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Paraparesis in an Adult Alpaca with Discospondylitis

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A 5-year-old domestic huacaya alpaca mare, with a body mass of 40 kg, was admitted to the Clinic for Ruminants, Vetsuisse-Faculty of Berne, Switzerland, with a history of acute onset of paresis in the pelvic limbs for one day without observed trauma. Previous treatments from the referring veterinarian included anthelmintics (ivermectin[a] 0.2 mg/kg of body weight) and antibiotics (ceftiofur[b] 1.0 mg/kg of body weight), resulting in no improvement in her clinical condition.

At examination, the alpaca was recumbent, but rectal temperature (37.8°C [100.04°F]; reference interval, 37.5–38.9°C [99.5–102.02°F]), heart rate (64 beats per minute [bpm]; reference interval, 60–90 bpm), and respiratory rate (24 breaths per minute; reference interval, 10–30 breaths per minute) were within the reference range.[1] The alpaca was in thin body condition. Examination of the cardiovascular, respiratory, digestive, and urinary systems did not reveal abnormalities.

A detailed neurologic examination was performed.[2] The animal was laying in sternal recumbency and was not able to stand up. When the alpaca was lifted from the floor, she showed a wide-based stance and slight stiffness in the thoracic limbs but was unable to support weight on the pelvic limbs. The wheelbarrowing test was slightly abnormal. This abnormality was induced because of perceived pain in the thoracic region and caused stiffness of the forelimbs while the animal was lifted up during the examination. Flexor reflex and extensor carpi radialis reflex were normal in both forelimbs. Proprioception was absent in the pelvic limbs, and a slightly increased patellar reflex was observed on both sides. Flexor reflex on pelvic limbs and perineal reflex were normal. Examination of the cranial nerves was normal. Generalized muscle atrophy was evident on palpation. The animal reacted with signs of severe pain to palpation of the thoracic vertebrae between Th5 and Th10.

Clinicopathologic tests included a complete blood cell count, blood chemistry panel, blood gas analysis, urinalysis, cytology of the cerebrospinal fluid (CSF), and parasitologic examination of the feces. The complete blood cell count revealed a leukocytosis due to mature neutrophilia compatible with stress and/or chronic inflammation of mild to moderate severity (27 $\times 10^9$/L; reference interval, 8–16 $\times 10^9$/L), or both.[3] The blood chemistry panel revealed increased concentrations of fibrinogen (6 g/L; reference interval, 1–5 g/L),[1] which were attributed to acute infection or tissue inflammation/damage. Blood gas values were within normal limits.[1] A urine sample was obtained by catheterization of the bladder, and no abnormality was detected at urinalysis. The CSF obtained aseptically by lumbar tap was clear,[1,2] the semiquantitative Pandy reaction for globulins was slightly positive, albumin was high with 100 mg/dL (reference interval, 17.9 ± 4.45 mg/dL),[4] and normal leukocyte counts (1 cell/μL; reference interval, 0.88 ± 1.11 cells/μL)[4] were present. Parasitologic examination revealed strongyle and *Dicrocoelium dentriticum* eggs in the fecal sample.

The clinical neurologic examination suggested an upper motor neuron lesion located cranial to the segments innervating the pelvic limbs; the observed palpation pain further suggested a lesion between Th5 and Th10. Laboratory workup indicated the presence of an inflammatory process in the central nervous system. Based on these results, an inflammatory process in the spinal cord (e.g., meningomyelitis, abscess in the spinal canal, discospondylitis, or larva migrans), intervertebral disc protrusion, neoplastic process, or spinal shock were considered as differential diagnoses.[5] A spinal cord trauma could not be excluded.

Because of the animal's ongoing and rapid deterioration, the severity of neurologic deficits, and arising expenses for further diagnostic examinations such as radiographs, magnetic resonance imaging (MRI) of the spine, and ultrasound of the muscles, the owner requested euthanasia of the alpaca.

