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AMERICAN COLLEGE OF VETERINARY INTERNAL MEDICINE

What Is Your Neurologic Diagnosis?

A 7-year-old 34.1-kg (75-lb) castrated male pit bull–type dog was evaluated because of sudden-onset, rapidly progressive tetraparesis. Approximately 48 hours earlier, the owner noticed that the dog had left thoracic limb lameness. There was no change in the lameness until approximately 12 hours prior to evaluation when the dog suddenly became unable to stand or walk. The dog had been previously healthy. There was no coughing, sneezing, diarrhea, or increase in volume or frequency of urination. The dog had apparently normal appetite and thirst. The owner did not report any trauma. One year earlier, the dog had undergone surgical treatment of a ruptured right cranial cruciate ligament.

Neurologic examination

Observation

Mental	Alert	X	Depressed		Disoriented		Stupor		Coma		
Posture	Normal		Head tilt		Tremor		Falling	X			
Gait	Normal		Ataxia	X	Pelvic limbs		All 4	X	Circling		
Paresis	Pelvic limbs		Tetra	X	Hemi		Mono				
Other	Despite provision of support of the dog's weight, minimal voluntary movements were observed in the thoracic limbs, whereas the pelvic limbs appeared to move almost normally. The pelvic limb gait had mild general proprioceptive (GP) ataxia and upper motor neuron (UMN) weakness. There was increased muscular tone in the thoracic limbs.										

Postural reactions

Key: 4 = Exaggerated, clonus; 3 = Exaggerated; 2 = Normal; 1 = Diminished; 0 = None; NE = Not evaluated.

	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Wheelbarrow	NE	NE		
Hopping	0	0	2	2
Ext postural thrust			NE	NE
Proprioceptive pos	0	0	2	2
Hemistand/walk	NE	NE	NE	NE
Placing–tactile	NE	NE		
Placing–visual	NE	NE		

Spinal reflexes

	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Quadriceps			2	2
Extensor carpi	NE	NE		
Flexion	2	2	2	2
Crossed extensor	NE	NE	NE	NE
Perineal			2	2

Cranial nerves

	L	R		L	R	Comments
II, VII–Vision menace	2	2	VIII–Nystagmus, resting	0	0	
II, III–Pupils resting	2	2	VIII–Nystagmus, change	0	0	
Stim L	2	2	V–Sensation	2	2	
Stim R	2	2	VII–Facial mm	2	2	
II–Fundus	2	2	V, VII–Palpebral flex	2	2	
III, IV, VI–Strabismus, resting	2	2	IX, X–Gag	2	2	
III, IV, VI, VIII–Strabismus, position	2	2	XII–Tongue	2	2	

Sensation (Locate and describe abnormal)

Hyperesthesia	0	No specific areas of discomfort but reluctant to turn the head and neck to the left
Superficial pain	NE	
Cutaneous reflex	2	
Deep pain	NE	

What is the problem? Where is the lesion? What are the most probable causes of this problem? What is your plan to establish a diagnosis? Please turn the page.

Assessment

Anatomic diagnosis

Problem	Rule out location
Tetraparesis	Focal or diffuse lesion affecting the C1 to C5 spinal cord segments, focal or diffuse lesion affecting the C6 to T2 spinal cord segments, or diffuse (generalized) neuromuscular disorder (lower motor neuron disorder)
Severe paresis of the thoracic limbs with increased extensor tone and normal withdrawal reflexes	Focal or diffuse lesion affecting the C1 to C5 spinal cord segments or focal or diffuse lesion affecting the brainstem between the midbrain and medulla
Mild GP ataxia and UMN weakness of the pelvic limbs	Focal or diffuse lesion affecting the spinal cord cranial to the L4 spinal cord segment

Likely location of 1 lesion

Taken in isolation, the deficits observed in the thoracic limbs reflected UMN weakness characterized by normal withdrawal reflexes and increased muscular tone. Likewise, the deficits in the pelvic limbs also reflected UMN paresis, albeit with lesser severity; this was characterized as GP ataxia because the dog could take steps with support. Therefore, the most likely location of a single lesion that would result in GP and UMN deficits in both the thoracic and pelvic limbs and not affect mentation or the cranial nerves was a focal or diffuse lesion affecting the C1 to C5 spinal cord segments.

Etiologic diagnosis—The differential diagnoses included degenerative intervertebral disk herniation, compressive hydrated nucleus pulposus extrusion, meningomyelitis (infectious, noninfectious, or immune-mediated), hematomyelia (acquired coagulopathies [eg, immune-mediated thrombocytopenia, toxicosis, or myelophthisis] or congenital coagulopathies [eg, von Willebrand disease or clotting factor deficiency]), and neoplasia (given the rapidity of development of signs, hematopoietic neoplasia [lymphoma or histiocytic sarcoma] was considered more likely than a glial neoplasm, meningioma, or primary vertebral neoplasm). Given the progressive course, ischemic myelopathy (fibrocartilaginous embolic myelopathy) and acute noncompressive nucleus pulposus extrusions were considered less likely.

