Suspected case of bovine spongiform encephalopathy

Although bovine spongiform encephalopathy (BSE) has been reported only from the United Kingdom,[1] and possibly the United States,[2] MAF's Animal Health Laboratories maintain surveillance for this disease in New Zealand.[1, 3, 4] In the case reported here a 4-year-old Friesian cow with nervous signs, which included aggression and an inability to walk properly, prompted a suspicion of BSE even though the onset of signs was apparently acute.

Clinical biochemistry examinations produced findings suggestive of end-stage liver disease. Notable were the increased liver enzymes, high globulins and low albumin (table 1).

TABLE 1: Serum biochemistry results on cow with nervous disease.

Analyte	*Units*	*Result*	*Reference Range*
PK	U/L 30	312	0 - 370
AST	U/L 30	99	25 - 120
AP	U/L 30	237	0 - 100
GDH	U/L 30	50	0 - 45
GGT	U/L 30	91	0 - 32
BIL	µMOL/L	38	0 - 13
TP	G/L	92	0 - 86
ALB	G/L	22	25 - 40
AG	ratio	0.31	0.5 - 1.2
CRE	µMOL/L	100	55 - 130
URE	MMOL/L	4.7	2.7 - 12.3
PO_4	MMOL/L	1.7	1.1 - 2.8
CA	MMOL/L	1.8	2.0 - 2.6
MG	MMOL/L	0.89	0.49 - 1.15
BOH	MMOL/L	0.2	0.2 - 1.0

Even though it was considered likely that this was a case of hepatic encephalopathy, a co-operative investigation was carried out by the veterinary practitioner and the Ruakura Animal Health Laboratory to rule out the possibility that this was a case of BSE (which we consider to be "bovine scrapie" in terms of notifiable diseases).

On-farm enquiry by the practitioner revealed that the cow was a pedigree animal, sired from imported British semen. In addition, it had recently been receiving a concentrate supplement to its diet.

The cow continued to be aggressive and declined into recumbency and apparent blindness over the next day. She was euthanased, and fresh and fixed liver and brain taken for laboratory examination.

Histology confirmed that there was severe chronic liver disease consistent with pyrrolizidine alkaloid toxicosis, as well as some acute hepatocellular degeneration and necrosis. Brain lesions were consistent with a hepatic encephalopathy, namely widespread spongy-vacuolation primarily affecting the white matter. There was also evidence of an acute polioencephalomalacia.

Table 2 compares and contrasts some aspects of this case and BSE, as reported in the literature.[1, 3, 5]

Because of the differences listed in table 2, and the critical examination of the brain, we believed we could confidently make a diagnosis of a hepatic encephalopathy, rather than BSE.

Because of some similarities in the presenting signs of both these diseases, it is important that cases are examined thoroughly. It is likely that cases similar to this will appear regularly because chronic liver damage due to pyrrolizidine alkaloids and sporidesmin is quite common in New Zealand.[6]

Table 2: Signs of nervous disease compared.

	This Case	*BSE*
Age	4 years	>3 years
onset	sudden	insidious
clinical course	a few days	progressive over 1-6 months
Nervous signs :		
altered behaviour	+	+
gout abnormalities	+	+
exaggerated reactions	-	+
recumbency	+	+
blindness	+	-
Clinical pathology :		
liver enzymes	elevated	normal
bilirubin	elevated	not given
glucose	not tested	elevated
globulins	high	normal
albumin	low	normal
Histopathology :		
liver	severe hepatopathy	No reported changes
brain intraneuronal vacuoles	absent	present
neuronal degeneration and necrosis	present in the cortex absent in brain stem nuclei	absent in the cortex present in brain stem nuclei
gliosis	absent	mild in some
spongy vacuolation of white matter	marked	some vacuolation of neuropil

References

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