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What Is Your Neurologic Diagnosis?

A 23-year-old Palomino gelding weighing 516 kg (1,135 lb) was evaluated by the emergency service of a veterinary teaching hospital because of sudden-onset tetraparesis and ataxia of < 24 hours' duration. The horse had no history of previous illness with the exception of fibrotic myopathy in the right pelvic limb years earlier. The horse was bright, alert, and responsive; rectal temperature was 37.3°C (99.1°F), pulse rate was 42 beats/min (second-degree atrioventricular block was suspected), and respiratory rate was 18 breaths/min. The horse had not received medication prior to the evaluation.

Neurologic examination

Observation

Mental	Alert	X	Aggressive		Depressed		Stupor		Coma		
Posture	Normal	X	Head tilt		Head press		Circling		Rigid Stance		
Other											

Key: 0 = No abnormality; 1 = Diminished; 2 = Exaggerated; NE = Not evaluated

Cranial nerves	L	R		L	R	Comments
II–Vision	0	0	V, VII–Palpebral response	0	0	
II, VII–Menace response	0	0	VIII–Nystagmus	0	0	
II, III–PLR, direct	0	0	IX, X, XI–Swallowing	0	0	
II, III–PLR, consensual	0	0	XII–Tongue tone	0	0	
III, IV, VI–Eye position	0	0	Slap test	1	1	

Key: 0 = None; 1 = Mild; 2 to 3 = Moderate; 4 = Severe; NE = Not evaluated

Gait analysis	Walk				Trot			
	Left forelimb	Right forelimb	Left hind limb	Right hind limb	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Paresis	4	4	3	3	NE	NE	NE	NE
Ataxia	3	3	3	3	NE	NE	NE	NE
Hypermetria	0	0	0	3	NE	NE	NE	NE
Hypometria	0	0	0	0	NE	NE	NE	NE

Postural reactions	Turns				Backing			
	Left forelimb	Right forelimb	Left hind limb	Right hind limb	Left forelimb	Right forelimb	Left hind limb	Right hind limb
Paresis	4	4	4	4	NE	NE	NE	NE
Ataxia	4	4	4	4	NE	NE	NE	NE
Hypermetria	0	0	0	3	NE	NE	NE	NE
Hypometria	0	0	0	0	NE	NE	NE	NE

- Does gait worsen with the head elevated? Yes ___ No _X_ NE ___
- Does gait worsen when walking on a slope? Yes ___ No ___ NE _X_

Key: 0 = Clinically normal; Abnormalities, 1 = Mild, 2 to 3 = Moderate, 4 = Severe

Other assessments	Neck and forelimbs		Trunk and hind limbs	
	Left	Right	Left	Right
Hoofwear	0	0	0	0
Conscious proprioception	3	3	3	3
Range of motion (neck)	1	1		
Muscle mass	0	0		
Cervicofacial response	0	0		
Panniculus response			0	0
Sway reaction	NE	NE	NE	NE
Sensation	0	0	0	0

Comments

There was firm swelling on the left side of the midcervical region.

- Localized sweating on neck or trunk?
No ___ Yes _X_
Where: Right midcervical region
- Tail/anal tone:
Normal _X_ Increased ___ Decreased ___

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
General proprioceptive ataxia in all 4 limbs	Cervical spinal cord segment or brainstem
Tetraparesis	Cervical spinal cord segment, brainstem, or lower motor neuron disease
Negative thoracolaryngeal reflex (slap test)	Cervical portion of the spinal cord, medulla, vagus nerve, or recurrent laryngeal nerve
Focal right-sided mid to caudal cervical hyperhidrosis	Sympathetic denervation in the mid to caudal neck region

Likely location of 1 lesion

The presence of general proprioceptive ataxia and tetraparesis without accompanying mentation changes or cranial nerve deficits is most consistent with myelopathy between the C1 and T2 spinal cord segments. The focal region of hyperhidrosis further localizes the lesion to a site in the mid to caudal neck region.

