Thiamine deficiency induced polioencephalomalacia (PEM)

THIAMINE DEPLETION AND POLIOENCEPHALOMALACIA: WHAT EVERY ALPACA OWNER NEEDS TO KNOW

Jill McElderry-Maxwell, September, 2011

OVERVIEW

The vitamin thiamine plays a critical role in alpaca health. Thiamine depletion can happen rapidly from a large number of causes and will lead to death unless promptly remedied by the immediate administration of injectable thiamine. Thiamine is inexpensive, but only available by prescription – and every camelid owner should have a bottle from their vet on hand at all times.

Any time an alpaca shows signs of lethargy, low appetite or neurological impairment, a shot of thiamine is a worthwhile precaution: "Thiamine is a safe and useful therapy any time we suspect neurological insult" (Evans, p. 39). It can never hurt, and it may help save your animal's life.

RUMEN FUNCTION AND THIAMINE PRODUCTION

A ruminant's digestive tract is an amazing system. Breakdown of a ruminant's diet begins in the mouth, where it is mixed with saliva and given a preliminary, brief chewing before being swallowed down to the reticulum, the first of a series of stomachs. After being later brought back up and chewed leisurely as cud, masticated food finally ends up in the rumen, or second stomach.

The rumen is a large organ that serves essentially as a fermentation vat. Much of the plant material eaten by ruminants consists of cellulose. Surprisingly, mammals are not capable of breaking down cellulose – at least, not on their own. Instead, a ruminant gets a little help from a diverse collection of microorganisms, including bacteria, protozoa and even viruses and fungi, that lives in their gut.

These microorganisms break down cellulose and other plant fibers and make their energy available to their host ruminant. The microorganisms also produce a number of substances critical to their host's survival and well-being, including the vitamin thiamine. Under normal conditions, a ruminant is able to synthesize all of the thiamine it needs for daily function without supplemental sources.

Thiamine is a B vitamin (B1). It is water-soluble and is manufactured constantly in the ruminant gut, as it is being continually depleted in turn. Thiamine plays an important role in energy metabolism for all body cells, but it especially critical in brain and heart cells. Without an adequate supply of thiamine, the brain ceases to function properly and actually begins to physically deteriorate.

Thiamine migrates from the gastrointestinal tract into the circulatory system via cellular mechanisms that are not fully understood. However, it is known that the half life of thiamine in sheep's blood is very short, typically under ten minutes (Harmeyer, 1989). Turnover in brain tissues is less rapid, but it is clear that a continuous supply of thiamine is necessary if the body's cells are to function properly.

WHAT HAPPENS WHEN THIAMINE LEVELS ARE LOW

In ruminants, the collection of symptoms brought on by low thiamine is called polioencephalomalacia (PEM). Symptoms are largely neurological in nature, as PEM results first in brain tissue swelling, and then in softening of brain tissue and the growth of brain lesions (it is also called cerebrocortical necrosis [CCN] in cattle and sheep). An animal with an advanced case of PEM will actually have holes in their brain visible upon necropsy.

Thiamine can be depleted in a myriad number of ways, and alpacas are much more sensitive to low levels than are cattle or other ruminants. They can also deplete their body's supply of thiamine much more rapidly than cattle, sheep or goats. While cattle may take weeks to show symptoms of PEM after a sudden feed change, alpacas can develop symptoms in as few as two (Evans, 2005). Why this is true is not known, but it is critical to be aware of this peculiarity. Veterinarians only familiar with PEM in cattle or other livestock may fail to appreciate just how quickly PEM can bring about the death of an alpaca without prompt and appropriate intervention. Although many cases of PEM in alpacas happen quite rapidly, prolonged periods of low thiamine availability can also lead to PEM, with animals exhibiting subtle signs of deficiency over an extended period of time.

There are many, many potential causes of thiamine deficiency. Some of the microorganisms in the ruminant gut make enzymes that break thiamine down, called thiaminases. An imbalance in gut flora may lead to a proliferation of these organisms beyond normal levels, with a resulting drop in thiamine availability to the alpaca host. Antibiotics and some wormers (levamisole, thiabendazole [Evans, 2005]) can cause rumen imbalances, as can the ingestion of feeds rich in carbohydrates. Animals experiencing lactic acidosis from eating too much grain or pelleted feeds frequently also suffer from PEM.

Thiaminases may also be ingested by an alpaca. Here in the United States, bracken fern (Pteridium aquilinum), prostrate pigweed (Amaranthus blitoides) and horsetails (Equisetum spp.) are common in many pastures and contain high levels of thiaminases (*Merck Veterinary Manual*). Thankfully, these plants generally taste bad and

have low palatability. Alpacas will seldom graze them unless there are few alternative food sources available, as may happen when pastures are overgrazed or in early spring when perennials emerge before grasses. In Australia and New Zealand, the Nardoo and rock ferns are of similar concern.

Another common cause of PEM in alpacas is coccidiosis, as coccidia rely on thiamine to reproduce and in doing so, reduce the amount available to the infected animal. Amprolium (Corid), frequently used to treat coccidiosis, is a thiaminase and its use may precipitate PEM unless supplemental thiamine is provided via injection into the blood stream. The alpaca has access to the injected thiamine but the coccidia in the gut do not. Clostridium and Streptococcus bacteria are also known producers of thiaminases (Harmeyer, 1989).

