CLOSTRIDIOSIS

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INTRODUCTION :

-Cause by **Clostridium**

Rods shaped, Gram positive, spores forming, anaerob

-Mechanisms of the disease are :

1. Infection

   Boutvur/ Black Leg ➔ *Cl. chauvoei*

   Paraboutvur/Malignant Edema ➔ *Cl. septicum*

   Pulpy Kidney ➔ *Cl. perfringens*

2. Entoxication

   Tetanus ➔ *Cl. tetani*

   Botulism ➔ *Cl. botulinum*

BOUTVUUR/ BLACK LEG

- **SYNONIM → Rauch Brand = Quarter ILL**
  Gangraena Emphysematosa
  Black Quarter = Charbon

- Acute fever Disease
- Attack : Cattle, Sheep, Goat, Swine and Deer
- Characteristic clinical sign are :
  serohemorrhagic inflamation
  crepitatant, spongy texture of the thick muscle

ETIOLOGY

Clostridium chauvoei ➔ Rods, Gram positive, spores form
spore at central or subterminal
Anaerobic
proteolitic and saccharolitic

EPIZOOTIOLOGI

This organism is the cause of **Black Leg in ruminants**

It occurs throughout the world

In Indonesia it occurs at Jogya, Solo, and Madiun

PATHOGENESIS

- **Transmission via** :
  - Wound ➔ castration, dehorning, injection tools, birth or calving help
  - peroral ➔ spores contamination food and drink

Spores invade at tissue ➔ vegetative form & multiply ➔ product toxin and spread at predilection (leg/thick muscle)

Proteolitic ➔ digest the muscle & collagen tissue ➔ black in color

Saccharolitic ➔ glycogen fermentation ➔ produce gas

Toxin effect ➔ increase blood vessels permeability ➔ fluid excretion ➔ edema

These are cause crepitant and spongy texture

CLINICAL SIGN

- Fever, depression, serohemorrhagic inflamation
- Crepitant and abortion in pregnant animal

Sheep (BRAXY) :

- paralyse, frequent respiration, sudden death
- incision of infected organ ➔ secreting dark reddish brown fluid.

DIAGNOSIS

-Clinical sign and pathological changes

-Bacteriology examination : Isolation & identification

Biology test & serology test

DIFFERENTIAL DIAGNOSIS

-Parabotvuur ➔ *Cl. septicum*

-Anthrax ➔ *B. anthracis*

PREVENTION AND CONTROL

Prevention : passive imunnisation (giving antiserum)

Vaccination and hygiene sanitation

Control of the disease :

- don’t slaughter the infected animals
- give antibiotic therapy and combined with antiserum

PARABOUTVUUR

SYNONIM ➔ Gas Gangrena = Malignant Oedema

Geburts Rausch Brand

Acute contagious disease in cattle, sheep, horse,goat, swine and human

Characteristic clinical sign ➔ emphysematous inflamation

Paraboutvuur can decrease population and death

ETIOLOGY

Clostridium septicum ➔ rods, spores at subterminal, motile (peritrichous flagella), Gram positive, anaerob

Produce collagenase and hyaluronidase enzyme

EPIZOOTIOLOGY

The disease occurs thoughout the world but sporadic in Indonesia

PATHOGENESIS

-Transmission via wound

Spore invade in tissue ➔ vegetative form ➔ multiply and toxin production ➔ toxin (blood stream) ➔ predilection (liver and ren) ➔ degeration

Toxin (toxaemia) ➔ cardiac failure and lysis of erythocyte ➔decrease oxygen ➔ hypoxia ➔ death

Infection *Cl. septicum* in sheep is called **Braxy / Bradsot**

CLINICAL SIGN

-At wound infection ➔ rapidly extending swelling with characteristic soft and pit in palpasion

-No crepitant

-General sign are fever, depression, increase puls, dyspneu, diarrhea, mucous membrane and muscular tissue are dark red and contains little ar no gas

-in pregnant animal ➔ abortion

Sheep (Braxy) :

suddendeath, paralysis and frequent respiration

Pathological changes ➔ incision of infected organ secreting dark red fluid and bad smell.

