What Is Your Diagnosis?

In cooperation with

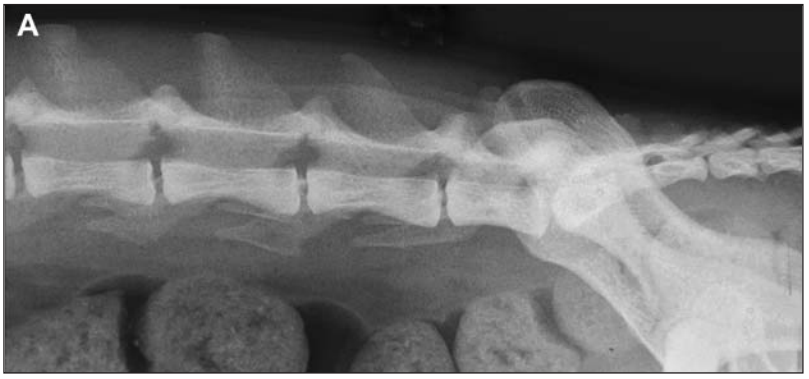

Figure 1—Right lateral (A) and ventrodorsal (B) radiographic views of the lumbar vertebrae and pelvis of a 3-year-old neutered male Burmese cat that was referred with a 3-month history of nonspecific hind limb gait abnormalities. The cranial aspect of the ventrodorsal image has been cropped so that the center of the image does not reflect the center of the x-ray beam.

History

A 3-year-old neutered male Burmese cat was referred with a 3-month history of nonspecific hind limb gait abnormalities. Seven days prior to referral, the cat had acutely developed reluctance to jump and a right-sided plantigrade stance. Clinical examination confirmed the plantigrade stance and moderate right hind limb muscle atrophy, predominantly in the gastrocnemius muscle. There were no signs of pain on limb or vertebral column manipulation, and results of an orthopedic and neurologic examination were otherwise unremarkable. No abnormalities were detected on CBC or serum biochemical analysis; serum electrolyte concentrations were within reference limits. The cat underwent general anesthesia, and electromyography of the right hind limb musculature was performed; no abnormalities were found on electromyographic evaluation. While the cat was under general anesthesia, radiographs of the lumbar vertebrae and pelvis were obtained (**Figure 1**).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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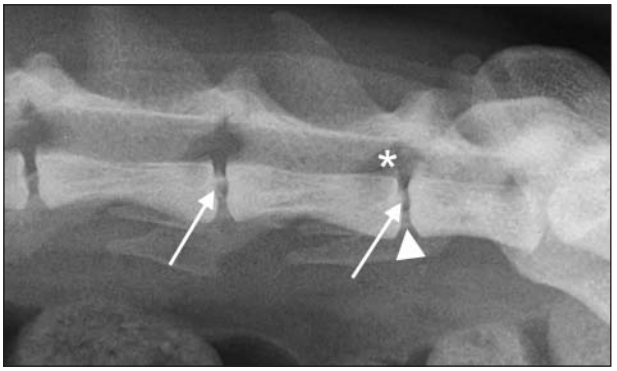

Figure 2—Same lateral radiographic image (magnified) as in Figure 1. Notice mineralization of the L5-6 and L6-7 disks (arrows). Additionally, narrowing of the L6-7 space (arrowhead) and mineral opacity at the L6-L7 neural foramen (asterisk) are evident.

Diagnostic Imaging Findings and Interpretation

On the lateral image, there is mineralization of the L5-6 and L6-7 disks and narrowing of the L6-7 space (**Figure 2**; magnified image). There is a mineral opacity at the L6-L7 neural foramen that could be material within the foramen or within the vertebral canal. Location of the mineralization cannot be confirmed on the ventrodorsal image. The cropped nature of the ventrodorsal view makes parallax error possible in the evaluation of the caudalmost disk spaces; thus, interpretation of disk space width is difficult from this image.

Magnetic resonance imaging of the lumbar portion of the vertebral column was performed. Images were obtained from sagittal T1-, T2-, and transverse T2-weighted sequences. The right parasagittal and transverse T2-weighted images demonstrated disk dehydration at L5-S1, evidenced by decreased T2-weighted signal intensity at each of the intervertebral spaces (**Figure 3**). A right-sided hypointense structure at the level of L6-7 with leftward displacement of the cauda equina and mild loss of the periradicular fat signal of the spinal nerve root was evident. These findings were consistent with lateralized Hansen type I disk extrusion.[1] Additionally, S2 and S3 were incompletely fused.

