Clinical and MRI Findings in Three Dogs with Polycystic Meningiomas

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ABSTRACT

One spayed female Labrador retriever and two castrated male golden retrievers were evaluated for chronic (i.e., ranging from 3 wk to 24 wk) neurologic signs localizable to the prosencephalon. Signs included seizures, circling, and behavior changes. MRI demonstrated extra-axial, contrast-enhancing, multiloculated, fluid-filled, cyst-like lesions with a mass effect, causing compression and displacement of brain parenchyma. Differential diagnoses included cystic neoplasm, abscess or other infectious cyst (e.g., alveolar hydatid cyst), or fluid-filled anomaly (e.g., arachnoid cyst). The cyst-like lesions were attached to the rostral falx cerebri in all cases. In addition, case 2 had a second polycystic mass at the caudal diencephalon. Surgical biopsy (case 3 with a single, rostral tumor via transfrontal craniectomy) and postmortem histology (in cases 1 and 2) confirmed polycystic meningiomas. Tumor types were transitional (cases 1 and 3) and fibrous (case 2), with positive immunohistochemical staining for vimentin. Case 3 was also positive for E-cadherin, s100, and CD34. In all cases, staining was predominantly negative for glial fibrillary acid protein and pancytokeratins, supporting a diagnosis of meningioma. This report describes the first cases of polycystic meningiomas in dogs. Polycystic meningiomas are a rare, but important, addition to the differential diagnoses for intracranial cyst-like lesions, significantly affecting planning for surgical resection and other therapeutic interventions. (*J Am Anim Hosp Assoc* 2012; 48:331–338. DOI 10.5326/JAAHA-MS-5774)

Introduction

Meningiomas are one of the most common primary intracranial tumors in dogs, and 33–49% of dogs with primary brain tumors have meningiomas.[1–3] Cystic meningiomas are a rare subtype of meningiomas, with few reports in the literature.[4–8] Although the prevalence of cystic meningiomas has not been established in dogs, between 2% and 4% of meningiomas in humans are cystic.[9] Polycystic meningiomas are reportedly even more rare in humans.[10,11] To the best of the authors' knowledge, this report describes the first three cases of polycystic meningiomas documented in dogs.

Case Report

Case 1

An 8 yr old spayed female Labrador retriever was referred to the Ontario Veterinary College Small Animal Clinic with a 1-mo history of behavior changes and increasing mental dullness. There was an initial increase in aggression toward people then a significant decrease in response to the owner and, increasingly, episodes of head-pressing, house-soiling, panting, pacing, circling to the right, and nightly vocalizations. The dog was receiving daily meloxicam[a] and glucosamine (brand unknown) for chronic degenerative disease of the hip and stifle joints.

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CSF *cerebrospinal fluid;* FLAIR *fluid-attenuated inversion recovery;* IHC *immunohistochemistry;* T1WI *T1-weighted images;* T2WI *T2-weighted images;* TE *time to echo;* TR *repetition time*

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Physical examination revealed no abnormalities. Neurologic examination revealed dullness and disorientation. The dog paced compulsively, circling to the right. The rest of the neurologic examination was normal. Based on the history and neurologic examination, the lesion was localized to the right prosencephalon. Complete blood count, serum biochemistry profile, and total thyroxine were normal.

MRI of the brain was performed under general anesthesia with a 1.5T GE Signa EXCITE II[b]. Fast spin-echo T1-weighted images (T1WI) used a repetition time (TR) of 600 msec and a time to echo (TE) of 12.4 msec. Fast spin-echo T2-weighted images (T2WI) used a TR of 4,250 msec and a TE of 88.9 msec. Fluid-attenuated inversion recovery (FLAIR) T2WI used a TR of 8,002 msec and a TE of 130.1 msec. Sagittal, dorsal, and transverse images were obtained. T1WI were repeated following the administration of a gadodiamide contrast agent[c] (0.1 mmol/kg IV).

In the rostral prosencephalon, centered on the falx cerebri, extending caudally from the cribriform plate and expanding into the olfactory bulb and frontal lobes, was a well-circumscribed, multiloculated structure that appeared separated from the ventricles by a thin wall. The structure appeared extra-axial, bulging out from the falx cerebri and exerting a mass effect with both caudal and lateral displacement and compression of the cerebral hemispheres and lateral ventricles. The contents of the structure were homogeneously hyperintense to cerebrospinal fluid (CSF) on T2WI (**Figure 1A**) and FLAIR images. On T1WI, the contents were homogeneously hypointense to the brain; however, the contents were hyperintense to CSF. On FLAIR images, there was a thin rim of increased signal intensity in the brain tissue around the lesion, consistent with vasogenic edema. Comparing pre- and postcontrast T1WIs, there was a focal area of marked contrast enhancement along the falx cerebri, extending caudally from the cribriform plate (**Figures 1B, C**).

