



What Is Your Diagnosis?

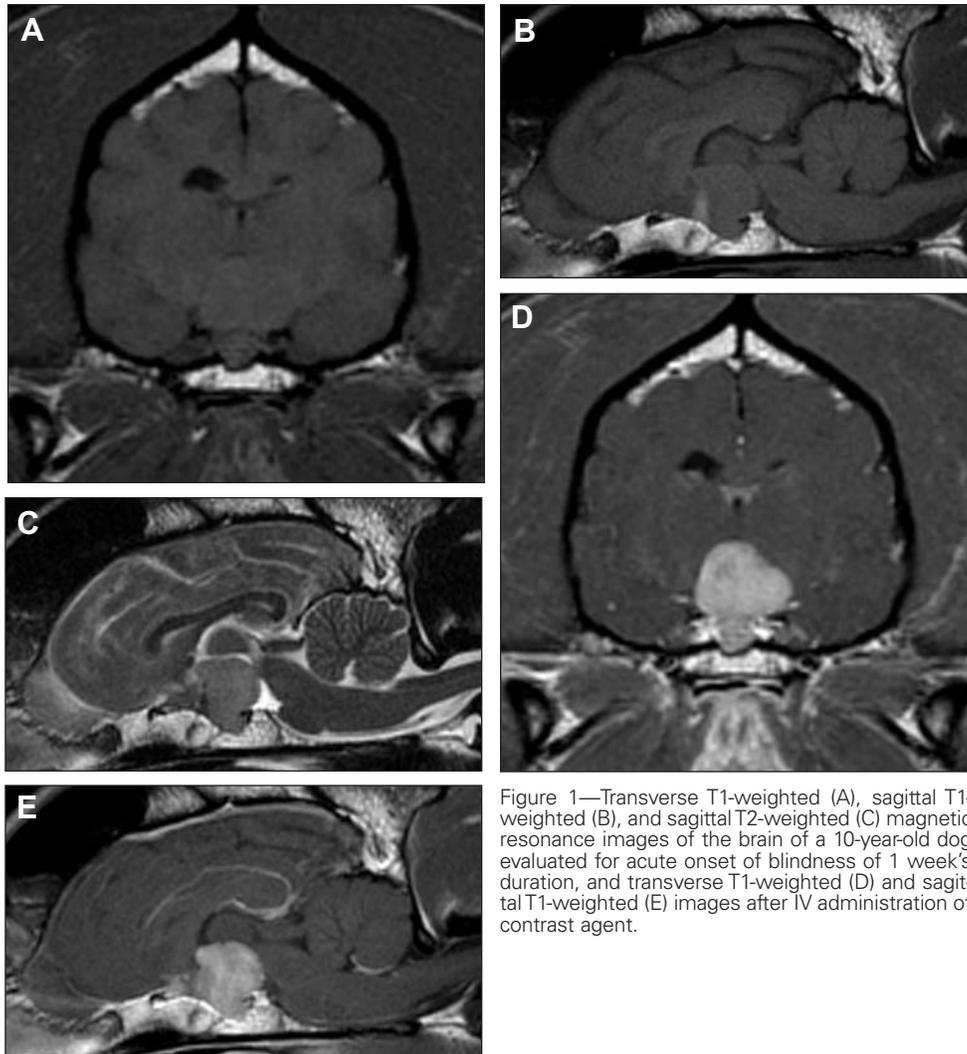


Figure 1—Transverse T1-weighted (A), sagittal T1-weighted (B), and sagittal T2-weighted (C) magnetic resonance images of the brain of a 10-year-old dog evaluated for acute onset of blindness of 1 week's duration, and transverse T1-weighted (D) and sagittal T1-weighted (E) images after IV administration of contrast agent.

