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ENTEROTOXEMIA
OF
SHEEP

Donald Wise

INFECTIOUS enterotoxemia is an acute, highly fatal toxemia of sheep, which is produced by the anaerobic bacillus, Clostridium perfringens (Clostridium welchii) type D, and is characterized by sudden prostration and congestion of the abomasum and small intestine. The exotoxin is absorbed into the body through the intestinal wall. A close relationship exists between the production of exotoxin by Clostridium perfringens and the heavy feeding of lambs, which is indicated by the fact that under average feeding conditions Clostridium perfringens lives in the intestinal tract as a non-pathogen. Heavy feeding is apparently a predisposing factor that is necessary in order that Clostridium perfringens may produce a fatal exotoxin that is absorbed by the body.

SYNONYMS

Enterotoxemia is known by other names, such as “pulpy kidney disease,” “milk colic,” “overeating disease,” “apoplexy” and “braxy-like disease.”

ETIOLOGY

Enterotoxemia is a combination of infectious and nutritional disease problems. The etiology of enterotoxemia is the presence of Clostridium perfringens, the active fermentation of carbohydrates and the production of type D exotoxin.

PATHOGENESIS

Enterotoxemia occurs in lambs that are receiving a large supply of their mother’s milk, feeder lambs on a high carbohydrate ration and under conditions where sheep are allowed to have access to large amounts of feed such as pasturing down wheat fields.

Mechanism of Invasion

Since Clostridium perfringens is a producer of an exotoxin, it follows that an invasion of tissues is not a prerequisite for the production of a diseased condition. An absorptive surface such as the mucosa of the intestine plus the exotoxin is all that is required to produce a toxic condition in the animal. Invasion of the intestinal mucosa does occur and its extent is usually limited by the muscularis mucosa.

The organisms are non-motile, thus motility is not involved in locomotion. Movement of the organisms is done entirely by peristalsis and other intestinal movements and movement of villi.

The organisms enter the lumen of the intestine and fill the intestinal crypts. If anaerobic conditions are present the bacteria may multiply. Large amounts of milk ingested at this time neutralizes the acidity and the bacteria multiply at a very rapid rate. Toxin which is produced is absorbed in very large amounts.
Natural cases of enterotoxemia in feeder lambs are due to the feeding of large quantities of corn, milk, barley, peas, cane and other feeds which tend to produce an alkaline medium. The large quantities of feed may cause engorgement of the intestine. The feeds swell as water is absorbed, and the result is a decreased rate of passage of ingesta through the gut or stasis which allows time for absorption of toxic quantities of the exotoxin. The stretching of the intestinal wall by the food produces atony of the musculature. Atony of the musculature results in a decrease of peristalsis and a stasis of intestinal contents.

Judging from Cole's statement the lessened exercise along with fatness encourages the lowering of the tonus of the intestinal musculature, thus favoring stasis of the ingesta.7

Experiments have shown that oral administration of Clostridium perfringens exotoxin to sheep will not produce symptoms of enterotoxemia. However, enterotoxemia is produced if opium or belladonna are administered with the exotoxin. These two drugs are capable of inducing stasis of the intestinal contents by decreasing peristalsis.

Iron is an essential mineral for the growth of Clostridium perfringens and the homolactic fermentation of this organism. Apparently the iron of foodstuffs supplies the necessary requirements for growth in the intestine. Investigators believe that the essentiality for iron is due to the need for the function of aldolase. Aldolase is the enzyme which converts hexose phosphate to triose phosphate in the formation of energy rich phosphate bonds.4

Magnesium is another mineral that is essential for the cell dividing mechanism of Clostridium perfringens. An excess or deficiency of magnesium results in the formation of filamentous forms of the organism.16

It has been suspected that enterotoximia may be set up by histamine of bacterial origin.

Australian experimenters have found that the main factors permitting absorption of epsilon toxin of Clostridium perfringens type D from the bowel were a high concentration of toxin, the presence of toxin in the anterior portion of the small intestine and some means of localizing the toxin for a period of time.1

Localization of Clostridium perfringens in the Intestine.

Clostridium perfringens is unable to multiply in wounds, tissues or the blood stream, which is due to the fact that the bacteria are anaerobic.11 The blood stream contains oxygen which is bound with hemoglobin; thus Clostridium perfringens is unable to produce a bacteremia.

Clostridium perfringens becomes localized in the intestinal tract because the portal of entry is the mouth and the mucosa of the intestine is the only place where the extensive absorption of toxin can occur. The spores of the bacteria are the forms that are ingested. The bacteria are unable to multiply in the rumen and duodenum, because the high acidity destroys all vegetative forms of the bacteria. After the spores enter the intestinal tract, multiplication of bacteria is very rapid in the alkaline medium, if anaerobic conditions are present.

The engorgement of the intestine with feed, especially carbohydrates, is another factor which is important in the multiplication of bacteria. Engorgement of the intestine decreases peristalsis and other intestinal movements, thus stasis of the food results. This condition allows impaction of the ingesta, which aids in the production of anaerobic condition. Stasis of the food also allows the necessary time which is required for the absorption of exotoxin into the blood vessels of the mucosa.

Another reason that explains why Clostridium perfringens exerts its action in the intestine is that trypsin is required to activate the exotoxin. A large portion of the exotoxin is in the form of a proto-toxin which is non-toxic.

Incubation Period.

