic lesion .

2- Bacillary hemoglobinuria liver infarct .

4- Lactation tetany .

5- Acute lead poisoning .

Treatment

Rx

1- Penicillin 10000 unit /kgbw iv ,some of it given in affecting tissue .

2- Black leg antiserum very large dose.

Control :

1- In enzootic area annual vaccination of allcattle between 6-2 years age at spring and Summer.

2- Vaccination of calves at 3 weeks of age ,formalin killed .

3- Sheep vaccination at 3 week befor lambing and before shearing.

4- The car cases ,destroyed by burning or deep burial to limit soil contamination ..

Malignant Edema (Gas gangrene)

Etiology :

Clostridium septicum , Clostridium chauvoei ,Clo. Perfringens

Clo. Sordellii , Clo. Novyi ,have all isolation from lesion .

Epidemiology :

- All age and species of animal are affected .
- The organism are common inhabitants of animal environment and intestinal tract , the disease occur sporadicaaly.
- The infection is usually soil-borne .
- Wound is portal of entry (deep puncture wound).
- Infection occurs through surgical or accidental wound ,following vaccination or through umbilical cord in newborn .
- After shearing,docking ,lambing in sheep.

Pathogenesis :

Local lesion, organism produce toxine –cause death locally the exotoxin cause extensive edema and necrosis followed by gangrene .

Clinical Finding :

- Clinical signs appear within 12-48 hr. of infection
- Local lesion at the site of infection consisting of soft,doughy ,swelling with local erythema with sever pain on palpation .
- Amphysema may or may not present .
- High fever,depression weak,muscle tremor,and lameness.
- When infection occur at parturition ,swelling of vulva with discharge of a reddish-brown fluid occur within 2-3 days.
- In swelled head of rams the edema is restricted initially to thehead ,it occur first under the eye ,spread to the subcutaneous tissue of the head and down the neck.

Clinical Pathology :

- Aspirated fluid from edema swelling or swabs from wound may give an early diagnosis .

Necropsy Finding :

Tissue chang es occur rapidly

- Gangrene of the skin with edema of subcutaneous and intermuscular connective tissue around the site of infection .

Differential Diagnosis :

-1- Black lege ,different by absence of typical muscle involvement and present of wound .

2- Anthrax in big .

Treatment :

Rx -pencillin heavy dose i/v repeated at 6 hr. intervas .

- Antitoxin
- Ingection of pencillin directly to lesion .
- Surgical incision to provid drainage and irrigation with hydrogen peroxide.

Control

-Hygien at lambing ,shearing ,castration and docking.

- Vaccination .

Braxcy

Etiology :

Clostridium septicum

EPiedmilogy :

- Disease occur only in mid winter when there are heavy frosts and snow .
- Organism is soil-borne ,and considered as normal inhabitant of ovine intestinal tract
- Case fatality rate 50%.

Pathogenesis :

Ingestion of frozen grass or other feed ,permit invasion by Clo.septicum

Clinical Finding

- Sudden onset of illness with segregation from the group .
- Compet anorexia ,depression ,high fever.
- The abdomen may be aistended with gas ,sheep becom recumbent and die within a few hours of first becoming ill.

Clinical Pathology : Ante mortem of till value .

Necropsy Finding

Localized area of edema congestion ,necrosis and ulceration of abomasal wall.

Differential Diagnosis :

1- Over eating on grain may couse local patches of rumenitis and reticulitis ,but no lesion in abomasums .
2- Infectious necrotic hepatitis ,but no liver lesion in braxy .

Treatment : No treatment

Control : - management of the flock is important .

-Vaccination with formalin killed ,two injection, two week apart .

- Sheep yarled at night and fed hay before being letout. to the frosted pasture each morning .

Infectious necrotic hepatitis (Black Disease)

Etiology :

Clostridium novyi type B

Epidemiology :

-well-nourished adult sheep in the 2-4 year age group .

- Seasonal occurrence because associated with liver fluck outbreak most common in summer or autumn month .

Fecal contamination of the pasture by carrier animal important source of infection .