History

A 10-year-old neutered male large mixed-breed dog was evaluated for acute onset of blindness of 1 week's duration. No abnormalities were detected on physical examination. The menace response and dazzle reflex were absent in both eyes, and the dog was unable to navigate a lighted examination room. The pupils were symmetrically dilated and unresponsive to direct and indirect light stimulation. Except for mild alterations in reflectivity within the tapetal retina, the remainder of the ophthalmic examination, including the appearance of the optic nerves, was considered normal. Abnormalities detected on serum biochemical analyses included high serum alkaline phosphatase activity (641 U/L; reference range, 5 to 131 U/L) and high serum triglycerides concentration (418 mg/dL; reference range, 29 to 191 mg/dL). A diagnosis of sudden acquired retinal degeneration syndrome was considered likely on the basis of these findings, and scotopic electroretinography was performed. The amplitudes were asymmetrical (80 and 153 μ V), but insufficiently low to account for the degree of observed vision loss. Ruling out a primary retinal disease in this manner suggested a retrobulbar nerve or brain lesion, which could be inflammatory or neoplastic in nature. Results of serologic tests for endemic infectious diseases were negative. Magnetic resonance imaging of the brain before and after IV administration of contrast agent (gadolinium) was performed during general anesthesia (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page ▶

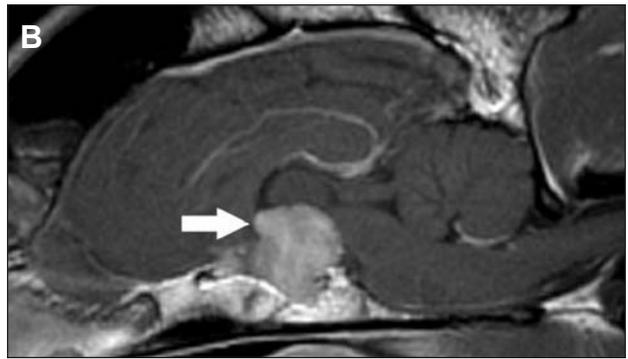
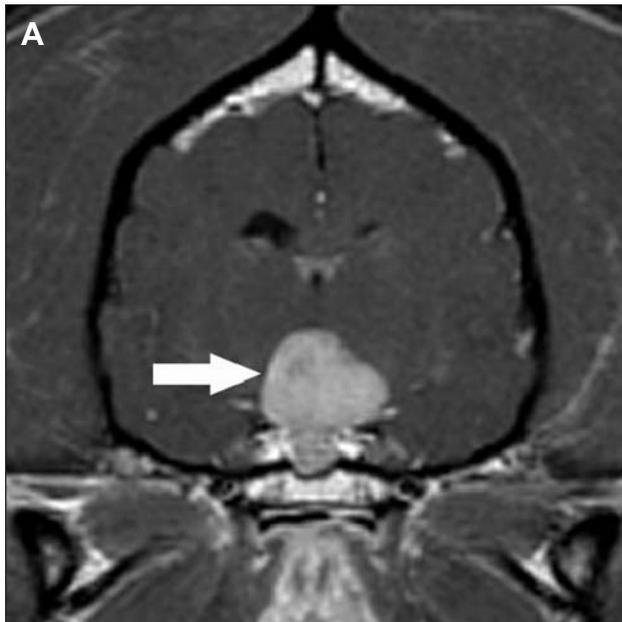


Figure 2—Contrast-enhanced transverse (A) and sagittal (B) T1-weighted magnetic resonance images as in Figure 1. Notice the sharply marginated mass originating from the area of the pituitary fossa and extending dorsally and rostrally to compress the diencephalon (arrow).

Diagnostic Imaging Findings and Interpretation

A sharply marginated mass originates from the area of the pituitary fossa (Figure 2). The mass is isointense to the surrounding brain parenchyma but homogeneously enhanced after administration of contrast agent. The mass extends dorsally and rostrally from the pituitary fossa and compresses the diencephalon. These imaging characteristics are most typical of a pituitary macroadenoma. In addition, a sharply marginated and somewhat angular area within the rostral aspect of the mass is hyperintense to the remainder of the mass and surrounding gray matter on T1-weighted images; it also has markedly reduced contrast enhancement, compared with the remainder of the mass. These imaging characteristics are most consistent with subacute hemorrhage and are believed to represent the magnetic susceptibility of intracellular methemoglobin.

