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Intracranial Arachnoid Cysts: Are They Clinically Significant?

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Intracranial arachnoid cysts constitute 1% of space-occupying lesions in humans.[1] The incidence of the condition in dogs is unknown. Only 6 reports describing radiographic findings of 13 affected dogs and 1 cat have been published, but detailed information about the clinical presentation of these patients is lacking.[2–7] The present report describes the clinical history, diagnostic findings, and long-term outcomes of 2 dogs in which the presence of arachnoid cysts was considered incidental.

Treatment options for arachnoid cysts include medical management or surgical intervention. In humans, surgical treatment consists of cyst fenestration or shunting and results in variable success rates.[8,9] In 5 dogs, fenestration was performed, and in 1 dog a shunt was implanted.[2,3,6,7] Clinical improvement was reported in 4 of the 5 dogs, with follow-up periods ranging from 2 months to 3.5 years.[2,3,6,7] One patient required a second fenestration procedure due to clinical deterioration after initial improvement. The dog that did not respond to surgery was euthanized after recurrent seizure activity. In humans, cysts have been reported as incidental findings at the time of autopsy. Intracranial arachnoid cysts also may be incidental findings in veterinary medicine, and affected patients should be evaluated carefully before surgical treatment is selected.

A 5-year-old male Shih Tzu dog was referred to the Ontario Veterinary College (OVC) with a 24-hour history of focal seizures characterized by facial twitching and excessive drooling. Before presentation, results of routine CBC and blood chemistry tests done by the referring veterinarian were within reference range. At that time, the dog was treated with IV fluids and methocarbamol[a] (22 mg/kg q8h). At admission to OVC, left-sided facial twitching was observed, and the dog reacted excessively to stimulation. Anisocoria, with the right pupil smaller than the left, was noted, with normal pupillary light reflexes. Fundic examination did not disclose any abnormalities. The seizures indicated a right-sided thalamocortical lesion, but the size of the right pupil could not be explained by this neuroanatomic localization and presumably was related to loss of left cortical inhibition over the right parasympathetic nucleus of the oculomotor nerve, indicating a left-sided lesion. Alternatively, irritation of the right parasympathetic nuclei could have resulted in anisocoria that, in combination with seizure activity, indicated a multifocal disorder. Cerebrospinal fluid (CSF) analysis identified a moderate pleocytosis with a white cell count of 18 cells/μL (reference range, ≤3 cells/μL). Cytology of 200 counted cells identified 62% small monocytoid cells, 20% lymphocytes, 17% large monocytoid cells, and 1% eosinophils. The protein concentration was slightly increased at 35 mg/dL (reference range, 0–30 mg/dL). Considering the acute onset of clinical signs and the results of CSF analysis, a tentative diagnosis of encephalitis was made. Differential diagnosis for the inflammation included infectious (eg, viral, rickettsial, fungal, protozoal) and noninfectious (eg, immune-mediated, idiopathic) causes. Serological titers for *Toxoplasma gondii*, *Neospora caninum*, and *Ehrlichia canis* were submitted and were negative.

