Cerebellar Abiotrophy in a Charolais Calf

Timothy J. Jones, *Class of 1988*
College of Veterinary Medicine
Kansas State University
Manhattan, Kansas 66506

Cerebellar abiotrophy is characterized by selective degeneration of Purkinjie cells of the cerebellar cortex.[1] It has been described in lambs, [4,7] dogs, [4,6] calves,[6,7,8] and horses.[9] Late onset cerebellar degeneration or abiotrophy has been observed in Holstein calves. Signs are first noted at 3 to 8 months of age and are progressive, with eventual loss of the ability to stand unassisted despite normal strength.[1] There are no gross lesions at necropsy, but histological examination shows degeneration of Purkinjie cells and neurons of the cerebellar nuclei. Affected calves have been shown to have common ancestry in the pedigree within three generations. It is important to know the genetic history since the condition is believed to be inherited as a recessive characteristic.[2] Convulsive seizures in cerebellar abiotrophy can be precipitated or enhanced by excitement or electrical shock. The seizures are of various intensity. Between seizures the calf appears normal but when moved, shows minor ataxia. Blood tests are normal and the cereberospinal fluid show no abnormalities.

Case History

A 6-month old Charolais calf was referred to Kansas State University Veterinary Hospital on September 5, 1987 with a history of convulsive seizures. The owner's observation was that the seizures were precipitated by coming in contact with an electric fence. On August 10, 1987 the calf became temporarily entangled in an electric fence. After release the calf proceeded to run through two additional electric fences and one barbed wire fence. The following day and thereafter the calf experienced occasional daily attacks where he would become stiff, topple backwards and froth at the mouth. During these attacks or when excited, the calf became aggressive and would charge people. For ten days the calf was treated with penicillin to prevent infection due to wire injuries on the legs. The animal had a vaccination history of being immunized with clostridial antigens. He was confined in a corral with his dam and 4 to 5 cow/calf pairs. Daily feed included 10 pounds of grain and alfalfa hay free choice.

The owner reported that the calf was hard to handle while halter breaking and he would display sudden aggression which would be immediately followed by a calm temperament. The owner also noted that the calf disliked the headgate as well as any handling near his head. The calf's hard handling characterisitic may have been due to his sire's similar disposition. No other problems were observed in the herd except for one heifer which died at the same age.

The calf was brought to Kansas State University Veterinary Hospital on September 5, 1987 as an emergency involving possible kidney stones and post electrocution neurological problems. A complete history and physical exam were performed with the following results: 104.4° F temperature, respiratory rate of 56/minute, heart rate of 66/minute and weight of 650 pounds. There were multiple lacerations on the legs, tail, back, pelvic region, head and face. The mucous membranes were pale and calf was breathing shallow and rapidly. There was a mucopurulent nasal discharge and the lung sounds were slightly harsh. There were periodic attacks of toppling backwards and upon walking the calf became ataxic, nervous, aggressive and would put his head down as if to attack.

The working problem list consisted of a neurological disorder and respiratory illness. The diagnostic procedure included a CBC, chemistry profile, urinalysis and CSF analysis. The calf was then evaluated using a television camera monitor. Initial treatment was LA-200® and vitamin B complex. The respiratory problem responded well to the medication as the lung sounds returned to normal and the nasal discharge ceased. The CBC showed an elevated fibrinogen possibly due to the right foreleg trauma acquired at the hospital when the calf tried to attack. The urinalysis and the CSF results were normal. The blood clotting time was somewhat delayed as observed by excessive hemorrhage when intramuscular injections were given. On September 9, 1987 Tribrissen® was administered through an intravenous catheter as a broad spectrum antibacterial. Five minutes later when the catheter was flushed with heparinized saline, the calf experienced an attack and a loud bang was heard from his stall. Observation of the situation showed he was lying down, front legs rigidly extended and head straight back. He then returned to a standing position displaying tenseness with severe muscle twitching, excessive salivation and was totally unaware of the environment. The entire episode lasted approximately five minutes. Only a few of these episodes were noticed by hospital personnel. The calf was observed for neurological problems and a brain tumor was suspected.

