*Spinal Dysraphism in a Two-Day-Old Aberdeen Angus

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Introduction

Spinal dysraphism is well documented in man and the Weimaraner breed of dogs. Clinical signs as well as morphologic changes observed in man and the dog are similar.

Bovine spinal dysraphism, arthrogryposis and cleft palate in newborn Charolais calves have been reported. In those cases dysraphism was confined to the cervical spinal cord and clinical signs paralleling those observed in dogs with spinal dysraphism were not reported. Recently, gross segmental lumbar dysraphia was reported in two Angus X Hereford calves on Kodiak Island.

The precise cause of spinal dysraphism is not known. However it has been suggested that anomalous vasculature in the ventral spinal arteries may be a cause in the dog. The purpose of this report is to document the occurrence of characteristic clinical signs including "bunny-hopping," and morphologic changes in a two-day old Aberdeen Angus calf. Additionally, this report suggests that abnormalities of the spinal ventral arteries may have played a role in the development of dysraphism in this animal.

Clinical Signs

A two-day-old female Aberdeen Angus calf was presented to the Washington State University Large Animal Clinic with a history of reluctant and difficult ambulation since birth. Physical examination of the animal revealed severe posterior ataxia, knuckling and extension of the hindlimbs. When the animal was assisted or forced to move, a characteristic symmetrical hopping motion (bunny-hopping) was observed. In addition, unilateral abduction of one hindlimb was noted when the animal was standing. Spinal flexor reflex elicitation induced a synchronous bilateral flexor response. Radiographs of the skeletal pelvic area were normal. The owner requested euthanasia and subsequently the animal was presented for necropsy examination.

Pathology

Upon opening the vertebral column, the vertebral canal appeared to have been larger than normally expected for this size animal. There appeared also to be excessive amounts of fluid resembling cerebrospinal fluid outside of the dura.

Significant gross findings were confined to the spinal cord and consisted of a prominent constriction of the posterior aspect of the cord. Immediately preceding and following the constricted area were bulb-like enlargements. The constricted area corresponded to L_1 through L_4 and was devoid of the ventral spinal artery and vein (Fig. 1). These vessels were prominent anterior and posterior to the constricted area. Incision of the bulb-like enlargements revealed a markedly dilated central canal and excessive amounts of cerebrospinal fluid (Fig. 2).

Significant light microscopic changes were confined to the spinal cord and consisted of central canal dilation (hydromyelia), absence and duplication of the canal, absence of an intact ependymal lining in the canal and mild degenerative changes in compressed grey and white matter. In addition, the ventral fissure was indistinct, and there were abnormalities of the dorsal septum and an asymmetry and lack of separation of the dorsal and ventral columns.

Tissue sections from the caudal aspects of the thoracic cord (T12 and T13) revealed a markedly dilated canal that was only partly lined by flattened ependymal cells (Fig. 3). Adjacent grey matter was compressed and neuronal degeneration was present. The neuropil of the surrounding white matter had a loose appearance and contained small vacuoles diffusely. Serial sections taken from the lumbar cord (contricted area) revealed a complete absence of the central canal in anterior sections (Fig. 4) and duplication of the central canal (Fig. 5) in posterior tissue samples.

Throughout the constricted area abnormalities of the grey matter were present. The entire grey matter mass was reduced and the dorsal and ventral columns were compressed. Large motor neurons were scattered through the grey matter and occupied the area corresponding to the nucleus dorsalis. A large vessel was present in the grey matter near the normal site of the central canal. Tissue sections from the posterior aspects of the constricted area revealed duplications of the central canal. The canals were often hypoplastic, but with intact ependymal epithelium.

Changes observed in the white matter throughout the constricted area consisted of a partial or indistinct dorsal septum and abnormalities of the ventral fissure. In no instances did the fissure extend to

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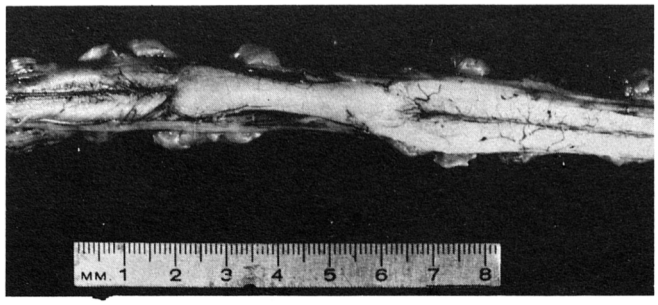

Figure 1. Gross photograph of the spinal cord demonstrating marked constriction of part of the lumbar segment. Notice the absence of the ventral spinal vessels in the constricted portion.

