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Abstract

In ruminants fed high grain rations, a condition called polioencephalomalacia (PEM), characterized by central nervous system derangement, can develop. It usually is noninfectious, responds to thiamin injections, and is characterized by sudden onset. Using artificial rumen techniques, we established that PEM results from a bacteria-produced enzyme in the rumen that converts the vitamin thiamin to a nonusable alternate form, pyrithiamin, which in turn caused severe metabolic disturbances in the central nervous system.

Keywords

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Polioencephalomalacia (PEM)--Current Research Status

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Summary

In ruminants fed high grain rations, a condition called polioencephalomalacia (PEM), characterized by central nervous system derangement, can develop. It usually is noninfectious, responds to thiamin injections, and is characterized by sudden onset. Using artificial rumen techniques, we established that PEM results from a bacteria-produced enzyme in the rumen that converts the vitamin thiamin to a nonusable alternate form, pyrithiamin, which in turn causes severe metabolic disturbances in the central nervous system.

Introduction

Polioencephalomalacia (PEM) is a central nervous system disorder that occurs most often in feedlot cattle on high grain diets. It responds to intravenous injections of large doses of thiamin. Signs include muscular incoordination, "circling" and pushing on feed bunks and fences with the head. Animals are sometimes (but not always) blind. If not treated, they die in a short time. If treated too late, they may die or suffer permanant derangements because of irreversable central nervous-system damage.

In 1972, we developed an experimental system that produced PEM in sheep (Kan. Ag. Exp. Sta. Bul. 557), which permitted research on other than spontaneously occurring cases.

Because cells in the central nervous system had to be thiamin deficient to produce the observable signs, thiamin had to be either destroyed or made unavailable. Normally, rumen bacteria produce all the B-complex vitamins needed by the host animal. The diet of a grain-fed ruminant should provide excess thiamin, even if none were produced in the rumen, because thiamin is plentiful in grain. How then, can a ruminant become thiamin deficient?

Experimental Procedure

We created polioencephalomalacia in three lambs, using the experimental model mentioned. We used rumen fluid removed from the lambs in "artificial rumen" studies, added thiamin (1 mg) to 200 ml of rumen fluid, and removed samples every five minutes to see how much thiamin remained. Half of the thiamin had disappeared in 14 minutes. In rumen fluid taken from a steer with spontaneous PEM, thiamin half-life was nine minutes.

To find out if the thiamin disappeared because it was being bound inside rumen bacteria, we harvested the bacteria from the artifical rumen and broke them up with ultrasonic sound waves. No thiamin was released, so we proposed that the thiamin was destroyed by a bacterial enzyme, thiaminase.

Earlier studies had indicated that PEM occurred (on the experimental model) after animals were switched rapidly to a high level of readily fermentable energy. To examine adaptation to concentrates more closely, we started lambs on all alfalfa hay diets (1200 g per day) and then switched them to 1200 g per day of whole oats or ground milo by replacing 100, 200, or 600 g per day of the alfalfa with an equal amount of grain. Thus, some animals made the transition from all hay to all grain in 12 days, and others had only two days to adapt. We used ground milo because of its high amount of readily fermentable energy, and whole oats because it contributes less readily fermentable energy. We then measured rumen pH, thiamin, and thiaminase activity (the ability of rumen fluid to destroy thiamin).

Results and Discussion

The more rapidly the change was made from hay to grain, the more acid the rumen microbes produced. When the lambs were changed to oats or milo at 600 g per day, lactic acid acidosis occurred, and on the milo diet, thiaminase, was produced. Thus thiaminase occurred only when animals were changed to grain rapidly, and then in conjunction with lactic acid acidosis.

As grain in the diets increased, the amount of thiamin in the rumen fluid decreased; there was no thiaminase activity (added thiamin was not destroyed), so less thiamin was being produced by the rumen microbes. Grains are higher in thiamin than alfalfa is, so we expected thiamin in the rumen fluid to increase with increasing grain.

Chemically, the vitamin, thiamin, is composed of two rings. One type of thiaminase destroys thiamin by simply spliting it into its two chemical rings. The other, more dangerous thiaminase, can substitute several nitrogen-containing rings for one of thiamin's rings, creating a new compound called pyrithiamin. Pyrithiamin is particularly dangerous to the animal. For thiamin to be used in the cells, it must be phosphorylated (by an enzyme) to thiamin pyrophosphate (TPP). Pyrithiamin blocks the phosphorylating enzyme. Thus, when the "substituting" type of thiaminase is present, less thiamin is available, and what is available cannot be phosphorylated, so it is not useful to the central nervous system cells. Pyrithiamin has special affinity for brain, heart, and kidney tissues where it exhibits its blocking phenomenon.

Our artificial rumen studies on rumen fluids known to contain thiaminase have shown that as thiamin disappears, pyrithiamin accumulates. So, the thiaminase in the rumen during PEM is the more dangerous variety.

Lactic acid acidosis and PEM both occur most often on high grain diets. Our early studies indicated that both occurred simultaneously.

Our early work showed that PEM often occurred about two weeks after an increase in ration concentrate--the type of change that leads to acidosis. Other workers have shown that when the rumen becomes acidotic, histamine is often produced. We believe that histamine may be the key to the PEM-acidosis relationship. Because histamine is a nitrogen-containing chemical ring, it might lead to pyrithiamin production.

Cattle with PEM will respond to large doses of thiamin, if they are treated early enough. A 600-lb. feeder calf with PEM needs about 600 mg thiamin hydrochloride intravenously (for immediate effect) and another 600 mg intramuscularly (for longer lasting effect). Give another 600 mg of thiamin intramuscularly daily for about three days to guard against a relapse when the first thiamin is used. Change the animals back to a higher roughage ration to relieve any lactic acid acidosis that might be present. Animals should start to recover from the PEM within one or two hours. However, the damage may be permanent so the animal dies or is permanently impaired, if found too late.

If, as we suspect, PEM is related to lactic acid acidosis, gradual ration changes should help in preventing it because lactic acid acidosis then would not develop.

Feeding thiamin, at the levels required to prevent PEM, probably is not feasible. Our work with sheep indicates that cattle would require as much as one gm per day. Injecting the high levels of thiamin probably "floods the system" and allows thiamin phosphorylation even when pyrithiamine is present.

Cattle feeders should keep injectable thiamin solution on hand, because PEM must be treated immediately to prevent death or permanent damage.