Comments

Complete blindness in an adult dog is most likely caused by bilateral retinal disease, bilateral optic nerve or tract disease, complete optic chiasmal disease, or extensive destruction of the occipital cortex. After retinal and intraocular optic nerve diseases are ruled out by electroretinography and ophthalmic examination, respectively, advanced imaging techniques are indicated to evaluate the remainder of the visual pathway. In the dog of this report, bilateral vision loss was caused by compression of the optic chiasm or the postchiasmal tracts, or both, by the mass originating from the pituitary fossa. The imaging characteristics of this mass are typical of pituitary macroadenoma, although histologic confirmation would be required for definitive diagnosis. Less likely differential diagnoses include pituitary carcinoma, parapituitary meningioma, astrocytoma, oligodendroglioma, granular cell tumor, myxosarcoma, granulomatous meningoencephalitis, multicentric lymphosarcoma, and other rare neoplasms.

An ACTH-secreting pituitary adenoma is the most common cause of hyperadrenocorticism in dogs. Most of these are probably microadenomas, but 10% to 50%

of the cases of pituitary-dependent hyperadrenocorticism in dogs may be attributable to macroadenomas,¹ which are visible by advanced imaging techniques. Dogs with pituitary macroadenomas commonly have polyuria, polydipsia, and polyphagia, and may also have neurologic signs, including blindness.^{1,2} Clinical signs of hyperadrenocorticism were not observed by the owners of the dog reported here; however, further questioning indicated that the dog had unlimited access to a swimming pool, from which it drank unknown amounts of water. The high serum alkaline phosphatase activity and triglycerides concentration were also compatible with hyperadrenocorticism.

Surgical intervention, in the form of microsurgical transsphenoidal hypophysectomy³ or radiation therapy,¹ has been performed with some success for pituitary tumors in dogs. Hypophysectomy in dogs has induced hypernatremia, keratoconjunctivitis sicca, diabetes insipidus, and hypothyroidism.⁴ Irradiation is effective for treatment of pituitary macroadenomas and complications are acceptably low with small tumor sizes. Larger tumor size and more severe preoperative neurologic signs adversely affect prognosis.⁵ Presently, radiation therapy is the treatment of choice and was offered to the owners of this dog. All therapeutic intervention was declined because the large tumor size resulted in a guarded overall and visual prognosis.

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2. Davidson MG, Nasisse MP, Breitschwerdt EB, et al. Acute blindness associated with intracranial tumors in dogs and cats: eight cases (1984–1989). *J Am Vet Med Assoc* 1991;199:755–758.

3. Meij B, Voorhout G, Rijnberk A. Progress in transsphenoidal hypophysectomy for treatment of pituitary-dependent hyperadrenocorticism in dogs and cats. *Mol Cell Endocrinol* 2002;197:89–96.

4. Meij BP, Voorhout G, van den Ingh TS, et al. Results of transsphenoidal hypophysectomy in 52 dogs with pituitary-dependent hyperadrenocorticism. *Vet Surg* 1998;27:246–261.

5. Theon AP, Feldman EC. Megavoltage irradiation of pituitary macrotumors in dogs with neurologic signs. *J Am Vet Med Assoc* 1998;213:225–231.

This report was submitted by Gwendolyn L. Lynch, DVM; Michael R. Broome, DVM, MS, DABVP; and Randall H. Scagliotti, DVM, MS, DACVO; from Eye Care for Animals, 3025 Edinger Ave, Tustin, CA 92780 (Lynch, Scagliotti); and Advanced Veterinary Medical Imaging, 3047 Edinger Ave, Tustin, CA 92780 (Broome). Dr. Lynch's present address is Eye Care for Animals at City of Angels Veterinary Specialty Center, 9599 Jefferson Blvd, Culver City, CA 90232. Address correspondence to Dr. Lynch.