CLOSTRIDIUM

- Anaerobic
- Spore forming
- Gram positive
- Resemble a spindle shape
- Clostridium is derived from Kloster meaning spindle

DISEASES

- Black Disease: Cl. Novyi (TYPE B)
- Big head disease: Clostridium novyi, Cl. Sordlli or rarely Cl. Chauvoei
- Red water: Cl. Hemolyticum
- Enterotoxemia: Cl. Perfringens type D
- Botulism: Cl. botulinum
- Braxy: Cl. septicum
- Tetanus: Cl. tetani

INFECTIONOUS NECROTIC HEPATITIS OR BLACK DISEASE

Etiology

- Acute
- Infectious
- Fatal
- Clostridium novyi type B
- Anaerobic
- 3 strains, A, B & C
- B is involved in this disease
- Under field conditions, it is associated with Fascioliasis

Prof. Ahrar Khan

5 June, 2015
Epidemiology

- Infectious disease of adult sheep (1 to 4 yr-old) and less frequently in young sheep and goats
- Morbidity rates: 5 to 10%
- Disease is always **FATAL**
- Most cases occur in **SUMMER** and **EARLY FALL** (High temperature and moisture)
- (Marshy areas, rainfall, summer - ideal conditions for the liver fluke development on pastures)

Pathogenesis

- *Clostridium novyi*
- **Soil borne**
- Through intestinal wall
- Fecal contamination
- Lodges in liver as latent infection
- By migration of liver (immature) fluke in liver
- Anaerobic environment (Hypoxia)
- Activates bacteria
- Release of toxin (Necrotizing toxin)
- **Hepatic necrosis & Fatal toxemia**

Antemortem findings

- May be sudden death in sheep
- Illness if observed is brief
- reluctance to move,
- drowsiness,
- rapid respiration,
- Fever (40 – 42°C)
- Affected animals tend to lag behind the flock (Segregation)
- Assume sternal recumbency

Postmortem findings

- Postmortem decomposition occurs rapidly.
- **Blood stained froth in airways**
- straw-coloured fluid in serosal cavities
- Dark brown swollen liver showing necrotic areas surrounded by a zone of hyperemia
- Most characteristic lesions are **grayish yellow multiple necrotic foci in liver that often follow the migration track of immature flukes**
- Enlarged pericardial sac, filled with straw colored fluid (**Hydropericardium**
Postmortem findings

- Petechial hemorrhages on pericardium and endocardium
- Darkened and cyanotic s/c tissue due to small blood vessel engorgement (dark appearance of the skin) – Hence the name “Black disease” derived
- Clear straw colored fluid in
  - Abdominal &
  - Thoracic cavities
  - Pericardial sac

Liver infarct

Large sharply demarcated infarct in the right parietal lobe of the liver (arrow).

Dark brown swollen liver showing necrotic areas (1–2 cm) in diameter surrounded by a zone of hyperemia.
Centrally located hepatocytes have undergone ischemic necrosis.

Differential Diagnosis

- **Black disease** (adult age; chronic; segregates, s/c cyanosis)
- **Enterotoxemia** (mostly young age; peracute, pulpy kidneys)
- **Bacillary Hb-uria** (Any age; Hb-uria; acute, Large anemic infarct in subterminal branch of portal vein)

Red water

- **Acute and fatal disease of cattle and sheep**
  - *Clostridium hemolyticum* type D
    - Anaerobic and spore forming
    - Can live for long time in contaminated soils and infected carcasses
    - High incidence in alkaline soils (pH 8)
- **Characterized by**
  - High fever
  - Hb-uria (in circulation: RBC and Hb. reduced)
  - Jaundice/Icterus
  - Abdominal pain, Labored respiration
  - Presence of necrotic foci in the liver
Pathogenesis: *Liver fluke + Cl. hemolyticum*

- *Cl. hemolyticum* → Through the intestines → Liver
- Organized thrombus formation in the subterminal branch of portal vein → Anaerobic conditions in liver due to migration of flukes
- Large anemic infarct: Bacteria found in the infarct that produce two types of toxins
  - Hemolysin: Acute hemolytic anemia
  - Necrotizing: necrotic lesions in liver

Lesions

- Anemia/icterus
- Dehydration
- Rigor mortis develop quickly
- Anemic infarct in liver:
  - Pale colored
  - Surrounded by zone of hyperemia
  - General appearance of necrosis
- S/c gelatinous edema becomes crepitant in few hours

Lesions

- Extensive
  - Petechial or diffuse hemorrhages in S/C tissue
  - Fluids in
    - Plural
    - Pericardial
    - Peritoneal cavities

S/C Edema

- Extensive
  - Petechial or diffuse hemorrhages in S/C tissue
  - Fluids in
    - Plural
    - Pericardial
    - Peritoneal cavities
- Red urine in kidneys & bladder
- Kidneys: dark, friable and studded with petechiae
- Small Intestines: hemorrhagic, feces + blood clots