The dog was treated with a constant-rate infusion of diazepam[b] (0.5 mg/kg/h for 12 hours) and phenobarbital[c] (1.5 mg/kg PO q12h). Clinical signs (eg, facial twitching, anisocoria) improved gradually, and 2 days later the dog was discharged from the hospital on prednisone[d] (0.5 mg/kg PO q12h), phenobarbital[c] (2 mg/kg PO q12h), and trimethroprim sulfamethoxazole[e] (20 mg/kg PO q12h). After discharge, the dog was somnolent, restless, and paced compulsively for 48 hours. The decision was made to perform a magnetic resonance imaging (MRI) scan of the brain. On the T1-weighted postcontrast sagittal images, a large circumscribed mass with sharply defined margins was observed between the caudal aspect of the cerebrum and the cerebellum. The lesion was hypointense relative to brain tissue and isointense relative to CSF (Fig 1). On transverse T2-weighted images, the mass was hyperintense relative to brain tissue and isointense relative to CSF. The primary differential diagnosis considered for this extra-axial CSF-filled mass was an arachnoid cyst of the quadrigeminal cisterna. Because of continued focal seizure activity and mental status deterioration, a caudotentorial craniotomy was performed, and the cyst was fenestrated. A sample of the cystic wall was submitted for histological evaluation and consisted of meningeal tissue with a mesothelial lining. No neoplastic or inflammatory cells were found. Evaluation of the fluid retrieved from the cyst revealed low cellularity with good cell preservation and no bacterial growth. Methylprednisolone[f] (30 mg/kg IV) was administered preoperatively 2 hours after the procedure started and again 4 hours later. Neurological signs improved dramatically postoperatively, and the dog was discharged on phenobarbital[c] (2 mg/kg/d PO) and prednisone[d] (1 mg/kg/d PO). No seizure activity, somnolence, or circling episodes were noted until 5 months after surgery. While on phenobarbital[c] treatment, the dog had 4 focal seizures, and treatment was begun again with prednisone[d] (1 mg/kg/d for 5 days followed by 0.5 mg/kg/d). Eleven months after surgery, while receiving phenobarbital[c] and prednisone,[d] the dog was euthanized because of recurrent seizure activity. At postmortem examination, the cystic lesion was identified as a thick-walled fluctuant mass overlying the cerebellum. Additionally, a mixed mononuclear inflammatory reaction (consisting of

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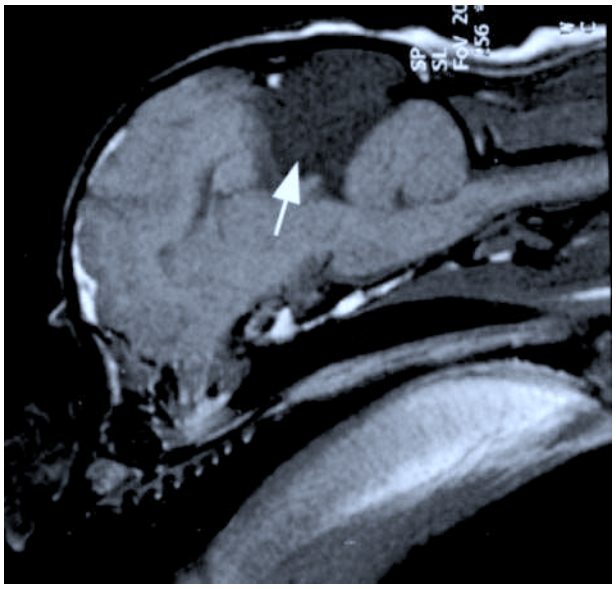

Fig 1. Dog #1: T1-weighted 3-mm–thick sagittal magnetic resonance imaging (MRI) image. Note the large hypointense cerebrospinal fluid (CSF)–filled cyst (white arrow). The intracranial arachnoid cyst is located in the quadrigeminal cisterna compressing the occipital lobe of the cerebrum rostrally and the cerebellum caudally.

