Polioencephalomalacia in goats

Polioencephalomalacia, or cerebro-cortical necrosis, is the most common nervous disease affecting goats in New Zealand. Although it is usually only recognsied as a cause of sporadic loss, subclinical disease may be significant and continued losses from affected mobs can be serious.

The lesion of laminar cortical necrosis is usually associated with induced thiamine deficiency in goats, although it can be produced by other agents in this and other species. Clinically it presents as a progressive nervous disease, characterised in the early stages by stargazing and twitching of the facial muscles, followed by blindness, recumbency, clonic convulsions, and death.

Young animals within the first year of life are most often affected, although all ages are susceptible. The morbidity is usually low within a group, but cumulative losses can be high over a period of time. In such groups it is common to find many animals with marginal thiamine levels. This finding, together with the observation that suboptimal thiamine levels in sheep are associated with production loss,[1] suggests that subclinical thiamine deficiency may be an unrecognised cause of poor growth rates in growing kids.

Indigestion appears to be an important predisposing factor which may be associated with meal feeding, especially if this is poorly controlled, as well as sudden changes in diet. In many cases, however, it is difficult to determine what it is that has precipitated an outbreak.

The most obvious lesions in the brain are those in the dorsal and dorso-lateral grey matter of the cerebral cortex. These are often discernible grossly as symmetrical yellow/brown areas of discolouration. In many cases, however, there are lesions in the paraventricular nuclei of the brain stem, the anterior and posterior colliculi, the caudate nuclei, and in the thalamus. These latter may be visible grossly as small haemorrhagic areas and are not unlike the lesions of thiamine deficiency in carnivores. It is probable that they precede the lesions of the cerebral cortex, which result from brain swelling and tentorial herniation.[2]

Polioencephalomalacia has been produced experimentally in lambs fed thiamine-free diets, but the natural disease is thought to be related to the production of thiaminase I in the rumen of affected animals. This enzyme is produced by a number of organisms, a common one being *Bacillus thiaminolyticus,* which has been shown to establish in lambs at a very young age. Why this or other thiaminase-producing organisms proliferate in some animals is not known for certain. Possibly indigetsion, with shifts in the pH of the ruminal liquor, may alter the balance of the ruminal flora, giving these species an advantage over others.

It has also been suggested that the normal production of thiamine by the ruminal flora may act itself to limit the proliferation of thiaminase-producing organisms by repressing the enzyme thiaminase I.[3] It is not known whether transmission of thiaminase-produing organisms takes place between animals grazing at pasture, or whether grazing with affected animals increases the risk of disease.

Diagnosis of the disease in the live animal is often made by observing the response to parenteral thiamine. If given early enough, many affected animals make a complete recovery. Such evidence can only be considered supportive because clinical recovery has been reported in ruminants suffering from lead poisoning that have been treated with thiamine.[4]

Blood thiamine analysis can be obtained through any of the MAFQual Animal Health Laboratories. Because of the progressive nature of the disease it is of most use in providing retrospective confirmation in cases that recover or those that cannot be necropsied.

Necropsy examination of affected animals often reveals a yellow discolouration of the cerebral cortex, with sometimes a clearly defined margin separating it from the normal tissue. A useful aid to diagnosis is the yellow-green fluorescence that is produced by affected areas of the brain when viewed under ultraviolet light, e.g. a Wood's lamp.

Histopathological examination of whole fixed brain is necessary to confirm the disease and to rule out the differential diagnoses, which include listeriosis, enterotoxaemia, toxoplasmosis, caprine arthritis encephalitis virus infection, copper deficiency, lead poisoning, and scrapie.

Parenteral thiamine given at the rate of 6–10 mg/kg intravenously has been recommended for the treatment of clinical cases.[5] This treatment attends to the immediate cause of the disease, but does not treat the underlying problem.

More recently oral treatment of goats with a high dose of thiamine (1 g) has been used with good effect in clinical cases,[6] and as a prophylactic measure.[6,7] High doses of thiamine repress thiaminase activity in the rumen.

Thiamine is available from the Whangarei Animal Health Laboratory. The price to veterinarians is $28.00 (incl. GST) for 200 g, plus $5.00 packaging and postage.

References

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