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Popular Article

Enterotoxaemia associated with *Clostridium perfringens* type D

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Abstract

Clostridium perfringens type D produces fatal enterotoxaemia in sheep and goat. It has worldwide distribution and economically important infectious disease. This disease has rapid progress of clinical signs and usually results in death in most cases prior to the treatment. The major pathogenic effect of bacteria is produced by epsilon toxin which increases the intestinal mucosa permeability and brings vascular changes. Treatment, prevention and control of disease depends on supportive treatment, anti-toxin, correction of diet and vaccination.

Introduction

Enterotoxaemia is also known as overeating or pulpy kidney disease. It is most common economically important infectious disease of sheep and goats throughout the world. It is most likely the significant cause of sudden death in goats. This condition is caused by absorption of large amount of toxins by *Clostridium perfringens type D* from the intestine. This bacterium is normally found in soil and gastrointestinal tract as a part of normal microflora. Enterotoxaemia is most common in young age or weaned on feed lots and adult on high carbohydrate diet or on lush green pasture. Sudden and high mortality rates are more in lambs and kids.

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Etiology

The gram positive, anaerobic, spore forming bacteria *Clostridium perfringens* type D is an important causative agent. This bacterium produces several toxins, among these epsilon toxins is most important cause of vascular damage. The ingestion of high level of carbohydrate or lush green pasture and sudden change of diet acts as predisposing factor.

Epidemiology

It is disease of ruminant animals chiefly in lambs and kids and worldwide in distribution. It rarely occurs in adult cattle, deer and domesticated camels. The highest incidence is seen between 3 to 10 weeks of age. Lambs weaned at 10 months of age are second most susceptible age and is generally associated with highly nutritious diet. A high frequency of infection has been linked with severe tapeworm infestation. Morbidity often affects less than 10% of herd, but it has a high mortality rate and typically results in the death of all affected animals (Miyashiro et al., 2007).

Pathogenesis

C. perfringens normally present in soil and as a part of intestinal microflora. Usually, toxemia does not occur because passage of ingesta keeps the bacterial population in control. In instances such as high carbohydrate or roughage diet, heavy milk feeding and sudden change in diet there will be effect on intestinal motility which gets reduces. This results in multiplication of bacteria and excess toxin accumulation. The most pathogenic toxin is epsilon toxin which increases the intestinal mucosa permeability. The receptor for epsilon toxin has been found on vascular endothelial cells and is associated with wide-spread vascular damage and hemorrhagic gastro-enteritis. Due to mobilization of hepatic glycogen, there is pronounced hyperglycemia.

Clinical findings (Sumithra *et al*., 2013; Jemal *et al.*, 2016)

- In per acute cases sudden death is the principal manifestation.
- In acute cases there is green or pasty diarrhea, frothy salivation, colic, bloat, recumbency, staggering, colonic convulsions, opisthotonos and death. In adult animals champing of jaw, blindness, rapid and shallow respiration, salivation, atonic rumen, pasty faces and animal lag behind the rest of flock.
- In sub-acute cases there is anorexia, intermittent diarrhea or dysentery.
- In chronic cases there will be progressive weight loss, emaciation and anemia.

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Diagnosis

- Diagnosis based on type of diet and clinical features.
- Clinical pathology: High blood sugar level up to 150-200mmol/L in terminal stages of enterotoxaemia.
- Necropsy finding: characteristic kidneys have mottled appearance and soft consistency (soft pulpy kidneys), petechial hemorrhages on intestine, abomasum, pericardium and pulmonary edema. Onset of carcass putrefaction is rapid. In nervous form focal symmetrical encephalomalacia.
- Laboratory diagnosis: toxins are detected by serum neutralizing test in mice and guinea pigs. Polyclonal capture ELISA and counter immunoelectrophoretic are advantageous as they require less sample and these are fast.

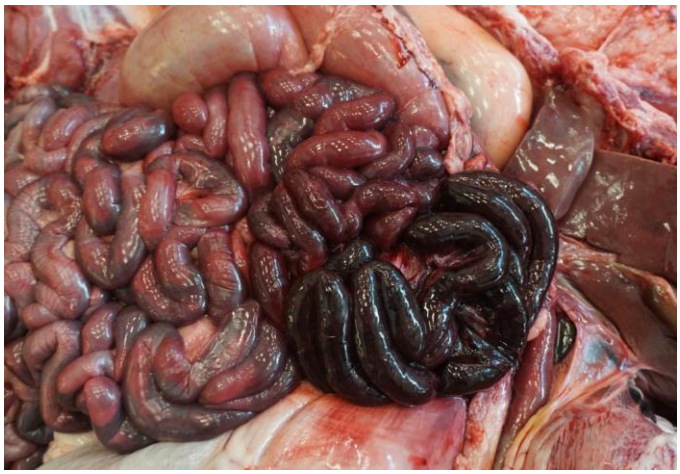

Fig.1: The intestines contain a segment of bowel that is dark red to purple marked necro hemorrhagic enteritis (Pfeifer, 2021).

Sample collection

a) Bacteriology: 20-30 ml of intestinal content
b) Clinical pathology: urine
c) Histology: fixed colon, ileum, jejunum, entire brain

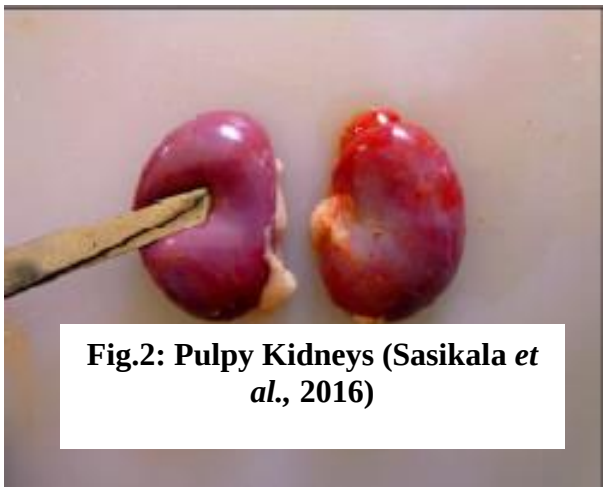

Fig.2: Pulpy Kidneys (Sasikala *et al.*, 2016)

Treatment

Due to rapid progression of disease treatment is usually ineffective. Hyperimmune serum can be given but is not of much value in acute of disease. Antitoxin in combination with orally sulphadimidine is found to be effective in goats (Jemal *et al.,* 2016). Therefore, the key to the treatment is prevention.

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Control and Prevention

- Reduction in food intake and increase the frequency of feeding.
- Avoiding heavy tapeworm infestation.
- In case of outbreak Epsilon antitoxin @ 200IU/kg bwt. can be administered.
- Vaccination: young ones can be vaccinated at 4-10 weeks of age and again a month later. Vaccine contains formalin inactivated toxoid and bacterin of *Clostridium perfringens* type D adjuvated with aluminum hydroxide. Dose = 2ml be S/C route.

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