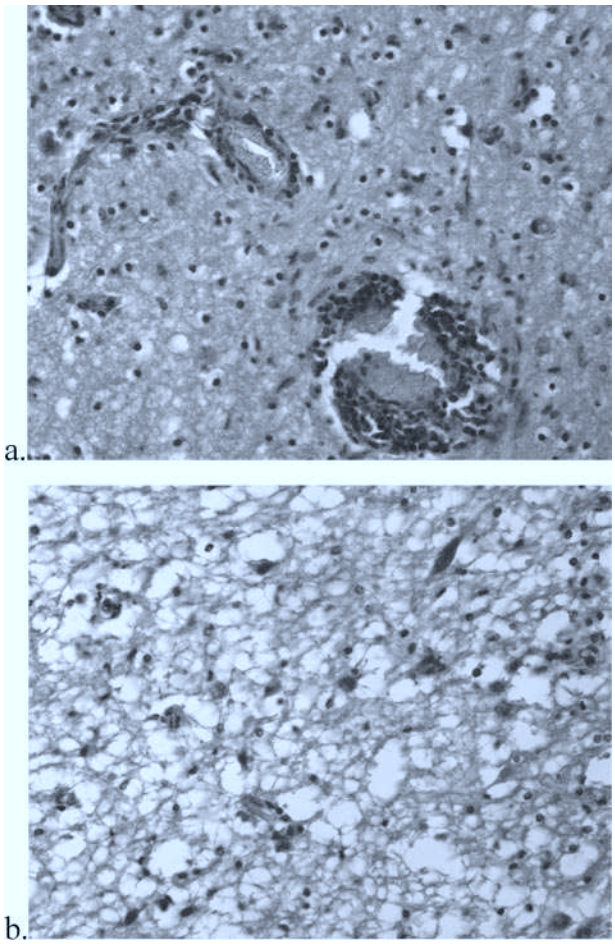

Fig 2. Dog #1: Hematoxylin and eosin (H&E) magnification 40×. **(a)** Note the nonsuppurative inflammatory infiltrate with presence of monocytic perivascular cuffs. **(b)** Diffuse necrosis with disruption of the normal cerebral architecture.

plasma cells, lymphocytes, and monocytes) involving the leptomeninges and thalamocortex was detected (Fig 2). A definitive diagnosis of necrotizing meningoencephalitis and intracranial subarachnoid cyst was made.

In the second case, a 9-week-old male Shih Tzu was presented to the OVC with a 3-day history of intention tremors and inability to walk. The dog had been treated with ampicillin[g] and diazepam[b] without improvement. Neurological examination identified an absent menace response (presumably age-related), hypermetria in all 4 limbs, and severe intention tremors. Routine CBC and blood chemistry results were normal. A CSF analysis disclosed a moderate pleocytosis with a white cell count of 27 cells/μL (reference range, ≤3 cells/μL). Cytology on 200 counted cells consisted of 42% monocytoid cells, 52% lymphocytes, 5% large foamy macrophages, and 1% neutrophils. Protein concentration was within the reference range at 26 mg/dL (normal, 0–30 mg/dL). Considering the acute onset of clinical signs, neurological abnormalities, and the results of the CSF analysis, a tentative diagnosis of encephalitis involving primarily the cerebellum was made. Differential diagnosis for the encephalitis included infectious and noninfectious causes. An MRI disclosed the presence of an intracranial arachnoid quadrigeminal cisternal cyst that was hypointense on T1-weighted images and hyperintense on T2-weighted images (Fig 3). Clinical improvement was noted after treatment with prednisolone acetate phosphate[h] (0.5 mg/kg PO q12h for 3 days, followed by 0.5 mg/kg/d for 7 days). The dog has remained normal for 29 months after completion of the anti-inflammatory treatment.

Despite numerous reports of human patients with improvement of neurological signs after treatment of arachnoid cysts, the disorder also has been recognized as an incidental finding at the time of autopsy.[10] The acute onset of neurological signs, CSF results, and postmortem diagnosis of necrotizing meningoencephalitis observed in dog 1 support the contention that the cystic structure may have been only an incidental finding. Although remarkable clinical improvement was noted after surgical fenestration, corticosteroid therapy at time of surgery may have been responsible for improvement. The second dog described in this report clearly supports the contention that the cystic structure was incidental, because the patient remained normal 29 months after stopping anti-inflammatory therapy. Failure of the clinical signs to localize the lesion to the site of the cyst in both of the dogs described here provides additional evidence that the radiological findings were not clinically relevant.

