Case Report
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Ultrasonographic Diagnosis of Syringohydromyelia and Segmental Hypoplasia of the Lumbar Spinal Cord in a Calf

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A 4-day-old, 33-kg female Holstein calf had not been able to stand since birth. The delivery, which was without complications, involved a primiparous dam.

On general physical examination, the calf appeared bright, alert, and had a normal appetite. The calf was in sternal recumbency with the hind limbs stiffly extended laterally and the forelimbs normally positioned underneath. Despite repeated attempts, it was unable to stand up. If passively positioned in a quadrupedal stance, the calf immediately fell down. The muscles were atrophic. Clinical examination of the respiratory and cardiovascular systems did not identify abnormalities that could explain the calf's inability to assume or maintain a quadrupedal stance.

A thorough neurological examination was carried out. Because of recumbency, postural reactions could not be tested. A delayed withdrawal reflex and absence of the patellar reflex were found in the pelvic limbs. The reflexes and musculature in the thoracic limbs were normal. There were voluntary tail movements, a normal perineal reflex, and normal anal tone. There were no signs of pain upon palpation of the vertebrae. Examination of the cranial nerves was normal. On the basis of the clinical and neurologic findings, the diagnosis was a paraparetic syndrome ascribed to a spinal lesion localized between the T3-S2 segments.

Inflammatory processes of the spinal cord were excluded on the basis of normal cerebrospinal fluid obtained from the lumbosacral space. BVD virus and *Neospora caninum*-induced malformation were excluded owing to the absence of the relevant antibodies. A tentative diagnosis of myelodysplasia at the level of the T3-S2 spinal cord segments was made. No additional diagnostic conclusions were possible regarding different types of myelodysplasia, such as diastenomyelia/diplomyelia complex (duplication of the gray matter at ≥ 1 segments), rachischisis (incomplete closure of the neural tube, with the central canal remaining open and communicating with the integument), meningocele (herniation of the dura matter through a spinal column defect), hydromyelia (dilatation of the central canal), syringomyelia (longitudinal cavitations of the spinal cord parenchyma), and hypoplasia (anomalous development of ≥ 1 segments of the spinal cord).[1,2]

Ultrasonography of the spinal cord was performed. The calf was placed in right lateral recumbency. No sedation was necessary. Only slight restraint was used to flex the pelvic girdle and the lumbosacral spine so as to enlarge the space between the vertebral arches for wider acoustic windows. Ultrasonographic examination was performed in sagittal and transverse orientations through the lumbosacral junction (L6-S1) as well as through more cranial lumbar intervertebral junctions (L5-L6, L4-L5, L3-L4, L2-L3). A 6–10 MHz linear 4-cm transducer[a] was used.

Ultrasonographic pathologic findings were observed only at the level of the L4-L5 and L3-L4 intervertebral junctions, whereas the L6-S1, L5-L6 and L2-L3 acoustic windows showed normal images of the spinal cord.

At the level of the L6-S1 acoustic window, in the sagittal orientation, the spinal cord appeared as a thin hypoechoic conus, 0.47 cm in thickness, with hyperechoic margins referable to the pia mater, and a central hyperechoic single line referable to the central canal. An anechoic cerebrospinal fluid-filled subarachnoid space and a hyperechoic dura-arachnoid layer were evident dorsally and ventrally to the spinal cord.

In the transverse orientation, the conus medullaris was round with a diameter of 0.47 cm; hyperechoic margins and a central hyperechoic dot also were visible, and again referable to the pia mater and the central canal, respectively. The spinal cord was surrounded centrifugally by an anechoic cerebrospinal fluid-filled subarachnoid space, and a hyperechoic dura-arachnoid layer. Two large hyperechoic nerve roots were evident, emerging symmetrically dorsolaterally and ventro-laterally. The aforementioned images were compatible with the normal features of the conus medullaris.[3]