Etiologic diagnosis—Initial examination findings for the horse indicated a neuroanatomic localization consistent with C1-T2 myelopathy. Differential diagnoses for clinical signs caused by a lesion in this region included cervical vertebral stenotic myelopathy (secondary to age-related degenerative joint disease of the articular facets), infectious disease (equine protozoal myeloencephalitis), previous vertebral fracture or luxation within the neck, trauma, compressive or infiltrating mass (abscess, neoplasia, or granuloma), or embolic myelopathy. The diagnostic plan included radiography (lateral and oblique views) of the cervical portion of the vertebral column to assess for a compressive lesion, mass, or fracture within the neck; myelography to assess for compression of the spinal cord; collection and analysis of a CSF sample to assess for inflammation, infection (including equine protozoal myeloencephalitis), or neoplasia within the CNS; CT of the cervical portion of the vertebral column to acquire additional information regarding any lesions identified radiographically and investigate soft tissue changes along the neck in cross-sectional planes; a CBC to assess for evidence of inflammation or infection; microbial cultures of blood and urine samples to assess for bacterial infection resulting in osteomyelitis; and biopsy of any affected vertebrae identified radiographically to determine cause.

Diagnostic test findings—The horse was initially admitted (day 1) to the hospital for monitoring and was treated with dexamethasone (0.23 mg/kg [0.1 mg/lb], IV) and phenylbutazone (4.4 mg/kg [2.0 mg/lb], PO, q 12 h). On day 2, the ataxia had improved but was still present. Lateral and oblique radiographic views of the cervical portion of the vertebral column from C1 to C7 were obtained. A moth-eaten lytic lesion was visible within the body of the fifth cervical vertebra **(Figure 1)**. Osseous proliferation or mineralization was present and bridged the cranial articular processes of the C5 vertebra and the caudal articular processes of the C4 vertebra. Mineralization was also present in the nuchal ligament at this level. Differential diagnoses for the lytic lesion included neoplasia or osteomyelitis. Osteomyelitis in adult horses generally develops as a result of either trauma or iatrogenic introduction of microbes during surgical procedures with or without implants.[1] Given that the horse of the present report had no evidence of penetrating trauma or history of surgery, neoplasia was considered the most probable cause for the lesion detected radiographically. Thus, additional diagnostic tests were not considered necessary.

The horse received a tapering dosage of dexamethasone (0.10 mg/kg [0.05 mg/lb], IV, on day 2 and 0.06 mg/kg [0.03 mg/lb], IV, on day 3) and phenylbutazone at the same dosage as initially administered. Although some improvement in ataxia was noted in response to treatment administered on day 1, the ataxia progressed after day 2 and the horse developed a repetitive cervical flexion and extension behavior that was interpreted as discomfort. The horse's condition continued to deteriorate despite medical management. General proprioceptive ataxia and upper motor neuron tetraparesis progressed, and the horse had difficulty rising from a recumbent position and standing. The severity and continued progression of signs despite treatment warranted a grave prognosis; in combination with a potentially neoplastic lesion and concern for progression to pathological fracture of the cervical vertebral column, the owners elected euthanasia by IV barbiturate overdose on day 6. Necropsy was permitted, during which a 4-cm-wide, blood-filled cavity that was within the body of the fifth cervical vertebra and that extended into the ventrally adjacent musculature was identified. The bony mass caused ventral extradural compression of the spinal cord and had narrowed the vertebral canal at this level. Histologically, the mass was consistent with hemangiosarcoma. There was mineralization of the ventral portion of the dura mater associated with the mass. The only histologic abnormalities within the spinal cord at this level were rare dilated

