PEER REVIEWED

Case Report – Diskospondylitis in Two First-calf Heifers

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Abstract

Two first-calf heifer Holstein herd mates were admitted to the veterinary teaching hospital with a one-to-two-week history of hind-limb weakness. Both heifers had recently freshened (25-30 days) and had their tails docked with castration bands at parturition. The chief presenting clinical signs for both cows were bilateral hind limb weakness, over-flexion of the hocks and occasional over-flexion (dorsal knuckling) of the fetlocks. Other pertinent physical examination findings included weak anal and tail tone. These clinical signs are diagnostic for tibial and sacro-caudal nerve damage. No significant abnormalities were found on preliminary blood work. Due to the poor prognosis for return to function, both cows were euthanized and a post-mortem examination was performed. Post-mortem examination revealed chronic suppurative diskospondylitis at the sixth lumbar vertebrae and first sacral vertebrae (L6-S1). *Escherichia coli* was cultured from the L6-S1 disk space of one cow (case 1); no other significant abnormalities were found on post-mortem examination. It was uncertain if the infection was secondary to an ascending infection from the tail-docking site, however, there was no evidence of generalized bacteremia or other sources of infection.

Résumé

Deux taures à leur premier veau issue du même troupeau ont été admises au centre hospitalier universitaire vétérinaire présentant des signes de faiblesse dans les membres postérieurs depuis 1 à 2 semaines. Les deux taures avaient vêlé récemment (25-30 jours) et été amputées de leur queue avec des élastiques de castration à la mise bas. Les signes cliniques principaux pour les deux vaches comprenaient une faiblesse bilatérale des deux membres arrière, une surflexion des jarrets et occasionnellement une surflexion des boulets (redressement dorsal du paturon). D'autres signes pertinents suite à l'examen incluaient un tonus faible de l'anus et de la queue. Ces signes cliniques permettent le diagnostic de dommages nerveux au niveau tibial et sacro-caudal. Des anomalies n'ont pas été détectées suite à l'analyse sanguine. Comme le pronostic d'un retour à la normal était plutôt mince, les deux vaches ont été euthanasiées et un examen post-mortem a été conduit. Cet examen a révélé une discospondylite suppurante chronique au niveau de la sixième vertèbre lombaire et de la première vertèbre sacrée (L6-S1). La bactérie *Escherichia coli* a été isolée dans l'espace entre les disques L6-S1 chez l'une des vaches. Aucune autre anomalie n'a été détectée. Il n'est pas clair si l'infection s'est propagée du site d'amputation aux vertèbres mais on note qu'il n'y avait pas de bactériémie généralisée ou d'autres sources d'infection.

Introduction

Diskospondylitis is a destructive, inflammatory and proliferative process involving intervertebral disks, their associated endplates and vertebral bodies.[9,13] Bacteremia is often implicated as the inciting cause of infection, but is seldom established as the inciting etiology.[17] Diskospondylitis has been well described in dogs,[10,11,13] cats,[12] horses,[1,2,4,7,9] and sheep,[15] however, little information exists on this condition in the bovine species.[16] This case report describes the clinical signs, diagnosis and post-mortem examination of two cases of diskospondylitis occurring in two Holstein herd mates.

History

The farm consisted of a closed herd of approximately 200 milking Holstein cows, 15 first-calf heifers, 45 heifers-to-calve and 35 dry cows. The cows were housed in a stanchion barn, but the heifers had occasional access to pasture. With the exception of the two sick heifers, no other cattle had shown evidence of systemic disease.

For the previous two months, both of the affected heifers had been housed in a stanchion barn. Neither heifer had difficulty calving and neither had received

any intramuscular injections within the previous 40 days. Immediately post-calving, castration bands had been placed on their tails, a procedure routinely performed on all first-calf heifers at freshening. Initially the owner had noticed that the heifers spent an increased percentage of time recumbent. When either heifer stood, she exhibited a hunched-back (dorsal-flexion) stance, and occasionally had episodes of buckling forward in the pelvic limb fetlocks. Both heifers had a healthy appetite, a normal temperature, and urinated and defecated normally.