At the level of the L5-L6 acoustic window, in the sagittal orientation, the spinal cord appeared as hypoechoic tube 0.90 cm in thickness, with hyperechoic margins (pia mater) and a central hyperechoic single line (central canal). An anechoic cerebrospinal fluid-filled subarachnoid space, and the hyperechoic dura-arachnoid layer were evident dorsally and ventrally to the spinal cord. In the transverse orientation, the spinal cord was ellipsoid (0.90 × 1.25 cm) with hyperechoic margins and a central hyperechoic dot. An anechoic cerebrospinal fluid-filled subarachnoid space,

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a hyperechoic dura-arachnoid layer, as well as the emergence of the hyperechoic dorsal and ventral nerve roots were visible as well. The findings were compatible with a normal lumbosacral intumescence, as confirmed by images obtained from a 7-day-old healthy Holstein calf (Fig 1). Contrary with what has been described above with respect to the L6-S1 and L5-L6 intervertebral junctions, the L4-L5 acoustic window, which also was expected to be consistent with the lumbosacral intumescence, generated abnormal images. In particular, in the sagittal orientation, the spinal cord had a sudden reduction in thickness, appearing as a small hypoechoic tube 0.38 cm in thickness with hyperechoic margins (pia mater), but without the hyperechoic line of the central canal (Fig 2). In the transverse orientation, it appeared round and measured 0.40 cm with partial hyperechoic margins, but without evidence of a central hyperechoic dot, as expected. Dorsal and ventral nerve roots also were not evident.

The L3-L4 acoustic window also indicated abnormal spinal cord features, but these were different from those described above. In fact, in the sagittal orientation, the spinal cord with its hyperechoic margins (compatible with the pia mater) regained the expected thickness, 1.0 cm, but showed a splitting of the central line into 2 hyperechoic lines, delimiting an anechoic space 0.30 cm in width (Fig 3), referable to a dilatation of the central canal (hydromyelia). In

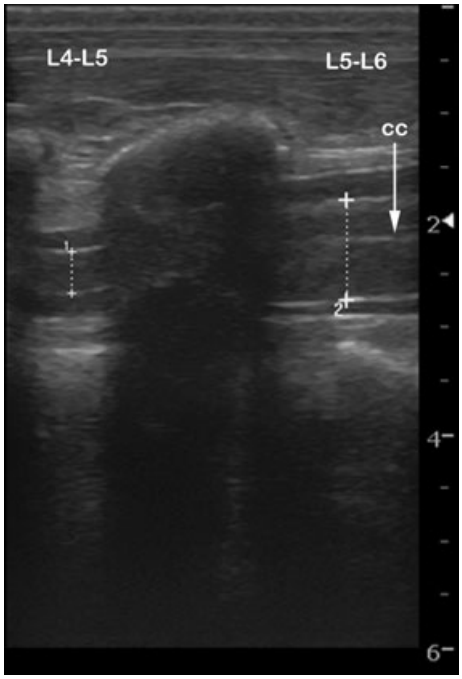

Fig 2. Sagittal sonogram at the level of the L4-L5 and L5-L6 acoustic windows. Note the different spinal cord thicknesses in the 2 acoustic windows, measuring 0.38 cm (broken line 1) and 0.90 cm (broken line 2) at L4-L5 and L5-L6, respectively. The absence of the hyperechoic line of the central canal in the L4-L5 acoustic window should also be noted, whereas it is present in the L5-L6 acoustic window (cc).

