University of Mosul
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Lecture title: ENTEROTOXEMIA ASSOCIATED with *Clostridium perfringens type D* (PULPY KIDNEY, OVEREATING DISEASE)

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Summary:

ETIOLOGY:

1- Enterotoxemia results from the proliferation of ***C. perfringens* type D** in the small intestine.

2- This organism produces a number of toxins, of which the **epsilon toxin** is the most important and results in **vascular damage** and the **damage to the nervous system** typical of this disease.

3- The presence of *C. perfringens* type D in the intestine does not in itself result in disease unless other factors intercede that promote proliferation and the production of toxin.

4- The natural habitat of the organism is in the intestine and in soil contaminated by feces, although it does not persist in soil for long periods of time.

EPIDEMIOLOGY:

- **Occurrence:**

1- Enterotoxemia associated with *C. perfringens* type D is a disease of ruminant animals, primarily of lambs, and is worldwide distribution.

2- The common practice of vaccination against this disease has reduced its prevalence, but it is still a common disease.

3- Although **most common in lambs,** it is also an important disease of **calves and goats**. It occurs **rarely** in adult cattle, deer, domesticated camels, and possibly horses.

4- **In pastured sheep**, it causes heavy losses, particularly in flocks managed for the production of lamb and mutton.

5- **The case–fatality rate approximates 100%.** In North America enterotoxemia ranks as one of the main causes of loss among feedlot lambs. It ranked third in importance as a cause of death despite a policy of vaccination, and the costs of prevention programs were the largest expenditure of all disease prevention programs in the feedlots.

- **Animal and Management Risk Factors:**

1- ***C. perfringens*** **type D** normally inhabits the alimentary tract of sheep and other ruminants but only in small numbers.

2- The organism does not persist for more than 1 year in the soil. Under certain conditions, the organisms proliferate rapidly in the intestines and produce lethal quantities of epsilon toxin. Mostly, the affected animals are on **highly nutritious diets** and are in **very good condition.**

3- **The husbandry conditions** in which the disease occurs include grazing on lush, rapidly growing pasture or young cereal crops, and heavy grain feeding in feedlots. Lambs on well-fed, heavy-milking ewes are particularly susceptible. The occurrence of the disease under these conditions has given rise to the name **"overeating disease."**

Sheep, Goats& Calves:

1- The highest incidence of the disease is in suckling lambs between **3 and 10 weeks of age**, although lambs as young as **1 to 5 days old can be affected. In calves**, the disease is most common between **1 and 4 months of age.**

2- The risk for disease in this age group is highest when ewes are grazed on lush pastures that result in profuse lactation. Larger and more rapidly growing single lambs are more susceptible than twins.

Weaned lambs up to 10 months of age are the second most susceptible age group, and again the occurrence of disease is associated with **highly nutritious diets.**

3- Feeder lambs are most commonly affected soon after they are introduced into feedlots.

4- **The peracute disease in goat kids** has the same age occurrence as in lambs, but less acute and chronic forms of enterotoxemia occur in adult goats. Sudden changes in diet appear to be the most common predisposing factor.

5- Outbreaks in sheep and goats have followed the administration of phenothiazine and other antihelmintics, and a high incidence has been observed in association with heavy tapeworm infestation.

Horses:

Type D enterotoxemia is rare in horses, but it has been suspected in mature horses fed concentrates during a drought.

PATHOGENESIS:

1- In certain circumstances, multiplication of the organisms and the production of toxin proceeds to the point in which toxemia occurs. The passage of large quantities of starch granules into the duodenum when sheep overeat on grain diets or are changed suddenly from a ration consisting largely of roughage to one consisting mainly of grain.

2- Factors such as **heavy milk feeding** may have the same effect. A slowing of alimentary tract movement has also been thought to permit excess toxin accumulation; also it can be any factor that causes intestinal stasis will predispose to the disease.

3- **The epsilon toxin of *C. perfringens* type D** is a pore-forming protein that increases the permeability of the intestinal mucosa to this and other toxins, facilitating its own absorption.

4- **Acute cases** are characterized by the development in the **brain of degeneration of vascular endothelium; perivascular and intercellular edema**, The damage to the vascular endothelium leads to the accumulation of protein-rich fluid effusions observable in heart, brain, and lung.

5- **The postmortem autolysis of kidney tissue** that occurs so rapidly and is the characteristic of **"pulpy kidney"**. There is a pronounced hyperglycemia caused by the mobilization of hepatic glycogen; severe hemoconcentration; and elevation of blood concentrations of pyruvate, lactate, and α-ketoglutarate.

CLINICAL FINDINGS:

- **Lambs:**

The course of the illness is very short, often less than 2 hours and not more than 12 hours. Many lambs are found dead without previously manifesting signs. In closely observed flocks the first signs may be dullness, depression, yawning, facial movements and loss of interest in feed. Acute cases may show little more than severe clonic convulsions with frothing at the mouth and rapid death.

