PEER REVIEWED

Posterior Paresis Resulting from a Vertebral Body Fracture in a Bull

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Abstract

A 2.5-year-old, mixed-breed rodeo bull was examined with a four-month history of ataxia and weakness in the pelvic limbs. The neurologic signs were noted while the bull was on pasture breeding cows. Spinal radiographs revealed a compression fracture of the 13th thoracic vertebrae, and myelography showed impingement of the spinal cord due to the fracture. Necropsy examination confirmed the diagnosis of a compression fracture of T13 with secondary damage to the spinal cord. Vertebral fractures are uncommon in adult cattle, yet this case appears to be unrelated to any previously published causes. Furthermore, myelography can be an effective diagnostic test in the evaluation of spinal cord disease in adult cattle.

Keywords: bovine, fracture, paresis, myelography

Résumé

Un taureau de rodéo de 2.5 ans de race mélangée montrait à l'examen des signes d'ataxie et de faiblesse dans les membres postérieurs qui duraient depuis quatre mois. Les signes neurologiques avaient été notés lorsque le taureau était au pâturage avec des vaches durant la saison de reproduction. Une radiographie de la moelle a montré une fracture de compression de la 13me vertèbre thoracique et une myélographie a montré un coincement de la moelle épinière causé par la fracture. L'examen à la nécropsie a confirmé le diagnostic de la fracture de compression de T13 avec dommage secondaire à la moelle épinière. Les fractures vertébrales sont rares chez les bovins adultes mais ce cas ne semble pas relié à aucune cause déjà publiée. De plus, la myélographie peut être un test diagnostic efficace dans l'évaluation des maladies de la moelle épinière chez des bovins adultes.

Introduction

Spinal cord disease in adult cattle can be due to a multitude of causes including spinal lymphosarcoma, trauma, parasitic migration, discospondylitis and vertebral body abscess.[5,6,7,11,14] Vertebral fractures are uncommon in adult cattle, but have been associated with poor facility design, nutritional deficiencies and estrus behavior in females.[2,8,12] Identifying the cause of the neurologic deficits can be significant as the treatment and prognosis for these diseases varies widely.[2,5,6,7,8,11,12,14] Myelography is commonly used in small animal and equine medicine to accurately diagnose diseases of the spinal cord, yet there is no published literature on the clinical evaluation of the adult bovine using myelography.

Clinical Findings and Case Management

A 2.5-year-old, mixed-breed rodeo bull weighing 2420 lb (1100 kg) was referred to the Ruminant Medicine Service at the North Carolina State College of Veterinary Medicine with a four-month history of neurologic deficits in his rear limbs. The bull was found to be acutely neurologic by the owner in a pasture he shared with 10 cows. The owner reported the bull appeared weak and occasionally stumbled and fell in the pasture. Upon examination by the referring veterinarian, the bull was diagnosed with a suspected thoracolumbar or lumbosacral lesion based on weakness in his hind limbs, with no obvious deficits in the forelimbs. The bull was treated with flunixin meglumine for three days (dose and route of administration unknown) and stall-rested for four months, but there was no change in clinical signs during this time. The bull had been actively competing in bull-riding competitions several months prior to the onset of clinical signs with no evidence of neurologic or orthopedic disease, and had been vaccinated with

a modified-live respiratory and leptospirosis vaccine (actual products unknown) six months prior to presentation. He had not been treated with an anthelmentic in the year prior to admission, and had no history of other significant medical problems.

On physical examination, the bull was bright, alert and moderately aggressive. The rectal temperature was 103°F (39.4°C). The mildly elevated temperature was attributed to transport and excitement, as it was normal when retaken later during hospitalization. The heart rate was 84 beats per minute, respiratory rate was 36 breaths per minute and there were two strong rumen contractions per minute. The bull's body condition was 6 (scale 1-9, with 9 being very obese), and he ate normally when offered hay. A rectal exam was not performed due to safety concerns for the examiner.

Although only a limited neurologic examination could be performed, the bull had significant posterior paresis and ataxia in the hind limbs. He appeared to have diminished conscious proprioception as he occasionally knuckled over on both rear feet. He would frequently cross his hind feet and circumduct each rear limb when walking. When standing, he pulled both hind limbs cranially underneath his abdomen. The front limbs appeared normal when walking and standing. A cranial nerve exam was within normal limits, and no pain could be elicited on palpation of his spine. Ideally, spinal reflexes would have been evaluated to more accurately differentiate between upper and lower motor neuron lesions. Results of these assessments would allow more precise focus of additional diagnostic tests to the thoracolumbar spine if an upper motor neuron lesion was found (normal to hyperreflexic) or the lumbosacral spine if a lower motor neuron lesion was identified (hyporeflexic). Due to the bull's size and aggressive nature, these tests could not be performed.

