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AMERICAN COLLEGE OF VETERINARY INTERNAL MEDICINE

What Is Your Neurologic Diagnosis?

A 13-year-old 4-kg (8.8-lb) neutered female domestic shorthair cat was presented for evaluation of acute nonambulatory tetraparesis. The cat was able to go outdoors and had seemed well 12 hours prior to presentation. At the time of presentation, the cat had to be carried into the hospital because it was unable to walk. General physical examination did not reveal any abnormalities except for low rectal temperature (36.5°C [97.7°F]). No heart murmur or pulse deficits were noted.

Neurologic examination

Observation

Mental	Alert	X	Depressed		Disoriented		Stupor		Coma		
Posture	Normal		Head tilt		Tremor		Falling		Other	X	
Gait	Normal		Ataxia		Pelvic limbs		All 4		Circling		
Paresis	Pelvic limbs		Tetra	X	Hemi		Mono				
Other	The cat was recumbent. No voluntary motor function was present in the hind limbs and right forelimb. The left forelimb was paretic but voluntary motor function was present. Muscle tone was increased in the hind limbs and decreased in the forelimbs.										

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

Postural reactions	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Wheelbarrow	1	0		
Hopping	1	0	0	0
Extensor postural thrust			0	0
Proprioceptive positioning	1	0	0	0
Hemistand/walk	NE	NE	NE	NE
Placing–tactile	2	0		
Placing–visual	2	0		

Spinal reflexes	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Quadriceps			3	3
Extensor carpi	2	0		
Flexion	2	1	3	3
Crossed extensor	0	0	4	4
Perineal			2	2

Cranial nerves

	L	R		L	R	Comments
II, VII–Vision menace	2	2	VIII–Nystagmus, resting	2	2	No abnormalities were evident
II, III–Pupils resting	2	2	VIII–Nystagmus, change	2	2	
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 any abnormality)

Hyperesthesia	3	Some cervical hyperesthesia and thoracolumbar hyperesthesia
Superficial pain	2	
Cutaneous reflex	1	Diminished (but this reflex is often not elicited reliably in cats)
Deep pain	NE	Not examined because of apparently normal superficial nociception

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
Nonambulatory tetraparesis with increased muscle tone in the hind limbs and decreased muscle tone in the forelimbs	C6-T2 spinal cord segments (or C1-C5 spinal cord segments, or both)
Right forelimb hyporeflexia	Right-sided C6-T2 spinal cord segments (or C1-C5 spinal cord segments, or both), lower motor neurons in those segments and corresponding nerve roots, peripheral nerves, or innervated muscles
Cervical and thoracolumbar hyperesthesia	Integument, muscles, ligaments, vertebrae, joints, intervertebral disks, nerves or nerve roots, or meninges

Likely location of 1 lesion

C6-T2 myelopathy, predominantly right-sided

Etiologic diagnosis—Differential diagnoses considered included traumatic spinal cord injury (internal [eg, traumatic intervertebral disk extrusion or acute noncompressive nucleus pulposus extrusion {ANNPE}] or external [eg, vehicle accident–associated trauma]), vascular incidents (ischemic myelopathy), infectious and noninfectious inflammatory disease, neoplasia (extradural [eg, osteosarcoma or lymphoma], intradural and extramedullary [eg, lymphoma or meningioma], or intramedullary [eg, glioma]), and degenerative disease (intervertebral disk disease [eg, protrusion]) with acute deterioration (so-called acute-on-chronic disease). Given the outdoor access of the cat, radiography of the vertebral column was performed and revealed no signs of vertebral trauma. Owing to the time of presentation, further diagnostic testing (to be performed after a reexamination 10 hours later) was considered for the next day. Intravenous fluid therapy was started, and an IV dose of meloxicam was given to provide analgesia for the cat's apparently mild spinal hyperesthesia.

Diagnostic test findings—Reexamination of the cat 10 hours later revealed loss of nociception in its hind limbs and the right forelimb. The left forelimb was also hyporeflexic at that time. The cervical hyperesthesia was markedly increased, despite analgesic drug administration. The prognosis was deemed grave, and the owner elected euthanasia (performed by IV administration of pentobarbital solution) and permitted postmortem examination of the cervicothoracic region.