Clinical Findings

Case 1

A two-year-old first-calf heifer, 30 days fresh, developed hind-limb weakness two weeks prior to presentation. Upon admission to the Cornell University Hospital for Animals (CUHA), she was bright, alert and responsive, but needed assistance standing up in the trailer. Shortly after unloading from the trailer, she became recumbent and unwilling to rise. On physical examination, all vital parameters were within normal limits. A neurologic examination revealed normal cranial nerve function, perineal and vulvar reflexes, but decreased tail tone and anal tone. After she was assisted to stand, the heifer had normal thoracic limb function and trunk stability. However, both pelvic limbs showed pronounced hock flexion and over-flexed, dorsal buckling of the fetlock. When she attempted to walk, she was weak on both pelvic limbs and kept them forward under the abdomen. No fractures, dislocations or other musculoskeletal abnormalities were detected.

Anatomic diagnosis for the neurological signs in this case was lesions or trauma of the sacral caudal spinal segments and roots, and tibial nerves. Differential diagnoses included spinal cord neoplasia, spinal cord trauma, spinal cord abscess, and spinal cord inflammation due to migration of *Hypoderma bovis* larvae. Other less-likely conditions on the differential diagnoses list included thromboembolic meningoencephalitis, and viral, bacterial and fungal meningitis.

Blood was submitted for a complete blood count (CBC) and serum chemistry panel. The CBC showed hyperproteinemia (9.1 g/dL; normal, 5.9 – 8.1 g/dL) and hyperfibrinogenemia (1200 mg/dL; normal, 100-600 mg/dL). The serum biochemistry panel demonstrated mild hypokalemia (3.6 mEq/L; normal, 3.9 – 5.3 mEq/L); hyperglobulinemia (6.7 g/dL; normal, 3.5 – 5.8 g/dL); hyperglycemia (116 mg/dL; normal, 31-77 mg/dL); increased aspartate transaminase (247 U/L; normal, 53 – 162 U/L); and increased creatine kinase levels (2408 U/L; normal, 77-265 U/L). A lumbosacral aspirate of the cerebrospinal fluid (CSF) was attempted, but the sample obtained was viscous and purulent. Cytological examination of the sample demonstrated many neutrophils, red blood cells and bacteria. Gram stain of the aspirate demonstrated many gram-positive cocci, and culture of the sample eventually yielded few colonies of *Escherichia coli*, suggesting a mixed bacterial infection. Due to poor prognosis, the heifer was euthanized and a post-mortem examination was performed.

Case 2

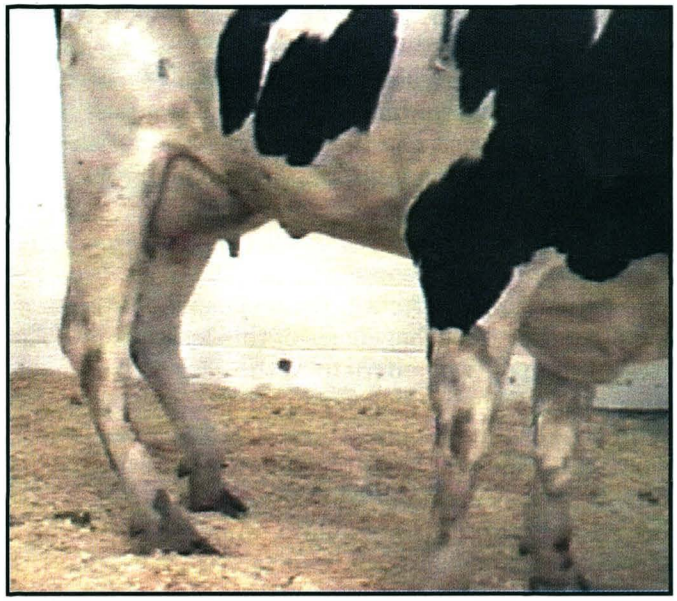
This first-calf Holstein heifer had freshened 25 days earlier, and was noticed to have developed hind-limb weakness one week prior to presentation. She was admitted to CUHA and found to be bright, alert and responsive. She was able to unload from the trailer successfully, but had difficulty walking. Enroute to the stall, she went down several times, but was able to rise using mainly her thoracic limbs. A neurological evaluation revealed normal cranial nerve function, normal perineal and vulvar reflexes, but decreased anal and tail tone. She appeared weak in both pelvic limbs. Both hocks were over-flexed and she buckled forward in both pelvic limb fetlocks (Photographs 1 and 2). Cutaneous sensation of the hind-limbs was evaluated using hemostats as a source for painful stimulus. Both hind-limbs appeared to have equal loss of skin sensation and analgesia that was localized distal to the stifle. Rectal examination and superficial palpation of the hind-limbs were within normal limits.

