Metabolic Diseases in Animals

1- Ruminal Acidosis

1.1. Definition and Pathogenesis

Sub-acute and acute rumen acidosis are very prevalent disorders of dairy herds. Two groups of cows at special risk for acidosis are early lactation cows and cows with high intake of grain in their diets. Acute acidosis is characterized by specific symptoms, which, if caught in time, can be treated directly; however, symptoms of subclinical acidosis are not very well defined. Often, subclinical acidosis is wrongly identified with poor forage quality or poor management. Because of the chronicity and unidentified signs subclinical acidosis inflicts major economic losses to dairy herds. The most typical clinical manifestation of subclinical acidosis is reduced or inconsistent feed intake. Other associated signs include decreased milk production, lowered fat content in the milk, poor body condition score, high culling rate, diarrhea, and laminitis. Subclinical acidosis occurs very often even in well managed and in high producing dairy herds.

On the other hand, acute acidosis results in very sick cows: physiological functions may be significantly impaired and death may occur. Acute acidosis is characterized by a significant decrease in ruminal pH ($\leq$ 5.0), enhanced concentrations of volatile fatty acids (VFA) and lactate in the rumen as well as a large decrease in the number of protozoa. Acidosis is most prevalent following intake of large amounts of grain or other rapidly fermented carbohydrates. Excessive intake of readily fermented starch often occurs immediately after calving when cows are first being adapted to a high-grain diet. For optimum ruminal fermentation and fiber digestion, ruminal pH should range between 6.0 and 6.4, although, even in healthy cows, ruminal pH will fluctuate below this level for short periods during the day.

Under normal conditions, bacteria in the rumen ferment the starch contained in cereal grains into weak organic acids such as VFA as well as glucose and lactic acid. Under normal feeding conditions, VFA are readily absorbed by papillae on the rumen wall.

Treatment

It is important to treat the sick cow as soon as possible. Selection of treatment for acidosis depends on the severity of the clinical symptoms. Producers need to consult a veterinary specialist for the most appropriate treatment. However, the following can be used as a guide until the veterinarian is contacted. If some cows show mild signs of

acidosis such as diarrhea, they should be fed a diet with smaller proportion of grain or increased amount of roughage. Cows with more severe signs such as severe diarrhea, off feed, and in a depressed state should be removed from the grain diet and fed roughage only. Cows should be given orally about 120 g of sodium bicarbonate and an electrolyte replacer dissolved in four to five liters of water. This treatment should be repeated three times per day if possible and the cows need to be encouraged to walk around. One should keep in mind that cows having very severe signs of acidosis such as lying down and being unable to stand are unlikely to respond to this type of treatment. A veterinarian should be consulted immediately.

Prevention

There are two commonly used management practices for prevention of ruminal acidosis:

(1) increasing the proportion of roughage in the diet.

(2) decreasing the intake of starch.

The reason for increasing the amount of roughage, in the form of hay or forages, is that it lowers the frequency of eating as well as the size of a meal. In addition, by increasing the proportion of roughage in the diet the time of chewing and the amount of saliva produced will be increased.

2- Laminitis

2.1. Definition and Pathogenesis

Laminitis is an inflammatory, non-infectious condition of the foot. The causative agents of laminitis are multiple and interrelated and they have not been fully explained. Feeding diets rich in highly fermentable carbohydrates that induce an acidotic state has been identified as one of the key factors in pathogenesis of laminitis. Digestive disorders such as acidosis, changes in the gastrointestinal bacterial flora, and translocation of endotoxin into the bloodstream predisposes cows to laminitis. Gramnegative-related infectious diseases such as mastitis, metritis, and foot rot also contribute indirectly to the etiology of the disease by providing large sources of endotoxin. Several environmental factors including hard surfaces, poor bedding, and lack of or excessive exercise have been blamed for the disease. Other contributing factors, such as body weight and feet and leg structure exacerbate the foot damage that is associated with laminitis.

Development of laminitis has been suggested to go through four consecutive stages: (1) transferring of endotoxin from gastrointestinal tract into the systemic circulation and damage of blood vessels, (2) lowering of the availability of nutrients and oxygen to the foot tissue, (3) breakdown and degeneration of the foot tissue, and (4) separation of bone and soft tissue with bleeding and bruises signs and development of inflammation (i.e. laminitis).

There are three main forms of laminitis recognized as acute, sub-acute, and chronic laminitis. During acute laminitis, although the cow is systemically ill and inflammation of the corium is evident, very few clinical signs are observed. However, local sign such as pain, swelling of the foot tissue and temperatures slightly greater than normal above the corium band are obvious. Conversely, sub-acute laminitis is more of a less apparent form of the disease. The most notable signs are softer horn, yellow coloration of the sole and bleeding stains in the solar area. On the other hand, chronic laminitis is associated with several typical changes to the hoof area.

3- Milk Fever / Hypocalcaemia / Parturient Paresis

At or near the time of parturition, the onset of lactation results in the sudden loss of calcium through milk.

The total circulating calcium in the blood of the cow is about 1.5 to 2.0 gm. The daily turnover of calcium within the body of a non-lactating and lactating cow is 10 gm. and 35

gm. respectively. Every 1 to 5 hours depending on the state of the mammary gland, total circulating quantity of calcium may be removed from the blood stream.
Most cases occur in the period immediately after calving and the incidence increases with age. Serum calcium levels decline from a normal of 10-12 mg/dl to 2-7 mg/dl. The disease may occur in cows of any age but is most common in high-producing dairy cows above 5 years old.

Milk fever is a condition of older, third to sixth lactation, high-producing dairy cows. It is associated with parturition, usually within 72 hours of giving birth. Because of the high volume of milk produced during this time, and subsequent demand for calcium, these cows often develop hypocalcaemia, or abnormally low levels of calcium in the blood.

Clinical Signs

There are three progressive stages of parturient paresis.
During stage one, cows are able to stand but show signs of hypersensitivity and excitability. Cows may appear restless and bellowing. If calcium therapy is not instituted, cows will progress to stage two.
In stage two, cows are unable to stand but can maintain sternal recumbency. Depression, anorexia, dry muzzle, subnormal body temperature, and cold extremities are seen. Cows often tuck their heads into their flanks or, if the head is extended, an S-shaped curve to the neck may be noted.
In stage three, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, unresponsive to stimuli, and can suffer severe bloat. Cardiac output worsens, heart rate can approach 120 beats/min, and pulse may be undetectable. Cows in stage three may survive only a few hours.

Treatment: Treatment is directed toward restoring the serum calcium level to normal as soon as possible to avoid muscular and nervous damage and recumbency. This would minimize the associated problems of hypocalcaemia.
Recommended treatment is IV injection of a calcium gluconate salt, although SC and IP routes are also used. A general rule for dosing is 1 g calcium/45 kg body wt.
The response to properly administered calcium therapy is quite characteristic. The cow's symptoms will appear to reverse themselves as they had previously progressed. The laterally recumbent cow will sit up to sternal position, and then it will often begin to have tremors over its body.

The Approach

Replacement of calcium by parenteral administration is the most important initial step, which should not be delayed in severely hypocalcaemic animals. Most solutions are available in single-dose, 500 ml bottles that contain 8-11 g calcium. The thumb rule is when the animal is showing signs of peripheral vascular failure, hypothermia and cold extremities; calcium borogluconate should be administered intravenously.