MRI of the thoracic and lumbar spine was performed immediately after the alpaca was euthanized (total examination time, 2 hours). Therefore, no special postmortem changes in signal intensity, except for missing intravascular flow, were expected. Sequences included a sagittal and transverse T2, a dorsal, high-resolution T1-weighting, and a dorsal STIR (short tau inversion recovery, fat suppression) of the thoracic spine (Table 1). The vertebral bodies of Th9 and Th10 showed slightly increased signal intensities (T2, STIR) adjacent to the intervertebral discs. The body of Th10 was shortened compared with Th9 and Th11 (Fig 1). The intervertebral disc between Th9 and Th10 showed an irregular shape and a low signal intensity in all sequences. The disc protruded dorsally and occluded about two-thirds of the vertebral canal. The disc material was surrounded by iso- to hyperintense material in T2 (Fig 1). A rounded structure of about 6 cm in diameter, which was hyperintense in all sequences (Fig 2), surrounded the intervertebral disc. The adjacent tissue showed a diffuse high signal of decreasing intensity toward the periphery in the STIR. The spinal cord was severely deformed and deviated

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Table 1. Sequences.

Sequence	TR/TE/(TI) (ms)	FA	FOV (mm)	Matrix	SlTh/SlSep
FSE T2 sagittal	2500/125	90	350 × 350	288 × 300	4/0.5
FSE T2 transverse	3540/100	90	230 × 230	160 × 160	5/0.5
FE 3D MPR dorsal	30/12	30	330 × 330	220 × 140	1.3/0
STIR dorsal	4000/25/(110)	90	350 × 350	256 × 152	5/0.5

TR, repetition time; TE, echo time; TI, inversion time; FA, flip angle; FOV, field of view; SlTh, slice thickness; SlSep, slice separation; FSE, fast-spin echo; FE, field echo; 3D, 3-dimensional; MPR, multiplanar reconstruction; STIR, short tau inversion recovery.

dorsally because of the protruded intervertebral disc and the surrounding material. The intramedullary signal was increased (T2, STIR) for about 2 segments caudally of the lesion (Fig 1). In the lumbar spine, a sagittal and transverse T2-weighting, dorsal STIR, and transverse T1-weighting were performed. A caudally diverging and diffusely increased signal intensity was observed in the left dorsal paravertebral musculature between L4 and tuber coxae. However, intervertebral discs and spinal cord were unremarkable in the lumbar spine.

The increased signal intensity of the vertebral bodies of Th9 and Th10 was consistent with bone marrow edema due to inflammatory reactions. High signal intensity changes of the bone marrow in STIR or T2 can be due to inflammatory edema, cellular infiltration, or bone marrow conversion.[6] Together with the deformed and protruded disc, as well as the reaction of the adjacent musculature, the tentative diagnosis based on MRI findings was spondylitis and discospondylitis of Th9 and Th10 with disc protrusion, associated hematoma, and severe spinal cord compression. The rounded inhomogenous structure surrounding the intervertebral disc space Th9 and Th10 was consistent with chronic inflammatory tissue or an abscess with a small amount of free fluid or inspissated debris. Caudally, spinal cord edema was visible adjacent to the compression. The diffuse increased signal intensity of the lumbar musculature was interpreted as lesions or diffuse myositis possibly provoked by biomechanical stress on the muscles due to pain in the thoracal region causing unphysiologic strain on the lumbar muscles.

Postmortem examination was performed immediately after the MRI procedure. A severe suppurative, chronic discospondylitis between Th9 and Th10 with abscess formation in the ventral intercostal subserosal space and massive compression of the spinal cord were the predominant macroscopic findings (Fig 3). Severe edema and