The anatomic diagnosis describing the neurologic signs in this case was sacral caudal spinal segments and roots, spinal nerves and tibial nerves. Differential diagnoses were similar to case 1.

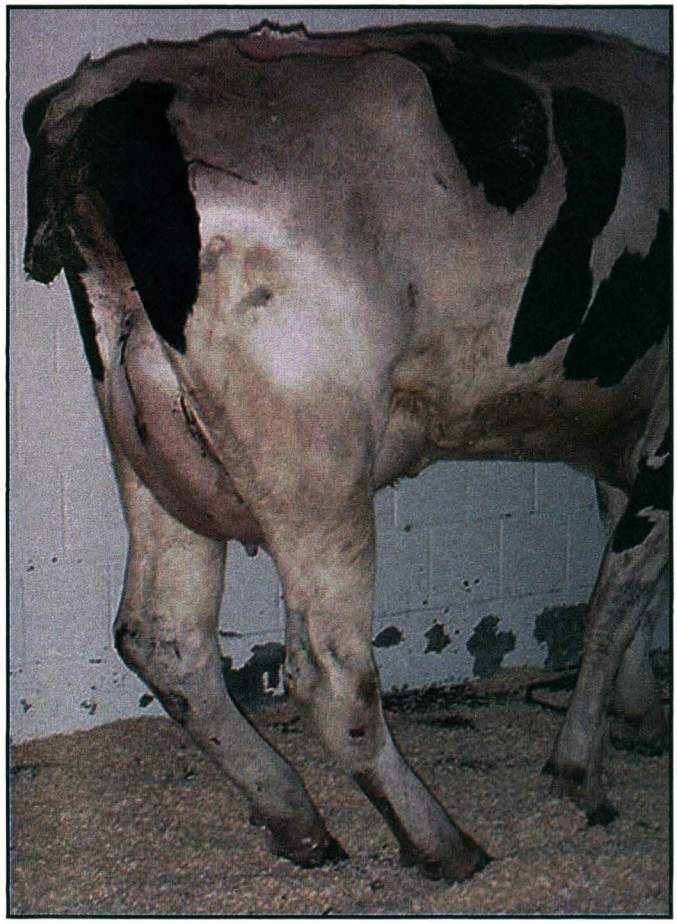
A complete blood cell count, serum biochemistry panel, and cerebrospinal fluid aspirate were obtained. The CBC showed neutrophilia (9 x 10^3 cells/μL; normal, 1.8 – 7.7 x10^3 cells/μL); hyperproteinemia (10 g/dL), and hyperfibrinogenemia (900 mg/dL). The serum biochemistry panel demonstrated mild hypokalemia (3.6 mEq/L), hyperglobulinemia (7.8 g/dL), hyperglycemia (101 mg/dL), increased aspartate transaminase (235 U/L) and increased creatine kinase (671 U/L). Cerebrospinal fluid cytology was normal. Due to the poor prognosis for return to normal function, the heifer was euthanized and a post-mortem examination was performed.

