Health Management: Polioencephalomalacia (Cerebrocortical Necrosis or Polio)

This is a fact sheet from the Health Management section of the *Alberta Feedlot Management Guide*, Second Edition published September 2000. The 1200 page guide is available for purchase on CD-ROM.

Take Home Message

- The major symptom of thiamin deficiency in cattle is polioencephalomalacia (polio).
- A low and variable percentage of feedlot cattle (1.7 to 6.6%) in Alberta on barley-based feedlot diets may have low thiamin levels indicative of the subclinical form of the disease and may be experiencing low productivity.
- Although the disease is sporadic and the incidence may be low, treatment is not particularly effective and mortality rate can be as high as 90% especially when cattle have been severely affected for more than 24 hours.
- It may be advantageous to add thiamin at 200-400 mg/head/day to feedlot rations especially when any wet-milled corn byproducts are fed.

Introduction

Polioencephalomalacia (polio) is a noninfectious disease of cattle characterised by reduced feed intake, impaired vision, muscle tremors and incoordination, head pressing against inanimate objects, grinding of teeth, groaning, convulsions and recumbency. There are two basic forms of the disease; 1) the acute form sporadically seen in feedlot cattle where affected animals are frequently found in a coma and 2) the mild or subacute form sporadically seen in animals on pasture. The incidence of the disease in Alberta is rather low (1.7 - 6.6%) (3). However, the death rate can be high (90%) in the acute case with death occurring in about 50% of the affected animals within a few days of the disease. In the subacute form, mortality is about 50%. Animals with the subacute form may recover completely or may never be completely better in terms of their low average daily gain.

Thiamin Deficiencies and Polio in Cattle

The B vitamin thiamin has two major roles in the metabolism of feedlot cattle. As thiamine pyrophosphate it is important in carbohydrate usage in the body of cattle. Thiamin is also believed to be an essential factor in the central nervous system, participating in the excitation of peripheral nerves. For the feedlot cattle, there are two sources of thiamin: 1) thiamin from the feed (both natural and supplemental) and 2) thiamin synthesized by the microorganisms in the rumen. Cereal grains and oil seed meals are comparatively rich in thiamin although about 40-70% of the thiamin from these sources is lost if they are stored for about a year.

The concept that B vitamins were microbially synthesized in the rumen and could meet the ruminant animals' requirements was generally accepted up to about the 1950's. However, from the early 1950's, cattle have been pushed to higher and higher levels of production, being finished faster by changes in diets and by the addition of ration additives which would be expected to change the requirements for the B vitamins (4). Indeed, thiamin supplementation at a level of 1.9 mg/kg diet of a concentrate barley-based diet resulted significant increases in average daily gain (ADG) of 0.22 kg in feedlot steers indicating that at least some of the steers were marginally deficient (3). This result suggests that decreased productivity in some feedlot cattle fed all-concentrate diets may be due to thiamin deficiency. However, the above suggestion is equivocal since daily gains of feedlot cattle upon thiamin supplementation has not been consistent.

Specifically, the initial discovery of polioencephalomalacia in Colorado cattle and sheep, the high economic loss, and the responsiveness of polioencephalomalacia to thiamin administration has led to a re-evaluation of the old concept that dietary supplementation is not necessary in cattle with functioning rumen. The causes of this serious nervous disease in cattle remain inconclusive but could result from long term thiamin deficiencies in rumen and other tissues due to an interplay of several factors. The following are some of the factors and conditions which may predispose your cattle to thiamin deficiency and subsequently to polio:

