*In cooperation with*

# What Is Your Neurologic Diagnosis?

**Signalment:** 12-year-old 20-kg (44-lb) spayed female mixed-breed dog.

**History:** The dog was examined because of what was described by the owners as a back problem. A gait disturbance in the pelvic limbs had become evident 3 days earlier; signs had worsened progressively, and the clients reported that the dog was having difficulty ascending steps or stairs and misjudged jumps onto furniture. On the day of the referral examination, the clients noticed additional stiffness associated with the thoracic limbs.

**Physical examination:** In general, physical examination findings (including mentation) were unremarkable. While standing, the dog's trunk swayed back and forth irregularly. The dog did not fall, had no head tilt, and did not drag any limb. At commencement of walking, the dog appeared spastic with signs of dysmetria (predominantly hypermetria) in all limbs.

**Neurologic examination**

**Observation**

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mental | Alert | X | Depressed | | Disoriented | | Stupor | | Coma | | |
| Posture | Normal | X | Head tilt | | Tremor | | Falling | | | | |
| Gait | Normal | | Ataxia | X | Pelvic limbs | | All 4 | X | Circling | | |
| Paresis | Pelvic limbs | | Tetra | | Hemi | | Mono | | | | |
| Other | Ambulatory in all limbs without support but with spasticity and dysmetria in all limbs. Truncal ataxia detectable when standing. Apparently normal conscious proprioception and strength in all limbs. No lateralization of signs was evident. During assessment of postural reactions, exaggerated responses were detected after initial delays. | | | | | | | | | | |

**Postural reactions**

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

| | LF | RF | LR | RR |
|---|---|---|---|---|
| Wheelbarrow | 3 | 3 | | |
| Hopping | 3 | 3 | 3 | 3 |
| Ext postural thrust | | | NE | NE |
| Proprioceptive pos | 2 | 2 | 2 | 2 |
| Hemistand/walk | 3 | 3 | 3 | 3 |
| Placing–tactile | NE | NE | | |
| Placing–visual | NE | NE | | |

**Spinal reflexes**

| | LF | RF | LR | RR |
|---|---|---|---|---|
| Quadriceps | | | 2 | 2 |
| Extensor carpi | NE | NE | | |
| Flexion | 2 | 2 | 2 | 2 |
| Crossed extensor | 0 | 0 | 0 | 0 |
| Perineal | | | 2 | 2 |

**Cranial nerves**

| | L | R | | L | R | Comments CN |
|---|---|---|---|---|---|---|
| II, VII–Vision menace | 2 | 2 | VIII–Nystagmus, resting | 2 | 2 | |
| 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 abnormal)

| | | |
|---|---|---|
| Hyperesthesia | 0 | |
| Superficial pain | 2 | |
| Cutaneous reflex | 2 | |
| Deep pain | 2 | |

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 |
|---|---|
| Gait disturbance in all limbs with dysmetria but apparently normal strength, proprioception, and reflexes | Cerebellum |

### Etiologic diagnosis

| Rule out disease process | Diagnostic plan (in order of priority) |
|---|---|
| Neoplasia | Emergency neuroradiography via magnetic resonance imaging—to confirm or rule out a macroscopic cerebellar lesion |
| Vascular (expanding hemorrhagic stroke with or without concurrent neoplasia; infarct) | Possible CSF analysis—to identify hemorrhage, inflammatory disease, or other microscopic lesions not detectable via magnetic resonance imaging |
| Inflammatory disease | General evaluation including clinicopathologic analyses, thoracic radiography, and abdominal ultrasonography—to assess for systemic disease or lesions outside the CNS |