Various diagnostic tests were considered, including a CBC (to provide evidence of thrombocytopenia or systemic inflammation related to an infection or immune-mediated disease), biochemical analyses (to evaluate the function of organs, such as the liver [ie, coagulopathy secondary to liver disease] or provide evidence of neoplastic involvement outside of the CNS), and 3-view thoracic radiography (to assess for metastatic neoplasia or multicentric hematopoietic neoplasia). Ultimately, MRI of the cervical vertebral column from the caudal aspect of the skull to approximately the T3 vertebra was deemed necessary to characterize the involvement of the spinal cord and provide a presumptive diagnosis.

Diagnostic test findings—Owing to financial constraints, the owner could not pursue diagnostic testing. Instead, the dog was hospitalized and treated with prednisone (0.37 mg/kg [0.17 mg/lb], PO, q 12 h). The dog's clinical signs remained static for 48 hours, at which time the dog acutely became tetraplegic. On the basis of the decline in the dog's neurologic status, the owner elected euthanasia by IV injection of an overdose of pentobarbital. Following euthanasia, the owner consented to CT[a] of the cervical portion of the vertebral column and necropsy. Postmortem CT was performed with the dog in dorsal recumbency and revealed a left-sided, hyperattenuating, extradural lesion extending from the level of the C2-3 intervertebral disk space to the level of the C6 vertebra **(Figure 1)**. A region of interest (centered on the extradural lesion at the level of the C4-5 intervertebral disk space) had a mean attenuation of 139 HU, which was compatible with hemorrhage admixed with mineralized intervertebral disk material. The lesion resulted in compression of the spinal cord, which was most severe at the level of the C4-5 intervertebral disk space where it occupied approximately 50% of the cross-sectional area of the vertebral canal. At necropsy, extradural extruded intervertebral disk material was identified at the level of the C4-5 intervertebral disk space along with extradural hemorrhage that extended from the C2 vertebra through the C6 vertebra.

Comments

For the dog of the present report, the disproportionate severity in neurologic deficits between the thoracic and pelvic limbs was best explained by a focal or diffuse lesion affecting the UMN tracts that are positioned medially in the white matter immediately adjacent to the gray matter of the spinal cord. In the cross-sectional view of a spinal cord, the affected UMN tracts are located in the central aspect of the spinal cord, likely in the medial aspect of the white matter in the lateral funiculi. Hence the clinical syndrome of disproportionately affected thoracic limbs, compared with findings for the pelvic limbs, has been termed central cord syndrome.[1] Alternatively, a ventral midline extradural lesion that primarily impacts the gray matter and the medial axons of the white matter tracts of the lateral funiculi would be

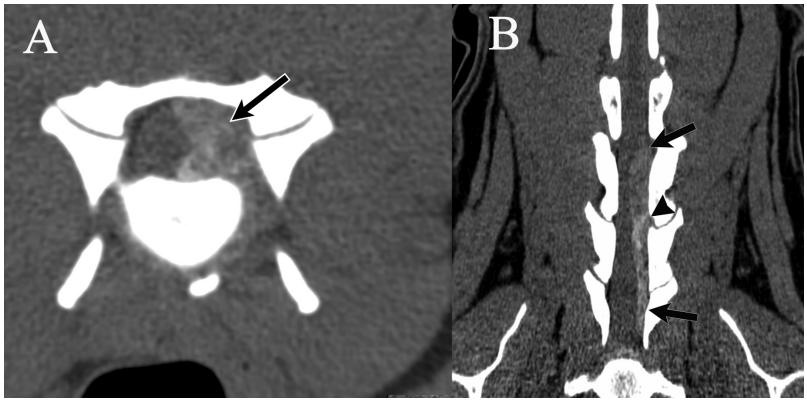

Figure 1—Computed tomographic images of the cervical portion of the vertebral column of a 7-year-old pit bull–type dog that was evaluated because of sudden-onset, rapidly progressive tetraparesis. Notice the left-sided, hyperattenuating extradural lesion causing compression of the spinal cord. A—Transverse plane CT image. The extradural lesion fills approximately 50% of the cross-sectional area of the vertebral canal at the C4-5 articulation (arrow). B—Dorsal plane reconstruction. The extradural lesion extends from approximately the C2-3 articulation (top arrow) to the midbody of the C6 vertebra (bottom arrow). At the C4-5 articulation, the extradural lesion has greater attenuation, consistent with hemorrhage admixed with mineralized intervertebral disk material (arrowhead).