Pathogenesis :

- under local anaerobic condition (occur in liver at migration of fluck) cause sever tissue destruction .

The organism proliferate ,then liberation toxin which cause local liver necrosis and more diffuse damage to the vascular system .

Clinical Finding :

- Affected sheep die during the night and are found dead without having exhibitedany signs .
- Sheepare seen to segregate from the flock ,fall down if drven .
- Fever which subsided to subnormal .
- Some hyperesthesia,respiration is rapids and shallow.
- Sheep remain in sterna recumency and die with in few minutes in this position .

In cattle :

- Sever depression reluctance to move ,coldness of the skin ,absence of rumin sound .
- Abdominal pain on palpation of liver.
- Periorbital edema .

Clinical Pathology:

- Blood stained froth may exude from nostril .
- Subcutaneous edema ,dark appearance of the inside of skin.
- Gray-brown appearance ,necrosis ,there are yellow area 1-2 cm in diameter and surrounded by zone of bright red hyperemia .
- There is usually evidence of recent invasion with liver fluck .

Differentia Diagnosis : sheep

1- Acute fascioliasis ,cause heavy mortality due to massive liver destruction , course of flock long .
2- Enterotoxemia .
3- black leg
4- malignant edema .
5- anthrax cause similar heavy mortality in sheep.

special lesion and necropsy use for diagnosis

Treatment : - No effective treatment.

-in cattlepencilline or wide spectram antibiotic .

Control:

-control liver fluke .

- burning the cadavers of infection animal .

-vaccination (alum precipitated toxin).

-vaccin as prophylactic in early summer.

Bacillary hemoglobiburia

Etiology :

Clostridium heamolyticum

Epidemiology :

- Disease is spread from infected to noninfected area by flooding ,by contaminated hay from infected area or by carrier animals .
- Carriage of bones or meat by dogs or other carnivores .
- Contamination of pasture may occur from feces or decomposing cadavers
- By contaminated material (ingestion) ,and the species involved usually cattle .
- Disease occur in summer and autumn .

Pathogenesis :

Invasion of flukes (cysticecercus tennicollus), induce liver damage then bacteria carriage to the liver causes hypoxia leading to development of an organized thrombus in subtermial branch of portal vain produce large anemic infarct (characteristic of disease)

- Two toxin hemolysin is responsible for hemolytic anemia.and Necrotizing agent .

Clinical Finding:

- Cattle in pasture may found dead without signs.
- Sudden onset of complete cessation of rumination ,feeding ,lactation and defecation .
- Abdominal pain , disinclination to move and arch back posture .
- Granting on walking .
- Respiration is shallow and labored .,fever, edema of brisket .
- Feces dark brown, mucus diarrhea some time with blood, urine is dark red, and jaundice .

- Duration of illness 12hr. in dairy cow. in advance pregnancy is abortion ,sever dyspnea just before death.

Clinical pathology :

- RBC count depress to$4x10^{12}$ cell/L.
- Glucose level elevated (100-120) .
- Blood culture during acute stage is positive .

Necropsy Finding :

-the perineum is soiledwith blood stained urine and feces .

- subcutaneous gelatinous edema which tend to becom crepitant in few hours or extensive petechial or diffuse hemorrhage in subcutaneous tissue are characteristic .

-jaundice ,fluid varying from clear to blood –stained and turbid are present in pleural ,pericardial and peritoneal cavities .

Differential Diagnosis :

Is differentiated from other disease which hemoglobinuria ,myoglobinuria and hematuria are cardinal signs .

-Acute leptospirosis ,sam clinical signs ,necropsy finging will differentiate the two and clinical pathology .

- Post parturient hemoglobinuria.

- hemolytic anemia caused by rabe ,kale.

-Babesiosis

-Anaplasmosis

- Enzootic hematuria

-Pylonephritis and cystitis ,present red cell in urine .

In sheep :

-Chronic copper poisoning

-Anthrax

- Black leg

-infectious necrotic hepatitis .