Based on clinical signs and neurologic examination, the lesion was localized to the thoracolumbar or lumbosacral regions. Differential diagnoses included spinal lymphosarcoma, vertebral and spinal trauma, discospondylitis, vertebral body abscess, spinal thromboembolism, parasitic invasion of the spinal cord by *Hypoderma bovis* and a congenital vertebral abnormality. Due to the bull's age and the lack of progression of clinical signs, injury was considered most likely. A complete blood count and chemistry panel were performed upon admission and were within normal limits. Collection of cerebrospinal fluid from the lumbosacral space was attempted, but a diagnostic sample could not be obtained due to blood contamination. Spinal radiographs and a myelogram were elected to further investigate the cause of paresis.

The following morning, after withholding feed and water for 24 hours, the bull was sedated with detomidine[a] (10 mg, IM) and acepromazine[b] (5 mg, IM). A 14-gauge, 5-1/2-inch jugular catheter was placed, and the bull was further sedated with detomidine (2.5 mg) intravenously. Anesthesia was induced with a combination of thiopental[c] (500 mg, IV) and diazepam[d] (50 mg, IV). After induction, the bull was orotracheally intubated and maintained on 2% isoflurane gas anesthesia.

Survey radiographs were taken of the thoracic, lumbar and sacral vertebrae. These radiographs indicated that the 13th thoracic vertebral body (T13) was shorter craniocaudally than other vertebrae, had an irregularly marginated caudal end plate and had mild bony proliferation ventrally, consistent with a compression fracture (Figure 1). There was increased lucency at the caudal aspect of the vertebral body, likely a result of bone resorption, however a concurrent infectious process could not be ruled out. A mineral opacity was seen ventral to the caudal aspect of the vertebral body, consistent with a bone fragment.

To confirm that the suspected compression fracture of T13 was impinging on the spinal cord, a myelogram was performed by removing 50 ml of cerebrospinal fluid (CSF) and injecting 50 ml of Iohexol[e] (300 mg/mL) into the subarachnoid space at the lumbosacral junction. The pelvis was elevated above the head to allow for cranial flow of the contrast material. Because the bull was intubated, the risk of aspiration was minimal if he had regurgitated.

There was an attenuation of the ventral aspect of the contrast medium column at the level of T13 (Figure 2), but the contour of the lateral aspects of the subarachnoid space could not be evaluated as dorsoventral radiographs could not be obtained. The CSF that was removed was submitted for evaluation. It appeared clear, and had a red blood cell count of 1865/µl, white blood cell count of 2/µl (10 large mononuclear cells and four small mononuclear cells) and a protein level of 38.4 mg/dl. This was considered normal CSF with minor blood

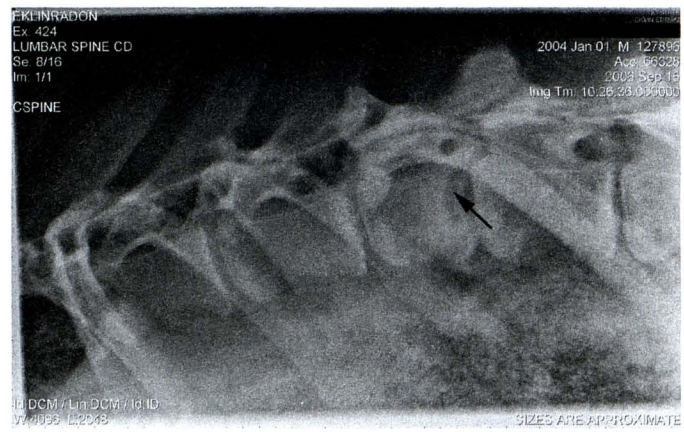

Figure 1. Radiographs of thoracolumbar spine showing compression of T13 and lucency (arrow) of the caudal endplate.

contamination. CSF chemistries were not performed, though finding elevated levels of creatine kinase would have provided further evidence of spinal cord damage as it increases with axonal degeneration.