Postmortem examination revealed macroscopically visible subdural hemorrhage from the level of C3 to T2 and patchy red-purple discoloration of the spinal cord in the same area. No compressive lesions were identified. Removal of the spinal cord revealed red discoloration of the spinal cord on the ventral aspect, dorsally adjacent to the C4-5 intervertebral disk. Sectioning of the spinal cord resulted in extrusion of malacic spinal cord tissue through the dural defect. The ventral aspect of the vertebral canal at the level of the C4 and C5 vertebrae contained red-black material (hemorrhage) and a gel-like substance. The intervertebral disk space of C4-5 was collapsed. The final diagnosis was a C4-5 ANNPE (sometimes also referred to as hydrated nucleus pulposus extrusion) with subdural hemorrhage and hemorrhagic ascending-descending myelomalacia.

Comments

Intervertebral disk disease in cats has been more frequently identified in recent years. Whereas feline infectious peritonitis and lymphoma were the most prevalent causes of spinal cord disease in cats in a 2004 study,[1] other contemporary and more recent studies[2–5] have identified several other causes of spinal cord disease in cats, many of which are more common nowadays than the still-encountered feline infectious peritonitis and lymphoma. Traumatic ischemic myelopathy, fibrocartilaginous embolism, and intervertebral disk disease (including ANNPE, disk extrusion [hernia nuclei pulposi Hansen type I], and disk protrusion [hernia nuclei pulposi Hansen type II]) in cats have all been reported.[2–6] Myelomalacia is defined as gross softening of the spinal cord characterized by hemorrhagic necrosis and liquefaction of spinal cord tissue[7]; affected cats have a very grave prognosis for recovery of function, and may die.[7–9] Focal ascending, descending, or progressive myelomalacia in dogs with acute intervertebral disk disease has been reported quite often, and several clinical and diagnostic findings have been shown to correlate to the risk of development of myelomalacia in that species.[9–12] For cats, myelomalacia as a consequence of intervertebral disk disease is uncommonly reported, although it is logical that it might indeed develop, as was evident in the case described in the present report. Myelomalacia is the result of primary and secondary injury to the structures of the spinal cord.[13] In instances of ANNPE, the primary injury is physical, and could theoretically range from concussion or

contusion to laceration depending on the velocity of the extrusion of the nucleus pulposus. Laceration of the dura and possibly the spinal cord could give rise to an intradural-intramedullary intervertebral disk extrusion.[6] Secondary injury, which develops after the primary insult through various mechanisms, resulting in further neuronal and neuroglial damage is still a subject of research but seems to be the result of a combination of several factors including excitotoxicosis, ischemia, and ischemic-reperfusion injury.[13] Such processes may be contained at the site of the primary injury or may spread cranially or caudally, for reasons that are not quite clear. Deterioration of the clinical status of dogs that develop myelomalacia usually ensues rapidly after onset of the first clinical signs, but a delay in onset of progressive deterioration (days after acute spinal cord injury) has been reported.[7–10]

In the case described in the present report, deterioration of the cat's clinical condition was noticeably swift within approximately 10 hours after presentation. Also, cervical hyperesthesia was mild at presentation (presence of cervical hyperesthesia is not consistently reported for veterinary cases of ANNPE) but increased in severity despite administration of an analgesic drug. The subdural hemorrhage encountered during postmortem examination might explain this finding because tension or pressure on the meninges would be expected to be quite painful.

As stated previously, ANNPE in cats has been reported,[6] and findings of more extensive postmortem and histopathologic examinations would have been ideal to confirm the nature of the gel-like substance encountered macroscopically in the cat of the present report. Such examinations could have confirmed that the gel-like substance was indeed nondegenerated nucleus pulposus material, and would have provided opportunities for investigation of possible concurrent pathological changes in the spinal cord (eg, vascular obstruction [fibrocartilaginous embolism]).

The MRI findings in dogs with ANNPE have been elaborately documented in the veterinary medical literature, and comparable findings would be expected in feline cases.[11,12] In dogs with thoracolumbar intervertebral disk extrusion, MRI findings have prognostic value related to development of myelomalacia.[10–12] However, the MRI findings of myelomalacia are nonspecific. In the case described in the present report, MRI would presumably have revealed extensive signal changes in and around the spinal cord, but findings certainly could not have definitively confirmed the nature of those changes. The clinically noticeable deterioration of the cat's condition (most importantly, loss of nociception) was crucial for establishing the cat's prognosis, and further diagnostic testing was declined by the owner after this information became available. Earlier diagnostic imaging was unfortunately not possible due to the out-of-hours presentation of the cat and logistic issues, but it would be unlikely to have influenced the outcome in this case. The case described in the present report has illustrated the importance of repeated clinical examination of cats with neurologic conditions and the importance of postmortem examination.

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