anticipated to cause a similar greater thoracic limb effect.[2]

The classic description of clinical signs in animals with a focal or diffuse lesion involving the C1 to C5 spinal cord segments include GP ataxia and UMN paresis that affect the thoracic and pelvic limbs equally.[2] Yet, a common finding in dogs with cervical spondylomyelopathy is the observation of a greater degree of GP ataxia and UMN paresis in the pelvic limbs, compared with that observed in the thoracic limbs.[3] One explanation contends that the extradural compression associated with cervical spondylomyelopathy primarily affects the superficial spinal cord tracts in the dorsal and dorsolateral white matter, which contain the cranially coursing GP fibers from the pelvic limbs and relatively spares the more medially located GP fibers coursing cranially from the thoracic limbs.[2] In contrast, the dog of the present report typified a less commonly encountered clinical case of a focal or diffuse lesion affecting the C1 to C5 spinal cord segments in which there was greater GP ataxia and UMN paresis in the thoracic limbs than in the pelvic limbs. Historically, the explanation for this has posited that the lesion must affect spinal cord tracts that are located medially (closer to the gray matter) in the spinal cord, thereby sparing the superficial spinal cord tracts conducting GP information coursing cranially from the pelvic limbs and primarily affecting the GP and UMN tracts involving the thoracic limbs. The converse to that explanation could account for the clinical signs associated with cervical spondylomyelopathy, in which the ataxia and weakness is greater in the pelvic limbs than in the thoracic limbs.

In humans, the clinical signs of a disproportionate degree of paresis of the upper limbs and variable sensory loss, compared with a limited involvement of the lower limbs, has been termed traumatic (acute) central cord syndrome (also known as man in a barrel syndrome).[4] Traumatic (acute) central cord syndrome often results from a hyperextension injury of the neck in which anterior-posterior compression results in great deformation of the central aspect of the spinal cord with fewer stresses being placed on the more superficial aspects of the lateral white matter.[5,6] The resultant injury to the central region of the cervical spinal cord would therefore impact the descending lateral corticospinal tracts, which are located in the lateral funiculus. Because the corticospinal tracts in people subserve fine motor movements of the arms and hands more so than providing control of the lower limbs, there is a disproportionate effect on the upper limbs with traumatic (acute) central cord syndrome.[7]

In veterinary (nonprimate) patients with central cord syndrome, wherein cervical spinal cord lesions cause paresis that is disproportionately worse in the thoracic limbs than in the pelvic limbs, attributing such disproportionate effects to dysfunction of the lateral corticospinal tracts, as in affected humans, is difficult. In dogs, experimental disruption of the motor cortex or pyramids (axons in the medulla that originate from the motor cortex and project into the spinal cord as the corticospinal tracts) results in a gait disturbance for a period of only a few days, after which there is little abnormality in the gait despite postural reaction deficits.[8] Thus, the pyramidal tract system appears less important for normal gait generation in nonprimates. Rather than ascribing a greater effect on a single UMN tract such as the corticospinal tracts, the greater degree of paresis in the thoracic limbs in nonprimate patients with central cord syndrome may broadly be the result of a disproportionate effect on the most medially positioned axons of multiple tracts that subserve thoracic limb function, compared with laterally positioned (superficial) axons that subserve the pelvic limbs. Although a more precise anatomic explanation for central cord syndrome in dogs and cats awaits description, the clinical recognition of central cord syndrome remains important.

As illustrated by the extradural location of the lesion in the dog of the present report, it should not

be assumed that the lesion responsible is located in the central aspect of the spinal cord of affected animals, despite the term central cord syndrome. Such an assumption may dissuade clinicians from performing diagnostic imaging because of the perception that an intramedullary lesion may not be amenable to surgery or may warrant a worse prognosis. Although the neurologic deficits may relate secondarily to spinal cord edema, ischemia, or another secondary consequence to a primary spinal cord injury, the underlying primary lesion may be an extradural compression as it was for the dog of the present report. In people with traumatic (acute) central cord syndrome, T2-weighted hyperintensity in the spinal cord detected during MRI may reflect edema or irreversible pathological changes (eg, microcavitation and gliosis).[5] Despite this, early decompression (ranging from 24 to 72 hours from the onset of signs), especially in individuals with progressive clinical signs, is recommended on the basis of favorable outcomes, compared with those achieved with nonsurgical treatment or late decompression.[9] The dog of the present report was euthanized (largely because of the owner's financial constraints), but decompressive surgery may have been a viable intervention with a resultant positive outcome. In the future, better characterization of the pathological changes associated with C1 to C5 myelopathies in which the thoracic limbs are disproportionately affected may lead to an improved understanding of optimal interventions and better prognosis for affected dogs.

Footnotes

a. Lightspeed 16, General Electric Medical Systems, Milwaukee, Wis.

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