The progression of clinical signs, together with the physical, neurologic, and laboratory findings, were suggestive of a neoplastic process, although an infectious or an inflammatory, noninfectious (e.g., granulomatous meningoencephalitis) etiology could not be excluded from consideration. Based on the MRI findings of a focal area of marked contrast enhancement in the region of the falx cerebri; the extra-axial, expansile, space-occupying nature of the mass; and its multiloculated appearance, polycystic meningioma or another cavitated neoplasm were the main differential diagnoses. Other possibilities, including abscess or other infectious cysts (e.g., cysticercus or alveolar hydatid cyst) or fluid-filled anomalies (e.g., dermoid or arachnoid cysts) were considered. An arachnoid cyst was considered unlikely because on FLAIR images, the structure's contents were hyperintense to CSF, suggesting a more proteinaceous fluid than CSF. Further diagnostics and therapy were declined, and euthanasia was requested due to the combination of both the severity of the clinical signs and the diagnosis of a probable neoplasia.

Postmortem examination revealed a focal, irregularly shaped, 2 cm × 1 cm × 1 cm cavitated structure in the rostral cerebrum that extended caudally to approximately 1–2 cm rostral to the corpus callosum that did not communicate with the ventricular system. Extending into the lumen of the structure was a flattened gray-brown isthmus of dura mater.

Microscopically, the tissue mass was densely cellular. An irregular network of anastomosing cords of variably sized cells was intermingled with collagenous stroma. The cell morphology varied from round to spindle-shaped, with cell sizes ranging from 10 μm to 20 μm × 20 μm to 40 μm. The cells had scant to moderate amounts of eosinophilic vacuolated cytoplasm, round to oval nuclei with stippled chromatin, moderate anisokaryosis, and a single nucleolus. The cell borders were indistinct and they were arranged in streams and whorls consistent with a transitional (mixed) meningioma. No mitotic figures were observed. At the junction between the neoplasm and brain, the brain parenchyma was irregular and had localized, multifocal, lymphocytic, perivascular cuffing, and gliosis.

Immunohistochemistry (IHC) was performed on the neoplasm. The cells showed diffuse and variably intense staining with antibodies to vimentin[d]. Using a pancytokeratin antibody[e], there was multifocal staining involving approximately 20% of the cells, typically those cells arranged in small clusters or whorls. This IHC pattern was supportive of the diagnosis of meningioma.[12]

Case 2

A 9 yr old castrated male golden retriever presented to the Ontario Veterinary College Small Animal Clinic, with a 3-wk history of abnormal behavior and pacing and a 1-wk history of right-sided circling and head pressing. Physical examination revealed no abnormalities. On neurologic examination, the dog displayed inappropriate behavior, decreased menace response in the right eye, compulsive circling to the right side, hypertonicity in the left pelvic limb, and delayed proprioceptive positioning in both pelvic limbs and in the left thoracic limb. Based on the neurologic findings, a predominantly right-sided prosencephalic lesion was suspected based on the compulsive circling to the right and the left-sided thoracic limb proprioceptive deficit. However, it was difficult to reconcile the bilateral postural deficits in the pelvic limbs and the right-sided menace deficit with a right-sided lesion. Therefore, the neurologic exam findings could also have been the result of either a right-sided prosencephalic lesion that also involved the midline

