Segmental Hypoplasia of the Spinal Cord in a Japanese Black Calf

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(Received 21 August 2008/Accepted 8 October 2008)

ABSTRACT. Segmental hypoplasia not associated with vertebral abnormalities was found in a Japanese Black calf that was unable to stand. Constriction occurred between the third and 5th segments of the lumbar spinal cord, and was most severe in the 4th segment. Myelodysplasia, such as hydromyelia and syringomyelia, absence or interruption of the central canal, dysplasia of the gray matter, and absence or divergence of the septal connective tissue at the dorsal median septum or the ventral median fissure, were confirmed histologically. These changes indicate hypoplasia of the segments affected following neural tube closure. Therefore, this case was suspected to be a closed neural tube defect.

KEY WORDS: Japanese Black calf, myelodysplasia, segmental hypoplasia of the spinal cord.

Congenital abnormalities of the spinal cord have been described collectively as myelodysplasia or dysraphic states [7, 10]. In cattle, the occurrence of spina bifida, a spinal neural tube closure defect, is common [8]. Hypoplasia of the spinal cord is defined as a reduced development of one or more segments of the spinal cord [10]. In cattle, this hypoplasia has been reported in Angus and other breeds [7, 8]. Although these reports described only the brief clinical signs of the affected animal [8], abnormalities in appearance, pathological changes and other details of the animals were not reported. Constriction or absence of the spinal cord have always been associated with vertebral abnormalities such as segmental aplasia of the spinal cord [4, 9], perosomus elumbis [6] and spinal stenosis [3].

In this study, we describe a rare anomaly of the spinal cord, namely, segmental hypoplasia of the spinal cord in a calf that was not associated with vertebral abnormalities. The morphological changes of the malformed spinal cord were examined, and their pathogenesis was discussed from an embryological viewpoint. The subject was a male Japanese Black calf that was unable to stand after birth. His left forelimb and right hindlimb were not flexible and exhibited only mild convulsion. In general appearance, spinal defects could not be confirmed. Although the calf was born 2 weeks before the expected delivery date, the general conditions, such as vigor and appetite, were fine. The calf was euthanized after one month according to the request of the owner of the animal, and a postmortem examination was performed. The weight of the calf before the necropsy was 42 kg, indicating a reduced growth compared to normal calves. It was the first delivery of the cow, which went through a normal pregnancy and did not receive the administration of any drugs. No similar cases have been reported from the maternal or paternal line of the calf.

The necropsy confirmed a constriction between the third (L3) and fifth (L5) segments of the lumbar spinal cord, with the constriction in the fourth segment (L4) being the most severe (diameter: approx. 6 mm) (Fig. 1). Despite a mild dorsal curvature of the spine, no spinal canal narrowing or abnormal vertebrae were observed from the maceration specimen. A cross section at the constricted region of the spinal cord revealed 1 to 3 spindle-shaped cavities with diameters of 1 to 2.5 mm between the dorsal marginal part and the central part near the dorsal median septum. The gray matter of the constricted region showed hypoplasia and poor delineation. At around L4, where the constriction was severe, the dorsal median septum and the ventral median fissure were not distinguishable by gross observation. Paraffin sections were prepared by isolating the section (approximately 3 mm thick) of spinal cord between the 10th thoracic spinal cord segment and the last lumbar spinal cord segment, which contained the constricted part. Serially sectioned samples were prepared as needed for histological examination.

The cavity observed in the center of the cord was partially lined by ependymal cells, indicating hydromyelia caused by the expansion of the central canal. The cavity present in the dorsal funiculi of the spinal cord was lined by altered myelin.
sheaths and nerve fibers, indicating syringomyelia (Fig. 2A). Mild expansion of the central canal began near T10 and gradually worsened caudally, forming a hydromyelic cavity (HC) which was most severe along the first half of L3. The shape of HC varied depending on the section, and in sections with severe expansion, ependymal linings were not observed around most of the lumen. The ependymal cells present were flattened, often forming small rosettes (Fig. 2B). Much of the central canal was absent from the second half of L3 to L4 (Fig. 3A), and only segments of the hypoplastic central canal were interrupted (Figs. 3B, 4). The syringomyelic cavity (SC) of the dorsal funiculi formed a tube divided into various lengths, and the adjoining white matter showed malacic changes with the occurrence of altered myelin sheaths and edema. SC occurring continually from T10 to L6 and was connected to HC in the center at L3 and L5 (Fig. 4). The gray matter, although being dissected around HC and exhibiting dysplastic lesions such as distortion influenced by the shape of the central canal and the poorly outlined ventral horn, was mostly symmetrical. In these regions, the neurons were scattered in low density and the large type neurons such as motor neurons were few. The dorsal median septum and the ventral median fissure were either absent or hypoplastic, and vascularity was low in parts divided by a small amount of connective tissue. In the ventral median fissure at L3, connective tissue that was poorly penetrated into the spinal cord was diverged, forming a Y-shape (Fig. 2A).

