Swans Veterinary Services

ASHEEP - PEM

Polioencephalomalacia (PEM) as a cause of neurological syndrome and sudden death in sheep and cattle.

Polioencephalomalacia (or PEM) is one of the common causes of neurological symptoms and sudden death in ruminant production systems. It is a disease that results in necrosis (or death) of cortical brain tissue and has a range of underlying causes. These include thiamine (vitamin B1) deficiency, lead poisoning and sulfur toxicosis. Thiamine deficiency is responsible for the majority of cases in sheep and cattle and will therefore be the focus of this article.

Rather than a primary nutritional deficiency of thiamine (vitamin B1), destruction by thiaminase producing bacteria in the rumen is the most common cause of PEM in Australian sheep and cattle. The increase of thiaminase producing bacteria is caused by changes in the rumen flora, most commonly due to high starch and low fibre diets (for instance, in feed lots where animals are being fed high concentrate feeds with inadequate roughage). Mass fatalities due to PEM appear to be most prevalent in feed lot animals; however it may also present sporadically in individual or multiple thrifty animals grazing lush pastures. We have observed twice this winter in the Esperance region where weaned lambs have been found dead on lush pastures. Significant Disease Investigations (SDI's), into both cases confirmed lesions in the brains of these lambs consistent with PEM. Less commonly, the ingestion of poisonous plants such as Bracken fern and sulfur containing feeds such as distillers grain have also been linked to cases.

In the early stages of the disease, affected animals will appear depressed, blind, separate from the mob, wonder aimlessly and stand in corners/press their heads against dead ends. As the disease progresses, they will then become recumbent and adopt an opisthotonic posture (this is the signature "star gazing" posture, whereby the animal will become rigid on its side and extend is neck towards the stars). Animals may also paddle their limbs from seizures and inevitably die without intervention. Groups of animals may simply be found dead if the early stages of the disease go unobserved.

Treatment of affected animals involves the aggressive supplementation of vitamin B1. In severe cases where the animal is already recumbent and stargazing, vitamin B1 is best administered intravenously, however, the prognosis for survival is poor. In moderately affected animals which are still standing, farmers can treat with twice to three times daily 10-15mg/kg injections of vitamin B1 into the muscle for at least 3 days or until symptoms resolve. Our treatment protocol for sheep is a 10ml injection of 125mg/ml vitamin B1 into the muscle followed by three times daily 4ml injections until symptoms resolve.

Careful dietary management is an important preventative strategy against PEM. Feedlots should slowly introduce high concentrate diets to new animals and ensure free access to good quality roughage. Thiamine can also be supplemented in concentrate rations and on a per-animal basis with the injectable preparation. Restricting access to thiaminase implicated plants (Bracken fern), sulfur containing substances (alkaline drinking water, brewers grain etc.) and lead (old car batteries, paint etc.) will also limit the risk of PEM.

Any cases of sudden death or neurological syndrome in multiple animals is reason to call a veterinarian, whereby the Department of Primary Industries and Regional Development's fees will be subsidized to the producer on a case by case basis.

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