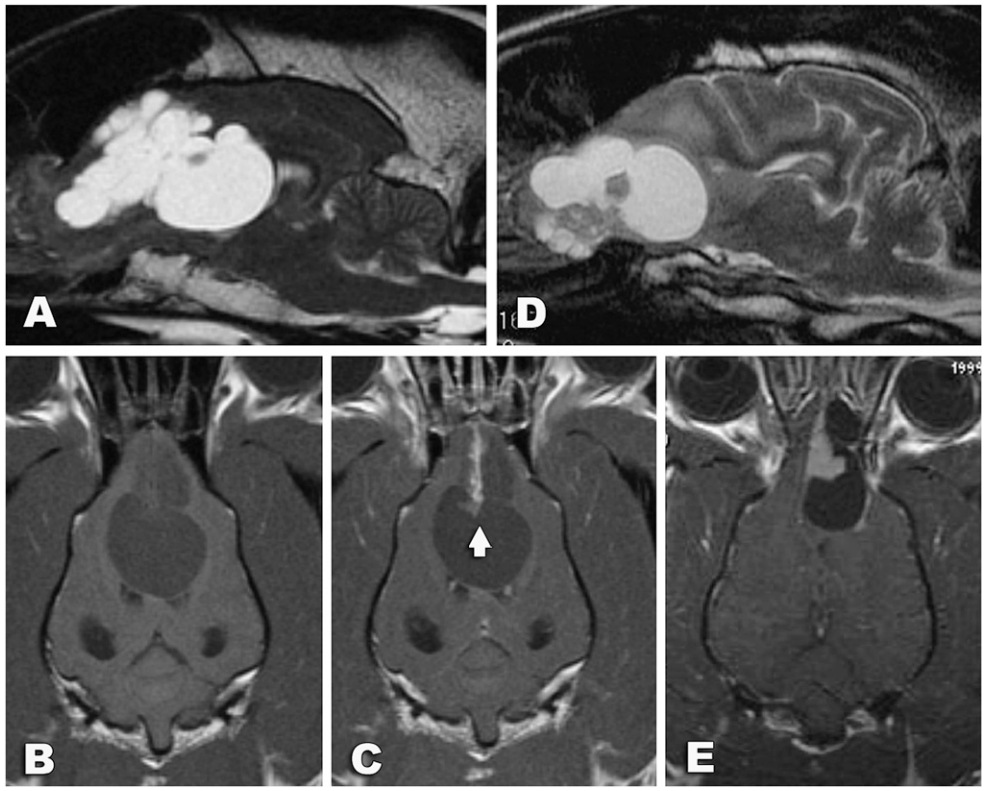

FIGURE 1 *MRI of the brain of the 8 yr old spayed female Labrador retriever described in case 1* (A, B, C) *and the 11 yr old castrated male golden retriever described in case 3* (D, E) *that presented with neurologic signs.* A*: A sagittal T2-weighted image (T2WI) showing a large, multiloculated, hyperintense structure occupying the frontal lobe.* B*: A dorsal T1-weighted image (T1WI) prior to IV contrast administration.* C*: Same image as in B, taken after IV contrast administration. Comparing images B and C, note the enhancement of the midline, suggesting involvement of the falx cerebri (arrow).* D*: A sagittal T2WI showing a large, multiloculated, hyperintense structure compressing the olfactory bulbs and frontal lobes.* E*: Dorsal plane T1WI postcontrast showing a hyperintense, well-demarcated structure present within a hypointense, well-demarcated mass on the left frontal lobe of the cerebrum.*

or a multifocal disease. Complete blood count, serum biochemistry profile, and urinalysis were normal.

MRI[b] was performed using similar protocols as in case 1. At the level of the caudal diencephalon and mesencephalon, a large, bipartite structure displaced the brain parenchyma dorsally and to the right side. The structure had a homogeneous intensity and was sharply demarcated from the surrounding parenchyma (**Figures 2A, B**). Ventral to this large structure, there were also three other smaller loculi. Ventrally, within the large structure, there was a smaller, circular structure that was isointense to normal brain parenchyma that had a well-delineated border with the larger structure (Figure 2B). Rostrally, at the level of the olfactory bulbs, two other sharply demarcated, oval structures were observed (**Figure 2C**). The largest was on the right side. All three structures had signal intensities similar to CSF on T1WI, T2WI, and FLAIR images. All imaging features were compatible with fluid-filled structures. Postcontrast T1WI revealed marked contrast enhancement ventral to the brainstem from the caudal diencephalon to the rostral medulla oblongata (Figure 2C). Contrast enhancement was also noted surrounding the olfactory lobe lesion, mostly dorsally and on the right side. The postcontrast images suggested that the structure was predominantly extra-axial. Differential diagnoses for these multiloculated, fluid-filled structures noted on MRI were identical to those of case 1, with the addition of arachnoid cysts due to the similarity in MRI signal characteristics between the CSF and the structures' contents.