Based on the poor prognosis for return to athletic performance or breeding soundness, the bull was euthanized and a necropsy was performed. On gross examination, the hemisected vertebral column revealed a T13 vertebral body fracture. The caudal portion of the T13 vertebral body was fractured centrally, and the adjacent intervertebral disc had ruptured and protruded into the fractured vertebral body. The affected T13-L1 intervertebral disc was focally red and firm, and the remainder of the disc material was pale yellow, firm and dry (Figure 3). The spinal cord at T13-L1 appeared normal grossly, but on histologic examination there were numerous multifocal, large open vacuoles (dilated myelin sheaths) predominantly within the ventral and lateral funiculi, some of which contained large foamy gitter cells and pale eosinophilic axonal remnants. In addition, there were moderate numbers of markedly swollen eosinophilic axons (spheroids) within some areas caudal to T13-L1 (Figure 4). Wallerian degeneration of the descending nerve tracts was present in the ventral and ventrolateral funiculi of L2, caudal to the site of primary insult. Lesions within the vertebral column and histopathology of the spinal cord were suggestive of an acute vertebral body fracture and disc rupture, leading to secondary spinal cord compression.

Discussion

There are retrospective studies of vertebral fractures in neonatal calves associated with dystocia,[1,15] but vertebral fractures in adult cattle appear to be relatively rare.[4] There are reports of vertebral fractures in juvenile cattle associated with poor facility maintenance or design,[2,7] but this bull had only been on pasture with cows in the weeks prior to the injury. The possibility of an injury occurring due to equipment in the pasture was deemed unlikely by the owner. Consequently, it was assumed that the bull was injured while breeding, but the exact nature of the injury remains unknown. Spinal injuries have been documented at artificial insemination centers, but these fractures are most often associated with bridging ventral spondylosis found almost exclusively in bulls greater than five years of age.[17] Sacral fractures potentially associated with estrus behavior have been reported in adult dairy cattle, but it is not clear if these are due to mounting other animals or being mounted.[12] We cannot rule out the possibility that the fracture could have been related to an injury suffered while bucking; however, the owner reported that the clinical signs appeared acutely several weeks after his last rodeo event.

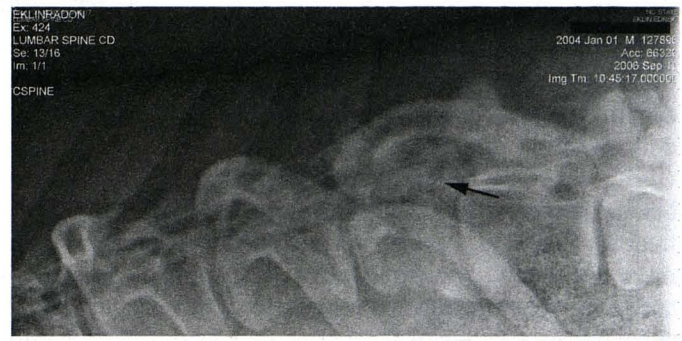

Figure 2. Myelogram showing attenuation of the dye column at the caudal edge of T13.

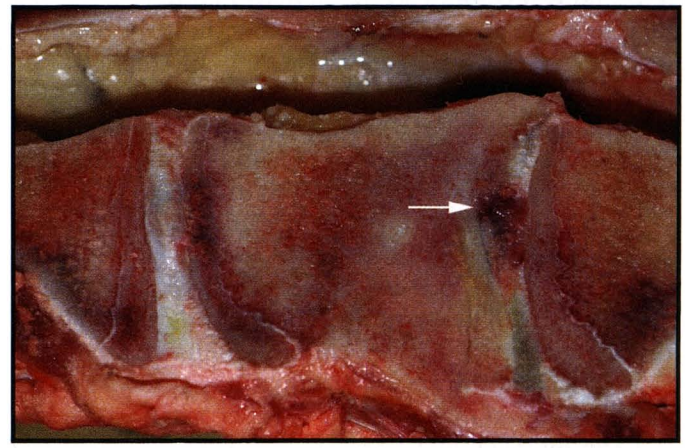

Figure 3. Hemisected vertebral column showing the compression fracture of the caudal endplate of T13.

