

METABOLIC DISEASES OF DAIRY CATTLE

Introduction

Dairy cattle require minerals in their diet for optimal productivity. These are derived from the feed and fodder. The input of minerals through feed and water must balance their output through faeces, urine and milk to maintain the animal's health. If the output exceeds input, the animals meet out their normal requirements by mobilization from its body reserves for a shorter period. But continuous imbalances develop into productivity related problems.

Nutritional imbalances, deficiencies, or erratic management of feeding programs for dairy cows can create large numbers and various types of health problems generally categorized as metabolic diseases. High producing dairy cows are most susceptible to metabolic diseases during the periparturient period.

In cattle, metabolic diseases which produces an acute, temporary, but potentially fatal deficiency includes

- ◆ Milk fever
- ◆ Hypomagnesaemia
- ◆ Ketosis
- ◆ Fat cow syndrome

MILK FEVER / HYPOCALCAEMIA / PARTURIENT PARESIS

Overview

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.

Since calcium is required for the release of acetylcholine at the neuromuscular junction, affected animals will begin to experience muscle weakness. As this hypocalcaemia worsens, the cow will become too weak to stand and will eventually become comatose over a matter of hours.

Calcium physiological functions

Calcium is the most common mineral in the body and the major extra-cellular divalent cation.

- Is a structural component of bones and teeth. Over 98% of Ca present in the body is found in bone.
- Is also important in muscle contraction (this is the main reason cows suffering from milk fever go down).
- Also plays a role in blood clotting and nerve impulse transmission.

- Calcium is critically important to normal nerve and muscle function. Acetylcholine, a neurotransmitter substance acting at the neuromuscular junction, requires calcium to properly stimulate muscle movement.

Etiology

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 yrs old. Incidence is higher in the Jersey breed

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.

Outcomes

- Milk fever is one of a number of conditions that leads to a "downer" cow.
- Mastitis (Decreased teat sphincter muscle tone and increased chance of bacteria entering the teat canal)
- Uterine prolapse (poor tone of uterine wall muscles make it flabby and tend to evert)
- Retained placenta / metritis (Reduction in uterine muscle contractile activity)
- Decreased productivity

Diagnosis

An older dairy cow near calving or that has recently calved that shows clinical signs and symptoms is highly diagnostic. Serum calcium levels will reveal hypocalcaemia, or low blood calcium. However, because of the rapid nature of this illness and the

often-slow return of laboratory results, treatment is usually initiated based on clinical signs only.

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.

As all bodily functions affected by hypocalcaemia begin to reverse, the affected animal may urinate, belch, and then begin the wobbly effort to rise. Cows generally rise within one hour. Repeated treatment may be necessary in 12 hours if the cow is still unable to rise.

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.

HYPOMAGNESAEMIA / LACTATION TETANY / GRASS TETANY

Magnesium is a widely distributed metal and is prominent body constituent. Almost half of the magnesium in the body is present in the bones. The normal concentration range in plasma is 1.8-3.5 mg dl⁻¹.

The magnesium ion is essential for normal bone metabolism, normal nerve function and muscle irritability. Magnesium also plays an essential part in the coenzyme system which links normal carbohydrate metabolism with phosphate metabolism and the provision of energy for muscle contraction.

Magnesium ions are by no means efficiently absorbed from the gastrointestinal tract and only 7 to 25% is absorbed under usual dietary conditions. But resorption mechanisms are very efficient.

Excretion of magnesium absorbed in excess of needs is renal. The kidneys exert an appreciable degree of control over magnesium excretion.

A deficiency of magnesium occurs commonly in cattle and to a lesser extent in sheep.

Predisposing causes

Grass tetany (or grass staggers) occurs in those cows, which are lactating heavily and are grazing on lush pastures.

All in all lactating cows on an unsupplemented diets is in a precarious position with regard to magnesium balance because of the narrow margin between daily intakes and overall needs exacerbated by variable bioavailability.

Clinical Signs

Low magnesium blood levels can be asymptomatic or be accompanied by tetany and convulsions – ‘milk tetany’ in calves, ‘grass tetany’, ‘stomach staggers’ or ‘Hereford disease’ in adult cattle. Serum magnesium levels fall below 1.5 mg. Percent.

The signs of magnesium deficiency are those of neuromuscular hyperirritability, culminating in titanic seizures and death.

The typical hypomagnesaemic milk fever case shows what is known in some areas as ‘the blinks’ – a fluttering of the eyelids. The animal is extremely hypersensitive, shows muscular tremors and may move in circles, occasionally appearing to ‘attack’ attendants. The pupil is often constricted and the anal sphincter flaccid. Convulsions may occur and the animal falls to the ground.

Hypomagnesaemia can be complicating factor in milk fever but is less commonly associated with calving than are hypocalcaemia and hypophosphataemia.