**Comments:** The rapid progression of signs over the preceding 72 hours warranted concern for a dynamic and worsening lesion. The hallmark of cerebellar disease is disruption of coordination that controls the rate and range of voluntary movement. Cerebellar disease is not associated with loss of strength. Postural reactions are typically exaggerated, and an affected dog cannot smoothly correct placement of its limbs. In many instances of severe cerebellar disease, the menace response is absent despite otherwise apparently normal vision; signs of vestibular dysfunction are also common. These latter signs were not detected in the dog of this report on initial examination. Although the absence of central vestibular signs does not in any way rule out cerebellar disease, this negative finding also suggests that the flocculonodular lobes and peduncles (archecerebellum) are not involved in the disease process because these structures represent the cerebellar connection to the vestibular apparatus. Dogs with cerebellar disease may also develop fine tremors or intention tremors. These signs were also not evident in the dog of this report. Truncal ataxia (swaying of the body at rest) is an inconsistent but nearly pathognomonic sign of cerebellar disease and was useful in differentiating the dog's syndrome from what is typically associated with dogs that have spinal cord lesions.

## Test results:

**Imaging procedures:** During anesthesia, magnetic resonance imaging of the brain was performed in the sagittal, transverse, and dorsal planes with T1 and T2 weighting and with a fluid attenuation inversion recovery (FLAIR) sequence by use of a 1.5-T magnet and human head coil. The T1-weighted imaging was repeated after IV administration of paramagnetic contrast agent (gadolinium). A lesion with mixed T2-weighted heterogeneity was identified in the cerebellum, and the entire cerebellum appeared large. The center of the mass had low T2-weighted signal intensity with some high–signal intensity areas around it. Perilesional edema was confirmed by use of the FLAIR sequence. After the administration of contrast agent, the periphery of the mass was enhanced and the center remained mostly unenhanced, suggesting a necrotic center. The mass was approximately 1.5 cm in diameter, but because of the perilesional edema, the mass appeared much larger (mass effect). As a result, compression of the caudal mesencephalic aqueduct and fourth ventricle was detected (**Figure 1**).

**Other diagnostic procedures:** Results of a CBC, serum biochemical analyses, urinalysis, thoracic radiography, and abdominal ultrasonography were within reference limits.

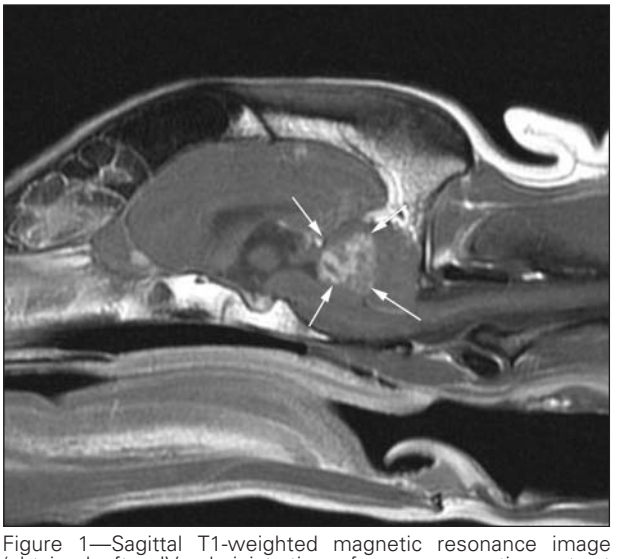

Figure 1—Sagittal T1-weighted magnetic resonance image (obtained after IV administration of a paramagnetic contrast agent) of the head of a dog with a progressive gait disturbance in the pelvic limbs. Notice the rostral cerebellar mass (rostral is to the left of the image). The arrows define the extent of the lesion, including perilesional edema. The signal within the cerebellum is heterogeneous. The center of the mass has low signal intensity with some high–signal intensity areas around it as a result of contrast enhancement, suggestive of a rich blood supply to the mass with necrosis centrally.

**Presumptive diagnosis:** The primary rule out was an intra-axial mass within the cerebellum that was associated with peripheral contrast agent enhancement; primary malignant glioma with secondary necrosis and mass effect were considered most likely. Infectious or inflammatory lesions such as toxoplasmosis were also considered, but the possibility of those diseases seemed remote.