Necropsy and Histological Examination

After the clinical examination, the calf was euthanized with T-61® and necropsied immediately thereafter. Gross

lesions showed lung abcesses and fibrous adhesions from pneumonia. The right foreleg had a massive infection and the right subscapular lymph node was enlarged due to trauma. Histological examination revealed selective degeneration of Purkinjie cells of the cerebellum as described in a previous case[1] (Figure 1).

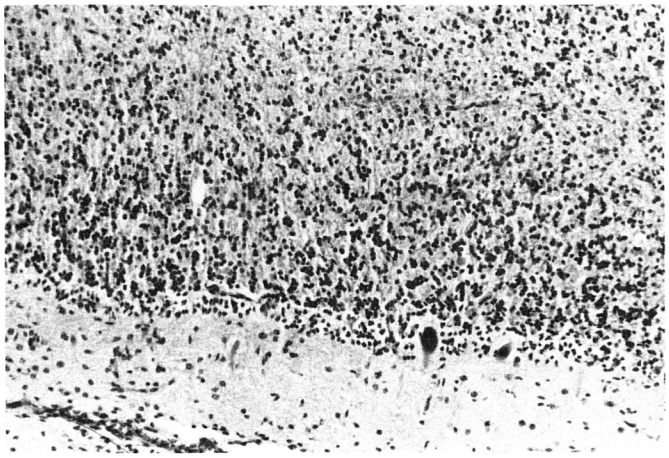

Figure 1.

Differential Diagnosis

Differential diagnosis on this case included electrocution, TEME (thromboembolic meningoencephalitis), polioencephalomalacia, streptococcal meningitis, enterotoxemia (type D), nervous coccididiosis, brain tumor, progressive ataxia of Charolais cattle, rabies, hepatic encephalopathy, hypomagnesemia, cerebellar hypoplasia, and congenital hypomyelinogenesis. Most may be differentiated by clinical and clinopathological examination, however some require histological examination.[1]

Summary

The Charolais calf presented was found to be experiencing convulsive seizures. These seizures continued to increase in severity therefore the calf was euthanized. The significant necropsy findings were: Histological examination of the brain demonstrated degeneration of the Purkinjie cells in the cerebellum. The brain stem and spinal cord revealed a moderate degree of Wallerian degeneration.

The calf's entanglement in the electric fence could have precipitated or exaggerated the convulsive seizures. This particular selective degeneration of the Purkinjie cells of the cerebellar cortex is pathognomonic for cerebellar abiotrophy due to the characteristic pathological features.

Acknowledgements

The author acknowledges the assistance of Dr. H.W. Leipold, Dr. K.B. Beeman, and Dr. B. Henning in preparation of this article.

References

1. Cho, D.Y.: Leipold, H.W.: Cerebellar cortical atrophy in a Charolais calf. Vet. Pathol. 15:264-266, 1978. 2. Howard, J.L.: Cerebellar Abiotrophy. Current Vet. Therapy 2 Food Animal Practice: 864, 1986. 3. Innes, J.R.M.; Rowlands, W.T.; Parry, H.B.: An inherited form of cortical cerebellar atrophy in ("draft") lambs in Great Britian. Vet. Rec. 61:225-228, 1949. 4. Cordy, D.R.; Sneebaker, A.H.: Cerebellar hypoplasia and degeneration in a family of Airedale dogs. J Neuropath Exp Neurol 11:324-2328, 1952. 5. Barlow, R.M.; Linklater, K.A.; Young, G.B.: Familial convulsions and ataxia in Angus calves. Vet Rec 83:60-65, 1968. 6. Jubb, K.V.F.; Kennedy, P.C.: Pathology of Domestic Animals. 2nd ed., vol 2: Academic Press, New York and London, 1970. 7. Van Vogaert, L.; Innes, J.R.M.: Cerebellar disorders in lambs: A study in animal neuropathology with some comments on ovine neuroanatomy. Arch Pathol 50:36-42, 1950. 8. White, M.E.: Whitlock, R.H.; DeLahunta, A.: A cerebellar abiotrophy of claves. Cornell Vet 6:476-491, 1975. 9. Beaty, M.T.; Leipold, H.W.; Cash, W.; DeBowes, R.: Cerebellar disease in Arabian horses. Prox. Am. Assoc. Equine Pract. 31:241-255, 1985.