Post-mortem Examination

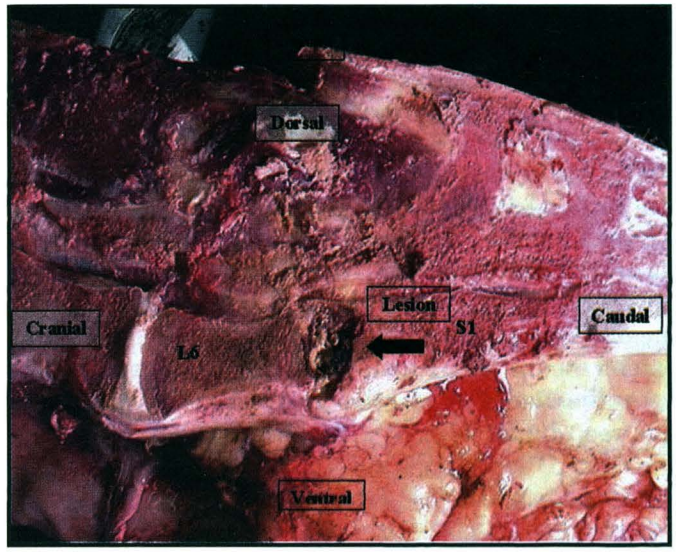
On the post-mortem examination of case 1, a band of white-to-yellow, thick, opaque material had completely replaced the intervertebral disk at L6–S1 (Photograph 3). The adjacent epiphyses of L6 and S1 were red, eroded and soft with thickening of the ventral vertebral ligament. Also evident on post-mortem examination was intramuscular hemorrhage involving both hind-limbs, possibly secondary to her inability to rise. In the right middle lung lobe, there was a mild focal bronchopneu-



Photograph 1. Case 2 - showing classical signs of tibial nerve paralysis with knuckling of the left lindlimb fetlock.



Photograph 2. Case 2 - showing clinical signs of bilateral tibial nerve paralysis with the left hindlimb being more affected than the right.



Photograph 3. Diskospondylitis between L6-S1.

monia. Careful dissection of the tissues of the tail beginning at Cd6 where the tail was docked, and extending cranial to L6-S1, did not reveal any abnormalities or any evidence of local extension of the infection from the tail-docking site. No other location of infection was identified during the post-mortem examination.

Similar findings were seen on the post-mortem examination of case 2. A band of thick tissue in the vertebral column, between L6 and S1, had completely replaced the intervertebral disk. This heifer also had her tail docked at the level of the body of the sixth caudal vertebrae, and minimal inflammation was associated within the area. No other pertinent necropsy findings were identified.

These heifers had similar histological findings. On the epiphyseal portions of L6 and S1, which bordered the lesion, there was evidence of bone lysis and inflammation. On histological examination the intervertebral disk and adjacent epiphyses in both cases were replaced by neutrophils, fibrin and scattered remnants of mineralized cartilage and necrotic bone. In the corresponding sections of the lumbar spinal cord and the cauda equina (the collection of spinal roots that stream caudally from the end of the spinal cord), there was chronic lymphoplasmacytic and neutrophilic meningitis, neuritis and perineuritis with endarteritis obliterans (occlusion of lumen of blood vessels). In the cauda equina, there was marked fibrosis that extended throughout the adipose tissue and entrapped the spinal nerves.

Discussion

For both heifers, the final diagnosis was chronic suppurative diskospondylitis at the level of the sixth lum-

bar and first sacral vertebral body, characterized by an accumulation of white-to-yellow, thick, opaque, purulent material that completely replaced the intervertebral disk and had lysed adjacent bone.