**Prognosis with treatment:** Guarded to poor. The presence of substantial mass effect and the concern for rostral tentorial herniation suggested that progression of signs would continue. The location of the mass made surgery an unlikely means by which treatment success (ie, survival with improved quality of life) could be achieved.

**Therapeutic plan:** Emergency treatment was initiated with an osmotic diuretic (mannitol; 20 g administered IV during a 20-minute period) and corticosteroid (methylprednisolone sodium succinate; 600 mg administered IV, followed 2 hours later by IV administration of 300 mg). If the dog's condition stabilized or improved following treatment, referral for external beam radiation therapy or possibly gamma-knife radiosurgery was to be discussed with the owner.

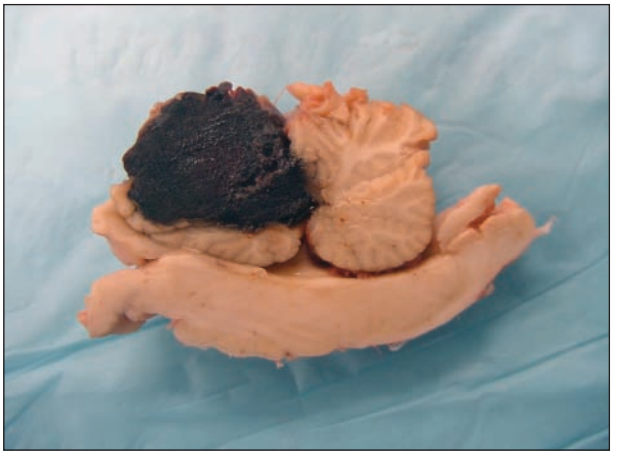

Figure 2—Photograph of the brainstem and overlying cerebellum (sagittal formalin-fixed section) of the dog in Figure 1. Notice the location of the tumor (dark tissue), which corresponds to the lesion detected via magnetic resonance imaging (rostral is to the left of the image). The tumor was approximately 1.5 cm in diameter. Histologically, the tumor was identified as a hemangiosarcoma.

**Outcome:** Despite the vigorous medical treatment of edema, the signs indicated rapid deterioration of the dog's condition. Several hours after the magnetic resonance imaging examination (approx 12 hours after the initial examination), the dog became nonambulatory. When supported, the dog continued to be spastic in all limbs and developed marked proprioceptive deficits in all limbs. The next morning, the dog was less responsive and alert and began assuming an opisthotonic posture. These signs were interpreted as a combination of worsening cerebellar deficits as well as the effects of brainstem compression and probable rostral tentorial herniation. On the basis of the worsened clinical condition, grave prognosis, and imaging findings, the dog was euthanized.

During a limited necropsy, protrusion of a dark mass through the cranial parenchyma of the cerebellum was detected (**Figure 2**). Microscopically, the mass had extensive hemorrhage and necrosis with scattered areas of intact neoplastic tissue that formed irregular vascular channels filled with blood. The cells had large nuclei with moderate nuclear size variation; mitotic activity was high (5 mitotic spindles/hpf). The histologic diagnosis was hemangiosarcoma. Because a complete necropsy was not performed, it was not determined whether the mass was a primary or metastatic lesion. However, metastatic hemangiosarcoma involving the brain is typically associated with many small lesions that are scattered throughout the brain tissue. The finding of a single large lesion and the inability during antemortem evaluations to identify lesions elsewhere made the probability of primary cerebellar hemangiosarcoma more likely in the dog of this report. Hemangiosarcomas can develop in any location.

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This feature is published in coordination with the American College of Veterinary Internal Medicine on behalf of the specialty of neurology. Contributors to this feature should contact Dr. Helen L. Simons (800-248-2862, ext 6692) for case submission forms. Completed forms will be sent to Dr. Karen Kline at Iowa State University for her review.