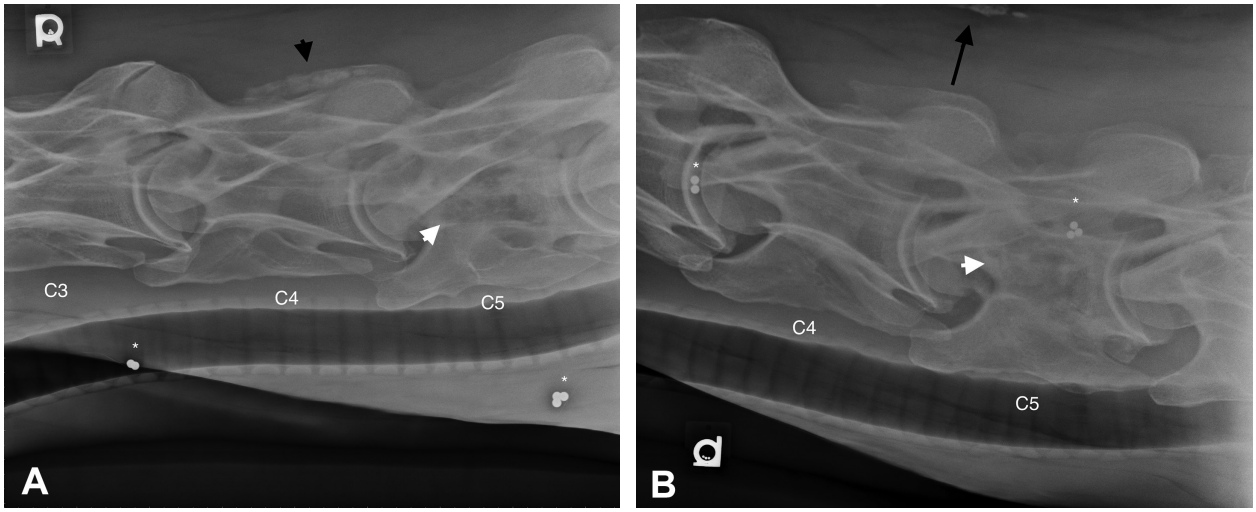

Figure 1—Right lateral (A) and right-to-left dorsoventral oblique (B) radiographic views of the fourth and fifth cervical vertebrae of a horse with sudden-onset tetraparesis and ataxia. Moth-eaten lysis is present within the body of the fifth cervical vertebra (white arrow). Osseous proliferation or mineralization is present bridging the cranial articular processes of C5 and the caudal articular processes of C4 (black arrow). Mineralization is present in the nuchal ligament (long-tailed black arrow). In both views, radiopaque markers are identified with an asterisk.

myelin sheaths, consistent with chronic compressive myelopathy.

Comments

Hemangiosarcoma in horses is rarely reported, but 2 case series[2,3] describing this type of neoplasm in this species have been published. In 35 horses (median age, 12 years) with disseminated hemangiosarcoma, clinical signs were most commonly referable to the musculoskeletal and respiratory systems.[3] At the time of initial evaluation, the most common clinical signs of those affected horses were dyspnea, swelling of the subcutaneous and muscular tissues, epistaxis, and lameness. Among the 35 horses, hemangiosarcoma was most frequently identified in the lungs and pleurae (77%), followed by the skeletal muscle (46%), spleen (43%), heart (34%), kidney (26%), and brain (26%).[3] In another report[2] that documented findings in 11 young horses (age, ≤ 3 years), the most common clinical signs at the initial evaluation were subcutaneous masses or swelling, diffuse limb swelling, or joint effusion. An antemortem diagnosis of hemangiosarcoma was made for 10 of those 11 cases,[2] compared with only 4 of 35 cases in the other report.[3] In both case series,[2,3] postmortem examination provided a definitive diagnosis for all remaining cases. Involvement of the vertebral bodies or spinal cord was not identified in any of those 35 horses with hemangiosarcoma. However, there are 5 reports[4-8] of single cases of hemangiosarcoma involving the cervical vertebrae of equids, including 1 donkey. An additional case involved a horse with hemangiosarcoma in the third thoracic vertebra.[9] Initial clinical signs for the 5 horses in which the cervical portion of the vertebral column was affected included ataxia (n = 3), difficulty raising the head (1), weight loss (2), lethargy (1), anorexia (1), nasal discharge (1), and bullous stomatitis (1).[4-8] For those reported cases, treatment of 1 horse was attempted; in that horse, the tumor did not directly involve the vertebral body but appeared to originate from the surrounding musculature and affected the spinous processes.[6] The horse underwent tumor excision and was reportedly doing well 14 months after surgery.[6] Euthanasia of the 4 other horses was elected because of grave prognoses.

