ENZOOTIC ATAXIA IN WAPITI

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CASE HISTORY:

In late December (1984) a 2-year-old wapiti-type stag, running with a group of 54 other stags of the same age, was noticed to have slight hind limb weakness and unsteadiness when running. It had been knocked over in the yards and was thought to have suffered a mild back or hip injury. Its condition gradually worsened and only when 5 other stags in the group gradually developed a similar gait was enzootic ataxia suspected. On January 22 the original stag, which was by now quite severely affected, was euthanased and a post mortem examination carried out. It was in light condition with no body fat reserves and no gross abnormalities. There was no evidence of *Elaphostrongylus cervi* in the C.N.S. and no *E. cervi* larvae were detected in a faecal sample. The serum Cu level was 2 umol/l and the liver Cu level was 20 umol/kg. Histopathological sections of mid-brain, cerebellum and spinal cord were examined and lesions indicative of enzootic ataxia were found in the spinal cord. In the cervical spinal cord there was massive demyelination in the ventral columns. In the thoracic spinal cord the changes were less severe.

A tentative diagnosis of enzootic ataxia was made immediately after the gross post mortem examination and the following day the rest of the group, comprising 20 red, 20 hybrid and 15 wapiti-type deer, were yarded. Five deer, all of which were wapiti-type, showed clinical signs of enzootic ataxia to varying degrees ranging from obvious unsteadiness (one animal) to a very mild loss of balance in the hind limbs when trotting over rough ground. When walking or galloping the gait of the mildly affected stags were apparently normal. Blood samples were taken from the 5 affected and the 1- unaffected wapiti and at random from 5 red and 5 hybrid stags. The serum copper levels were as follows:

Breed	Mean serum Cu (umol/l)	Range
Red (n = 5)	4.6	(3.8-5.8)
Hybrid (n = 5)	1.6	(1.5-2.5)
Wapiti		
unaffected (n = 10)	2.5	(1.0-5.5)
affected (n = 5)	1.5	(0.5-2.3)

The following day (January 24) all the stags in the group, except for 6 of 12 stags due to be slaughtered for meat studies 2 weeks later, were injected with "Coprin" (Glaxo): 1 ml (= 50 mg Cu) to red, 1.5 ml (= 75 mg Cu) to hybrid and 2 ml (= 100 mg Cu) to wapiti deer by subcutaneous injection in the neck. No further cases of enzootic ataxia developed.

At slaughter on February 8 the following results were obtained from the 12 stags (6 untreated and 6 treated):

Untreated Stags	Serum Cu (umol/l)	Liver Cu (umol/kg)
Red G216	2.7	62
Red G250	4.7	38
Hybrid 0214	3.5	36
Hybrid 0247	(not done)	31
Wapiti B218	2.7	50
Wapiti B222	3.1	20
Treated Stags		
Red G207	6.7	692
Red G244	11.4	268
Hybrid 0224	10.8	571
Hybrid 0243	9.4	623
Wapiti B221	16.9	318
Wapiti B224*	10.2	353

*The Wapiti B224 was slightly ataxic at the time of slaughter and histopathological examination of the spinal cord showed occasional distended pale axons in the grey matter and slight loss of myelin in the ventral columns. Histological examination of the spinal cords of 4 stags (0214, 0243, B221, B222) showed no definite abnormalities.

Unfortunately samples were not available from the other animals in the slaughter trial.

Pasture samples were coincidently being taken from the deer farm from May 1984 to March 1985 and spanned the period of the outbreak. They were not the specific paddocks grazed by this group of stags but are probably fairly representative of the pastures on the deer farm during this period. The analyses are as follows:

	Paddock No.	Major elements % DM S	Trace elements ppm. DM Mn	Zn	Cu	Mo
May 84	25	.33	290	26	7	.28
	26	.29	122	19	6	.16
July 84	24	.29	211	28	8	.25
	40	.29	304	18	6	.42
Nov 84	27	.45	86	28	5	.19
	28	.54	253	31	5	.34
	38	.43	254	23	4	.05
	39	.47	403	20	5	.08
Mar 85	30	.41	251	32	6	.12
Adequate/normal range (Grace, 1983)		(0.18-0.5)	(50-500)	(25-50)	(6-10)	(0.2-1.0)

DISCUSSION

Enzootic ataxia in red deer has been diagnosed on a number of occasions both in N.Z. (Wilson *et al.*, 1979; Familton *et al.*, 1985; Clark and Hepburn, 1986) and overseas (Barlow *et al.*, 1964; Terlecki *et al.*, 1964; McTaggart *et al.*, 1981).

We have presented this case-study in order to provide more information about the possible predisposing causes and breed effects as well as providing results of copper analyses of serum, liver and pasture samples.

Although the group of 55 stags comprised approximately equal numbers of red, hybrid and wapiti only the last group was clinically affected. This suggests that wapiti have a higher dietary requirement for Cu and are probably more susceptible to enzootic ataxia.

Another possible factor is that as the 3 breeds were grazing the same short pasture the larger wapiti may not have been able to compete as well against the smaller red deer. It was notable that the worst affected stag was in poor condition. Reid *et al* (1980) found that liver Cu levels in mature feral wapiti in Fiordland were lower than those of feral red deer in the same area (327 and 613 umol/kg respectively).

The pastures on which the group had grazed were similar to those from which pasture samples had been taken. Prior to the outbreak pasture Cu levels of 6-8 ppm were recorded in May and July while the spring levels were 4-5. Mo levels were low, Mn and Zn were in the normal range and S was in the high end of the "normal" range. Grace (1983) suggests that, provided the availability of Cu is not greatly influenced by dietary factors such as Mo and S then pasture Cu levels of 5-6 and 7-10 ppm DM are adequate for sheep and cattle respectively. The sheep grazed on similar pasture at Invermay do not appear to suffer from Cu deficiency. Thus it appears likely that under these conditions the deer's Cu requirements are probably higher than those of sheep and that the pasture on the Invermay deer farm is probably marginally deficient and there may be some interaction with moderately high S levels. This was probably compounded by the effect of the drought on pasture quality in the spring and summer of 1984-85. The first effects were felt in late spring when the grass growth slowed and the quality deteriorated. At this time the animals were not growing as well as expected and were having to eat fairly rank poor quality feed. Under these conditions the deer appear to have been unable to replete their Cu reserves following their normal winter/spring depletion (Mackintosh *et al*, 1986).

In previous years all the deer have been drenched for lungworm using a benzimidazole anthelmintic to which Cu and Se were added. Weaners are drenched regularly throughout the autumn and then every 2 or 3 months in the following winter/spring. Yearlings and adults are drenched twice yearly in spring and autumn. In 1983 Invermay changed to Ivomec to which the manufacturers advised against adding Cu. This group of 2-year-old stags were the first group for some years not to get Cu regularly as weaners and yearlings. This could also have contributed to their deficiency.

The treatment of the 6 slaughtered stags 2 weeks before with injectible Cu appears to have resulted in a rapid repletion of the liver Cu reserves; their mean liver Cu level was 470 umol/kg compared to 40 umol/kg in the untreated stags. No further cases of enzootic ataxia developed and the condition of the 3 remaining mild cases did not deteriorate further although a residual mild ataxia remains.

SUMMARY

Wapiti deer appear to be more prone to Cu deficiency and enzootic ataxia than red deer when grazed on pasture with marginal Cu levels. The outbreak

of enzootic ataxia at Invermay appears to have been precipitated by the very dry late spring/early summer with poor pasture growth, poor quality and feed shortage. The administration of injectible Cu halted the outbreak and restored adequate liver Cu reserves.

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