Cases that survive for a few hours show a green, pasty diarrhea, staggering, recumbency, opisthotonus, and severe clonic convulsions. Death occurs during a convulsion or after a short period of coma.

Adult sheep:

These usually survive for longer periods of up to 24 hours. They lag behind the flock and show staggering and knuckling; champing of the jaws; salivation; and rapid, shallow irregular respiration.

There may be bloat in the terminal stages. Irritation signs, including convulsions, muscle tremor, grinding of the teeth, and salivation, may occur but are less common than in lambs.

Calves:

1- The syndrome is similar to that seen in adult sheep, with nervous signs predominating. Peracute cases are found dead without having shown premonitory signs of illness and with no evidence of struggling.

2- The more common, acute cases show a sudden onset of bellowing, mania, and convulsions, with the convulsions persisting until death occurs 1 to 2 hours later.

3- Subacute cases, many of which recover, do not drink, are quiet and docile, and appear to be blind.

- They may continue in this state for 2 to 3 days and then recover quickly and completely.

Goats:

1- Diarrhea is a prominent sign in affected goats, especially in those that survive for more than a few days.

2- In the peracute form, which occurs most frequently in young kids, there are convulsions after an initial attack of fever (40.5°C) with severe abdominal pain and dysentery; death occurs in 4 to 36 hours.

3- In the acute form, which is more common in adults, there is usually no fever, and abdominal pain and diarrhea are prominent with death or recovery within 2 to 4 days.

In chronic cases, the goats may be ill for several weeks and show anorexia, intermittent severe diarrhea and, in some cases, dysentery and the presence of epithelial shreds in the feces. Chronic wasting, anemia, and eventual emaciation also occur with chronic disease in goats.

CLINICAL PATHOLOGY:

A high plasma glucose concentration of 8.3 to 11.1 mmol/L (150 to 200 mg/dL) Hyperglycemia, and marked glycosuria are characteristic of the terminal stages of Enterotoxemia in sheep, and are supportive for a diagnosis but are not pathognomonic.

Bowel filtrates can be tested for toxicity by injection into mice. If the filtrate is toxic, the type of toxin can be determined by protection of the mice with specific antisera.

Commercial enzyme-linked immunosorbent assay (ELISA) kits and multiplex PCR assays have become available for toxin detection.

NECROPSY FINDINGS:

1- The body condition of the animal is usually good, but there is often fecal staining of the perineum and rapid decomposition of the carcass.

2- In peracute cases there may be no gross lesions. More frequently, there is an excess of clear, straw-colored pericardial and thoracic fluid that clots on exposure to air.

3- Many petechiae are present in the epicardium and endocardium, and there is pulmonary edema. Patchy congestion of the abomasal and intestinal mucosae is usual, and the small intestine often contains a moderate amount of thin, creamy ingesta.

The content of the large intestine may be watery and dark green. The characteristic finding of soft, pulpy kidneys is only useful in animals necropsied within a few hours after death because it is nonspecific and merely correlates to a more rapid rate of autolysis. The liver is dark and congested. The rumen and abomasum of feedlot lambs may be overloaded with concentrates.

Samples for Confirmation of Diagnosis:

- **Bacteriology**: 20 to 30 mL of intestinal content, frozen in a leak-proof glass or plastic container (ELISA, latex agglutination, bioassay, anaerobic culture, PCR
- **Clinical pathology**: urine (assay–glucose) (best performed at time of necropsy)
- **Histology**: fixed colon, ileum, jejunum, entire brain

Differential Diagnosis:

Lambs:

- Acute pasteurellosis
- Septicemia associated with *Histophilus somni* (formerly *Haemophilus agni*)
- *Clostridium sordellii*
- Polioencephalomalacia
- Rumen overload

Sheep:

- Hypocalcemia
- Hypomagnesemia
- Focal symmetric encephalomalacia (chronic enterotoxemia)
- Rabies
- Pregnancy toxemia
- Louping-ill

Calves:

- Lead poisoning
- Polioencephalomalacia
- Hepatoencephalopathy
- *H. somni* (formerly *Haemophilus somni*)

Goats:

- Salmonellosis
- Coccidiosis

TREATMENT:

In general, the clinical course of the disease is too acute for effective treatment. In goats the course is longer, and antitoxin in combination with orally administered sulfadimidine may be effective in treatment.

CONTROL:

There are three major control measures available: reduction of the food intake, administration of antitoxin, and vaccination. These may be used individually or in combination.

- **Reduction in Food Intake:**

Reduction in food intake is the cheapest but least effective in control and is used as a short-term control while waiting for immunity to develop after vaccination.

- **Antitoxin:**

Antitoxin can be administered to all sheep as soon as an outbreak commences. The administration of ε-antitoxin 200 IU/kg BW will provide for protective circulating antitoxin levels for 21 to 29 days. Immediate losses are prevented, and in most instances the disease does not recur.