



What Is Your Neurologic Diagnosis?

An 8-year-old 40.8-kg (89.8-lb) castrated male Labrador Retriever was evaluated because of sudden onset of bilateral blindness 9 days earlier and worsened dyspnea. Seven days prior to the evaluation, a veterinary ophthalmologist had examined the dog. At that time, a diagnosis of bilateral retrobulbar optic neuritis was made. During the 4 to 6 days prior to the referral evaluation, treatment of the dog included oral administration of doxycycline, enrofloxacin, prednisone, and famotidine. The dog was seronegative for canine distemper virus.

During the initial evaluation, the dog was bright, alert, and responsive. The dog's rectal temperature (38.6°C [101.5°F]) and heart rate (60 beats/min) were within reference ranges. Physical examination revealed dyspnea with very little air movement out of the nares. Thoracic auscultation revealed loud respiratory noises, which became very quiet once the mouth was held closed. The remainder of the physical examination findings were unremarkable. A follow-up ophthalmic examination was completed; there was no evidence of optic neuritis or other cause of blindness.

Neurologic examination

Observation

Mental	Alert	X	Depressed		Disoriented		Stupor		Coma	
Posture	Normal	X	Head tilt		Tremor		Falling			
Gait	Normal	X	Ataxia		Pelvic limbs		All 4		Circling	
Paresis	Pelvic limbs		Tetra		Hemi		Mono			
Other	The dog bumped into objects when ambulating.									

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

Postural reactions

	LF	RF	LR	RR
Wheelbarrow	2	2		
Hopping	2	2	2	1
Ext postural thrust			2	2
Proprioceptive pos	2	2	2	2
Hemistand/walk	2	2	2	2
Placing–tactile	2	2		
Placing–visual	0	0		

Spinal reflexes

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

Cranial nerves

	L	R		L	R	Comments CN
II, VII–Vision menace	0	0	VIII–Nystagmus, resting	2	2	Pupils were fixed and dilated. Menace and following responses were absent. Direct pupillary light response was present in the left eye, but consensual response was absent. In the right eye, direct pupillary light response was absent, but consensual response was present.
II, III–Pupils resting	2	2	VIII–Nystagmus, change	2	2	
Stim L	0	0	V–Sensation	2	2	
Stim R	0	0	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
Acute blindness (bilateral)	Retina, cranial nerve II, or midbrain
Absent pupillary light reflex (bilateral)	Retina, cranial nerves II or III, or midbrain
Absent menace response (bilateral)	Retina, cranial nerve II, forebrain, or cerebellum
Dyspnea	Laryngeal or nasopharyngeal region

Likely location of one lesion

CNS disease involving the cerebrum, with potential involvement of the nasopharynx

Etiologic diagnosis—Rule out disease processes included neoplasia (primary [focal or multifocal]), infectious or inflammatory disease, or laryngeal paralysis. The diagnostic plan included performing a CBC, serum biochemical analyses, urinalysis, and evaluation of a CSF sample (to rule out optic neuritis or other generalized inflammatory disease); laryngeal examination under sedation (to rule out laryngeal paralysis as the cause of dyspnea); thoracic radiography (to identify any evidence of metastatic disease); magnetic resonance imaging of the brain and caudal portion of the nasal cavity (to rule out any masses or pathological change within the brain or nasal cavity); and endoscopy of the caudal portion of the nasopharynx (to obtain biopsy specimens of any abnormal tissue for histologic examination).

Diagnostic test findings—Results of a CBC indicated mild neutrophilia and mild monocytosis consistent with a stress leukogram. Serum biochemical abnormalities included high alkaline phosphatase activity (2,532 U/L; reference range, 4 to 95 U/L), high alanine aminotransferase activity (254 U/L; reference range, 26 to 200 U/L), and hypocalcemia (8.7 mg/dL; reference range, 9.5 to 11.8 mg/dL). Urinalysis results were unremarkable. Laryngeal examination under sedation revealed bilateral laryngeal paralysis. Findings of thoracic radiography were considered normal for a dog of that age and body condition.