Treatment

Rx

- Pencilline or tetracycline in full dose .
- Antitoxic serum 50-100 ml.
- Blood transfusion , fluid therapy .
- Hemopoiesis should be facilitated by provision of mineral supplemints containing iron, copper and cobalt .

Control :

-use formalin killed culture give good protection for ayear in cattle .

- vaccination (sc, I p and im) carried out 4-6 week before the expected occurrence of the disease .

-annual revaccination of all animal over 6 month of age .

The carcasses of animal dying from the disease should be disposed by burning or deep burial.

Enterotoxaemia

Entertoxemia caused by Clostridium perfringens type A.

- The organism is part of bacterial flora of alimentary tract of normal animal.
- In sheep highly fatal hemolitytic disease .
- Acte hemorrhagic enteritis in calves and adult cattle .
- The organism produce toxin :alfa toxin ,
- Sever depression ,collaps ,mucosal pallor ,jaundice ,hemoglobinuria ,dyspnea and sever abdominal pain.
- The disease is highly fatal ,most affected animal die within 12hr.of onset of the disease.

Clinical Pathology

- Fecal culture .

Necropsy Finding :

- Pallor ,jaundice ,kidney look swellen dark brown with area of infarction ,intestine show area of necrosis .

Differential Diagnosis :

- Chronic copper poisoning .
- Leptospirosis In calves .

Treatment :

Rx

- Antiserum is effecting in prevention of the disease in calves .
- Formalized vaccine shown some immunity in sheep .

Enterotoxemia Caused by Clostridium perfringens type B,C and E

Etiology :

- The organism occur in soil and alimentary tract of normal animal.
- Lamb dysentery caused by Cl. Perfringes type B and C in young lamb Up to 3 weeks of age .

-Struck Clostridium perfringes type C ---occur in adult sheep

- Goat Enterotoxemia caused by Cl. Per. Type C .
- Calf Enterotoxemia caused by Cl. Per. Type B and C in young . Calves up to 10 days of age.

Foal Enterotoxemia caused by Clostridium perfringens type C

Etiology :

-occur in first few days of life .

- the bacteria capable of forming spores .

-rapidly growing ,well nourished animal are most susceptible .

- the toxin produce are alpha ,beta and epsilon in type B. ,and alpha and beta in type C ,and iota and alpha in type E.

Epidemiology :

- Lamb dysentray is most prevalent in cold weather .
- Fatality rate is 100 %,and mortality rate 20-30 %..
- Cause of infect small animal :the intestine is not well developed and the toxin killed or inhibit by trypsin , and the colostrums rich in substances cause inhibition for trypsin (trypsin inhibitor) .

Pathogenesis :

- Ingestion of organism (soil fecal contamination of udder) then proliferates and attaches to the surface of epithelial cell of intestinal villus .
- Produce beta toxin which responsible for disease syndromes .
- Beta toxin is necrotizing and produce damage to microvilli ,cause destruction and desquamation of intestinal epithelial cell.

- Hemorrhagic enteritis and ulceration of intestinal mucosa .

Clinical Finding :

- Lamb dysentray in lamb less than 2 weeks of age .
- Sudden death without signs in per acute case.

Acute form **:**

- Loss of sucking ,sever abdominal pain manifest by bleating ,stretching and looking at abdomen .
- Brown fluid feces some time with blood .,defecation with painful straining .
- Death after decumbency and coma within 24hr.of illness .
- Cases may occur in older lamb up to 3 weeks of age **.**

Chronic form :

- In older lamb called "Pine" ,chronic abdominal pain ,reluctant to suck but no diarrhea .

Struck in adult sheep :

-sudden death .

- befor death there is abdominal pain and convulsion .

- In calves :

The disease occuras out break of sever dysentery with some death in calves 7-10 day old , diarrhea dysentery and acute abdominal pain ,there may be nervous signs like tetany **.**

Clinical Pathology :

1 -mad on necropsy material .

2-Antitoxin in sera of recovery animal .