Further diagnostic tests were performed to search for systemic disease. Ophthalmic examination revealed only nuclear sclerosis in both lenses and incipient cortical cataracts. Thoracic radiographs and an abdominal ultrasound were unremarkable. The analysis of CSF obtained from the cerebellomedullary cistern was normal. No ova were identified in a modified Cornell-Wisconsin centrifugal fecal floatation test. Based on the MRI findings and results of the

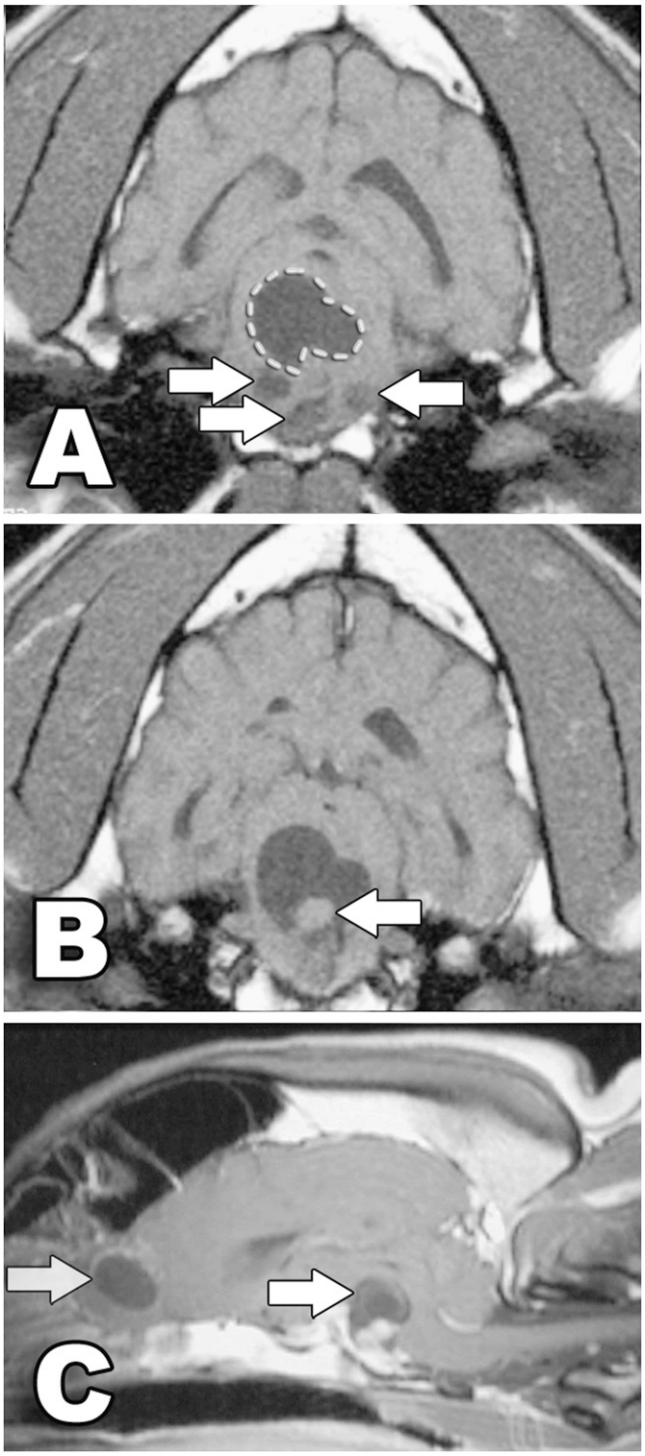

FIGURE 2 *MRI of the 9 yr old castrated male golden retriever described in case 2 with neurologic signs.* A*: Transverse T1WI showing a large, hypointense, well-delineated, bipartite structure at the level of mesencephalon. This large hypointense structure extended mostly to the right (*dotted outline*). Ventrally, three other small structures can be observed (*arrows*).* B*: Transverse T1WI showing the same hypointense structure at the level of mesencephalon as in* A*. At the ventral aspect of the large structure, an isointense circular area is seen (*arrow*). This image of a well-defined circular area, with the isointense area inside, was considered compatible with the descriptions of "hole with a dot".* C*: Sagittal T1WI postcontrast showing two large, hypointense, well-delineated structures. One was at the level of the olfactory bulbs and the other was in the brainstem (*arrows*). Ventral to the structure on the brainstem, marked contrast enhancement can be seen. Heterogeneous contrast enhancement is also obvious dorsal to the olfactory bulb structure.*

additional tests, the primary differentials were parasitic or neoplastic cysts.