Magnetic resonance images^a of the brain and caudal portion of the nasal cavity were acquired by use of T1-weighted and T2-weighted fast spin echo sequences and a T2*-weighted gradient echo sequence. Additionally, T1-weighted sequences in transverse, sagittal, and dorsal planes were also acquired after IV administration of gadoteridol.^b In T2-weighted transverse images, hyperintense material was evident in the caudoventral portion of the nasal cavity, within the nasopharynx and sphenoid sinus, and lateral to the caudal aspect of the nasal cavity; the left side was affected more than the right side (Figure 1). In T2*-weighted images, portions of the more lobular-appearing regions of the material were hypointense, compared with surrounding tissues, indicative of mineralization in these regions. In T1-weighted transverse images, the sphenoid bone appeared discontinuous

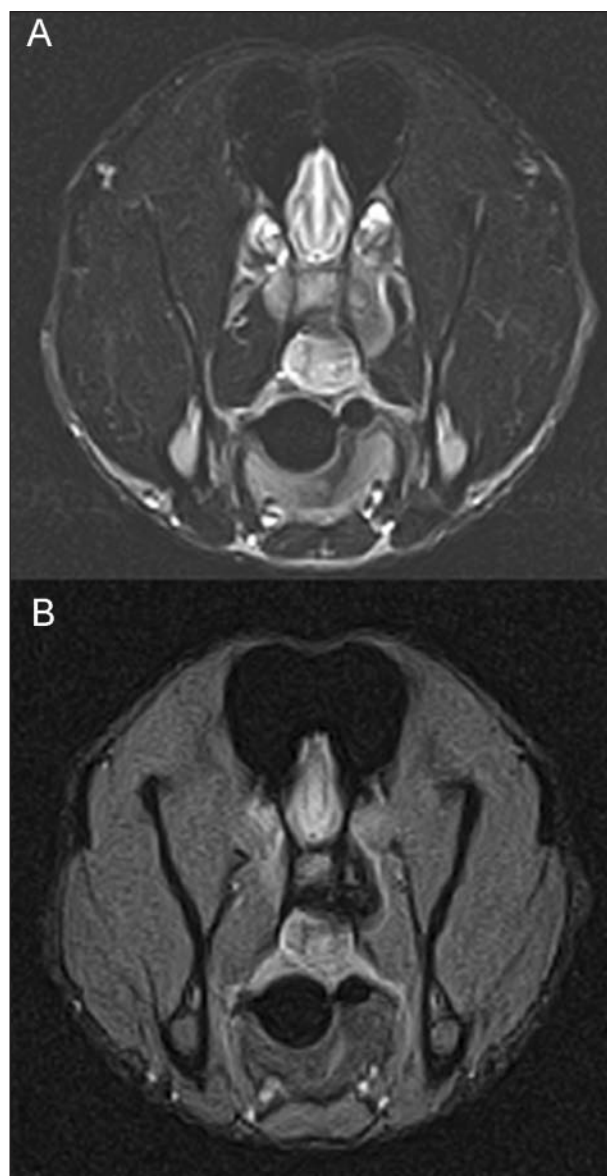


Figure 1—T2-weighted (A) and T2*-weighted (B) transverse magnetic resonance images of the brain of a dog that was evaluated because of sudden onset of bilateral blindness 9 days earlier and worsened dyspnea. Notice the regions that are hypointense, compared with surrounding tissues; these are likely areas of mineralization.