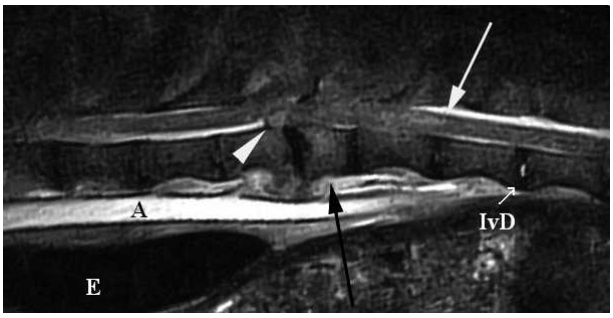

Fig 1. Fast-spin echo T2 sequence sagittal. Midsagittal slice through the caudal thoracic spine. Cranial is left, and dorsal is on top of the image. Th10 (black arrow) is shortened, and both Th9 and Th10 reveal slightly increased signal intensities. The spinal cord is deviated dorsally by inhomogenous material (white arrow head) consistent with hematoma and shows increased signal intensity until the caudal end of Th10 (white arrow). A, aorta; E, esophagus; IvD, intervertebral disc Th12/13.

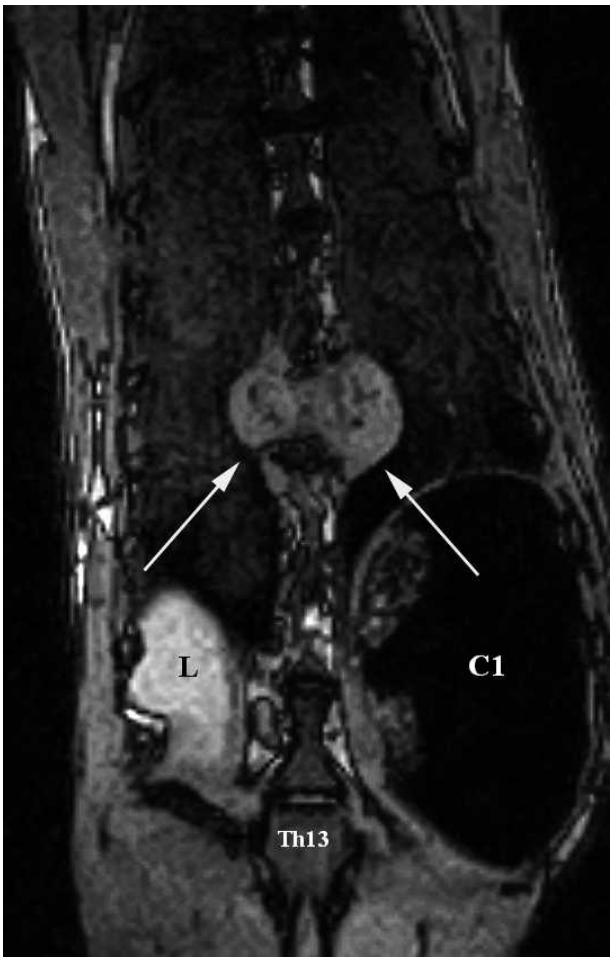

Fig 2. Field echo sequence dorsal. Dorsal plane image at a level slightly ventral to the vertebral bodies. Cranial is on top, and the right of the animal is on the left side of the image. There is a well-delineated, rounded structure with high signal intensity (white arrows) consisting of reactive tissue with abscess formation. L, liver; Th13, thoracic vertebral body 13; C1, lumen of the first compartment.

