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Brief Communication

Enzootic Ataxia in a Norwegian Red Deer Herd

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In the 1960s, a disease characterized clinically by ataxia and posterior paresis that had for a long time been recognised in park herds of red deer *(Cervus elaphus)* in Britain, and in park and wild red deer in Germany, was shown to be associated with demyelination in the spinal cord (*Barlow & Butler* 1964, *Terlecki & Done* 1964, *Knösel & Schulz* 1968). Some of the cases also showed nerve cell degeneration and necrosis in the brain tissue. Later, enzootic ataxia was reported in deer farms in New Zealand (*Wilson et al.* 1979), Australia (*Peet & Hepworth* 1993), China (*Yoshikawa et al.* 1996) and Germany (*Geisel et al.* 1997). The disease occurs in young adult deer. The aetiology is considered multifactorial, but copper (Cu) deficiency appears a central factor, and the disease can be efficiently prevented through Cu supplementation (*Wilson et al.* 1979). In New Zealand, Cu deficiency has also been suggested as a cause of osteochondrosis in farmed red deer calves (*Thompson et al.* 1994, *Audigè et al.* 1995).

This paper reports the occurrence of enzootic ataxia in a small red deer herd located on a dairy farm in the municipality of Ølen, Hordaland county, Norway. The herd, comprising about 15 animals, was established in the late 1980s based on locally wild-captured animals. The animals grazed unfertilised, semi-cultivated pasture in a 10 hectare enclosure. From October to April they also had access to silage *ad libitum,* and, once a week, the animals were given a mineral mixture produced for cattle and goat (Felleskjøpet) mixed with concentrates. The mineral mixture contained 600 mg Cu per kilogram.

During winter 1996/97 a 1.5-year-old female developed signs of posterior locomotor disturbances. The condition remained stable for about 8 months after which the animal was destroyed without veterinary inspection. In November 1997 a 2.5-year-old female showed signs of hind limb weakness and unsteadiness while running. Signs progressed, and the animal was euthanized 1 month after onset of disease. Its head, cervical columna and samples of liver and blood were available for laboratory examination.

The skull was split in the sagittal plane and the cervical vertebral column was opened from the dorsal. No gross lesions could be found on the brain and spinal cord, and the organs were fixed in 10% buffered formalin. After fixation, tranverse tissue blocks of the cerebral hemispheres, pons, cerebellum and medulla oblongata and 2 tranverse tissue blocks per segment of the cervical spinal cord were embedded in paraffin, cut at 5 µm, and stained with haematoxylin and eosin (HE) for histopathological examination. Selected sections were stained with luxol fast blue. The concentration of Cu in the liver tissue

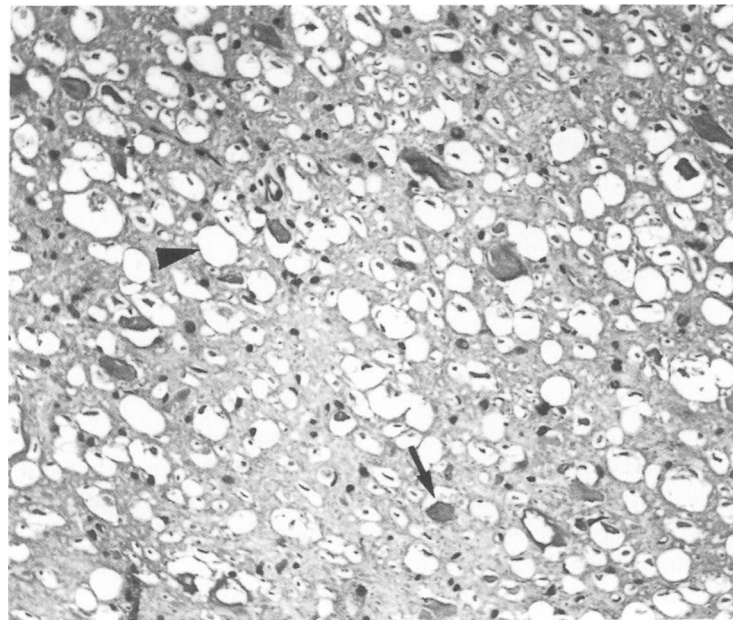

Figure 1. Cross section of the cervical spinal cord. Deficit of stainable myelin, and vacuoles with swollen (arrow) or disintegrated (arrowhead) axons and scattered macrophages. HE × 200.

and serum were measured using atomic absorption spectroscopy (*Jorhem & Engman* 1996).

Histopathological examination revealed bilaterally symmetrical demyelination and axon degeneration in the ventral and lateral columns of the cervical spinal cord (Fig. 1). Vacuoles or distended myelin sheats surrounding swollen or disintegrated axons and scattered macrophages frequently gave affected areas a spongy appearence. Similar lesions also occurred in white matter of the medulla oblongata, but no consistent histological findings could be detected elsewhere in the brain tissue. The concentrations of Cu in the liver tissue and blood serum were 47 μmol/kg wet matter basis (WMB) and 6.3 μmol/l respectively.

Our diagnosis of enzootic ataxia was made on the basis of typical clinical signs and histopathological spinal cord lesions (*Barlow & Butler* 1964, *Terlecki & Done* 1964, *Knösel & Schulz* 1968), combined with abnormally low Cu levels in the liver and serum.

The liver is the central storage organ for Cu in the body and therefore analysis of liver samples gives the best indication of the Cu status (*Grace* 1983). The level of Cu in serum is controlled by homeostatic mechanisms and can be normal also when liver Cu levels are low. Thus, the level of Cu in serum declines only after the Cu concentration in the liver has been depleted (*Mackintosh et al.* 1986). The Cu liver concentration found in our animal was below the concentration of 100 μmol/kg WMB regarded as deficient in farmed deer, and lower than 60 μmol/kg considered the «critical» concentration below which enzootic ataxia may occur (*Clark & Hepburn* 1986, Mackintosh et al. 1986). The serum Cu concentration found was

below 8 μmol/l, which is regarded as indicative of Cu deficiency following liver depletion (*Clark & Hepburn* 1986, *Mackintosh et al.* 1986).

Cu deficiency can occur as a result of ingesting pasture containing an inadequate level of Cu (simple deficiency), or as a result of high levels of molybdenum, sulphur, iron, and cadmium in the diet reducing the absorption or availability of Cu (induced Cu deficiency) (*Grace* 1983). A more complete evaluation of causes requires pasture and soil analyses that were not carried out in our study. However, the reported cases occurred during winter, when Cu liver concentrations in farmed deer have been found to decrease due to low Cu contents in pasture (*Mackintosh et al.* 1986). In addition, feeding on silage during winter may have contributed to Cu deficiency, as this is usually made from rapidly growing spring grass which tends to be very low in Cu (*Mackintosh* 1998).

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