DIAGNOSIS

-Clinical sign and pathological changes

-Bacteriology examination ➔ Isolation and identification

or biology test with rabbit and guinea pig

DIFFERENTIAL DIAGNOSIS

1. Boutvuur ➔ *Cl. chauvoei*
2. Anthrax ➔ *B. anthracis*
3. Streptococcosis in Horse

PREVENTION AND CONTROL

-Prevention : - immunization / vaccination

- hygiene sanitation

-Controlling : - don’t slaughter the infected animals

- discard the visceral & carcas totally

-Treatment : - antiserum

- chemoterapy ➔ Sulfathiazol

- antibiotic ➔ penicillin, tetracycline

- combination of antiserum and antibiotic

INFECTION OF *Cl. novyi* :

1. *Cl. novyi type A* ➔ Big Head = Swelled Head (sheep) and Gas Gangrena (cattle)
2. *Cl. novyi type B* ➔ Black Disease (cattle and sheep)
3. *Cl. novyi type C* ➔ Osteomyelitis (Buffalo)
4. *Cl. novyi type D = Cl. hemolyticum* ➔ Icterohemoglobinuria = Red Water Disease (cattle, sometimes sheep and swine).

BIG HEAD = SWELLED HEAD = SWOLLEN HEAD

ETIOLOGY

Clostridium novyi type A

➔ rods, the size is larger than the others Clostridium, motile (peritrichous flagella), Gram positive, spores shaped are oval and at subterminal

This organism is more strickly anaerobic than others

EPIZOOTIOLOGY

The disease was found in Indonesia, United States (USA), Europe, Australia and New Zealand

PATHOGENESIS

Sensitive animals : cattle, sheep, goat, dog and horse

Transmission via wound especially infection of the head and neck area result from fighting trauma.

Invasion of bacteria ➔ multiply and toxin production (alpha toxin) ➔ oedema and gas gangrene of the head and neck (Oedema malignant)

CLINICAL SIGN : - swelling of the head and neck area

- suddendeath

Pathological changes : hemorrhagic in lung

DIAGNOSIS

- Clinical sign and pathological changes
- Laboratory examination : Isolation and identification
- Serology test ➔ FAT

PREVENTION AND CONTROL

Prevention : Vaccination with attenuated toxin

In endemic area ➔ hyperimun serum (antiserum)

Controling the disease ➔ hygiene sanitation (*Cl. novyi type A* is found in the soil and intestinal tract herbivora animals)

Treatment : combination of antibiotic (Penicilline) and chemoterapy (Sulfadiazine) or combination of antibiotic and antisera

RED WATER DISEASE

Infectious Icterohaemoglobinuria= Haemorrhagic Disease

INTRODUCTION :

- The disease attack ➔ cattle, sheep and swine
- The characteristic symptom ➔ urine is a dark red

ETIOLOGY :

Clostridium hemolyticum = Cl. novyi type D

rods, singly, or forming a short chain, Gram positive

motile and oval spores (sub terminal)

Produce lechitinase and necrotic toxin.

EPIZOOTIOLOGY :

The disease was found in United States & New Zealand, occurs during the summer and early fall season

In Indonesia the disease has not been found.

Sensitive animals ➔ cattle, sheep and swine

Experimental animals ➔ rabbit, guinea pig and mice

PATHOGENESIS

Peroral (spores) ➔ tract digestivus ➔ germination of spores ➔ vegegative bacteria ➔ portal vein in liver ➔ toxin production cause liver necrotic and subcutaneus & visceral haemorrhagic

CLINICAL SIGN :

-Anorexia

-Rumination and lactation are decrease

-Fever and mucous membrane icterus

-The temperature to become subnormal before death and pulsus frequent

-Urine port-wine in color or dark red and foamy

-Feses to become soft, dark red

-Death occurs 36 hours after the first symptoms appears.

