International Dairy Topics

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Clostridial enterotoxaemia

Introduction

A common fatal disease of calves is enterotoxaemia caused by Clostridium perfringens type C. Enteric diseases caused by Clostridium perfringens types A, B and D have been reported but are quite rare. Neonatal calves are the most commonly affected but older calves up to three months old can sustain significant losses from this disease.

Cause and predisposing factors

For the production of type C enterotoxaemia the Clostridium perfringens needs to go through a rapid growth and reproduction spurt that results in a superinfection of the intestinal lumen and toxin production. This spurt in clostridial numbers is often stimulated by an intake of a large volume of soluble carbohydrate and/or protein, for example feeding large quantities of milk or milk replacer. In older calves it can be associated with heavy feeding of grain or sudden access to high quality silage. It can also be triggered by a period of overfeeding followed by one of relatively low feed intake.

β-toxin is the principle major lethal toxin of Clostridium perfringens type C although this bacterium also produces variable amounts of α-toxin.

β-toxin induces a necrosis of the cells lining the intestine (enterocytes) and this then allows the toxin access to the deeper layers of the intestinal wall, thereby causing a widespread submucosal necrosis and haemorrhaging into the intestinal lumen.

The β-toxin is a protein that is inactivated by trypsin. As this enzyme is minimally present in the neonate because of minimal pancreatic activity, the action of trypsin inhibitors in colostrum counter the lethal effects of the β-toxin.

Ingestion of large quantities of milk/concentrate can be sufficient to dilute the pancreatic enzymes, including trypsin, to a point at which they can no longer inactivate the β-toxin. An alternative theory is that the bacterium may proliferate to such an extent that the amount of β-toxin released into the gut simply exceeds the amount of trypsin.

Clinical signs

Enterotoxaemia produces acute signs such as colic, abdominal distension, dehydration, depression and diarrhoea. In the peracute form the clinical signs may not be seen as death can occur so quickly. Colic and abdominal distension occur before the diarrhoea. The faeces are not as voluminous or watery as those seen in other conditions but may contain obvious mucus and blood. Occasionally neurological signs may be seen terminally.