**University of Mosul**
**Lecture No.:**
**College of Veterinary Medicine**
**Date:**
**Unit of Scientific Affairs**
**Website:**

## Lecture title: Enterotoxaemia

**Lecturer Affiliation: Department Of Pathology And Poultry Diseases**

**Summary:**

Enterotoxaemia Caused By Clostridium Perfringes Types B And C. Clostridium perfringens type B causes lamb dysentery and enterotoxaemia in goats. Clostridium perfringens type C also causes acute enteritis in lambs.

### Pathogenesis

Following ingestion, the organism colonise and proliferate on the intestinal mucosa. Cl. perfringens type B producing alpha, beta and epsilon toxins whereas, Cl. perfringens type C produce alpha and beta toxins only. The beta toxin causes a severe haemorrhagic and ulceration of the intestinal mucosa. In adult animals the toxins produced are inactivated by trypsin but the neonate animals do not produce enough quantities of trypsin to destroy the beta toxin. Hence the disease is restricted to lambs under 1-2 weeks old. Necrosis and desquamation of the mucosa occurs resulting into ulcers. The irritation of the intestinal mucosa caused by presence of the organisms and toxins increases peristaltic movements and disturb the absorptive mechanisms on the mucosa resulting in loss of water and electrolytes from tissues into the gut. This is followed by dehydration, and acidosis. toxemias which is caused by presence of toxins in the circulation cause shock which progress into death.

### Clinical signs

Peracute, acute, subacute and chronic syndromes may occur. The peracute disease is characterized by sudden death without any premonitory signs. The acute syndrome is characterized by depression, failure to suckle, severe abdominal pain, bleating and lagging behind the flock or recumbency. A brownish or bloody diarrhoea is also present. The lambs then become comatose and die within 24 hours from the onset of the disease

subacute syndrome is manifested by dullness, sluggish movements and abdominal pains. Tenesmus and a mucoid yellowish or blood-stained diarrhoea are observed. Death follows after prostration and coma. The chronic disease is mainly characterized by unthriftiness and a mucoid or blood-stained diarrhoea may be observed.

### Pathological features

The acute and subacute cases are characterized by haemorrhagic enteritis, congestion and ulceration of the mucosa which becomes dark red. The intestinal contents are bloodstained and the peritoneal cavity contains excess serous fluid. Adhesions of intestinal loops and presence of

frank blood in the intestinal lumen have been reported. Perforation of the ulcerated intestine may occur resulting in peritoneal effusion, fibrinous peritonitis and adhesions. Subepicardial and subendocardial haemorrhages and, hepatic and renal degeneration may be evident. In chronic cases, there may be splenomegaly and clotted milk may be found in the abomasum. Large numbers of Cl. perfringens can be demonstrated in smears made from the affected portions of the intestine.

In adult sheep, CL. perfringens type C causes a condition known as struck which is often characterized by sudden death although the affected animals may exhibit abdominal pain and convulsions. Ulcerative and haemorrhagic enteritis, fibrinous peritonitis, petechiae in serosal tissues and transudation in the peritoneal, pleural and pericardial are observed at post mortem. If the examination is delayed, rapid putrefaction of the carcass occurs.

## ANTHRAX

This is a peracute, acute or subacute and often fatal disease of animals and man and, in small ruminants it is characterized by septicaemia, splenomegaly and gelatinous infiltration of subcutaneous and subserosal tissues.

### Etiology:

The disease is caused by a large Gram-positive, spore-forming bacterium, *Bacillus anthracis.*

### Pathogenesis

The pathogenicity of B. anthracis is related to the presence of the antigenic capsule and the ability of the organism to produce a leucocidal protein toxin which is anti phagocytic, increase vascular permeability, delays blood clotting and produces capillary thrombosis. Increased capillary permeability causes leakage of body fluids into tissues and body cavities causing edema and haemonconcentration. Edema of the lungs interferes with pulmonary perfusion leading to hypoxia, respiratory distress and inadequate supply of oxygen to the central nervous system. Leakage of body fluid into body tissues also results in decreased serum calcium and increased serum potassium leading to hyperirritability and convulsions which are observed in some animals. Presence of the toxin in the circulation causes severe anoxia, hypoglycaemia, alkalosis and shock which terminate into death.

### Clinical features

The incubation period is 1-3 weeks Peracute and acute forms of the disease occur in sheep and goats. The peracute disease is characterized by sudden death without premonitory signs, although there may be fever, dyspnoea, muscle tremors, congestion of the mucosae and terminal convulsions in few animals. The course of the acute disease takes about 2 hours and it is initially

characterized by severe depression and listlessness. Fever (42 °C), anorexia, laboured breathing, congested and haemorrhagic mucosae, increased heart rate, rumenal stasis and reduced milk production are common features. There may blood discharges from the mouth, nostrils, anus and vulva. Diarrhoea or dysentery and edema of the tongue, sternum, flanks and perineum have been observed. Pregnant animals abort and blood-stained or reddish-yellow milk is produced. Animals then collapse and die after terminal convulsions.

## Pathological features

Post mortem examination of carcasses suspected to have died fromanthrax is not recommended because of the risk of exposure of thevegetative organisms to air which triggers the formation of endospores and,hence contamination of the environment. The common gross post mortemfeatures of anthrax in goats or sheep include complete absence of rigormortis and rapid putrefaction and bloating of the cadaver. Non-clottingdark tarry blood oozes from the mouth, ears, nostrils, anus and vulva. Thespleen is grossly enlarged with softening and sometimes liquefaction. Severeenteritis, ecchymotic haemorrhages throughout the body tissues andblood-stained fluid in body cavities are frequently observed.

**Histopathologically** there is widespread necrosis and haemorrhage intissues and, capillary thrombosis. Large numbers of vegetative B. anthracis can be demonstrated in peripheral blood during the terminal stages of the disease.

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