RYEGRASS STAGGERS IN DEER

Marjorie B. Orr, Invermay Animal Health Laboratory, Private Bag, Mosgiel.

Colin G. Mackintosh, Invermay Agricultural Research Centre, Private Bag, Mosgiel.

Abstract

Ryegrass staggers (RGS) in deer can cause major losses through stock deaths.

When the season and climate favour RGS, farmers and practitioners should be on the lookout for it, and at the first sign they should take prompt action to minimise losses.

RGS is common in sheep and cattle in New Zealand, but little has been published on its incidence in deer.

RGS is caused by neurotoxins produced by an endophyte in ryegrass.

The clinical signs of RGS are head tremor, fasciculation of the muscle along the back, convulsions and sometimes death.

Wapiti deer may be more susceptible than sheep, cattle or red deer.

Diagnosis is based on histological examination of the cerebellum.

Control can be achieved by preventing the close grazing of endophyte infected pasture by deer.

Introduction

Ryegrass staggers (RGS) occurs in the drier east coast areas of the North and South Islands, especially in summer and autumn. It is common in cattle and sheep (6), but it's incidence in deer is largely unknown.

RGS is caused by the neurotoxins (lolitrems A and B) produced by an endophyte which grows in perennial ryegrass (*Lolium perenne* L). These neurotoxins are concentrated in the seed heads and leaf sheath of the grass, with lesser amounts in the leaf blades (3).

Some ryegrass cultivars, for example Ellet, have a large proportion of infected plants; some, e.g. Nui species have variable levels, while others, notably Ruanui, have low levels (12).

The endophyte protects the grass against attack by the Argentinian stem weevil (8). Consequently in areas where the weevil occurs high endophyte ryegrass cultivars produce more pasture than the endophyte-free cultivars. The farmer must balance the advantages of increased grass production against the greater risk of RGS in stock.

An outbreak in wapiti deer at Invermay

A recent outbreak in imported Canadian wapiti at Invermay provided the first published report of RGS in deer (5). The outbreak occurred in February 1983 in a group of 23 wapiti deer. A stag and a hind became slightly ataxic, and the whole group was brought in for examination. During this procedure the stag went down twice and thrashed in lateral recumbency for 10-15 seconds before regaining its feet. It had a continuous head and body tremor.

The affected stag was left in the yards with hay and water while the rest of the group were moved into a second paddock. The following day the stag's velvet was removed under local anaesthetic to prevent damage but the stag died a few hours later.

About four days later, the group of deer was yarded again. The original affected hind was still ataxic. It had a head tremor, twitching eyelids and ears and continuous muscle fasciculations along it's back. Three or four other animals seemed more apprehensive than normal and appeared to have a slight tremor. The group was moved into a third paddock where there was little ryegrass, and lucerne hay and concentrates were fed.

Five days later the group was yarded again and all seemed normal.

Pasture samples from the three paddocks were sent to Lincoln College, for estimates of the proportion of endophyte infected plants, and to Ruakura for extraction of the neurotoxins, lolitrem A and lolitrem B, and for mouse bioassays of lolitrem. Pasture samples from all 3 paddocks showed 90% infection of ryegrass with endophytes. Pasture from the first paddock caused protracted tremors in mice, pasture from the second paddock caused a moderate tremor and pasture from the third paddock produced only a short duration marginal tremor.

We suspect that high levels of neurotoxins in the first paddock caused RGS in several deer, and in the third paddock the relatively small proportion of toxic ryegrass plants plus supplementation with hay and concentrates allowed recovery.

There have been two similar outbreaks of RGS in wapiti deer on Invermay since the original outbreak in 1983/84, with a total of 4 deaths and 10 to 15 clinical cases in a mob of 30 deer. Only one red deer has ever shown signs of RGS at Invermay, and that was a mild case. Cattle on Invermay have never shown signs of RGS, and cases in sheep have been uncommon in recent years. We believe that wapiti deer may be more susceptible to RGS than red deer or cattle. Anecdotal evidence from practitioners in Wanaka and Timaru supports this observation.

The main cost of the outbreaks resulted from animal losses. Four wapiti deer worth $10000 to $15000 each died in 2 years. Supplementary feed had to be provided and there may well have been additional hidden losses through impairment of growth of young wapiti and reduced fertility of stags.

Lesions

The usual cause of death of sheep and cattle with RGS is misadventure. Apart from the lesions relating to this, they show no characteristic gross pathological changes. In the deer we have necropsied, there has occasionally been a haemorrhagic enteropathy, possibly stress induced (5).

There are usually characteristic histopathological changes in RGS (7, 10). These changes are restricted to the cerebellum. In cases which have persisted for over three weeks, there is Purkinje cell degeneration. There are scattered round and elongated homogeneous eosinophilic bodies up to 30 µm in diameter in the granular layer of the cerebellum near the Purkinje cells. These are thought to be beaded swellings of Purkinje cell axons and they are characteristic of RGS, but not pathognomonic. Purkinje cells may also show chromatolysis or pyknosis - possibly the result of repeated anoxia during seizures.