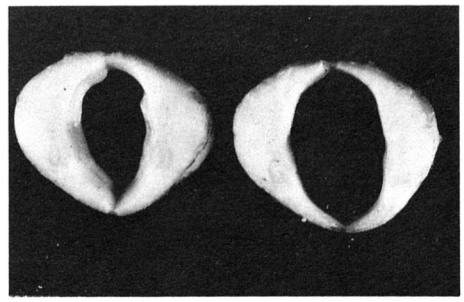

Figure 2. Cross sections through the bulb-like enlargement anterior to the constricted lumbar segment revealing extreme hydromyelia.

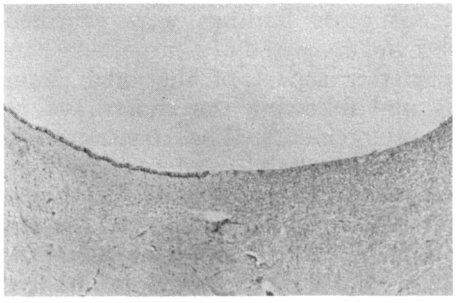

Figure 3. Photomicrograph of markedly dilated central canal and partial lining by ependymal cells.

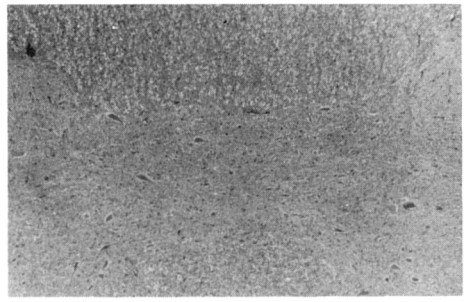

Figure 4. Photomicrograph of section taken through constricted portion of the lumbar cord. Notice the absence of a central canal and the scattered motor neurons.

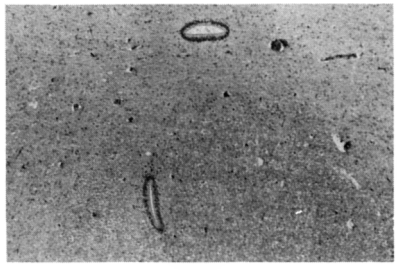

Figure 5. Photomicrograph of section taken through the posterior aspect of the constricted lumbar segment. Notice central canal duplication.

the vicinity of the central grey matter.

Tissue samples taken from the enlargement posterior to the constricted area contained changes similar to those of the anterior enlargement but to a lesser degree. Central canal dilation was much less and ependymal epithelium lined a larger portion of the canal wall.

Discussion

Clinical signs observed in this animal were similar to those observed in two Angus X Hereford calves on Kodiak Island (1). These authors also reported segmental lumbar dysraphia consisting of a grossly dilated central canal and two dorsally located cystic cavitations. In addition, the extreme narrowness and dilation of the central canal, a partial ependymal lining of the wall of the canal, compression of the ventral grey horns, and neuronal degeneration observed in this calf have been reported in Charolais calves (2). However, the marked gross constriction of the lumbar segment of the cord with associated absence of the ventral spinal vessels, and the absence and duplication of the central canal have not been reported in the bovine.

It has been suggested on the basis of preliminary observations (3) that an anomalous vasculature in the ventral spinal arterial extrinic vessels as to size and pattern may be the cause in dogs affected with dysraphism. In this calf, the absence of the ventral spinal vessels in their normal location in the affected lumbar segment and the presence of these vessels within the neuropil suggest to us that the abnormal vasculature could well account for the observed abnormalities. Irrespective of the cause, lesions observed in this calf are consistent with those reported in the dog (3) and man (4) with spinal dysraphism.

We observed several channels in the spinal cord substance preceding the lumbar constriction that may well represent syringomyelia. Utilizing serial sections, we were unable to discern a direct communication of the channels and the central canal. For that reason we have chosen not to include syringomyelia in the diagnosis.

References

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