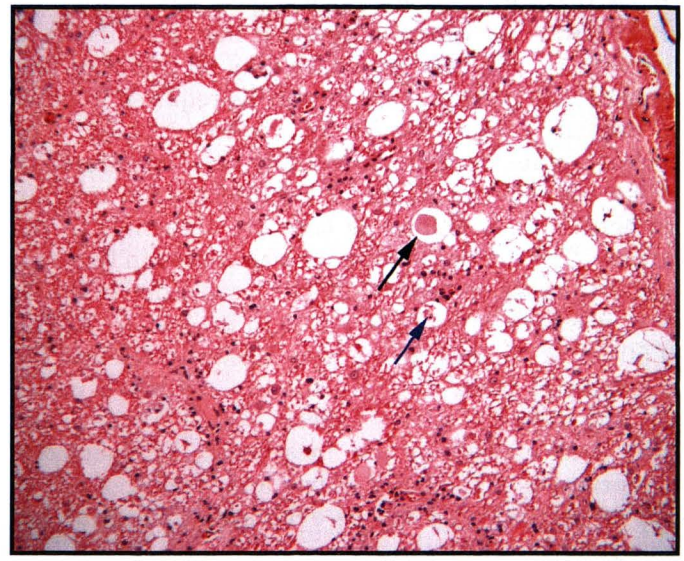

Figure 4. A photomicrograph at 200X demonstrating numerous markedly dilated myelin sheaths containing swollen axons (black arrow) and variable numbers of phagocytic cells (blue arrow) intermixed with cellular debris.

Primary pathology of the vertebra leading to a pathologic fracture could have contributed to the compression fracture, but this was unlikely. Spinal fractures secondary to osteodystrophy due to dietary mineral deficiencies have been reported,[8] but necropsy findings were not consistent with an osteodystrophy as the bone breaking strength and cortical thickness were normal at necropsy. Histopathology of the affected vertebrae would have been useful to further rule out a pathologic fracture, but was not performed. Although not commonly reported as a cause of fractures, discospondylitis could lead to compression of the affected vertebrae and a similar lucency on radiographs. Radiographs of cattle with discospondylitis consistently show lysis of the endplates of both the vertebrae cranial and caudal to the affected disk, and the cervical vertebrae are the only reported locations of the lesion in cattle.[5,9,14] In this case, no culture of the disk material was performed at necropsy, however both radiographs and necropsy suggested that the adjacent vertebral body (L1) was normal and that only a thoracic vertebra (T13) was involved.

Spinal radiographs are useful for diagnosis of many diseases of the spine and vertebrae in cattle, but are often normal in cases of soft tissue compression of the spine.[10,16] Myelography is commonly used in small animal medicine and equine medicine to diagnose compression of the spinal cord, but has rarely been reported in ruminants. There are reports of postmortem and antemortem myelography in calves,[3,13,16] but to our knowledge this is the first report of a myelogram being performed in a live adult bovine.

Understandably, myelography may be cost-prohibitive in most production animals, and meat and/or milk withdrawals for contrast material may be difficult to determine. Yet, this procedure may be useful in animals of high genetic merit or may be required in order to confirm a diagnosis for insurance purposes. The contrast material could potentially be administered in either the cisterna magna or the lumbosacral space in an anesthetized animal as both are accessible in the bovine. The lumbosacral space was chosen in this case because, based on neuroanatomic localization of the lesion, it was believed to be closer to the lesion. A total of 50 mL of contrast media were administered into the lumbosacral space with no acute adverse effects noted, but any potential adverse effects such as seizure may have been masked by the anesthesia. Furthermore, exacerbation of the bull's ataxia and weakness due to the procedure could not be evaluated as he was not recovered from anesthesia.

Conclusions

Based on the findings in this case, vertebral body fracture should be included as a differential in cases of acute pelvic limb paresis in adult cattle. Radiographs may show evidence of the fracture in these cases, but myelography can be performed in a referral setting to confirm compression of the spinal cord. Myelography may also be useful in diagnosing other causes of spinal cord compression, such as spinal lymphosarcoma in dairy cattle, that are more difficult to diagnose with survey radiographs alone.

Endnotes

[a]Dormosedan®, Pfizer Animal Health, New York, NY
[b]Acepromazine maleate, Vedco, Inc., St. Joseph, MO
[c]Pentothal, Abbott Laboratories, Chicago, IL
[d]Diazepam, Hospira, Inc., Lake Forest, IL
[e]Omnipaque, Amersham Health Inc., Princeton, NJ

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