The round, isointense area inside of the large cavity (Figure 2B) was consistent with a parasite scolex.[13] Therefore, a therapeutic trial was started with oral praziquantel[f] (5 mg/kg *q* 24 hr for 2 wk) followed by oral albendazole[g] (25 mg/kg *q* 12 hr for 10 days). Oral prednisone[h] (1 mg/kg *q* 24 hr for 2 wk then 0.5 mg/kg *q* 24 hr for 2 wk) was prescribed at the same time as the praziquantel. Two weeks after treatment initiation, the dog had improved remarkably; however, 4 days after the albendazole was initiated, the dog became markedly lethargic, weak, and started vomiting. Complete blood count showed pancytopenia consistent with bone marrow suppression secondary to albendazole therapy. All medications, including both the praziquantel and albendazole, were discontinued, and the dog was treated with a proton pump inhibitor (omeprazole, 0.7 mg/kg q 24 hr for 10 days) [i], a gastrointestinal promotility agent (metoclopramide 0.02 mg/kg/hr intravenous constant rate infusion pro re nata) [j], and antibiotics (ampicillin 22 mg/kg intravenous q 8 hr[k] and enrofloxacin 5 mg/kg intravenous q 24 hr) [l]. He also received IV fluid therapy[m], a transfusion of fresh frozen plasma[n], and a granulocyte colony-stimulating factor (filgrastim, 5 ug/kg subcutaneously q 24 hr for 7 days)[o]. The dog improved progressively and was discharged 8 days later. Specifically, he stopped vomiting on the third day of hospitalization, started eating and drinking on the sixth day, and had a normal neutrophil count on the seventh day. After the dog had fully recovered from bone marrow suppression, prednisone therapy was reinstituted (1 mg/kg *q* 24 hr for 2 wk then 0.5 mg/kg *q* 24 hr indefinitely)[h].

A follow-up MRI was performed 6 wk after the antiparasitic treatment was originally prescribed. The lesions were present and similar as in the first MRI, except a more uniform and intense contrast enhancement surrounding the fluid-filled lesions was noted. The dog's clinical signs varied over the next few weeks, and the prednisone dosage was adjusted accordingly until a more severe deterioration occurred 3 mo after initial presentation. Euthanasia was elected.

At necropsy, an irregular structure measuring approximately 2.5 cm × 1.5 cm was identified at the base of the mesencephalon, with a large cyst-like structure dorsal to it. Histologically, the structure was composed of variably sized, fusiform to polygonal cells with oval vesicular to euchromatic nuclei. Those cells had an abundant, pale eosinophilic cytoplasm. The nucleus-to-cytoplasmic ratio was approximately 1:2, and there was mild anisokaryosis and anisocytosis. Mitotic figures were not evident. In some areas, the cells were densely packed in sheets, occasionally in syncytial whorls. Some of the larger sections of the structure that had small cavitations also had poorly cellular areas at their periphery characterized by abundant, pale plaques of eosinophilic stroma and scant clusters of fusiform cells. The neuropil surrounding the large cavity was vacuolated. The histologic diagnosis was fibrous meningioma.

IHC was performed. More than 75% of the cells within the neoplasm stained strongly with antibodies to vimentin[d]. The cells did not stain with antibodies to cytokeratins (pancytokeratin[e], and the high molecular weight cytokeratin[p]), or glial fibrillary astrocytic protein[q]. The IHC staining pattern was supportive of the diagnosis of meningioma.[12,14]

Case 3

An 11 yr old castrated male golden retriever presented to Carolina Veterinary Specialists with a 6-mo history of seizures and a recent episode of cluster seizures. An oral melanoma had been treated with surgical excision and follow-up melanoma vaccine 2 yr prior to presentation. The dog was being treated with phenobarbital[r] for seizures and deracoxib[s] for chronic osteoarthritis affecting multiple joints.

Physical examination revealed no abnormalities. The dog reportedly had nine seizures over the 24 hr prior to presentation and experienced a seizure on initial presentation. Diazepam[t] (0.5 mg/kg) was administered IV, and the seizure activity ceased. On initial neurologic examination, the dog was sedate and ataxic. There was an absent menace response in both eyes. The remainder of the neurologic examination was normal. Based on the history of seizures, the lesion was localized to the prosencephalon. Results of a complete blood cell count and serum biochemistry profile were normal. The serum phenobarbital level was 32.6 mg/mL (reference range, 15–45 mg/mL).

