

RADIOGRAPHIC DIAGNOSIS: INTRAMEDULLARY EXTRUSION OF AN INTERVERTEBRAL DISC

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Signalment

An 11-year-old, neutered crossbred terrier.

History

The terrier presented with a 12-h history of acute asymmetric paraparesis. The dog was ambulatory but had marked pelvic limb ataxia, bilateral proprioceptive deficits in the pelvic limbs (worse on the right side), increased pelvic limb myotatic reflexes, deep pain sensation, and loss of cutaneous trunci reflex at L2-L3. Cranial nerve examination was unremarkable. Spinal hyperesthesia was not detected. A right-sided thoracolumbar (T3-L3) lesion was suspected. The differential diagnoses included intervertebral disc disease, fibrocartilagenous embolism, neoplasia, and trauma.

The dog was managed conservatively with strict cage rest. However, after 7 days there was no improvement in neurological signs, and spinal radiography and myelography were recommended.

Radiographic Findings

Spondylosis deformans was evident on survey radiographs and involved many vertebral elements, principally the caudal cervical, mid-thoracic (T5-T11), and lumbar (L1-L7) vertebrae. There was significant reduction in the width of the intervertebral space at C6-C7 with concurrent ankylosing spondylopathy. At the thoracolumbar junction, disc spaces T12-13 and T13-L1 were narrower than those at T10-T11 and T11-T12.

A myelogram was performed using iohexol (4 ml; 240 mg/ml iodine) injected into the subarachnoid space at L5-L6. The contrast medium extended from S2 cranially to C1. Mild attenuation of the contrast medium column was evident at C6-C7, T11-T12, T12-T13, and L2-L3. Extradural attenuation of the subarachnoid space was observed at these intervertebral spaces, signifying the presence of either soft

tissue mass or disc prolapse. A more dramatic lesion was evident at T13-L1, with a billing defect within the contrast medium in the ventral subarachnoid space and splitting of the contrast medium ("golf tee") both cranially and caudally. On oblique projections, the right ventrolateral margins of the spinal cord were displaced by an intradural-extramedullary mass (Fig. 1). Furthermore, intramedullary swelling of the spinal cord was noted adjacent to the intradural mass. The intradural and intraparenchymal masses, considered together, affected approximately a 7 mm × 3 mm segment of spinal cord.

The radiographic interpretation was a right-sided intradural-extramedullary mass at T13-L1 with secondary swelling of the cord. Neoplasia was suspected, although the acute onset of neurological signs and location of the myelographic lesion over a disc space was compatible with an intervertebral disc extrusion that had penetrated the dura mater.

Surgery

An exploratory hemilaminectomy was performed on the right side at T13-L1. The ventral aspect of the vertebral canal was examined by probing and gentle flushing with isotonic saline. Intervertebral disc material was not detected. Following durotomy, the spinal cord was swollen with a purplish-discoloration. The dog was euthanatized at the request of the owners as a surgically correctable lesion was not detected. The T11-L3 spinal cord segment was removed.

Pathology

Grossly, there was moderate swelling with pitting and hemorrhage along the dorsal aspect of the spinal cord (Fig. 2A). A firm mass (3 mm × 6 mm), adherent to the dura, protruded 2 mm from the ventral aspect of the spinal cord (Fig. 2B). On transverse section, the mass extended dorsally and laterally, resulting in obliteration of the majority of the spinal cord parenchyma at this level. Histologically, the mass was composed of fibrocartilagenous material containing multiple areas of focal calcification (Fig. 3), consistent with the lesion originating from degenerate intervertebral disc material.

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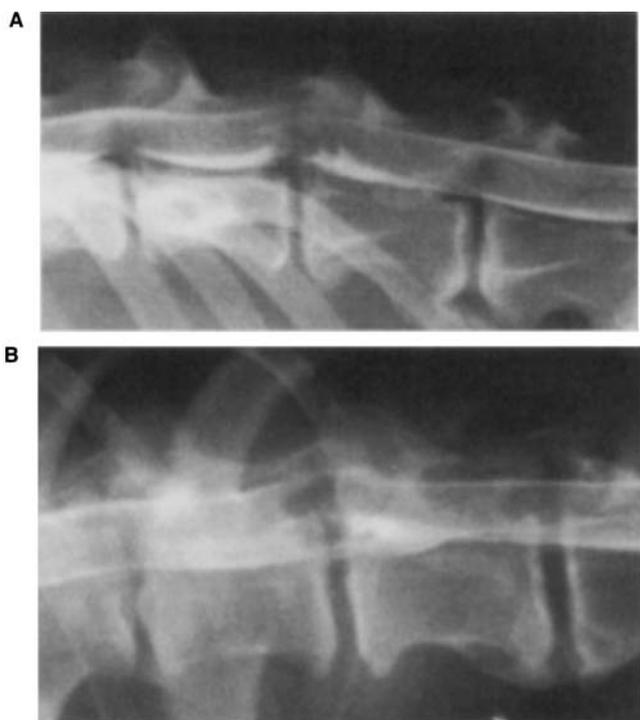