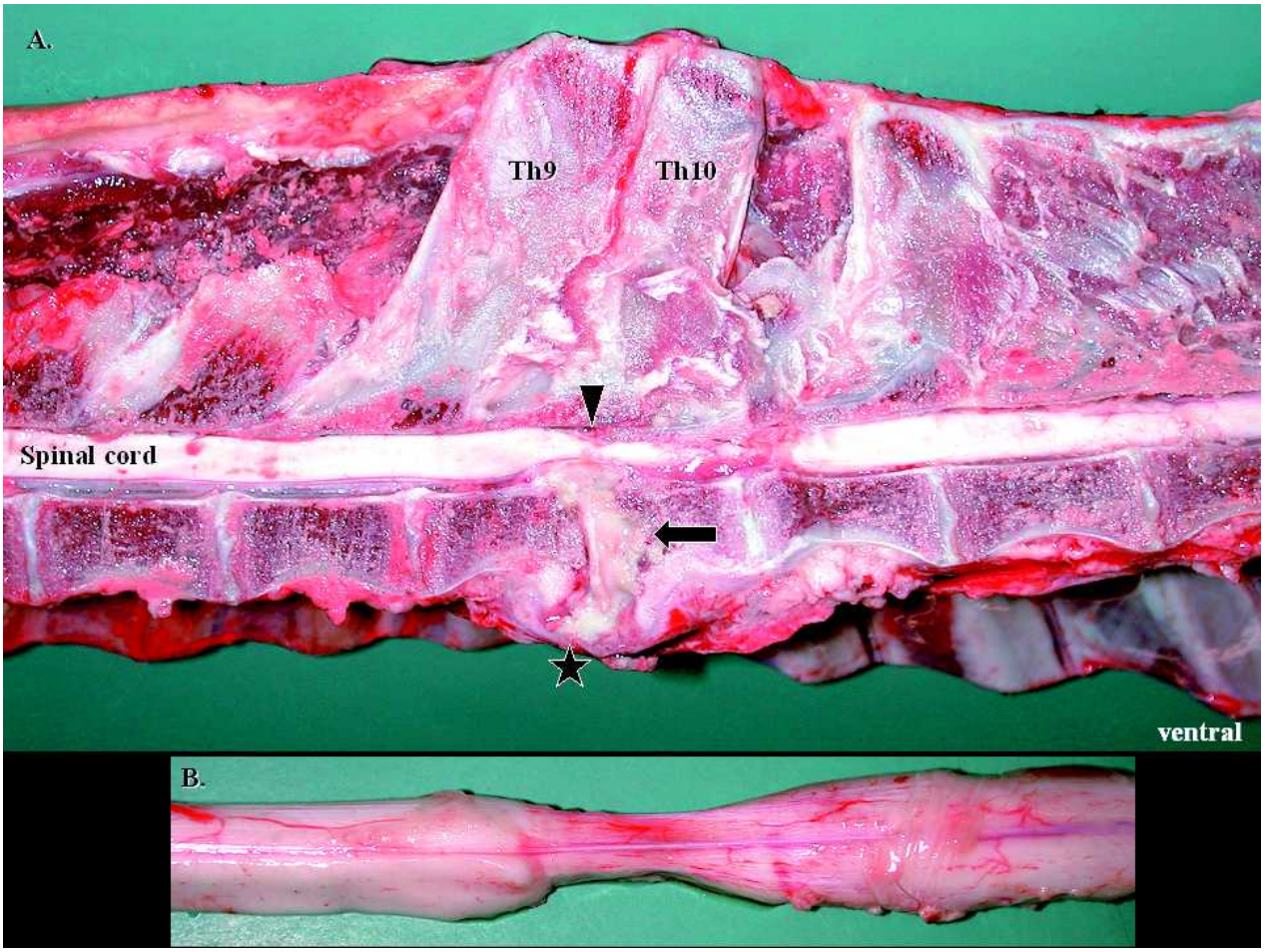

Fig. 3. **(A)** Suppurative discospondylitis (arrow) between the thoracic vertebral bodies Th9 and Th10, with ventral and dorsal herniation/expansion. Abscess formation in the intercostal, subserosal space (star). Note the severe compression of the spinal cord due to the protrusion into the vertebral canal (arrow head). **(B)** Segmental compression of the spinal cord at the level of the narrowing of the vertebral canal.

contusion of the right lumbar musculature was present. Histologically, degenerative lesions were seen in all funiculi (ascending and descending tracts) of the spinal cord that were characterized by dilated axons forming spheroids and ballooned myelin sheaths in the white matter (Wallerian degeneration). These changes were associated with hemorrhage, few myelophages, and chromatolysis in the grey matter. The severity of the Wallerian degeneration decreased cranially and caudally to the injury.