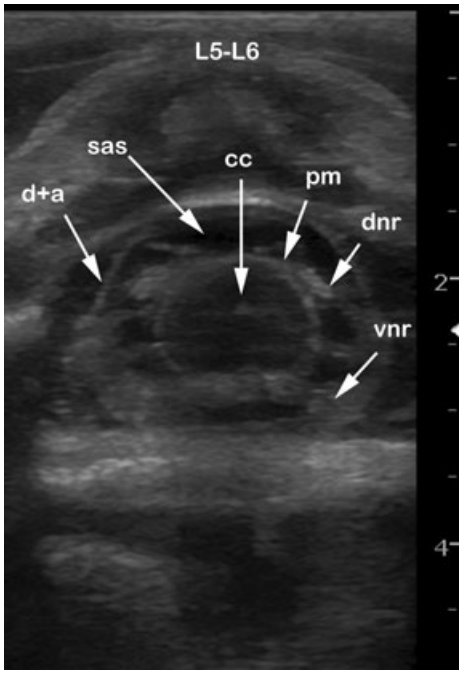

Fig 1. Transverse sonogram at the level of the L5-L6 acoustic window in a 7-day-old healthy Holstein calf. The following anatomical structures can be recognized: central canal (cc), well-defined central dot; pia mater (pm), thin, but strongly echogenic layer; subarachnoid space (sas), anechoic zone; dura-arachnoid layer (d + a), echogenic line; dorsal and ventral nerve roots (dnr and vnr), hyperechoic branches.

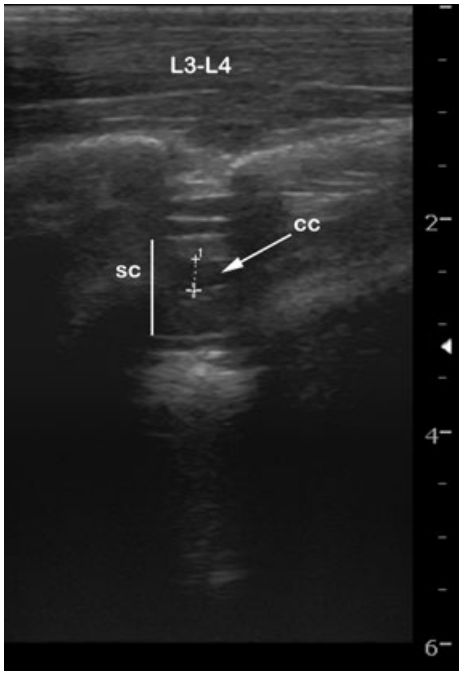

Fig 3. Sagittal sonogram at the level of the L3-L4 acoustic window. Note the presence of 2 hyperechoic lines delimiting the dilated (broken line 1) anechoic central canal (cc). The solid line indicates the spinal cord (sc).

the transverse orientation, the spinal cord appeared ellipsoid (1.00 × 1.46 cm) with hyperechoic margins and, similar to what was observed in the sagittal orientation, a hypoechoic lumen confirmed the dilatation of the central canal, which measured 0.30–0.40 cm in diameter. The dorsal and ventral nerve roots were not evident. The L2-L3 acoustic window, in both the sagittal and the transverse orientations, again showed the characteristic features of a normal spinal cord. Therefore, the ultrasound findings localized at the L4-L5 and L3-L4 junctions were consistent with a diagnosis of lumbar hypoplasia (L4-L5 junction) and syringohydromyelia (L3-L4 junction) of the spinal cord.

The calf was euthanized because of the poor prognosis, and a postmortem examination was carried out. The vertebral column, including the lumbar vertebrae, was normal. On the contrary, the segments of the spinal cord from L3 to L5 showed abnormal morphology. In particular, the lumbar intumescence had an hourglass appearance because of the presence of a 1.50 cm-long thinning at the level of the L4-L5 segment, up to 0.30 cm in diameter (Fig 4). Moreover, the segment of the spinal cord between L3 and L4 showed a fluctuation upon palpation. At this level, sagittal sectioning disclosed a fluid-containing 1.50 cm-long, 0.30 cm-wide, spindle-shaped cavity.

