BRIEF COMMUNICATION

Cerebellar Cortical Atrophy in a Charolais Calf

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Cerebellar cortical atrophy is characterized by selective degeneration of Purkinje cells of the cerebellar cortex. It originally was described in lambs as “daft” lamb disease [4, 7] and recently in Aberdeen Angus calves as “familial convulsions and ataxia” [1]. Clinical and pathological findings were similar in both daft lambs and familial convulsions and ataxia, but mode of inheritance differed. Daft lamb disease is inherited as a simple autosomal recessive, while familial convulsion and ataxia in Angus calves may be inherited as a dominant trait.

A 9-month-old purebred female Charolais calf that had been on pasture since birth had normal growth rate and behavior until found recumbent when 6 months old. She was alert but unable to get up for several hours and then recovered spontaneously. Later she had sudden convulsive seizures of various intensities. The seizures occurred at about 2- to 3-week intervals and lasted 1 to several minutes. Clinical signs diminished to a spastic residual ataxia. Between attacks the calf seemed nearly normal to ataxic, to have lack of proprioception at times and had a fine tremor. The attacks could be precipitated or exaggerated by excitement such as electric shock. The head and tail were in normal position, with the limbs slightly bent and rigid. When she fell, she struggled and had difficulty getting up. The clinical signs were solely neurological. Numerous tests on blood and cerebrospinal fluid gave no abnormality. The owner said there had been several other calves in the herd with similar signs. The dam of the affected calf had six other calves previously, but none had been affected.

After the clinical examination, the calf was killed with phenobarbital sodium and necropsied immediately. There were no gross lesions. Microscopically there was selective widespread cerebellar cortical degeneration of Purkinje cells almost exclusively. Degeneration of Purkinje cells was characterized by various stages of chromatolysis, by shrunken and hyperchromatic or pale cytoplasm, by swelling and vacuolation (fig. 1). Chromatolysis occurred around nuclei of most Purkinje cells, but in some it was between the nucleus and axon hillock (fig. 2). Many degenerated Purkinje cells had disappeared, leaving large empty baskets (fig. 3). There were swollen axonal torpedoes of Purkinje cells in the granular layer (fig. 4). A few vacuoles were in the molecular layer. Neither neuronophagia nor glial nodules were seen. No substance could be demonstrated in the vacuoles or in the

264
cytoplasm of the affected Purkinje cells by the stains used. The foliate and central white matter of the cerebellum were not affected.

Many diseases of cattle are manifested by convulsions and ataxia. Among them
are meningoencephalitis, polioencephalomalacia, hypomagnesemia, internal hydrocephalus, cerebellar hypoplasia and degeneration, and congenital hypomyelination. Most may be differentiated by clinical and clinicopathological examination, but some require histologic evaluation. A selective Purkinje-cell degeneration is pathognomonic of a cerebellar cortical atrophy. It has been described in lambs [4, 7], dogs [2, 6] and calves [1, 6, 8]. The condition seemed to be inherited and had almost identical pathological changes in all three species, but mode of inheritance differed. The disease’s mode of inheritance in dogs is undetermined and may be recessive in Holstein calves [8]. The condition called cortical cerebellar disease in an Ayshire calf [5] seemed to differ histologically from the cerebellar cortical atrophy we described; it was considered to be cerebellar hypoplasia.

The clinical and pathological features in our Charolais calf were similar to those of familial convulsions and ataxia in the Angus breed although the intensity varied [1]. The owner’s statement suggested genetic disease, but no clear evidence of this could be demonstrated.

The suggestion that degeneration of Purkinje cells probably results from, rather than causes, acute convulsive attacks [1, 3] does not explain why the changes are selective and affect Purkinje cells almost exclusively.

The characteristic pathological features led to our diagnosis of cerebellar cortical atrophy.

References
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