

Progressive Ataxia in Charolais Cattle

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Progressive ataxia in pure-bred Charolais cattle first was seen in England [1] and has since been recognized in Canada, New Zealand [3] and France [2]. Neurologic disturbances usually begin between 1 and 2 years old but have been seen as early as 6 months. There is progressive incoordination. Inability to rise develops over a period of months; urinating in an uneven flow is an early abnormality in females [3]. Vision, alertness, appetite and appendicular muscle tone and mass remain normal.

In sections stained with hematoxylin and eosin (HE), myelinated parts of the optic nerves, brain and spinal cord have ill-defined foci of fine filaments separated by a pale-pink ground substance (fig. 1, 2). The foci or plaques tend to be more numerous in specific anatomic areas [1]. Normal axons are within these foci of disordered myelin (fig. 3). Oligodendroglia adjacent to the plaques may be hypertrophied with a round vesicular nucleus, dense peripheral chromatin and granular cytoplasm, which gives them an epithelioid appearance (fig. 2). Ultrastructurally, the lesion of the myelin sheath is adjacent to nodes of Ranvier and consists of hypertrophied and hyperplastic oligodendrocytic projections from which arise many small processes. There also are incomplete myelin lamellae and paranodal myelin figures [1]. These changes reflect a defect of unknown cause in oligodendroglia.

Progressive ataxia has been seen in twin purebred Charolais heifers born in Oklahoma. Twin 1 at 6 months had posterior incoordination; by 8 months this had progressed to walking with a stilted gait and legs spread apart. At rest the rear legs were held forward. At 1 year old she could not rise on her hind legs without assistance. She was killed and necropsied. Peculiarities in urination were not recorded. No gross lesions were seen at necropsy except a torn round ligament and subluxation of the right femoral head.

At 8 months twin 2 had posterior incoordination. She died when she was 2 years old. During the last weeks she could rise only with assistance; bloat had to be relieved several times. Two weeks before she died she had a heifer calf which remains unaffected at the age of 8 months. A limited postmortem examination showed subdural hemorrhage of the ventral brain stem and anterior cervical spinal cord.

Microscopic changes of the central nervous systems were similar to those in previous reports and have been verified [2]. Moderate perivascular infiltrates of

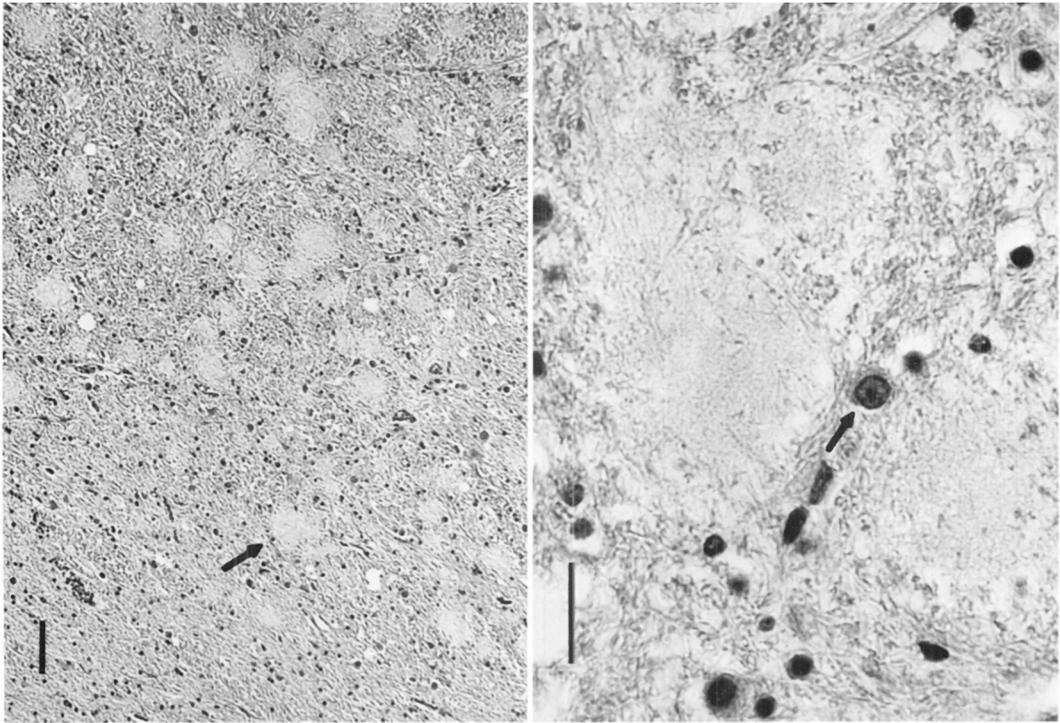


Fig. 1: Numerous pale foci or plaques (↑) in internal capsule of cerebrum of twin 2. Line = 200 μm . HE.

Fig. 2: Three adjacent plaques almost surround hypertrophied oligodendrocyte (↑). Line = 25 μm . HE.

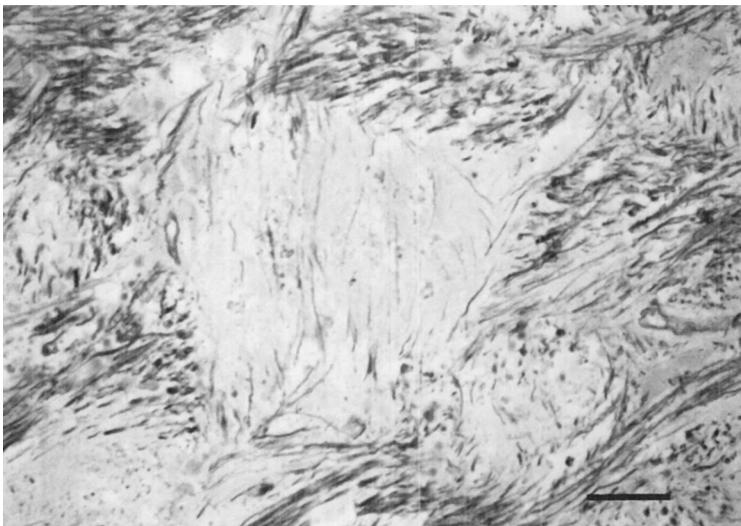


Fig. 3: Axons traverse pale focus of disordered myelin. Line = 50 μm . Luxol Fast Blue – Holmes' Silver Nitrate.

lymphocytes, monocytes and a few neutrophils were seen in a small region of the thalamus from twin 2.

The oligodendroglial defect has not been fully characterized. The occurrence of this unique entity in one breed suggests an hereditary basis but a pattern of inheritance has yet to be determined.

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References

- 1 BLAKEMORE, W.F.; PALMER, A.C.; BARLOW R.M.: Progressive ataxia of Charolais cattle associated with disordered myelin. *Acta Neuropathol (Berl)* **29**:127-139, 1974
- 2 PALMER, A.C.: Personal commun., 1976
- 3 PALMER, A.C.; BLAKEMORE, W.F.: Progressive ataxia of Charolais cattle. *Bov Pract* **10**:84-85, 1975
- 4 PALMER, A.C.; BLAKEMORE, W.F.; BARLOW, R.M.; FRASER, J.A.; OGDEN, A.L.: Progressive ataxia of Charolais cattle associated with a myelin disorder. *Vet Rec* **91**:592-594, 1972