PEM may be caused by a change in an animal's ability to absorb thiamine from the gut, or by the too rapid removal of thiamine from the body. Possible causes for metabolic disruption along these lines may include changes in the weather, forages and stress levels. In short, it sometimes seems like almost anything can cause polioencephalomalacia in an alpaca.

Excess sulfates or sulfides in the diet may also cause polioencephalomalacia, but this form of PEM is not treatable with thiamine. Possible sources of excess sulfur compounds may be well or untreated water (especially in times of drought), concentrated feeds (particularly grain by-products such as distillers grains, and feeds containing molasses) and some plants under particular growing conditions. There is no currently known treatment for sulphur-induced PEM other than the removal of the sulphur source, which may save those animals in which the symptoms are less advanced. Sulphur-induced PEM should be suspected in animals which do not respond to thiamine therapy. Lead poisoning may also produce the symptoms of PEM, and can be detected by sampling blood lead levels.

SYMPTOMS OF POLIOENCEPHALOMALACIA

Animals with PEM may have diarrhea, are typically at least somewhat listless or lethargic and exhibit unusual neurological symptoms. Signs of subacute PEM may be subtle, but often include

- decreased appetite;
- failure to remain with herdmates;
- staggering or unsteady gait;
- elevated head or stargazing;
- head or ear twitching;
- excess salivation and drooling.

The acute stage of PEM is typically characterized by

- increased severity of symptoms seen in subacute PEM;
- blindness;
- grinding teeth;
- opisthotonos (spasming or arching of the back and neck – the "death arch");
- seizures and muscle spasms;
- recumbency and failure to rise.

Untreated acute PEM will lead to coma and death. Untreated subacute PEM will result in animals that fail to grow and thrive, and may also ultimately progress to death.

There are a number of other conditions with symptoms similar to PEM. While PEM should always be suspected and thiamine administered if any of the above symptoms are seen, breeders should also consult with their veterinarians in order to rule out additional potential diagnoses. Conditions that may produce symptoms similar to PEM include, but are not limited to:

- listeriosis
- grain poisoning
- rabies
- tetanus
- lead or heavy metal poisoning
- vitamin A deficiency
- ryegrass staggers
- meningeal worm parasitism
- heat stress
- sulphur-induced polioencephalomalacia

Reviewing an animal's recent history and environment may be helpful in ruling the above conditions in or out. In all cases where neurological symptoms are seen, aggressively treating with thiamine while pursuing a definitive diagnosis is recommended.

TREATMENT OF POLIOENCEPHALOMALACIA

Any alpaca breeder suspecting that one of their alpacas may be suffering from PEM should immediately reach

for the bottle of thiamine that absolutely should be in their medicine cabinet at all times. Thiamine is unfortunately a prescription item that must be sourced through a veterinarian – it cannot be obtained at the feed store when an emergency arises. The standard B complex vitamins available over the counter are not an adequate substitute for pure thiamine, preferably the 500mg concentration if possible.

Since thiamine is a water soluble vitamin, it is essentially impossible to overdose when given by injection, as the alpaca will simply excrete anything it does not need. For this reason, there is no need to be precise in dosing as long as the required minimum dose is met – "too much" is as good as "just enough". Dr. Evans recommends 6-11mg/kg (3-5mg/lb) every 8 hours for 24 hours in his Field Manual. Other veterinarians have recommended a wide variety of treatments ranging from 10mg/kg (4.5mg/lb) every three hours until symptoms are gone to 5mg/kg (2.25mg/lb) every six hours for 24 hours (Jensen, 2006).

Many experienced breeders feel that these amounts are all too low, particularly if given SQ. If at all possible, the first dose of thiamine should be administered IV, but since this is beyond the reach of many breeders, increasing both the amount of thiamine and frequency of dosing may be enough to compensate. Note that the *Merck Veterinary Manual*recommends for cattle:

Therapy must be started early in the disease course for benefits to be achieved. If brain lesions are particularly severe or treatment is delayed, full clinical recovery may not be possible. The dosage of thiamine is 10-20, mg/kg, IM or SC, tid. Initial treatment may be administered IV.

If we double Dr. Evans' dose recommendation to 20mg/kg (9mg/lb) to match the higher end of the Merck recommendation, and give the thiamine twice as often (every four hours for 24), the dosages for a 100 pound alpaca are:

➢ 4.5 ml of 200mg concentration thiamine or

➢ 1.8 ml of 500mg concentration thiamine to deliver 900mg of thiamine

Contrast this with the amount of B complex that would be required, using Agri-Labs products from Valley Vet for an example. The same 100 pound alpaca would need:

➢ 72 ml of B complex or

➢ 9 ml of fortified B complex to deliver 900mg of thiamine

Fortified B complex is seldom sold at farm stores and generally must be ordered. Clearly, given the volume of B complex required to administer the necessary thiamine dose, it makes sense to obtain a bottle of pure thiamine from your vet before an emergency arises.

Oftentimes, if an animal just seems slightly "off," one or two SQ injections of thiamine over the course of a day will be enough to bring the animal back into balance. Some animals seem more prone to thiamine depletion due to stress, and an injection of thiamine prior to or just following a stressful event such as shearing may prevent greater problems later.

In summary, alpacas are extremely sensitive to changes in thiamine availability and can deplete their body's resources rapidly. The potential causes of PEM are almost infinite, and any time an alpaca exhibits neurological symptoms, the possibility of PEM should be considered. Immediate administration of thiamine is easy, inexpensive and appropriate any time an alpaca seems "off," and while a more definitive diagnosis is sought.

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