Subacute Necrotizing Encephalopathy of Simmental Calves

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Introduction

Neurologic disorders in feedlot cattle are common. These "brainer" cases often occur sporadically and do not warrant diagnostic investigation. The occurrence of this recently recognized neurologic disorder warrants investigations to establish a definite diagnosis when purebred Simmental calves are affected and parentage data may be available.

In excess of 30 cases have been confirmed since this disorder was first reported in the USA in 1991.[3] Many more cases were reported by owners but no data was available to confirm the diagnosis. Due to its clinical similarity to many currently recognized conditions and lack of familiarity with the disease it likely goes undiagnosed in most feedlots. The disease was first described in Australia in 1989 and Australian cases were clinically and pathologically similar to those reported in North America.[2,3,4,5] The American cases have been reported in Oregon, Iowa, Kansas and North Dakota. As diagnostic criteria were well established by 1994, cases may have been diagnosed at other laboratories and not reported to the program.[5] Most recent cases were examined in the fall of 1997 indicating the problem persists in North American cattle populations.

Clinical Findings

This syndrome occurs in 5-12 month old Simmental calves. Symptoms often occur a few weeks but occasionally a month or more after entering the feedlot. Ataxia is the first symptom noted by the pen riders. The ataxia ranges in severity at onset, likely dependent on the astuteness of the observer. Toe dragging, knuckling and difficulty rising is first seen. Mild weakness and swaying of hind quarters is common. Symptoms could be exacerbated by turning calves in a tight circle. One owner noted that several affected calves could be captured after a short and vigorous run when they would collapse and remained recumbent for approximately 5 minutes. Calves then regained the ability to rise and attempt escape. This was not observed in all cases and could be an unusual expression of the condition. Affected calves became recumbent and would remain down and most often sternal (Fig. 1). Cranial nerve function appeared intact and calves appeared aware of surroundings. Difficulty rising was expressed as rising on the front limbs first and extreme difficulty was encountered as calves attempt to move the rear limbs beneath the body. The disease is slowly progressive and all calves eventually became recumbent and were destroyed. Calves have survived 1 month or longer with moderate clinical signs. Generally calves were sacrificed 3-6 weeks from onset. A few calves succumbed faster but perhaps early signs were missed. Mild fever, tachycardia and tachypnea were seen in calves but may have been related to struggle, anxiety and resisting restraint or concurrent disease.

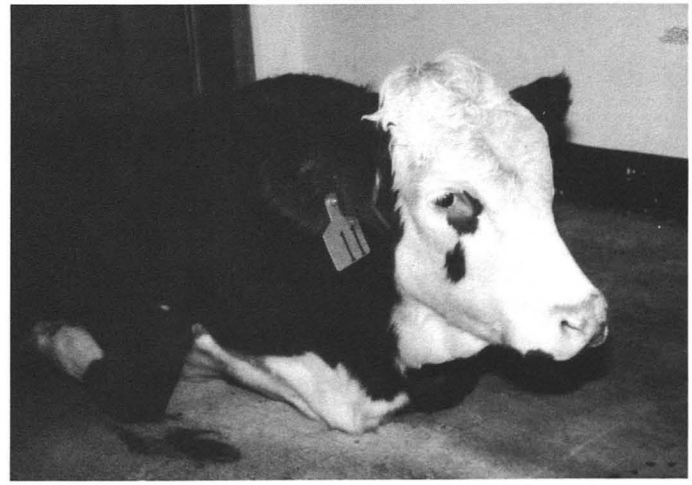

Figure 1. Clinically affected 10 month old Simmental calf. Calf is sternal and unable to rise.

Contrary to one published report reporting high aluminum and low copper levels in affected calves, trace mineral status appeared unrelated as accessed by ICP analysis on liver.[3,5] Elevated blood lactate has been reported in affected calves.[5] The elevated lactate suggested possible disruption of aerobic metabolism. Cytochrome C oxidase activity was assayed in brain and cardiac muscle of three calves and was normal.

The syndrome can be differentiated clinically from polio, *Hemophilus somnus*, nervous coccidiosis, and organophosphate toxicosis by the absence of cranial nerve involvement, blindness, opisthotonos, and spontaneous seizure activity. It may be more difficult to distinguish from spinal rabies, spinal injury, grub reactions, or a vertebral body abscess. The disease has only been described in Simmental cattle and their crosses. The breed background was a good indicator of when to include the syndrome in the differentials. The clinical signs initially were those of spinal origin. The nuclei involved coordinate fine motor control of cerebral-spinal impulses and lesions within them affected coordination.

