University of Mosul
Lecture No.:
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Website:

Lecture title: Pulpy kidney

Synonyms : Enterotoxemia, overeating diseases.

Lecturer Affiliation: Department of Internal and preventive medicine

Summary:

Definition:

An acute infectious toxemia disease of ruminants caused by the toxin of *Clostridium perfringens type D*, and characterized clinically by diarrhea , convulsion ,paralysis and sudden death.

Etiology: *Clostridium perfringens type D*

C. perfringens type D normally inhabits the alimentary tract of sheep and other ruminants but only in small numbers. Under certain conditions, the organisms proliferate rapidly in the intestines and produce lethal quantities of epsilon toxin.

Epidemiology

Distribution: Occurs world wide.

Transmission and mode of infection:

The feces of animals or carcasses of dead animals are considered the most important sources of contamination of pasture and soil.

Susceptible host : mainly sheep , to lesser extent goats and occasionally cattle and camels.

Factors influencing susceptibility :

1- It occurs in all breeds and sexes of sheep, but is more common in animals 1-12 months of ages.

2- Lambs on well fed heavy milking ewes or feedlot lambs after they start fattening at 2-3 weeks are more susceptible" overeating" .

Pathogenesis :

A. *Cl. perfringens* type D spores are ingested with contaminated feed and water.Under normal conditions, some viable organisms are destroyed in the rumen and abomasums.Some survive to reach the small intestine , where rapid multiplication and production of toxin occurs , especially epsilon protoxin is converted to the active epsilon toxin by trypsin.Under normal conditions, the movement of ingested keeps the bacterial population and toxin content down to a low level, but if intestine stasis occurs , this may be due to the presence of toxins themselves or due to overeating and passage of undigested food particles to the small intestine , accumulation of the bacteria and toxin occurs.

B. The epsilon increases the permeability of the intestinal mucosa to this and other toxins.The first effect of the toxin is to cause a profuse mucoid diarrhea. After absorption it produces a stimulation and depreesion of the central nervous system.There is severs hyperglycemia , glucouria, hemoconcentration and elevation of blood concentrations of pyruvate , lactate and Gamma Ketoglutrate.Death comes from shock and toxemia. The rapid postmortem autolysis of kidney tissue is characteristic.

Clinical signs:

In lambs the morbidity rate is 10% .The mortality rate approaches 100%. The course of the disease is very short (usually less than 2 hours and never more than 12 hours), and the disease is characterized by sudden death or signs of illness such as dullness , depression , anorexia , convulsions , salivation , diarrhea , staggering , recumbancy , opisthotonus and death.

In adult sheep, the course may extend to 24 hours. The animals isolate themselves , staggering , knuckling , champing of the jaws, salivation , shallow, rapid respiration, neurologic signs, including convulsions, muscle tremors , grinding of the teeth , salivation and death.

Postmortum lesions:

The rumen and abomasums may be overloaded with concentrates and show patchy congestion of the abomasal and intestinal mucosae.The liver may be dark and congested. There is an excess of clear straw colored pericardial fluid, sometimes gelatinous and blood stained.The characteristic change is the presence of soft, pulpy kidneys a few hours after death.

Diagnosis:

A- Field diagnosis:Clinical signs and typical postmortum lesion.

B- Laboratory diagnosis: depending on demonstration *Cl. perfringes* toxins in the intestinal contents by :

1- A polyclonal capture ELISA (Direct ELISA)could detect lower concentrations of epsilon toxin in intestinal fluid, pericardial fluid.

2- Mouse neutralization tests

Bowel filtrates can be tested for toxicity by injection into mice.

If the filtrate is toxic, the type of toxin can be determined by the protection of the mice with specific antisera, but this does not determine the type of Clostridia. The detection of beta toxin indicates the presence of types B or C, and epsilon toxin the presence of types B or D.

3- A high blood sugar level and marked glycosuria are characteristic of the terminal stages of enterotoxemia in sheep, and are supportive for a diagnosis but are not pathognomonic.

Prognosis : unfavourable

Differential diagnosis:

1- Anthrax

2-Black disease

3- Bloat.

4-Pregnancy toxemia

Treatment.

In general, the clinical course of the disease is too acute for effective treatment. Hyperimmune serum, an efficient short- term prophylactic, is unlikely to be of much value in sick animals because of the acute nature of the disease. In goats the course is longer, and antitoxin combination with orally administered sulfadimidine may be effective in treatment.

Control: There are three major control measures available:

1. Reduction of the food intake.
2. Hygienic disposal of carcasses.
3. Vaccination.

A multivalent bacterin-toxoid containing antigens to all of the clostridial diseases is commonly used in sheep in these circumstances or where all of these diseases are likely to occur.