MRI of the brain was performed under general anesthesia with a 1.5T GE Signa Horizon[u]. T1WI used a TR of 600 msec and a TE of 18 msec. T2WI used a TR of 4,716.7 msec and a TE of 83.8 msec. FLAIR T2WI used a TR of 9,002 msec and a TE of 127.5 msec. Both the sagittal and transverse planes were imaged. T1WI in transverse and dorsal planes were obtained following the IV administration of 0.1 mmol/kg gadopentetate dimeglumine[v].

One 3.6 cm × 1.6 cm × 2 cm, well-demarcated, multiloculated structure was present in the left olfactory and frontal lobes of the cerebrum, displacing the left frontal lobe caudally and causing deviation of the falx cerebri to the right. Similar to the other two cases described above, the structure had imaging features consistent with fluid content (**Figure 1D**). The structure did not appear to be associated with the ventricles. On transverse images, the structure appeared homogeneous and was situated dorsally at the level of the olfactory bulb and frontal cortex. Postcontrast T1WI revealed a well-demarcated 1.5 cm × 0.8 cm × 1.8 cm contrast-enhancing mass that appeared to be associated with the falx cerebri (**Figure 1E**). The mass was surrounded laterally and dorsally by the hypointense structure.

Based on the MRI findings, the differential diagnoses were similar to cases 1 and 2. A transfrontal craniectomy was performed, and a moderate amount of gray, glistening soft tissue was removed from adjacent to the left side of the falx cerebri. There was a large amount of fluid surrounding the mass, and the fluid was passively drained and suctioned during surgery. When no additional abnormal tissue could be palpated along the falx, blood-soaked cellulose sponges were placed over the craniectomy site, and the surgical site was closed routinely. The dog recovered uneventfully from anesthesia and was discharged from the hospital 3 days following surgery with oral phenobarbital[r] (4.5 mg/kg *q* 12 hr), oral tramadol[w] (2.8 mg/kg *q* 8–12 hr *pro re nata*), and oral prednisone[h] (1 mg/kg *q* 24 hr for 3 days then decreased to 0.5 mg/kg *q* 24 hr for 11 days).

The dog presented 2 wk later for a scheduled reevaluation. The dog had not had any seizures and was ambulatory, but the owner reported that the dog had difficulty rising and had had intermittent episodes of urinating in the house. On examination, the dog seemed painful on manipulation of the coxofemoral joints and elbows. The tramadol was discontinued, and the prednisone was decreased to 0.25 mg/kg *q* 24 hr for 3 days then discontinued. The phenobarbital was continued as previously prescribed. Following the cessation of the prednisone, the dog was prescribed deracoxib[s] for the osteoarthritis.

The surgically excised tissue was submitted for histopathologic evaluation. Microscopically, the tissue had moderate to dense cellularity with polygonal to spindloid cells arranged mostly in a random fashion with scattered whorls. The cells had an oval hyperchromatic nucleus with moderate variation in nuclear diameter and either a small or indistinct nucleolus. The cells had ample eosinophilic, lacy cytoplasm with indistinct cell borders. A few papillary projections at the periphery of the larger tissue

specimens were bordered by these cells. In scattered areas of the neoplasia, vacuolar degeneration with pyknosis was observed. There were no mitotic figures in 10 high-power (400×) fields. Vascularity was prominent. The histologic diagnosis was meningioma, and transitional meningioma was considered most likely given that the cells ranged from polygonal to spindloid and formed a few whorls.

IHC was performed, and neoplastic cells diffusely and strongly expressed E-cadherin[x]. Neoplastic cells were also positive for vimentin[d] and CD34[y]. Many cells were immunoreactive for S100[z]. The cells were uniformly negative for glial fibrillary astrocyte protein[q] and pancytokeratins[e]. The IHC results supported the diagnosis of meningioma and ruled out other possibilities, such as astrocytoma and metastatic carcinoma.[12,14]

Discussion

The three dogs in this report were retrievers (two goldens and one Labrador). Golden retrievers and Labrador retrievers are predisposed to brain tumors.[7,15,16] Other cases of cystic meningiomas were reported in individuals of the following breeds: Scottish terrier, mixed-breed, American Eskimo, and Maltese.[4–6,8] One large retrospective case series reported 29 meningiomas with large, cyst-like structures, but did not find an association with anatomic location, signalment, tumor grade, histologic subtype, or surrounding meningeal involvement.[7]