Seizures appear to be a common manifestation in human patients and animals with intracranial cysts.[2,4,5,7] According to the literature, 7 of the 14 affected animals (1 cat, 6 dogs) had seizure activity. Resolution of seizures was attributed to surgical intervention in 3 of the 6 affected dogs. In the first dog described here, seizure activity could have been due to underlying inflammatory disease, despite the cystic lesion identified on MRI. Unfortunately, the results of the CSF analysis have only been described in 3 of the 13 pre-

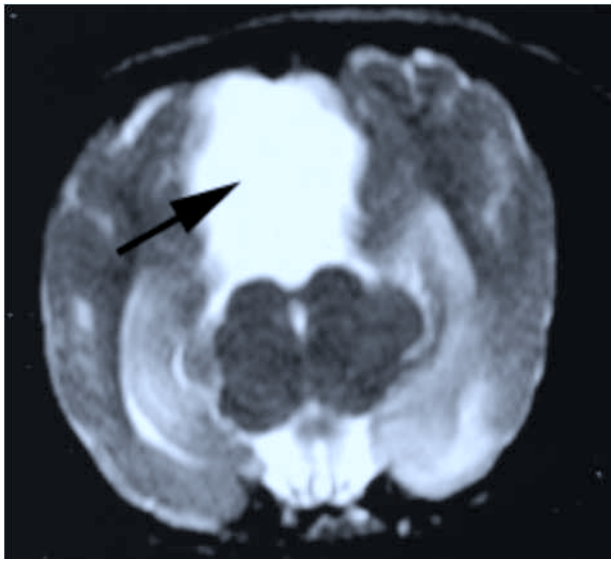

Fig 3. Dog #2: T2-weighted 3-mm–thick transverse magnetic resonance imaging (MRI) image at the level of the midbrain. Note the intracranial arachnoid cyst indicated by the arrow. The cyst is hyperintense to brain tissue, isointense to cerebrospinal fluid (CSF), and is located dorsally to the midbrain.

viously reported dogs.[6,7] Moreover, in 2 of these 3 dogs, intracystic hemorrhage was suspected and made interpretation of the sample difficult.[6] The third animal reported had an increase in CSF protein concentration with normal cell count.[7]

It is unknown if a breed predilection exists for this condition. Interestingly, 5 of the 13 dogs previously reported as having intracranial cysts were Pugs or Shih Tzus.[2,6] Similarly, the 2 dogs in the present report were Shih Tzus.

Clinical improvement (decreased seizure frequency, improved learning abilities) is reported in some children when intracranial cysts are treated surgically early in life.[8,9] In humans, surgical treatment of large arachnoid cysts is recommended after inflammation, neoplasia, or other pathology has been ruled out as the cause of clinical signs.[11] Guidelines are less clear for asymptomatic patients with cysts. Some neurologists advocate prophylactic fenestration to prevent traumatic rupture of veins crossing the cyst that could cause neurological impairment.[10]

In human patients, intracranial arachnoid cysts may increase dramatically in size, leading to CSF flow obstruction and clinical manifestation of prosencephalic signs. This outcome does not appear to occur in dogs. Therefore, when deciding about the clinical relevance of intracranial cysts in dogs, the signalment, CSF analysis, and correlation of clinical and neuroimaging findings must be thoroughly evaluated.

Footnotes

[a] Robaxin-V, Fort Dodge Animal Health, Fort Dodge, IA
[b] Diazepam, Roche Laboratories, Toronto, Ontario, Canada
[c] Phenobarbital, Pharmascience, Montreal, Quebec, Canada
[d] Apo-prednisone, Apotex, Toronto, Ontario, Canada
[e] Septra, Apotex, Toronto, Ontario, Canada
[f] Methylprednisolone, Pharm & Chem Co Ltd, Toronto, Ontario, Canada
[g] Ampicillin, Pharm & Chem Co Ltd, Toronto, Ontario, Canada
[h] Prednisolone acetate, Pharmascience, Montreal, Quebec, Canada

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