The destruction of thiamin

1. Thiamine may be destroyed through an increase in the amount of thiaminase in the rumen. Thiaminase is an enzyme in the rumen and there are two types of this enzyme; thiaminase type I and type II. These exoenzymes (bound to the cell surface of certain types of ruminal bacteria) destroy thiamin in the rumen. An increase in the amount of thiaminase may occur when: A) the diet is abruptly changed to concentrates. This change may cause the amount of thiamin to fall rapidly and the decrease is usually associated with an increase in certain types of ruminal bacteria with thiaminase type I activity. Apparently, the sudden decrease in rumen pH (increase in rumen acidity) releases the enzyme. Therefore, acidosis (grain overload) has been linked to polio. B) Certain species of fungi from mouldy feed produce high amounts of thiaminase and can lead to increased destruction of thiamin in the rumen.
2. Thiamine in the rumen may be destroyed by amprolium (a coccidiostat) which acts as an anti-thiamine substance (antimetabolite) and may accentuate the destruction of thiamin in the rumen.
3. A cobalt deficient diet may also lead to a thiamin deficiency although the evidence for this is unclear.
4. Excessive sulphur intake, as a consequence of high amounts in either the feed and /or the drinking water of cattle can lead to the destruction of thiamin in the rumen.

Inadequate amounts of thiamin in the rumen

The concept that there may be inadequate amounts of thiamin synthesized in cattle is only a theoretical concept at this time. Blood and Radostits (2) reported in their book that in theory there may be inadequate amounts of thiamin in the rumen if bacterial production is hindered as may be the case in cattle fed high amounts of concentrate with inadequate roughage. Indeed, considerable evidence seems to point to the fact that there is an inverse relationship between amount of thiamin synthesized in the rumen and the amount of thiamin contained in the diet. This has led to suggestions that the rumen microorganisms do not synthesize thiamin unless required or they degrade excess thiamin (1).

Clinical Recognition

It is critical that when you suspect a case(s) of polio in your herd of cattle you should call your vet. However, in case you want to diagnose this disease yourself, these are the signs (condensed from Blood and Radostits, 1989) (2) that you should be looking for. In acute cases, the most common sign of polio is an animal found dead or a downer in the pen. Cattle that are alive that may have polio may shown a sudden onset of blindness, muscle tremors, especially of the head, frothy salivation and head pressing. In the beginning, irritation and convulsions may be sporadic, but within an few hours, the irritation and convulsions may be continuous. The animal then may become recumbent. The temperature may remain normal except after extreme muscle tremors and convulsions. In the less severe cases, cattle with polio show head-pressing and remain stationary for several hours or a few days and will show anorexia. Clinical cases of polio are commonly seen in yearling feedlot cattle. Older cattle are less susceptible and recovery is more common in the older age group of cattle.

The disease should be differentiated from acute lead poisoning, hypomagnesemia, vitamin A deficiency, infectious meningoencephalitis (ITEME) among other diseases of cattle with clinical findings referable to brain dysfunction or malfunction.

Treatment

Treatment should begin immediately after the onset of signs. Treatment of choice is thiamin hydrochloride at 10 mg/kg body weight given intravenous initially and followed by similar doses every three hours for a total of five treatments. Those animals that are clinically subpar and are still anorexic by the third day after the initiation of treatment will most likely not respond to treatment and should be put down. For the rest of the cattle, a dietary change and supplementation of thiamin at 1 gm/head/day for about 2 to 3 weeks are the preferred prophylaxis.

Control and Prevention

In total, the present state of knowledge indicates that polio is linked to a thiamin deficiency cause either by its destruction or inadequate amounts in the diet and production in the rumen. It is therefore prudent to indicate supplementation of the diets of concentrate-fed cattle as a method of preventing and controlling the disease. As indicated above, 1 gm/head/day of thiamin may be necessary to control polio in the presence of thiaminase especially on high concentrate diets. Protective levels have not been established and research is needed to establish protective levels necessary to control polio in feedlot cattle. However, to prevent polio, it is becoming commonplace to supplement feedlot diets with thiamin at 200-400 mg/head/day. Thiamin hydrochloride and mononitrate are the two commonly used forms for supplementation. The cost of supplementing at these levels which hopefully would prevent polio is rather small (0.77 - 1.5 cents/head/day) and with an added benefit of an increased average daily gain of feedlot cattle in some cases, supplementation with thiamin should become a common event in the feedlot.

References

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