Comments

Dorsal laminectomy of L6 and L7, including right facetectomy, was performed. A large volume of degenerate mineralized nucleus pulposus was removed from the right ventrolateral aspect of the spinal canal at the level of the L6-7 space. At 6 months after surgery the referring veterinarian reported that the cat had recovered well and the plantigrade stance had improved greatly, although not completely.

Survey radiography of the vertebral column is generally not a sensitive enough imaging modality to identify potential spinal cord compression or structures within the vertebral canal unless the structure is mineralized, as was true for the cat of the present report. Although intervertebral disk disease is relatively common in dogs, its incidence in cats is much lower, although it has been recognized with increased frequency in recent years.[2] A substantial proportion of older cats without clinical signs of neurologic disease have incidental findings of Hansen type II disk protrusions on necropsy. However, Hansen type I lesions predominate over type II lesions in clinically affected cats.[3]

Although myelography can be performed in cats, identification of lumbar extradural compression on a myelogram can be difficult because the lesion may be more

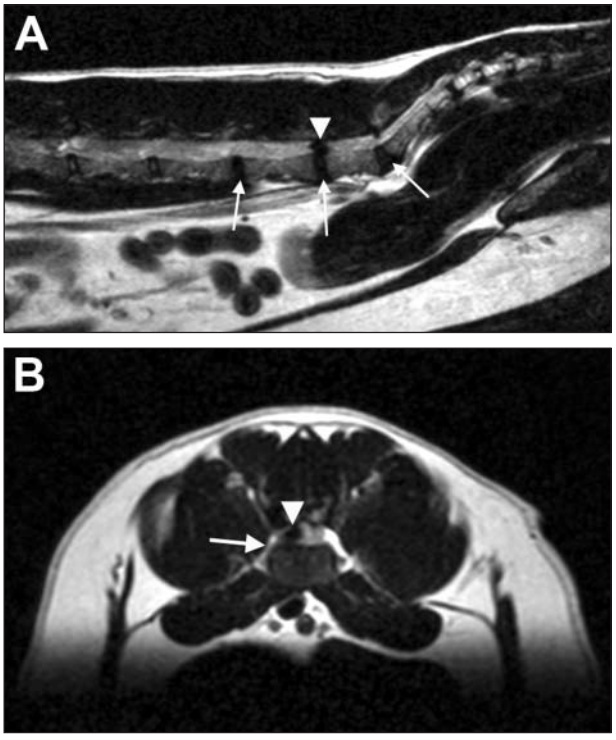

Figure 3—T2-weighted right parasagittal (A) and transverse (B) MRI images of the lumbar vertebrae of the same dog as in Figure 1. A—Notice the hypointense disk material within the vertebral canal (arrowhead) and reduced signal intensity at the intervertebral disk spaces (arrows). Also notice that S2 and S3 are incompletely fused. B—Image obtained at the level of the L6-7 space. Notice the right-sided hypointense mineralized disk material displacing the cauda equina to the left (arrowhead). Also notice the reduced periradicular fat signal on the right (arrow).

caudally located than the extent of the thecal sac. However, the sac does extend more caudally in cats than in dogs.[3] Epidurography may facilitate diagnosis, but this technique is difficult in cats because of the small size of the extradural space, making interpretation of images challenging.[4] Computed tomography can also be useful to identify extradural compression, but MRI was preferred for the cat of the present report because of the high sensitivity of MRI in the detection of spinal cord compressive disease and the ability to assess intraparenchymal spinal cord changes.

Mineralization of the L6-7 disk coupled with collapse of the disk space and dorsal migration of the mineralized material into the vertebral canal made extradural compression of the spinal nerves within the canal supplying the right L6-L7 nerve root the most likely cause of the unilateral plantigrade stance of the cat of the present report. Pathological changes were confirmed and spatially defined on MRI. It is also likely that the muscle atrophy was primarily neurogenic in origin because the muscles primarily affected (gastrocnemius) are innervated by the tibial nerve. Lateralized intervertebral disk extrusion should be considered as a rare but possible cause of isolated peripheral neuropathies in cats.

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3. Negrin A, Schatzberg S, Platt SR. The paralyzed cat. Neuroanatomic diagnosis and specific spinal cord diseases. *J Feline Med Surg* 2009;11:361–372.
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