In cooperation with

AMERICAN COLLEGE OF VETERINARY INTERNAL MEDICINE

What Is Your Neurologic Diagnosis?

A 9-year-old 27.1-kg (59.6-lb) neutered male Labrador Retriever was examined because of progressive lethargy, signs of pain, anorexia, pyrexia, and pelvic limb weakness of 3 days' duration. The owner also noticed progressive weight loss during the preceding 3 months. On initial examination, the dog had a rectal temperature of 40°C (104°F), and the remainder of the physical examination findings were unremarkable. A neurologic examination was performed. Postural reactions in the right thoracic limb and both pelvic limbs were delayed; spinal reflexes were exaggerated. The dog collapsed and assumed sternal recumbency on elevation of the head.

Neurologic examination

Observation

Mental	Alert		Depressed	X	Disoriented		Stupor		Coma		
Posture	Normal		Head tilt		Tremor		Falling		Kyphosis	X	
Gait	Normal		Ataxia	X	Pelvic limbs		All 4	X	Circling		
Paresis	Pelvic limbs		Tetra	X	Hemi		Mono				
Other	Severe thoracolumbar kyphosis with low carriage of the head was present. A short-strided gait in all 4 limbs was evident.										

Postural reactions

Key: 4 = exaggerated, clonus; 3 = exaggerated; 2 = normal; 1 = diminished; 0 = none; NE = not evaluated

	LF	RF	LR	RR
Wheelbarrow	NE	NE		
Hopping	2	1	1	1
Ext postural thrust			1	1
Proprioceptive pos	2	1	1	1
Hemistand/walk	2	1	1	1
Placing–tactile	NE	NE		
Placing–visual	NE	NE		

Spinal reflexes

	LF	RF	LR	RR
Quadriceps			3	3
Extensor carpi	2	2		
Flexion	2	2	1	1
Crossed extensor	2	2	2	2
Perineal			2	2

Cranial nerves

	L	R
II, VII–Vision menace	2	2
II, III–Pupils resting	2	1
Stim L	2	2
Stim R	2	2
II–Fundus	2	2
III, IV, VI–Strabismus, resting	2	2
III, IV, VI, VIII–Strabismus, position	2	2

	L	R
VIII–Nystagmus, resting	2	2
VIII–Nystagmus, change	2	2
V–Sensation	1	1
VII–Facial mm	2	2
V, VII–Palpebral flex	2	2
IX, X–Gag	2	2
XII–Tongue	2	2

Comments CN

Anisocoria (right pupil smaller than left) and mild ptosis and enophthalmos in the right eye were detected. Decreased nasal sensation was also identified.

Sensation (Locate and describe abnormal)

Hyperesthesia	3	Cervical spinal column, evident on extension of the neck and along the midthoracic vertebrae during palpation of spinous processes.
Superficial pain	2	
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
Decreased level of consciousness	Diffuse forebrain or reticular activating substance within the caudal portion of the brainstem
Decreased nasal sensation	Result of stress or lesion of the trigeminal nerves (ophthalmic or maxillary branch), trigeminal ganglion, or trigeminal tract in the pons and medulla
Tetraparesis and ataxia	C1-T2 spinal cord segments or multifocal disease of the spinal cord
Short-strided gait in all 4 limbs	Diffuse lower motor neuron disease (polyneuropathy) or multifocal spinal lesion affecting both intumescences (C6-T2 and L4-S1)
Partial right-sided Horner syndrome (miosis, enophthalmos, and ptosis of the right eye)	Right-sided cervical or T1-T3 region of the spinal cord, right T1-T3 ventral root and proximal spinal nerves, or less likely, cranial thoracic sympathetic trunk, cervicothoracic ganglion, middle cervical ganglion, cervical sympathetic trunk, middle ear, internal carotid nerve, or retroocular space[1]
Kyphosis with hyperesthesia over cervical and thoracic portions of the vertebral column	Postural position that alleviates pain secondary to cervical and thoracic lesion affecting meninges or nerve roots
Exaggerated quadriceps reflex	True hyperreflexia (upper motor neuron lesion affecting T3-L3 spinal cord segments) or pseudo-hyperreflexia (attributable to removal of tonic antagonistic influence by disruption of the innervation of caudal thigh muscles [ie, L6-S1 spinal cord or nerve root lesion])[2]
Diminished flexion of pelvic limbs	L4-S1 lesion

Likely location of 1 lesion

Meninges and spinal cord (multifocal or diffuse problem), most likely C5-T2 (on the right) and L6-S1 spinal cord segments or nerve roots, and possibly brain.