3-Serological test **.**

Necropsy Finding :

- Hemorrhagic enteritis , ulceration of mucosa.
- In Type B the lesion occur as localized area of necrosis .,blood stained contents are present in the intestine and excess of serous fluid in peritoneal cavity .
- Sub endocardal and subepicardial hemorrhages are present

Differential Diagnosis :

-enteritis and septicemia caused by *E coli and Salmonella* spp.

- *Actinobacillius equuli* and porcine transmissible gastroenteritis(pig).

Confirm the diagnosis by laboratory examination of fecal material or intestinal contents .

Treatment :

Rx

-hyperimmune antiserum is the only treatment of value .25 ml of type C antiserum have been used in calves .

- Oral administration of penicillin ,prevent proliferation of organism .

Control :

- Vaccination ,with type specific toxoid or bacterin .
- When out break occur all pregnant animal should be vaccinated to provide colostral immunity .
- Antiserum protect susceptible animal ,after birth .
- Administration of benzathine or benethamine penicillin G. or amoxicillin at birth .
- -2injuction of vaccine are necessary 1 month apart, the second injection being give 2-3 weeks before parturition .

Enterotoxemia Caused by Clostridium ***perfringnes*** **type D**

Etiology :

- *Clostridium perfringnes* in the small intestine .

- organismproduce number of toxin which epsilon toxin ,is most important and result in vascular damage and nervus system damage .

Epidemiology :

- The disease of ruminant primarily of lamb .
- The incidence rate 10%, case fatality 100%.
- The organism is normal inhibits of alimentary tract of sheep, and produce lethal quantities of epsilon-toxin ,the affected animal in good condition and occur in lamb 3-10weeks.
- The weaned lamb up to 10 month of age can affected .

Pathogenesis:

- Ingestion of organism then multiplication and production of toxemia .
- Slowing of alimentary tract movement permit excess toxin accumulation .
- Toxin produce increase permeability of intestinal mucosa to this and other toxin .
- Profuse mucoid diarrhea , stimulation and the depression of central nervous system .
- Vascular damage lead to accumulation of protein rich fluid in heart,lung and brain.
- The postmortem autolysis of kidney tissue which occur so rapidly it's the characteristic of pulpy "kidney"

Clinical Finding :

- The courseof the disease in lamb very short less than 2hr. and many animal found dead without signs .
- Dullness ,depression ,yawning ,facial movement and loss of interest in feed .
- Green pasty diarrhea ,staggering ,recumbency,sever convulsion .
- Adult sheep usually survive longer periods up to 24hr. showing staggering and knuckling ,champing of the jaws ,salivation and rapid shallow ,irregular respiration .

- Convulsion ,muscle tremor,grinding of teath and salivation .

Clinical pathology :

- **Hi**gh level of sugar 8.3-10 mmol/l
- Glucosuria are characteristic
- Detection of organism or toxin in feces .

Necropsy Finding :

The carcass usually in good condition

- In peracute form : no gross lesion ,there is an excess of clear ,straw colored pericardial fluid petechial present in pericardiaum and endocardium
- The intestine usually containe moderated amount of thin custared ingesta
- Acharacteristic chang is soft pulpy kidney afew hours after deatg.

Differential diagnosis :

-sudden death

-Acute pasteurellosis

-hypocalcemia

Hypomagnesemia

-septicemia caused by *Haemophilus agni*

- focal symmetrical encephalomalcia

-Acute rumen impaction due to over eating , no convulsion

In adult sheep and calves

-Rabies .

- acute lead posioining

-hypomagnesemic tetany .

-pregnancy toxemia

-louping ill .

Treatment :Rx

- Hyper immune serum as prophylactic .
- Antitoxin in combination with orall administration of Sulfadimidin **.**

Control :

-reduction of food intake .

- vaccination ,killed vaccine ,give at 3day of age , the vaccine produce good immunity, second injection at 1 month of age
-simultaneous administration of hyper immune serum .
-Revaccination at 6 month intervals .
-Fat lamb:
-in outbreak occur ,administration of antiserum and toxoid and repeat the toxoid in amonth.