In the bovine species, the sciatic nerve, which includes the peroneal and tibial nerves, is formed by the ventral branches of L6, S1 and S2 spinal nerves. These nerves leave the vertebral column between L6 and S1, S1 and S2, and S2 and S3, respectively. The sciatic nerve innervates the caudal thigh muscles (biceps femoris, semimembranosus and semitendinosus), which function to extend the hip and flex the stifle. The tibial nerve innervates the caudal leg muscles (gastrocnemius, superficial and deep digital flexors), which extend the hock and flex the digits. The peroneal nerve innervates the cranial leg muscles (cranial tibial, long digital extensor and peroneus longus) that flex the hock and extend the digits.[3,6] In diagnosing neuropathies, sensory deficits may also aid in localizing the neuroanatomic deficit. There are several autonomous zones of cutaneous innervation in the pelvic limb. These are areas that are solely innervated by a nerve, and lack of or decrease in sensation usually indicates damage to a particular nerve. The cutaneous sensory distribution of the tibial nerve is to the caudal surface of the leg and plantar surface of the foot. The peroneal cutaneous sensory distribution is to the cranial and lateral surfaces of the leg and the dorsal surface of the foot. The saphenous nerve, branch of the femoral nerve, has an autonomous zone on the medial side of the crus and metatarsus.[5]

These heifers presented with clinical signs attributable to tibial and sacro-caudal nerve damage. The over-flexion of the hocks and dorsal buckling of the fetlocks is typical for tibial nerve damage. A more thorough examination could be obtained in the heifer of case 2 when she stood. She demonstrated weak anal sphincter tone and tail tone, suggesting sacro-caudal nerve damage. Minor cutaneous sensation deficits in the autonomous zones for the peroneal and tibial nerves were also observed.

The diskospondylitis and extension of inflammation to surrounding neuronal tissues clearly correlates with the clinical presentation of these heifers. The neurologic deficits seen with diskospondylitis result from dorsal expansion of the inflammation into the vertebral canal and compression of the spinal cord. The cerebrospinal fluid is usually normal if the infection and compression are extradural.[5,14] It is possible that, due to positioning of the recumbent heifer in case 1 and awkward insertion of the needle, extradural suppurative material associated with the diskospondylitis was aspirated instead of the CSF. In case 1, the Gram stain of the CSF revealed gram-positive bacteria, whereas a gram-negative organism, *E. coli*, was cultured. This suggests a mixed bacterial infection. Unfortunately, aerobic and anaerobic cultures of the purulent material were not performed on the post-mortem examination to confirm these suspicions. The most common infectious agents isolated from spinal abscesses of ruminants include *Corynebacterium pseudotuberculosis, Arcanobacter pyogenes* (formerly *Actinomyces pyogenes*), *Pasteurella haemolytica, Staphylococcus aureus* and *Fusobacterium necrophorum*.[8]

Although the effects of the diskospondylitis lesion are clear, the etiopathogenesis of this lesion is not. The presentation of two heifers from the same farm, simultaneously developing diskospondylitis at the level of L6-S1, questions the possible pathogenesis. Potential routes of infection include hematogenous spread; migrating foreign bodies; previous trauma to the intervertebral disc, vertebral body or endplate; immunosuppression; and direct extension of paravertebral infections. Hematogenous spread of organisms is thought to be the most common pathogenesis in dogs.[13,18]

Hematogenously derived abscesses in the vertebrae can arise because of embolization of septic thrombi into the metaphyseal arteries. The blood flow through these vessels is sluggish because of the circuitous route the vessels assume in their approach to the physis. The metaphyseal vessels communicate with the ventral vertebral plexus, which in turn drains into the vena cava and the azygous vein. The ventral vertebral plexus lacks valves, thus blood flow reverses with an increase in abdominal or pleural pressure as occurs during calving. Regurgitated blood from infected sites in the body cavities may shower the vertebrae and spinal cord with bacteria.[8] This would suggest that tail docking prior to parturition is not ideal.