Histological examination of the spinal cord at the level of the above-mentioned lesion (L3-L4) showed an optically empty cavitary lesion in the medial part of the gray matter. The cavity was lined by ependymal cells only in its ventral and caudal parts. The surrounding neuroparenchyma had architectural alterations caused by compression exerted by the above-mentioned space-occupying lesion. Blood vessels of the nervous tissue and meninges were congested. A progressive reduction of the dorsal column system, together with neuronal hypoplasia, gliosis, and an increased number of blood vessels, was evident caudal to the cyst. Moreover, there was a loss of the anatomical structure of the gray matter, which ended abruptly at a distance of 1.5 cm from the caudal blind sac of the cavity (L4-L5). More distally, the spinal cord contained only myelinic nerve fibers. The final diagnosis was syringohydromyelia associated with segmental hypoplasia of the lumbar spinal cord.

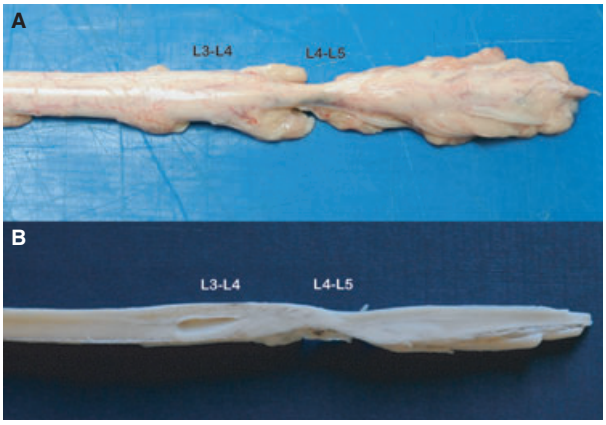

Fig 4. Spinal cord: fresh anatomical specimen (a) and formalin-fixed, sagittally-sectioned, specimen (b). Note the hourglass aspect of the lumbar intumescence with thinning at the level of the L4-L5 segment. The spindle-shaped cavity between L3 and L4 should also be noted.

Syringohydromyelia and segmental hypoplasia are 2 developmental disorders of the spinal cord rarely diagnosed in living calves. Myelography, MRI, or both are considered essential for diagnosing such conditions, but neither is readily available to bovine practitioners. This study showed that ultrasonography (an ancillary diagnostic technique that has been gaining wider use in bovine veterinary practice) is able to detect such congenital anomalies in living calves.

Hydromyelia and syringomyelia constitute a complex of spinal cord abnormalities characterized by the presence of fluid-filled cysts. In particular, hydromyelia is a simple distension of the central canal, with the accumulation of cerebrospinal fluid. Therefore, it is lined by ependymal cells. On the contrary, syringomyelia is a neoformed, usually tubular, paracentral cavitation within the neuroparenchyma (syrinx). It is lined by glial cells and contains a liquid consistent with cerebrospinal fluid.[4] The all-inclusive term syringohydromyelia has been proposed because hydromyelic cavities often include a contiguous pocket of neuroparenchyma lined by glial cells and, in addition, syrinxes within the neuroparenchyma often rupture into the central canal.[5] However, the term syringomyelia also is generally accepted for all clinical conditions characterized by spinal cord cavitations containing fluid identical with or closely resembling cerebrospinal fluid.[6] Moreover, hydromyelia and syringomyelia may be difficult to distinguish, even after detailed histologic examination. Segmental hypoplasia of the spinal cord is a rare dysraphism caused by an anomalous development of ≥ 1 segments of the spinal cord, generally associated with vertebral abnormalities.[7,8]

Both syringohydromyelia and spinal segmental hypoplasia, along with associated congenital or progressive neurologic symptoms, have rarely been described in cattle.[9,10] Even rarer are reports on the use of ultrasonography in cattle for assessing myelodysplasia in general.[3,11] In fact, whereas in human medicine spinal ultrasound examination constitutes the initial screening test in infants suspected of spinal dysraphism,[12] in veterinary medicine, especially in bovine medicine, it still is rarely used. Moreover, in young calves, the acoustic shadowing created by the ossified dorsal vertebral elements does not create the same favorable circumstances that are present in young human infants where the posterior arch of the spine only becomes ossified beginning at 6 months of age.[12]