FIG. 1. (A) Lateral myelogram of the thoracolumbar junction. The ventral subarachnoid space is interrupted by an expansile lucent mass at T13-L1. A billing defect, with splitting of the contrast medium ("golf-tee"), at the cranial and caudal margins of the lucent mass are typical of an intradural-extramedullary lesion. The spinal cord is displaced dorsally over the intervertebral disc spaces at T12-T13 and T13-L1. There is mild attenuation of the ventral contrast medium column over both of these disc spaces, indicating concurrent extradural compression and displacement of the spinal cord. (B) Right oblique myelogram of the thoracolumbar junction. The ventrolateral subarachnoid space is dilated and contains a spherical lucent mass. The spinal cord is displaced dorsolaterally.

Discussion

Extrusion of disc material through the dura and into the spinal cord is a rare manifestation of intervertebral disc disease.^{1,8} The severity of neurological signs in dogs with intervertebral disc disease depends on the nature of disc degeneration, spinal cord-to-vertebral canal diameter, velocity of disc extrusion, and volume of the compressive mass.⁸ In the current case, neurological abnormalities were remarkably mild, considering the velocity presumably required for extruded disc material to penetrate the dura and spinal cord, and the volume of disc material within the spinal cord parenchyma. Spinal hyperesthesia was not present as there was no focal mass compressing and irritating the meninges.⁸ Importantly, the radiographic and myelographic features typically associated with intervertebral disc disease were absent.

There was no evidence of reduced intervertebral foraminal size, increased opacity within the intervertebral foramen, mineralized disc material in the vertebral canal, or significant narrowing of the T13-L1 disc space on survey

radiographs.⁹ The only myelographic feature consistent with intervertebral disc disease was that the lesion was centered over an intervertebral disc space. Typically, intervertebral disc disease results in extradural compression of the spinal cord.⁹ An intradural-intramedullary pattern can also be observed in acute intervertebral disc disease due to spinal cord swelling.⁹ However, in the current case, myelographic findings included an intradural-extramedullary lesion with swelling of the adjacent spinal cord at T13-L1. Neoplasia, such as meningioma or nerve root tumor,⁵ is the most common cause of an intradural-extramedullary lesion.⁹ Advanced imaging, using either magnetic resonance or computed tomography, may have been beneficial in the diagnosis of intervertebral disc disease in this dog.^{2,4,6}

Extrusion of intervertebral disc material has been proposed as a cause of dural laceration in dogs.^{3,7} Laceration of the dura mater was evident in the current case, although extradural leakage of contrast material was not seen on the myelogram, presumably because the dural tear was chronic and had healed to the extruded intervertebral disc material.