Unlike other veterinary species, horses are unique in that spinal cord injuries can lead to focal sweating that can aid clinicians in neurolocalization. Although the mechanism behind this phenomenon has not been firmly established, it is hypothesized that an interruption of the sympathetic innervation to the skin leads to vasodilation and an increase in blood flow to the affected skin. This consequently exposes the sweat glands, which in horses are highly sensitive to norepinephrine, to higher circulating concentrations of norepinephrine and results in excessive sweating. The affected region of skin is generally hyperthermic, compared with adjacent unaffected areas, which supports the hypothesis of sympathetic denervation and vasodilation and can be confirmed with thermography.[10] This phenomenon was identified in the horse of the present report, in which a discrete region of hyperhidrosis was identified in the region of C5 on the right side of the neck. Sympathetic postganglionic axons that supply the neck travel in the vertebral nerve, which is a branch from the cervicothoracic ganglion. The vertebral nerve follows the vertebral artery through the transverse foramen of C6 and courses cranially through each transverse foramen to C1. The vertebral nerve supplies a ramus to each cervical spinal nerve at the intervertebral foramen.[10] In the case described in the present report, the localized sweating was suspected

to have developed secondary to damage to the ramus from the vertebral nerve or cervical spinal nerve at the level of the C5 vertebra through compression or invasion by the tumor. If the tumor had been larger or more invasive, thereby involving the vertebral nerve and sympathetic axons traveling cranially, the horse would have been expected to have sweating over the entire right cervical region cranial to C5, instead of the observed focal area of sweating.

Cervical radiography proved to be a key diagnostic technique in the case described in the present report. Radiography of the cervical portion of the vertebral column of horses is relatively simple to perform, even in ambulatory practice, and should not be overlooked in the evaluation of horses with ataxia. Computed tomography would have provided a more detailed image of the lesion, and CT following contrast medium administration would likely have identified the extent of the tumor ventral to the vertebral body. However, the horse's degree of ataxia in combination with severe lysis of the fifth cervical vertebra led to safety concerns regarding anesthetic recovery. Thus, the risk of performing additional imaging was considered to outweigh the benefit of any information that may have been gleaned from CT.

References

1. Goodrich LR. Osteomyelitis in horses. *Vet Clin North Am Equine Pract* 2006;22:389–417.
2. Johns I, Stephen JO, Del Piero F, et al. Hemangiosarcoma in 11 young horses. *J Vet Intern Med* 2005;19:564–570.
3. Southwood LL, Schott HC, Henry CJ, et al. Disseminated hemangiosarcoma in 35 horses. *J Vet Intern Med* 2000;14:105–109.
4. Kennedy FA, Brown CM. Vertebral angiosarcoma in a horse. *J Vet Diagn Invest* 1993;5:125–127.
5. Berry S. Spinal cord compression secondary to hemangiosarcoma in a Saddlebred stallion. *Can Vet J* 1999;40:886–887.
6. Williams MA, Dowling PM, Angarano DW, et al. Paraneoplastic bullous stomatitis in a horse. *J Am Vet Med Assoc* 1995;207:331–334.
7. Newton-Clarke MJ, Guffoy MRG, Dykes NL, et al. Ataxia due to a vertebral haemangiosarcoma in a horse. *Vet Rec* 1994;135:182–184.
8. MacGillivray KC, Sweeney CR, McLear R, et al. Vertebral hemangiosarcoma in a 16-year-old miniature Sicilian donkey. *Vet Radiol Ultrasound* 2003;44:429–432.
9. Lombardo de Barros CS. Vertebral hemangiosarcoma as a cause of spinal cord compression in a horse. *Ciência Rural* 1997;27:503–504.
10. de Lahunta A, Glass E, Kent M. Lower motor neuron: general visceral efferent system. In: de Lahunta A, Glass E, Kent M, eds. *Veterinary neuroanatomy and clinical neurology.* 4th ed. St Louis: Elsevier Saunders, 2015;204–210.

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