Recently, 2 ultrasound acoustic windows for spinal cord examination have been proposed for calves: 1, cervical, is the atlanto-occipital junction[11] and the other, lumbar, is the lumbosacral L6-S1 intervertebral space.[3]

In this study, despite the ossification of the spinous elements, in addition to the lumbosacral junction L6-S1, it was possible to use other new lumbar acoustic

windows, namely the more cranial intervertebral lumbar spaces. The best sonographic images were obtained at the level of the L6-S1 and L5-L6 acoustic windows. However, the acoustic windows cranial to the L5-L6 junction also offered satisfactory images, enabling the detection of spinal cord segmental hypoplasia at the L4-L5 junction and of syringohydromyelia at the L3-L4 junction. In fact, all the above-mentioned acoustic windows allowed pattern recognition, as well as the assessment of abnormalities of the spinal cord (hypoechoic structure with no discernible interface between the gray and the white matter), the central canal (well-defined linear central echo or dot – depending on the scanning plan – hyperechoic to the surrounding spinal cord parenchyma), the pia mater (thin but strongly echogenic layer enveloping the cord parenchyma), the subarachnoid space (further peripheral anechoic zone), and the dura mater together with the arachnoid layer (a prominent echogenic line representing a combination of the 2 structures, superficial to the spinal cord). Moreover, in the transverse orientation, the nerve roots were evident as 2 large hyperechoic branches emerging symmetrically dorso-laterally and ventro-laterally.

With respect to the central canal, the limited amount of cerebrospinal fluid in the unaffected portions of the spinal cord did not allow the central canal to have an anechoic appearance, as might be expected for a fluid-filled structure. Although still under debate in the literature, the central cord echo was attributed to the difference in acoustic impedance between the neural tissue of the cord and the fibrous ependyma lining the canal, or to the interface between the myelinated ventral white commissure and the central portions of the anterior median fissure.[13] The increased amount of fluid filling the central canal in the spinal cord tract affected by hydromelia (L3-L4) allowed recognition of an anechoic lumen within a double-lined central canal. Similar considerations can be made with respect to the transverse orientation; a single echoic dot and a hypoechoic lumen, respectively, demonstrated the scant quantity and increased quantity of cerebrospinal fluid in the normal and in the hydromelia-affected spinal cord tracts, respectively. How much cerebrospinal fluid is necessary to ultrasonographically transform the echoic image into an hypoechoic lumen might be the subject of future investigation, but, currently, to the authors' knowledge, remains unknown.

The hyperechoic aspects of the pia mater (the meningeal layer adhering to the surface of the spinal cord) were very useful in sagittal orientation for depicting the narrowing of the spinal cord at the L4-L5 acoustic window. The hyperechogenicity that delimits the dorsal and ventral borders of the spinal cord is, however, attributable to not only to the pia mater itself, but also to the acoustic impedance differences between the spinal cord parenchyma and the subarachnoid fluid. Because of the presence of the acellular cerebrospinal fluid, the subarachnoid space is seen as an anechoic zone between the pia mater and the more superficial dura-arachnoid layer. The thin net-like structure of the arachnoid cannot be detected ultrasonographically as a separate structure. Because of its close contact with the inner surface of the dura mater, it is ultrasonographically included in the prominent hyperechogenic line that defines the strong and dense structure of the most superficial meninge.

With respect to the execution of the technique, the examination was favored by lumbosacral flexion, which enabled widening of the ultrasound windows. Unfortunately, the particular anatomical shape of the spinous processes of the thoracic vertebrae did not allow scanning the more cranial portions of the spinal cord. In conclusion, this study showed the ultrasonographical features of 2 rare spinal cord malformations that may be underreported because of the current lack of diagnoses made on living calves. This study, therefore, definitively demonstrated that ultrasound examination can be useful ancillary diagnostic tool for assessing diseases of the spinal cord in young calves.

Footnote

[a] Logiq P5, GE Healthcare, Milano, Italy (General Electric Company, Fairfield, CT)

Acknowledgment

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Conflict of Interest: Authors disclose no conflict of interest.

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