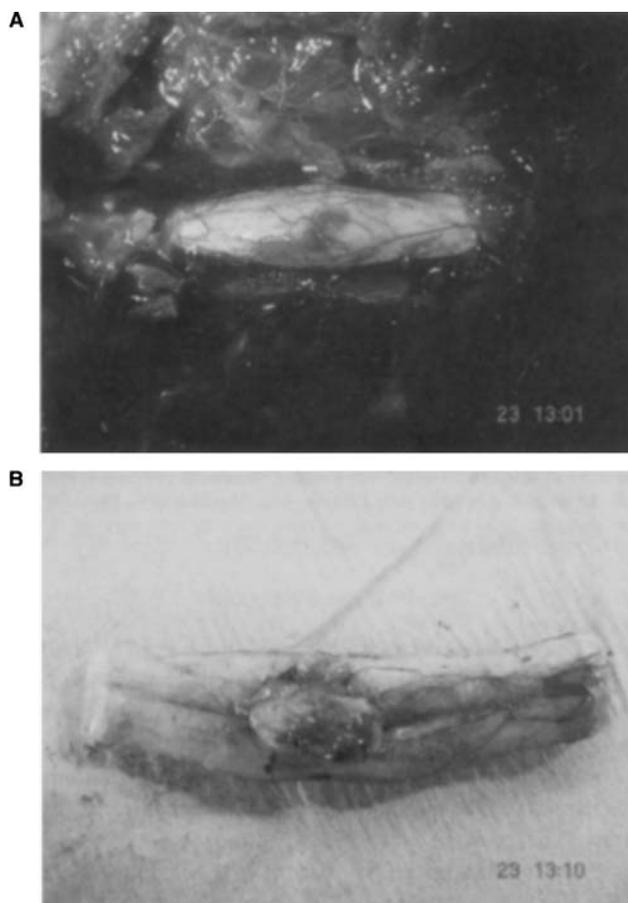


FIG. 2. (A) Postmortem photograph of the dorsal aspect of the spinal cord. There is swelling and parenchymal hemorrhage. (B) Postmortem photograph of the ventral aspect of the sectioned spinal cord following removal of the dura mater. There is a mass penetrating the ventral median fissure.

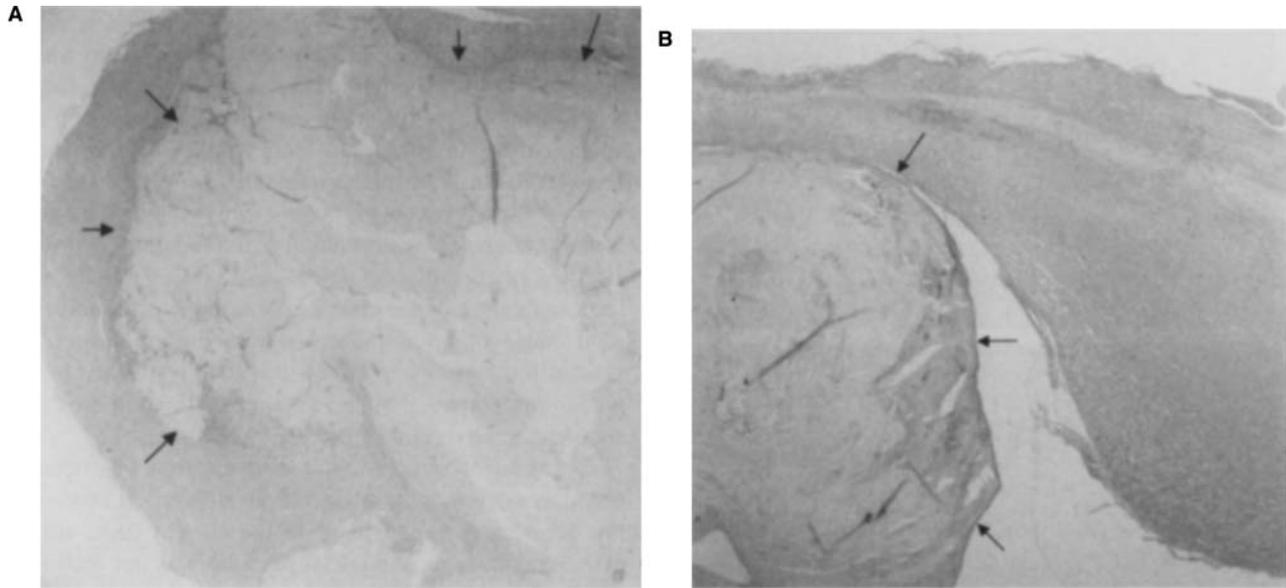


FIG. 3. (A) Transverse section of the spinal cord. (B) Longitudinal section of the spinal cord. The fibrocartilaginous material is indicated by the arrows. In the transverse section, the fibrocartilaginous material occupies the majority of the spinal cord. In the longitudinal section, there is little normal spinal cord dorsal to the mass.

Due to the mild neurological signs, decompressive surgery and appropriate postoperative management may have been successful if further treatment was permitted.

Extrusion of disc material into the spinal cord paren-

chyma is rare. Nonetheless, in patients with an acute onset of neurological signs, intervertebral disc disease should be considered when an intradural myelographic lesion or mixed pattern is centered over an intervertebral disc space.

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