Etiologic diagnosis—The differential diagnoses for a subacute progressive painful multifocal CNS condition associated with pyrexia in a dog include inflammatory or infectious diseases (eg, bacterial, viral, protozoal, fungal, or sterile or immune-mediated meningoencephalomyelitis) or neoplastic (either primary [eg, CNS lymphoma or histiocytic sarcoma] or metastatic conditions [eg, hemangiosarcoma, other sarcomas, or carcinomas]). Metabolic, toxic, or degenerative diseases are less likely because they are not associated with signs of pain or pyrexia. The diagnostic plan included hematologic and serum biochemical analyses and urinalysis (to assess the dog's general condition), possible serologic testing for detection of antibodies against common infectious agents (to rule out toxoplasmosis and neosporosis in a dog with access to farmland in Scotland), and plain survey radiography of the vertebral column and abdomen (to identify a primary focus of infection or neoplasm). Following radiography, a CT[a] assessment of the cervical and thoracolumbar vertebral column was planned because of CT's increased sensitivity (compared with that of radiography) and ability to reformat the images of the vertebral column in multiple planes. Magnetic resonance imaging was also indicated but was not available at that time. Analysis of a CSF sample was planned to rule out meningeal inflammation if the diagnostic imaging procedures did not reveal any neoplastic lesions.

Diagnostic test findings—The dog had mild normocytic normochromic anemia with mild erythrocyte anisocytosis and neutrophilic leukocytosis with a mild left shift, suggestive of anemia of chronic disease and a superimposed inflammatory response. Serum biochemical abnormalities included mildly high creatine kinase activity, indicative of muscle damage, and mild hyperlipidemia with high cholesterol and triglyceride concentrations, indicative of an underlying condition such as pancreatitis, cholestasis, hypothyroidism, hyperadrenocorticism, or diabetes mellitus. The dog was seronegative for anti-*Neospora caninum* and anti-*Toxoplasma gondii* antibodies. Results of the urinalysis were unremarkable.

Radiography of the vertebral column revealed spondylosis deformans on the ventral aspects of T9 and T10 and of the L7 and S1 vertebral bodies (considered incidental age-related findings). Abdominal radiography revealed diffuse loss of abdominal serosal detail, consistent with peritoneal effusion. With the dog anesthetized, CT of the vertebral column revealed a well-defined, ovoid, non–contrast-enhancing region of decreased attenuation within the right epaxial lumbar musculature. An additional similar well-defined area was detected dorsal to the transverse processes of the lumbar vertebral bodies with a non–contrast-enhancing lesion within the sub-lumbar musculature **(Figure 1)**. A subtle enhancement of the meninges and spinal cord at the level of the L3

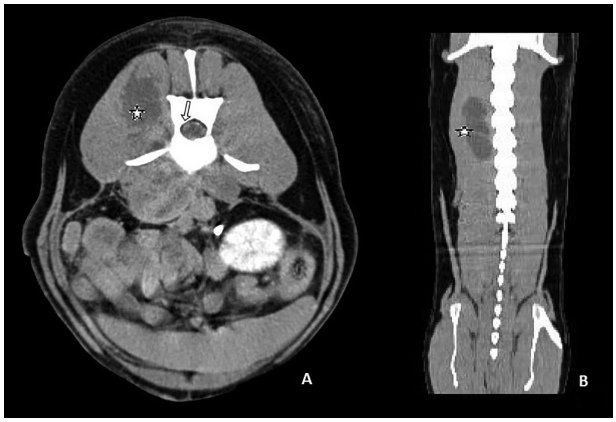

Figure 1—Transverse CT image at the level of the L3 vertebra (A) and dorsal CT reconstruction of the thoracolumbar spinal column (B), both obtained after contrast agent administration, of a dog that was examined because of progressive lethargy, signs of pain, anorexia, pyrexia, and pelvic limb weakness of 3 days' duration. There is a well-demarcated, ovoid, non–contrast-enhancing region of decreased attenuation within the right epaxial lumbar musculature (star) and enhancement of the meninges and spinal cord (arrow).

vertebra was visible after contrast agent administration, suggesting continuity of the sub-lumbar lesion with the meninges. No foreign body was identified. A CSF sample was collected at the level of cisterna magna and submitted for analysis.