Conclusion

These heifers lacked evidence of significant infection in any other site within the body other than L6-S1 diskospondylitis. It was uncertain if the diskospondylitis was secondary to an ascending infection from the tail-docking site in caudal vertebrae. However, it is unlikely that internal organs were the source of the bacteria needed to set up a hematogenously derived abscess, and consequently the diskospondylitis lesion. Multiple heifers on this farm have had their tails docked at roughly the same age, and in the same manner, without any reported ill effects until the presentation of these two heifers. The infection was located at L6-S1 in both cases, however the reason for this is unknown. A possible hypothesis is that there is subclinical injury or repeated mechanical stress at this site, for example, with mounting behaviors, and use of restraint, such as the tail jack. That would promote localized inflammation, subsequently altering microvasculature and blood flow, and

creating a more favorable condition for bacterial colonization. The final post-mortem diagnosis was suppurative diskospondylitis localized at L6-S1 with no significant evidence of infection anywhere else in the body. These findings give rise to the conclusion that the diskospondylitis lesion may be secondary to ascending infection from the tail-docking site.

References

1. Adams SB, Steckel R, Blevins W: Diskospondylitis in five horses. *J Am Vet Med Assoc* 186(3):270-272, 1985.
2. Chaffin MK, Honnas CM, Crabill MR, Schneiter HL, Brumbaugh GW, Briner RP: Cauda equina syndrome, diskospondylitis, and a paravertebral abscess caused by *Rhodococcus equi* in a foal. *J Am Vet Med Assoc* 206(2):215-220, 1995.
3. Ciszewski DK, Ames N: Diseases of the peripheral nerves. *Vet Clin North Am Food Animal Pract* 3(1):193-212, 1987.
4. Colbourne CM, Raidal SL, Yovich JV, Howell JMcC, Richardson JL: *Aus Vet J* 75(7):477-479, 1997.
5. de Lahunta A: *Veterinary Neuroanatomy and Clinical Neurology,* (ed 2). Philadelphia, WB Saunders Co, 1983, pp 61-63, 70-74.
6. de Lahunta A, Habel RE: *Applied Veterinary Anatomy*. Philadelphia, WB Saunders Co, 1986, p 221.
7. Furr MO, Anver M, Wise M: Intervertebral disk prolapse and diskospondylitis in a horse. *J Am Vet Med Assoc* 198(12):2095-2096, 1991.
8. George LW: Spinal Abscesses, in Smith BP, (ed): *Large Animal Internal Medicine,* ed 3. St. Louis, Mosby, 2002, pp 982-984.
9. Hillyer MH, Innes JF, Patteson MW, Barr ARS: Discospondylitis in an adult horse. *Vet Rec* 23:519-521, 1996.
10. Hurov L, Troy G, Turnwald G: Diskospondylitis in the dog: 27 cases. *J Am Vet Med Assoc* 173(3):275-281, 1978.
11. Kornegay JN, Barber DL: Diskospondylitis in dogs. *J Am Vet Med Assoc* 177(4):337-341, 1980.
12. Malik R, Latter M, Love DN: Bacterial diskospondylitis in a cat. *J Small Animal Pract* 31:404-406, 1990.
13. Moore, MP: Diskospondylitis. *Vet Clin North Am Small Animal Pract* 22:1027-1034, 1992.
14. Rebhun WG, de Lahunta A, Baum KH, King J, Roth L: Compressive neoplasms affecting the bovine spinal cord. *Compend Contin Educ Pract Vet* 5 (Suppl) 5:396-400, 1984.
15. Scott PR, Penny CD, Murray LD: A field study of eight ovine vertebral body abscess cases. *NZ Vet J* 39:105-107, 1991.
16. Sherman DM, Trevor RA: Vertebral body abscess in cattle: A review of five cases. *J Am Vet Med Assoc* 188(6):608-611, 1986.
17. Turnwald GH, Shires PK, Turk MAM, Cox HU, Pechman RD, Kearney MT, Hugh-Jones ME, Balsamo GA, Helouin CM: Diskospondylitis in a kennel of dogs: clinicopathologic findings. *J Am Vet Med Assoc* 188:178-183, 1986.
18. William BT: Diskospondylitis and other vertebral infections. *Vet Clin North Am Small Anim Pract* 30(1):169-181, 2000.