The use of the terms "cyst" and "polycystic" may be misconstrued as these fluid-filled cavities are not true cysts with epithelial linings. However, the term "cyst" has been associated with meningiomas since the earliest descriptions.[17] In those descriptions, the term "cyst" was used to describe a fluid-filled cavity grossly resembling a cyst. This broader sense of the term is likely how it has been perpetuated in the literature. More recent uses of the term "polycystic" seem to stem from the multiloculated appearance of the mass on imaging.[10,11]

Referred to as the Nauta classification scheme, cysts associated with meningiomas have been classified into four configurations: those located centrally within the tumor (type I), peripherally within the tumor (type II), within the adjacent brain (type III), and at the interface of tumor and brain (type IV).[18] In all three cases described herein, the cysts were located peripherally within the meningioma (Nauta type II) (**Figure 3**). Theories for cyst formation within the meningioma include tumor necrosis and aggregation of microcytes, entrapment of a portion of the ventricular system, CSF imbibition by the tumor, or fluid production by the tumor itself.[1,8,19] The mechanisms for Nauta type II cyst formation likely vary from case to case. For example, case 1 in this report had MRI characteristics that were distinct from CSF,

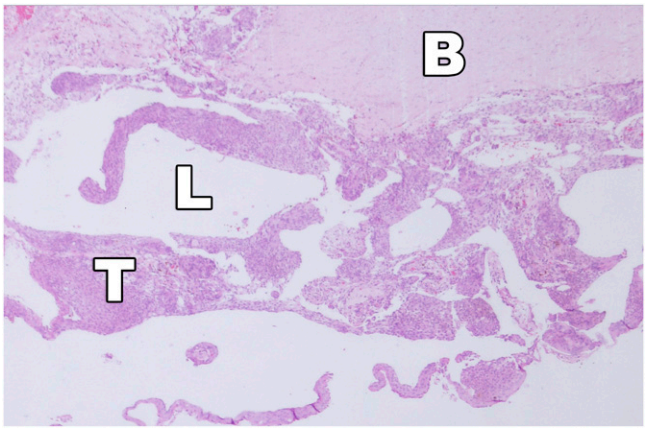

FIGURE 3 *Histopathology of the meningioma diagnosed in the 8 yr old female spayed Labrador retriever (case 1). Neoplastic cells (*T*) are obvious between the brain parenchyma (*B*, the pale pink region at the top right of the image) and the cyst lumen (*L*, the white space), forming tendrils and sheets that extend into the cyst lumen. Hematoxylin and eosin staining, original magnification ×2.*

suggesting a more proteinaceous fluid than CSF. The cyst contents in the other two cases displayed similar MRI characteristics to CSF. However, it is notable that all three cases described herein were classified as Nauta type II, as were two of the reported cases of polycystic meningiomas in humans.[10] The third reported polycystic meningioma in a human did not describe the cyst location relative to the tumor.[11] The pathophysiology question may be better settled by more detailed investigation of tumor contents and surrounding tissue as further cases of this rare tumor phenotype are discovered.

To the best of the authors' knowledge, case 2 in this report is the first example of multiple polycystic meningiomas in a dog. Only six cases of multiple meningiomas have been reported in dogs, none of which were polycystic.[7,20,21] Multiple meningiomas are more common in cats, comprising approximately 17% of meningiomas.[22]

MRI is an ideal modality to identify polycystic meningiomas. The low signal intensity on T1WI and FLAIR images, together with the high signal intensity on T2WI, point toward the fluid nature of the mass, and gadodiamide enhancement highlights the tumor core. These characteristics are consistent with those published for human cystic meningiomas in which Nauta's types II and III do not show enhancement of the cyst wall, but the solid tumor component does display enhancement.[9] With surgical resection of meningiomas becoming more common, attention should be given to classification of the cystic meningioma subtypes and to the constitution of the cyst wall because these factors may impact totality of resection.[23]