On macroscopic evaluation, the CSF had a blood-tinged, turbid appearance with neutrophilic pleocytosis and a high protein concentration. Microscopic examination of prepared smears revealed many small cocci and coccobacilli within neutrophils. A CT-guided fine-needle aspirate of the affected epaxial musculature was obtained for bacterial culture, as were blood samples collected from the right jugular and left saphenous veins. A slow-growing anaerobe with characteristics similar to those of *Bacteroides* spp was isolated from both the fine-needle aspirate and the blood sample cultures.

A diagnosis of suspected right epaxial myositis with concurrent acute septic meningomyelitis and peritonitis was made, probably resulting from an occult migrating foreign body. The owner was informed that the prognosis for the dog was poor. Aggressive treatment involving surgical removal of the abscess and exploration of the possible area of entry of the bacterial infection within the dura (at L3) combined with long-term antimicrobial administration was offered but declined by the owner. With the owner's consent, the dog was euthanized by means of IV injection of pentobarbital sodium solution. Postmortem examination was performed.

On gross pathological examination, unilateral and regionally extensive suppurative paravertebral lumbar myositis was identified and histopathologic findings confirmed acute peritonitis and meningomyelitis. No gross evidence of diskospondylitis, osteomyelitis, or a foreign body was found. Microscopic changes of the psoas muscles were consistent with necrotizing and suppurative myositis with moderate fibrosis. The main histologic finding in the brain was chronic-active leptomeningitis.

Comments

Epaxial suppurative myositis with extension in the vertebral canal is uncommon in dogs. It has been previously described in dogs[3-9] with hunting breeds being most frequently affected.[5,6,7] The most common clinical findings include lethargy, anorexia, spinal hyperesthesia, and acute progressive para- or tetraparesis.[3,4,9,10] In affected dogs, pyrexia is frequently reported; however, it is not a consistent finding and dogs with bacterial meningitis might be normothermic,[4,6] especially if they have received antimicrobials prior to evaluation.

The initial infection may result from direct or indirect contact with an infectious agent (associated with foreign bodies, trauma [eg, bite wound[3]], or extension from an adjacent suppurative focus [eg, diskospondylitis][4,6]) or more rarely from hematogenous spread.[4,5,9,11,12] In the dog of the present report, we speculated that the inflammatory process, most likely of bacterial origin, resulted from direct extension of an infection causing paravertebral epaxial myositis into the vertebral canal and CNS. On the basis of the clinical history and absence of diskospondylitis, the presence of a small migrating foreign body was suspected. Entry of plant-derived foreign bodies via the respiratory tract and their subsequent migration to the ventral lumbar region in dogs has been previously reported[5,6]; however, in the case described in this report, there were no gross signs of pleural or diaphragmatic involvement.

The management of paraspinal abscesses with extension into the CNS remains controversial in veterinary and human medicine. Most clinicians opt for aggressive surgical treatment in combination with antimicrobial administration.[3,4,6,8,11,13,14] Percutaneous ultrasound-guided drainage of a paraspinal abscess followed by medical management in a dog has also been reported.[5] In general, spinal epidural empyema in dogs has been associated with a high mortality rate,[3,4,6,9,10] although good outcomes for individual cases have been sporadically reported.[3,4,13,15] In both human and veterinary medicine, early diagnosis of paraspinal abscesses is closely associated with a favorable outcome.[3,12,14,16]

Bacteroides spp have previously been identified as pathogens in canine cases of spinal epidural empyema and meningoencephalomyelitis.[4] These organisms are anaerobic, bile-resistant gram-negative bacilli found among the oral and the colonic microflora.[17,18] For the dog of the present report, it is possible that the pathogen was carried by a grass awn migrating out of the intestinal tract to the epaxial musculature. Common bacteria associated with paraspinal abscesses include *Staphylococcus* spp, *Streptococcus canis, Escherichia coli, Pasteurella* spp, *Actinomyces* spp,

Fusobacterium spp, and *Klebsiella* spp.[3,4,14,15,19] Magnetic resonance imaging is the most sensitive imaging technique for diagnosis of spinal epidural empyema of the CNS,[4–6] but in the case described in the present report, a CT scan allowed identification of a soft tissue lesion compatible with a paraspinal abscess[10] and revealed focal enhancement of the meninges and spinal cord, supporting the clinical suspicion of meningomyelitis.

Footnotes

a. Helical 4-slice CT unit, Somatom Volume Zoom, Siemens, Munich, Germany.

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