Histological changes observed in the constricted part of the spinal cord, such as the HC, the SC, the absence or interruption of the central canal, the dysplasia of the gray matter, and the absence or divergence of the septal connective tissue at the dorsal median septum or the ventral median fissure, were consistent with the characteristics of myelodysplasia and dysraphism indicated [7, 10]. From the affected region and the degree of the anomalous changes, the localized constriction of the spinal cord confirmed that the subject calf was diagnosed as segmental hypoplasia of the spinal cord.

Segmental defects of the spinal cord in cattle are often accompanied by vertebral defects, and cases such as perosomus elumbis, in which the caudal spinal cord (the lumbosacral cord) is completely absent [6] and partial aplasia...
undergoes in segments of the cranial thoracic, lumbar and cervical spinal cord [4, 9]. These cases were classified as segmental aplasia of the spinal cord and were accompanied by myelodysplastic lesions, similar to those observed here, near the areas where the spinal cord was absent. Congenital spinal stenosis was reported to be associated with multifocal stenosis of the vertebral canal caused by the metaphyseal and epiphyseal enlargement of the affected vertebral bodies, also to be accompanied by myelopathy including the occurrence of dilated, empty axon cylinders, myelin loss and swollen axon [3]. In regard to the cavitation of the spinal cord, only a hydromyelic cavity was formed in the region of segmental aplasia of the spinal cord and no cavity formation was observed in the region of the congenital spinal stenosis.

The cavitation of the spinal cord without vertebral defects was reported in spinal dysraphism at the cervical spinal cord [11] and syringomyelia at the thoracic spinal cord [1]. However, in the case of spinal dysraphism, which closely resembles our case in the histological appearance of the spinal cord, a direct communication between the hydromyelic and syringomyelic cavities was not observed. The case of syringomyelia, which had syringomyelic cavity with the duplication and blind ending of the central canal, was associated with dominant inflammatory changes such as gliosis and perivascular cuffing was observed, suggesting viral infection. Furthermore, a constriction of the affected spinal segments was not seen in these cases. From the affected site and the degree of the defective changes, the morphological aspects in this study suggest that the present case is different from the cases described above. In humans, segmental hypoplasia of the spinal cord is associated with segmental spinal dysgenesis, which is often accompanied by focal vertebral malformations including narrowing of the spinal canal. In this case, the spinal cord at the level of the abnormality was thinned or, in some regions, indiscernible, and a bulky, low-lying cord segment was present caudal to the focal abnormality [12, 13]. While a segmental hypoplasia of the spinal cord has been reported to be accompanied by spina bifida occulta and hypoplasia of the vertebral body, but not by the narrowing of the vertebral canal, the case also exhibited meningocele, hydrocephalus, hypoplasia of the corpus callosum and Chiari I malformation [2]. Therefore, no cases similar to the present case have been reported in either cattle or humans.

The confusion surrounding the use of the terms “neural tube defect” and “spina bifida” has been discussed, and it has been proposed from the embryological and pathological viewpoints that failure of primary neurulation and neural tube closure should be classified as open neural tube defects and that, after neural tube closure, they should be classified as closed neural tube defects caused by deficient development of the structures around the spinal cord [5]. The hypoplasia of the spinal cord in the calf reported here is a closed neural tube defect because it was not accompanied by bone or skin lesions such as the cleavage of the vertebral neural arch or skin. The absence or interruption of the central canal in its constricted section indicates a developmental disturbance of undifferentiated epithelial cells of the neural tube lining the lumen of the post-closure neural tube, suggesting that the normal development of ependymal cells was inhibited. This resulted in incomplete compartmentalization of the wall of the neural tube (insufficient formation of basal plate, alar plate, roof plate, and floor plate) and thus caused dysplasia or alterations in the gray matter, dorsal and ventral median septa. In addition, blind endings formed by the absence or interruption of the central canal caused abnormal

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**Fig. 4.** Distribution of the spinal cord lesion along sections cranial and caudal to the constricted section. The HC in the center and the SC in the dorsal funiculi are connected at L3 and L5, and small interrupted segments of the central canal exist from the second half of L3 to L4 (Bold lines indicate the lining of ependymal cells). The outline of the gray matter becomes unclear at L4, exhibiting dysplastic changes. The ventral median septum, which is diverged at L3 forming a Y-shape, gradually disappears toward the caudal severer constricted region.
expansion and consequent rupture of the canal through obstruction of cerebrospinal fluid flow, dissecting of parenchyma and the creation of cavities in the spinal cord. Low vascularity in the dorsal and ventral septa indicating a poor blood supply to the affected spinal cord segments may be related to its pathogenesis. The etiology of the segmental hypoplasia of the spinal cord in the calf reported here remains to be fully defined.

REFERENCES