Discospondylitis, caused by a bacterial infection or occasionally, by trauma of the spinal vertebrae and the adjacent intervertebral discs, is a rare but life-threatening condition, which has been reported in large[7–9] and small animals.[10–16] Discospondylitis can be caused by local bacterial infection after hematogenous spread of organisms from infected sites elsewhere in the body, e.g., after direct injury to a vertebral endplate or intervertebral disc causing vascular disruption and increased susceptibility to infection. The infectious process starts either in the vertebral body or in the intervertebral disc, with subsequent spreading to the adjacent tissue.[12] Frequently, the inflammatory reaction extends dorsally into the spinal canal causing compression of the spinal cord, or ventrally or laterally, leading to the development of paravertebral abscesses.[17] The Wallerian degeneration of the white matter and the chromatolysis of the grey matter observed in the present case is a change typically after spinal cord compression.[18] The neurologic signs observed at clinical examination were in accordance with the histologic changes.

Clinical signs of discospondylitis are often nonspecific and may include inappetence, weight loss, depression, fever, and reluctance to move. In early stages, many animals have either no neurologic deficits at all or only mild disturbances, such as proprioceptive deficits or mild paresis.[19] Infection is often caused by coagulase-positive staphylococci, streptococci, or *Brucella* spp., likely as a result of bacteremia in small animals.[12,17,19–22] Less frequently, fungal infections, injury by migrating foreign bodies, or as described by Remedios et al,[23] bacterial contamination after unsterile administration of lumbosacral epidural analgesia[24] can cause discospondylitis. No specific causes have been described for new world camelids.

Spinal radiography of the involved region, revealing, eg., disc space collapse, bone lysis, and sclerosis in the

vertebral endplate, is usually diagnostic.[19] Computed tomography or MRI can be performed to evaluate patients with only subtle radiographic changes in the spine.[12]

Cases of vertebral infections, if diagnosed early, can reportedly be treated successfully with appropriate medication in humans and small animals.[16] Medical treatment of discospondylitis can be guided by culture and antibiotic sensitivity testing. Samples for bacterial culture may be obtained from the affected disc spaces by fluoroscopically guided needle or surgical aspiration.[12,15,24] Because the isolated organism in small animals is usually a *Staphylococcus* spp., cephalosporins or beta-lactamase–resistant penicillins are often effective. Antibiotic therapy is typically required for several months to eliminate the infection, and relapses are common despite long-term treatment.[9,16,19,21] In the present case, microbial culture of abscess material collected at postmortem examination was sterile, and no bacteria were seen in the affected tissues at histologic examination, possibly because of previous treatment with antibiotics. No growth of *Brucella* was detected, and serologic testing for *Brucella abortus* (enzyme-linked immunoabsorbent assay and complement-binding reaction) and immunohistochemical examination of the abscess capsule were negative.

Surgical intervention in small animals with discospondylitis is required when no response to initial medical therapy occurs, if evidence of spinal cord compression is present, or if the animal shows severe and progressive neurologic deficits.[15,20] Lesions are curetted, and necrotic bone and disc material is harvested for bacterial and fungal cultures. Surgical stabilization of the vertebrae may be necessary after decompression.[12,21]

To our knowledge, discospondylitis has not been reported in new world camelids to date. Nevertheless, discospondylitis should be considered as differential diagnosis in cases of paraparesis. The described clinical signs of paraparesis were very advanced because of severe compression of the spinal cord between Th9 and Th10; therefore, no treatment could be considered for this case.

The use of MRI techniques for diagnostic purposes is still not readily available for large animals. High investment, maintenance, and running costs, as well as the costs for the animal's owner, limit the routine performance of MRI (US$600–700 per examination at our institution). Furthermore, MRI examination requires general anaesthesia and can carry a high risk in neurologically impaired animals.

Nevertheless, the present case confirms that MRI is the diagnostic procedure of choice for the evaluation of the full extent of spinal lesions in new world camelids. This procedure can allow an exact intra vitam diagnosis and gives valuable information for treatment and prognosis.

Footnotes

[a] IVOMEC (ivermectin), Biokema, Crissier-Lausanne, Switzerland

[b] Excenel RTU (ceftiofur hydrochloride), Pfizer AG, Zurich, Switzerland

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