Muscle necrosis has also been reported in cases of RGS, but this is probably a secondary lesion.

Differential diagnosis

There are several conditions in deer with nervous signs similar to those of RGS.

Malignant catarrhal fever: Tremor and hyperexcitably before death may be clinical features of MCF, but usually any nervous signs are accompanied by pyrexia. Histopathological examination of the brain will aid diagnosis.

Enzootic ataxia: In farmed deer, EA is characterised by apparent hind limb weakness in young adult or mature animals. Histological demonstration of spinal cord demylination and the demonstration of low liver copper levels enable a diagnosis of EA to be made.

The epidemiology of other diseases which theoretically could cause nervous signs in deer is virtually unknown. Such diseases include annual ryegrass staggers, paspalum staggers, polioencephalomalacia, parapituitary abscess, focal symmetrical encephalomalacia, hypomagnesaemia and meningitis.

In making a differential diagnosis of any disease of deer in which there are nervous signs the practitioner should consider all of these.

When submitting material to the Animal Health Laboratory for diagnosis, the practitioner will find that he gets as good as he gives! He should provide a full and relevant history, and if he suspects RGS this should include descriptions of the clinical signs and of the pasture and recent weather conditions. His description of post-mortem changes should include any gross abnormalities in the brain or spinal cord.

Fixed brain should be submitted for examination under ultraviolet light (polioencephalomalacia) and fixed brain and spinal cord should be submitted for histological examination. It may not always possible to remove the brain and spinal cord intact, but portions of cerebrum, cerebellum and spinal cord should always be included. Fixed small and large intestine, mesenteric lymph node, liver, kidney, lung and heart are always useful for histological examination.

Whole and clotted blood samples from live affected animals can be useful for serum Cu, Ca, Mg, ketones, SGOT, CPK assays etc.

Fresh liver could be provided for Cu assay. Small intestine contents could be submitted for counter-immuno-electrophoresis (focal symmetrical encephalamolacia), fresh brain should be submitted if there is any sign of infection, e.g. abscess, meningitis.

Treatment

Epsom salts may help through a laxative effect in 'flushing out' toxins from the intestine. Lime, massive doses of vitamin B1 and seaweed extracts are said by some to protect against RGS. None of these treatments has been subjected to scientific tests.

Control

As the risk of RGS is greatest when animals are grazing short ryegrass dominant pastures in summer and autumn, pasture longer than 3 cm should be provided. Set stocking should be avoided if it forces animals to eat close to the ground - the leaf sheath contains high levels of fungus. The seed head may also contain neurotoxins so pastures should not be allowed to run to seed. Daily shifts if possible may help to prevent close grazing of pasture (9).

Endophyte-free ryegrass seed or other species of grass could be sown and the white clover component of pasture could be encouraged (9).

At the first sign of trouble, stock should be moved quietly to paddocks sown with low endophyte ryegrass, onto a crop or onto pastures where there is little ryegrass or where supplementary feed can be provided.

Affected animals should be moved in the early morning when signs are generally milder than later in the day.

Control measures may be impractical in dry summer conditions when alternative feed is not available. It may be worth spraying pasture with systemic fungicide but the quantity required makes this an expensive procedure (4).

The 'take home' message is this. When the season and climate favour RGS, be on the lookout for it. At the first sign of trouble take measures to minimise the impact on the herd.

References

1. Boland, C.J. (1982). Surveillance **9**: 2.

2. Fletcher, L.J. and Barrell, G.K. (1984). N.Z. vet. J. **32**: 139-40.

3. Harvey, I.C. (1983). Proc. of N.Z. Grassland Assoc. **44**: 234-6.

4. Latch, G.C.M. (1983). Proc. of N.Z. Grassland Assoc. **44**: 251-3.

5. Mackintosh, C.G., Orr, M.B., Gallagher, R.T., Harvey, I.C. (1982). N.Z. vet. J. **30**: 106-7.

6. Manktelow, B.W. (1984). The Veterinary Handbook. Foundation for Cont. Educ. N.Z. V.A., Palmerston North.

7. Mason, R.W. (1968). Aust. vet. J. **44**: 428.

8. Mortimer, P.H. and di Menna, M.E. (1983). Proc. of N.Z. Grassland Assoc. **44**: 240-3.

9. Mortimer, P.H., Fletcher, L.R., di Menna, M.E., Harvey, I.C., Smith, G.S., Barker, G.M., Gallagher, R.T. and White, E.P. (1982). Proc. Ruakura Farmers Conf. **34**: 71-4.

10. Munday, B.L. and Mason, R.W. (1967). Aust. vet. J. **43**: 598-9.

11. Peterson, A.J., Bass, J.J. and Byford, M.J. (1984). N.Z. vet. J. **32**: 36-7.

12. Scott, D.J. (1983). Proc. of N.Z. Grassland Assoc. **44**: 264-6.