Two types of clinical hypomagnesaemia may be distinguished.

There is an acute, often fatal type, which usually follows abrupt changes of diet, especially from indoor feeding to outdoor grazing on fresh leys (hence the term ‘grass staggers’).

The subacute type is usually recurrent and indicates borderline deficiency of magnesium. In both cases the condition is one of inadequate intake and/or absorption of magnesium.

Treatment and prophylaxis

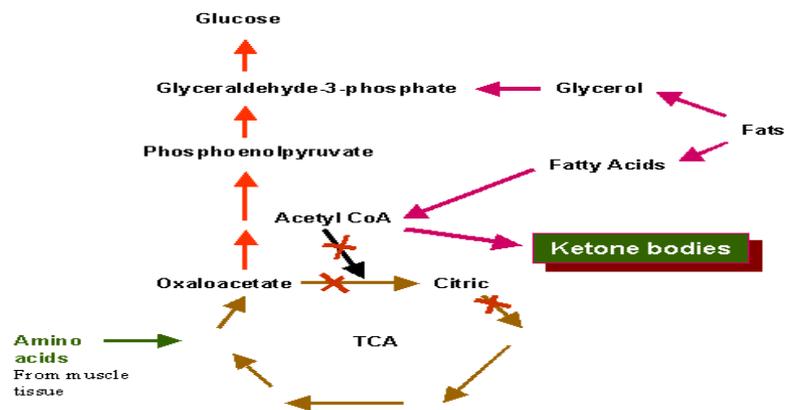
In most cases of grass tetany moderate hypocalcaemia accompanies the hypomagnesaemia. Grass tetany may also accompany ketosis.

Hypomagnesaemia cases can be treated by combine calcium, phosphorus and magnesium injections, by magnesium-dextrose injections, by calcium borogluconate-magnesium injections or by plain 25% magnesium sulphate injections.

KETOSIS OR ACETONEMIA

Ketosis or acetonemia is a common metabolic disease of lactating cows occurring during the first 10 to 60 days after calving in high-producing cows. The three-week period after calving seems to be the most critical time. The disease results from a lowered blood sugar in the circulating blood, which causes the formation and release of ketone bodies. Ketone bodies (specifically acetone) are volatilized and account for the "sweetish" smell detectable on the breath, and in the milk or urine of affected cows.

The incidence of ketosis is higher in older cows and high-producing cows. As cows produce milk, they become more susceptible.



Clinical Signs

Symptoms of ketosis in dairy cattle include dullness, depression, a staring expression, rapid loss of weight, a drop in milk production, constipation, mucus covered feces, incoordination and partial paralysis. A few cows may become highly excitable. Breathing is shallow with an acetone smell in the breath. Cows will usually consume hay, straw or other roughage but generally refuse grain or concentrates. About two-thirds of the cases are primary or uncomplicated ketosis. The other third are secondary cases, complicated by such things as retained placenta, metritis, displaced abomasum, nephritis, hardware or the other problems causing cows to go off-feed. An elevated temperature may indicate that other factors are involved.

Rumen Fatty Acids

Since ketosis is only a practical problem in ruminants, changes in the rumen have been investigated. Fatty acids (acetic, propionic and butyric) arising from microbial rumen fermentation furnish from 40 to 70 percent of a ruminant animal's energy requirements. Of these acids, propionic is by far most vital to the prevention of ketosis, and high-energy rations favor propionate production. An increase in butyric acid would be undesirable since this acid is a potential source of ketone bodies.

Other suggestions for the prevention of ketosis include the addition of sodium propionate and propylene glycol to the dairy ration. Generally, the response to either system is slow and treatment must be extended over a period of time. Sodium propionate creates a palatability problem whereas propylene glycol is completely palatable. Twice daily feeding of 120ml of propylene glycol, beginning 14 days prior to the anticipated calving date and continued for 7 weeks postpartum, reduced the incidence of ketosis by 18 percent.

Diagnosis

The ketone test is a simple diagnostic tool for determining the presence of ketone bodies and is used by veterinarians and is also available to dairymen. The test is used for determining the presence of acetone in milk and urine. Colostrum milk does not give accurate results. The urine test shows positive results before the milk test does. Even so, do not be concerned until a positive test is obtained from milk. The blood level of ketone bodies is the best test for determining the degree of ketosis.

Treatment

Most accepted ketosis treatments attempt to increase blood sugar levels. Usually, about 500 ml of a 50 percent glucose solution is used. When this is the sole treatment, relapses are frequent. As a result, most veterinarians recommend intravenous injection of glucose with the incorporation of insulin as a part of the therapy. Also, some veterinarians supplement corticosteroids for a few days following treatment to boost blood glucose levels.

For Further information please refer to our Product details of **Calcium Borogluconate and Calcium Magnesium Borogluconate**