DIAGNOSIS :

1. Clinical sign and pathological changes
2. Bacteriology examination : Isolation and identification
3. Biological test ➔ guinea pig
4. Clinical pathology : Erythrocyte 1.200.000

   Hb 3,5 and Leucocyte count increase

DIFFERENTIAL DIAGNOSIS :

1. Anthrax
2. Septicaemia Epizootica
3. Black Leg = Boutvuur

CONTROL AND TREATMENT

1. PREVENTION

-Immunisation ➔ Immune serum with preventive dose

Vaccination with “Phenolized whole culture vaccine” or “Formolized Bacterin”

-Sanitation ➔ pasture in wet pastureland

- Isolation and treatment of infected animals

2. TREATMENT

Antiserum every 2 weeks and symptomatic therapy

INFECTION OF *Cl. welchii = Cl. perfringens* ➔

1. Cl. welchii type A ➔ Anemia hemolytica = “Yellow Lamb” ➔sheep (ichterus and hemoglobinuria)
2. Cl. welchii type B ➔ Dysentri = “Lamb Dysentri”
3. Cl. welchii type C ➔

   Struck (adult sheep) ➔ suddendeath after convulsion

   Enterotoxaemia (cattle) ➔ death after hours diarrhea

   Enteritis haemorrhagia (young swine and fowl) ➔ death, 72 hours after neonatus
4. Cl. welchii type D ➔ Pulpy Kidney & Enterotoxaemia (sheep) ➔ similar with dysentri

PULPY KIDNEY
Enterotoxaemia= Over eating disease

- **INTRODUCTION**

Acute and fatal Entoxication in sheep

Cause Epsilon toxin which producing by *Cl. Welchii type D* in intestine

This organism is found in the soil and in the alimentary tract animals

More toxigenic varieties of the organism cause fatal toxaemia in sheep, calves, young pigs and man

ETIOLOGY

-Clostridium welchii = Clostridium perfringens

-Rods, singly or in pairs, non motile, capsule, Gram positive

-The spores are oval at central or subterminal

-Produce 4 toxins ➔ alpha, beta, epsilon & iota

-Toxins are heat labile

-If toxin + chemicals ➔ toxoid (antigenicity but have no toxicity)

EPIZOOTIOLOGY

-The disease in Indonesia ➔ has not been found

But occurs in Australia, New Zealand, England & USA

-Sensitive animals : sheep, calves, goats, foals and man and young animals more sensitive than the old ones

PATHOGENESIS

The bacteria invade in tissue tract digestivus ➔ produce toxin in small intestine ➔ absorbtion ➔ mucous membrane

CLINICAL SIGN :

Incubation periode : 2 – 3 hours

Convulsion with agonal struggling, dyspneu and death

Type of the disease are :

1. Peracute (neuro type) ➔ convulsion, dyspneu & death
2. Subacute ➔ deprresion
3. Chronic (digestive type) ➔ diarrhae and recovery after 1 week

PATHOLOGICAL CHANGES :

Inflamation, ulceration and hemorrhagic of small intestine

Congestion and oedematous of caecum and colon.

DIAGNOSIS :

1. Clinical sign and pathological changes
2. Isolation and identification of bacteria
3. Identification toxin by Serum-netralization test

DIFFERENTIAL DIAGNOSIS :

- Black Disease (*Cl. novyi type B*)
- Black Leg (*Cl. chauvoei*)
- Anthrax (*B. anthracis*)

CONTROL AND TREATMENT :

1.PREVENTION :

Don’t giving highly and suddenly of protinaceous diets

Immunization ➔ vaccination and antiserum

2. TREATMENT :

Antibiotics (Chlortetracycline and Penicilline in feed)

Animals that suffer Pulpy Kidney do not enter to slaughterhouse.


Thankyou