Differential Diagnosis
Parturient Hb-uria

- An acute disease
- High yielding buffaloes and cows
- History of parturition
- Characterized by
  - Hypophosphataemia,
  - Intravascular hemolysis,
  - Hemoglobinuria and
  - Anemia

Differential Diagnosis
Parturient Hb-uria

ANTHRAX:
ZOONOTIC AS WELL AS BIOTERARISM IMPORTANCE

Other biothreat agents: Brucella, tularemia, plague, botulism, hemorrhagic fevers, smallpox, aflatoxicosis

Prof. Ahrar Khan
Anthrax

- highly pathogenic for most herbivorous animals and human
- Disease occurs worldwide
- Alkaline soils favorable for the survival of the spores
- *Bacillus anthracis*
- Large Gram positive, spore forming, non-motile bacillus
- Spores remain viable in soil for decades

Anthrax

- In ruminants, the disease is usually brief and septicemic;
- in horses, pigs, and dogs, it is frequently localized to the throat or intestine and may be fatal before invasion of the blood occurs.

*Bacillus anthracis*: Virulence Factors

- Edema Factor
- Protective Antigen
- Lethal Factor
- Capsule inhibits phagocytosis
How does anthrax manifests itself?

- **Incubation period**: 1-14 days
- **A common feature**: Animal in apparently good condition die suddenly without showing signs of ill health
- **Acute cases**: Fever, depression, difficult breathing and convulsions
- **Animals may die within 2-3 days**
- **Bloody (tarry red color)** discharge from all natural openings of the body

The pathogenesis of anthrax is an initial lymphangitis and lymphadenitis, which develops into septicemia.

- Spores that are inhaled are ingested by cells lining alveoli and transported to the tracheobronchial nodes,
- Spores vegetate in lymph nodes and spread to blood is via lymphatics as well as by lymphovenous connections within lymph nodes, and numerous bacilli spread in the lymph node to node
- Bacilli that enter the blood are taken up by mononuclear phagocyte system, especially the spleen, where secondary infection is established
- **Physiological disturbances, clinical signs and death** depend on the development of a massive septicemia
Once the degree of bacteremia passes a certain threshold, which is about 0.3% of maximum, death will occur even though all bacilli may have been destroyed by antibiotic therapy.

The toxin consists of three complementary components designated factors I, II, and III, or edema factor, protective antigen, lethal factor, respectively.

The combined effects of the three toxins are injury and inactivation of phagocytes, increased capillary permeability, anticomplementary activity, and impairment of coagulation.

Bovine anthrax is usually septicemic, and sudden death is usually the first indication of its presence in a herd.

Even when cattle are observed closely, they may be dead within 1 hour of showing signs of illness, although some will show general signs of illness for about 24 hours before death.

No post mortem of animal suspected died of anthrax: Reason

- Source of spreading infection: zoonosis
- Environmentally resistant spores when bacterium comes in contact with air (oxygen); source of re-infection of susceptible hosts
- Putrefactive processes kill most, if not all, the vegetative B. anthracis cells in the carcass within a few days
- Characteristic terminal bloody exudate from body opening, some organisms may escape and sporulate

The signs of illness vary with the route of entry and when, as usually happens, entry is by inhalation or ingestion with no area of localization, the animals are depressed and listless.

On examination there is high fever, increased heart and respiratory rates, and congested and terminally cyanotic mucosae that show evidence of bleeding.

Animals that survive for a day may have dysentery, abortion, edematous swellings of the perineum, throat and abdominal wall, and blood-stained milk.

Spread to Humans

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**Forms of Anthrax**

- **Cutaneous:** Spores enter the broken skin, germinate and rapidly proliferate at the site of entry
- **Pulmonary:** Spore inhalation and phagocytosis. Hemorrhagic necrosis of lymph nodes
- **Gastrointestinal:** Spreads to mesenteric lymph nodes, septicemia, shock, and death

**Inhalational Anthrax Victim**

*view of chest cavity*

- Lung
- Heart

**Anthrax: Enlarged, dark & softened spleen**
Control measures

- Aimed at breaking the cycle of infection depicted (Previous Figure).
- Correct disposal of anthrax carcasses (point X)
- Correct disinfection, decontamination and disposal of contaminated materials (point Y)
- Vaccination of exposed susceptible animals (point Z1) and humans in at-risk occupations (point Z2).

Proper disposal of carcass

- Incineration
- Burial
- The last resort in such situations is to leave the carcass unmoved and adequately closed off from other animals, particularly scavengers, or people.
- The carcass should be fenced off and covered using branches of trees, corrugated iron or any other available materials, and hazard signs should be posted around the site.