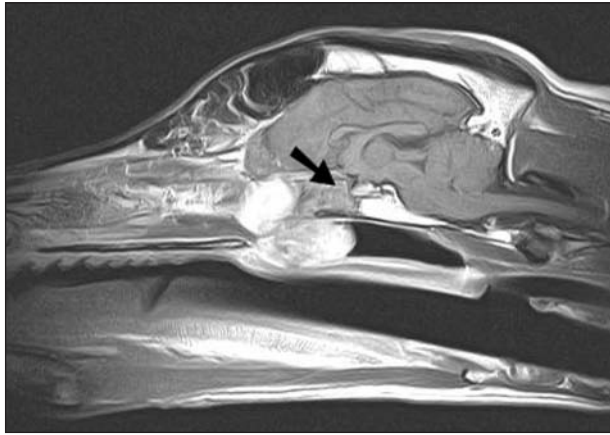


Figure 2—Sagittal T1-weighted magnetic resonance image of the brain of the dog in Figure 1 obtained after administration of contrast medium. Notice the mass that extends to the level of the optic chiasm (black arrow). Rostral is to the left.

and indistinct rostrally. After administration of contrast medium, there was strong contrast enhancement of the portion of the mass within the nasopharynx on T1-weighted images. Contrast-enhanced lobulated portions of the mass extended dorsally through the sphenoid bone into the sphenoid sinus, caudoventral portion of the nasal cavity, and ventral portion of the calvarium. The mass extended caudally to the region of the optic chiasm and appeared compressive in that region (Figure 2). Ventrally, the mass extended to the soft palate and appeared to mildly deviate the rostral portion of the soft palate ventrally.

A retroflexed endoscope was inserted into the caudal portion of the nasopharynx, and examination revealed that the mass occluded most of the caudal portion of the nasal cavity. Multiple biopsy specimens of the mass and surrounding tissues were obtained, and sections were examined histologically. The histopathologic diagnosis was a highly aggressive osteoblastic osteosarcoma of the basisphenoid bone. Although collection of a CSF sample for analysis was initially planned, this was not undertaken because the cause of the clinical signs had been determined.

The therapeutic plan for the dog included evaluation for possible radiation therapy. However, in the interim, the dog's respiratory condition declined and it was euthanized. Necropsy was not performed.

Discussion

Intranasal tumors are quite uncommon in dogs, accounting for only 1% to 2% of all neoplasms in that species.¹ Carcinomas compose 70% of the intranasal tumors; adenocarcinoma is the most commonly reported intranasal carcinoma, with sarcoma representing a smaller proportion; the exact percentage is unknown.²⁻⁴ However, investigation of 285 dogs revealed that nasal osteosarcomas (most commonly fibroblastic osteosarcomas) composed approximately 6% of nasal neoplasms.^{4,5} Nasal sarcomas are typically locally aggressive but generally nonmetastatic. Microscopic examination of lymph node aspirates may yield a diagnosis but usually has limited success.

Attempts to treat nasal and intranasal tumors often include chemotherapy, radiation therapy, and surgery, but the long-term prognosis is usually poor. The median survival time of untreated patients is approximately 3 to 6 months, and cytoreductive surgery alone does not increase survival time.⁶⁻⁸ Improvement of survival time by use of multimodal treatments has been investigated.^{1,9} The longest survival times have been associated with the use of radiation treatment, which can increase survival time to 8 to 25 months when combined with other treatments such as debulking surgeries or administration of chemotherapeutic agents.^{1,9} Approximately 70% of dogs with nasal osteosarcomas respond positively to radiation therapy, but the response rate among dogs with intranasal osteosarcomas is not known.⁹ The use of chemotherapeutic agents has been investigated, particularly the use of platinum agents. The use of cisplatin and radiotherapy has been reported to help increase survival times with nasal tumors.¹⁰ Administration of alternating IV doses of doxorubicin and carboplatin with the use of orally administered piroxicam is fairly efficacious in achieving complete or partial remission.¹

- a. Vista (I.OT), Picker International Inc, Highland Heights, Ohio.
- b. ProHance, Bracco Diagnostics Inc, Princeton, NJ.

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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. Submissions will be sent to Dr. Karen Kline, DVM, DACVIM, for her review.