The incubation period for Clostridium perfringens is very short in most cases of the disease. Since Clostridium perfringens is a normal inhabitant of the intestine, all that is required to produce toxemia are ideal conditions for multiplication.
Engorgement of the intestine in sheep occurs very rapidly, which is due to large quantities of the ration and the atony of the intestinal musculature. As soon as the stasis of the food occurs, the ideal conditions are present for the growth and rapid multiplication of bacteria and the absorption of toxin are present. The ideal conditions referred to above are fermentation of carbohydrates and the anaerobic conditions that are produced by impaction.

**Symptoms.**

Most of the symptoms of this disease are the result of lesions of the central nervous system. Orthotonus, a form of tenteric spasms in which the neck and limbs are held in a fixed straight line, is present. Other symptoms are incoordination of movement, walking in a circle, head thrown back on the side, pushing and straining against fences, blindness, recumbent coma during the later stages of the disease, rise in temperature of two or three degrees and glucosuria of two to six percent sugar. The animal may jump into the air, fall upon the ground and go into convulsions. Injury of the renal corpuscle and renal tubule cells by the exotoxin produces a condition whereby the kidneys are unable to prevent the escape of glucose into the urine. Degenerate changes of the liver permit glycogenolysis, which is also a contributory factor to glucosuria. Less acute attacks show regurgitation, diarrhea, inappetence and loss of condition.

**Nervous Lesions.**

Lesions of this disease are produced as a result of injury of the endothelial lining of the blood vessels and the characteristic inflammatory reactions which accompany the hemorrhages.

The nervous lesions were studied by administering the exotoxin to a dog. The lesions were hyperemia with small perivascular hemorrhages and larger extravasations accompanied by lesions in the nervous tissue. Perivascular infiltrations of mononuclear cells were present in both the white and gray matter. All the animals showed uniformly distributed and scattered foci of edema throughout the nervous tissue. Diseased nerve cells were found in foci of edema, hemorrhagic foci and in small groups in apparently normal tissues in the cerebrum, cerebellum, spinal bulb and medulla. Cells of the spinal medulla and bulb showed "acute swelling." Scattered foci of the cerebral cortex showed "cellular liquefaction." There were a few shrunken, deeply colored cells with agglutinated Nissl's granules and sinuous dendrites, "wrinkled cells" and some lightly stained "shadow cells." Proliferation of neurophages and peripheral glial cells were present around the diseased foci. The nerve fibers showed marked changes of the myelin sheathes, which were sometimes unevenly stained, sometimes fragmented and sometimes distorted into a balloon shape. The neuroglial proliferations and the myelin sheath changes were more extensive under the conditions of chronic intoxication.

**Other Microscopic Lesions.**

The pulpy kidney is brownish yellow to a distinct yellow in color. The yellow discoloration is due to the fatty degeneration of the kidney. The kidney is also soft, enlarged and friable, which is the reason for the name "pulpy kidney." The liver undergoes fatty degeneration changes, and the release of glucose occurs. The liver is yellow in color. Gas bubbles may form under the capsule. The capsule may be friable, and tawny areas appear on the surface of the engorged liver. Degenerative changes of the liver and kidney are considered to be pathognomonic for the disease. These changes appear three or four hours after death and may be both ante-mortem and post-mortem in nature.

**Gross Lesions.**

Gross hemorrhagic lesions may be lacking in acute cases of the disease. Petechial and echymotic hemorrhages may be present under the epicardium. Subendocardial and subepicardial hemorrhages may be present. Petechial hemorrhages are present under the serosa of the small intestine. Contents of the ileum resemble mayonnaise in color and consistency.
abomasum may become distended with gas and rupture. It is impossible to strip the mesentery from the entire intestinal wall without tearing the intestine every few inches. Intramuscular hemorrhages are present in the diaphragm and abdominal muscles. The pericardial sac contains a straw colored fluid which may be coagulated. The lymph nodes are edematous and hyperemic. The small intestine and abomasum are hyperemic. Rare cases may show mild enteritis and gastritis.

Hemorrhagic septicemia and paratyphoid dysentery may be confused with this disease. Lack of pneumonia rules out hemorrhagic septicemia. Paratyphoid disease may result in diarrhea which is seldom seen in enterotoxemia. Ecchymoses on the intestine and diaphragm and coagulated fluid in the pericardial sac will rule out listeriosis.

**Cause of Death.**

Death of the animal may be due to paralysis of the respiratory center which leads to cardiac failure. The symptoms of heart insufficiency and respiratory difficulties are observed.

**References**

10. Merchant, I. A. Veterinary Bacteriology and Virology.

*Tibial Injuries*

(continued from page 103)

be reduced by manipulation of the patella. Force is applied by the hand to the medial side of the patella as the affected leg is further extended. This will usually allow the patella to move back to its normal position. However, the luxation often recurs immediately. In some instances blistering the stifle following manual replacement have proven successful. Usually, sectioning the medial patellar ligament will be found to be the most successful treatment. Following desmotomy, several months of stall rest along with a fortified ration is indicated.

Estrogenic substances, Classy related to the stilbestrol used as a beef growth stimulant, has been found in high levels in the following forage plants: subterranean clover, red clover, ladino clover, alfalfa, birdsfoot trefoil, wheat, rye, oats, and beets.

Air-conditioning and heating to guarantee temperatures between 50 and 70 degrees the year round would put hogs on the market 100 days after weaning on 600 pounds of feed or less, experts say.

A recent study revealed that mow-cured hay retained 64 per cent more of the carotene of the green crop than sun-cured hay.