Pathology

The characteristic lesion can be seen grossly. The lesions were quite small and most easily observed after formalin fixation. The lesions were 4-6 mm and occurred in a bilateral symmetrical pattern just cranial to the obex (Fig. 2). The lesions were grey, depressed foci and affected the olivary and other brainstem nuclei (Fig. 3). The lesions were just lateral to the 4th ventricle in the dorsal brainstem and progressing caudally lesions were present in the ventral lateral brainstem. If the appropriate level of brainstem was sectioned, 4 distinct foci were found. Lesions in the spinal cord and thalamus were seen but were far less common. The lesions near the obex were always present and are considered diagnostic for this disorder.

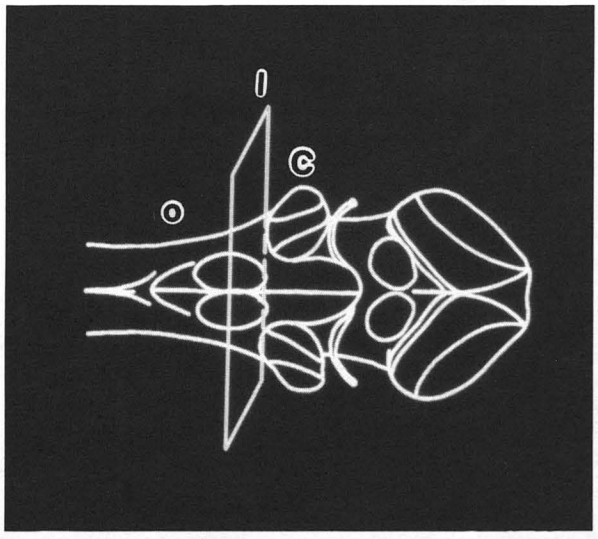

Figure 2. Locations of lesion near the obex. Serial sections 0.5 cm apart should reveal gross lesions in affected calves. c - cerebellar peduncle, l - level of the lesion, o - obex.

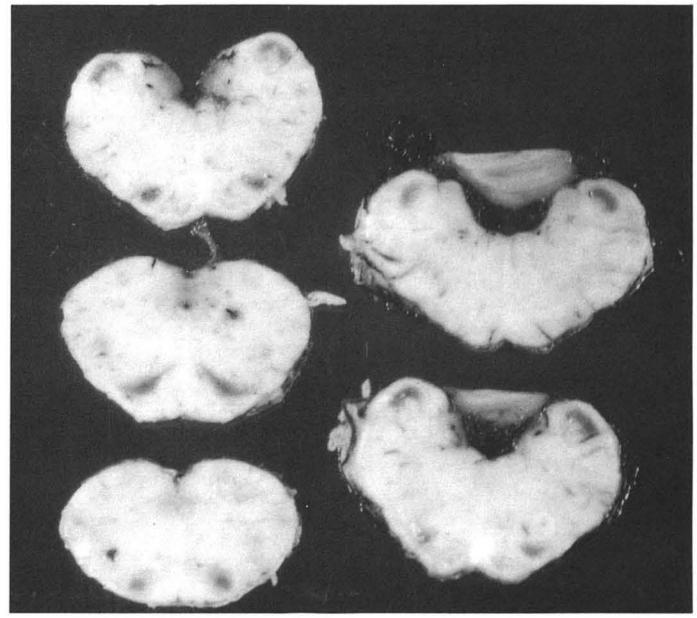

Figure 3. Gross lesions near the obex. Dorsal lesions are from anterior segments and ventral lesions from nearest the obex.

Histologic examination revealed liquefaction and rarefaction of neuropil. Neurons were present within the lesions and appeared intact with loss of extended cell processes. Vascular structures were present within the rarefied foci and appeared reactive. Endothelial cells were hypertrophied. Occasional spongiosis was present in adjacent white matter.

Conclusion

The cause of the syndrome remains a mystery. The single breed affected suggests a genetic etiology. The mechanism of inheritance has not been proven. Low incidence in affected populations would suggest a recessive, or perhaps maternal inheritance. Leighs' syndrome in children has similar clinical and pathological features. The syndrome in children is caused by a variety of metabolic defects. Cytochrome C oxidase deficiency, most commonly, and biotinidase deficiency have been described.[1] Several cases of Leighs' syndrome appear to be maternally inherited. Generally I have recommended culling dams of affected calves and consideration be given to culling sires. Herds which have experienced cases in the recent past were surveyed regarding management changes and recurrences. Culling has

generally been effective in reducing recurrences. More data need to be gathered regarding the genetic relationships of affected calves so that science-based breeding management changes can be recommended. If the syndrome is maternally inherited, culling sires may be unjustified. Samples of brainstem from any suspect cases should be submitted for histologic confirmation.

References

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