Differential diagnoses for intracranial polycystic lesions found on MRI include cystic meningioma or other cystic neoplasms, fluid-filled anomalies (e.g., dermoid or arachnoid cysts), and abscesses or other infectious cysts (e.g., cysticercus or alveolar hydatid cyst). The diagnosis of neurocysticercosis has, as an absolute diagnostic criterion, appears as a "hole with a dot" (i.e., a cystic lesion showing the scolex) on MRI.[13] It was believed that the dog in case 2 had imaging findings possibly consistent with neurocysticercosis; therefore, a fecal floatation was performed. A fecal floatation positive for *Taenia*-type eggs would have provided potential confirmatory evidence of infection when occurring simultaneously with central nervous system signs and a consistent MRI. In contrast, a negative result would not have ruled out this differential diagnosis. A polymerase chain reaction test on the eggs would have been required to definitively identify the parasite.[24] For the dog in case 2, an additional consideration was that it resided in a geographical region in Ontario where neurocysticercosis caused by *Taenia crassiceps* had been reported in a woodchuck.[25] Neurocysticercosis had been previously diagnosed in a dog using MRI.[26] As a result, neurocysticercosis due to this parasite was tentatively diagnosed, and a therapeutic trial initiated. With the follow-up MRI and the waxing and waning evolution of clinical signs during treatment, it became apparent that the cysts were more likely associated with a neoplasm like a meningioma. This suspicion was later confirmed on histologic and IHC evaluations. Other antemortem diagnostic tests to help differentiate between the possible etiologies of the cystic lesion included CSF analysis (as performed in case 2) and lesion biopsy for histopathology (as performed in case 3).

Conclusion

This report describes the MRI and histopathology characteristics of three cases of canine polycystic meningiomas. Although rare, polycystic meningioma is an important differential diagnosis for multiloculated, fluid-filled intracranial lesions in dogs. They should be considered along with parasitic cysts (e.g., neurocysticercosis) in the differential diagnoses, especially in light of geographic location and travel history. Other diagnostic tests to potentially help differentiate between possible etiologies include fecal floatation, CSF and analysis, and lesion biopsy for histopathology and IHC. This report aims to increase awareness of this presentation because the management of parasitic and neoplastic lesions differs greatly.

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FOOTNOTES

[a] Metacam; Boehringer Ingelheim Vetmedica Inc., Burlington, Ontario, Canada

[b] 1.5 T GE Signa EXCITE II; GE Healthcare, Milwaukee, WI

[c] Omniscan; Amersham Health Inc., Oakville, Ontario, Canada

[d] Dako mouse monoclonal antibody clone Vim 3B4 diluted 1:200; Dako Canada Inc., Burlington, Ontario, Canada

[e] Dako mouse monoclonal antibody clone AE1 AE3 diluted 1:100; Dako Canada Inc., Burlington, Ontario, Canada

[f] Droncit; Bayer HealthCare, Bayer Inc., Toronto, Ontario, Canada

[g] Valbazen; Pfizer Animal Health, Pfizer Canada Inc., Kirkland, Quebec, Canada

[h] Apo-prednisone; Apotex, Toronto, Ontario, Canada

[i] Losec; AstraZeneca Canada Inc., Mississauga, Ontario, Canada

[j] Metoclopramide Hydrochloride Injection Sandoz Standard; Sandoz Canada Inc., Quebec, Canada

[k] Ampicillin Sodium for Injection; Novopharm Ltd, Toronto, Ontario, Canada

[l] Baytril Injectable Solution; Bayer Inc. Animal Health, Toronto, Ontario, Canada

[m] Plasma-Lyte A; Baxter Corporation, Mississauga, Ontario, Canada

[n] Fresh frozen plasma; Ontario Veterinary College Veterinary Teaching Hospital Canine Blood Donor Program, Guelph, Ontario, Canada

[o] Neupogen; Amgen Inc., Amgen Manufacturing Ltd, Thousand Oaks, CA

[p] Dako mouse monoclonal antibody clone 34βE12 diluted 1:100; Dako Canada Inc., Burlington, Ontario, Canada

[q] Dako rabbit polyclonal antibody pre-dilute (ready-to-use); Dako Canada Inc., Burlington, Ontario, Canada

[r] Phenobarbital; Qualitest Pharmaceuticals, Huntsville, AL

[s] Deramaxx; Novartis Animal Health Inc., Greensboro, NC

[t] Diazepam; Qualitest Pharmaceuticals, Huntsville, AL

[u] 1.5 T Signa Horizon; GE Healthcare, Milwaukee, WI

[v] Magnevist, Bayer HealthCare Pharmaceuticals, Bayer Inc., Montville, NJ

[w] Tramadol, Amneal Pharmaceuticals, Hauppauge, NY

[x] E-cadherin antibody; BD Transduction, Franklin Lakes, NJ

[y] CD34 antibody; Santa Cruz Biotechnology, Santa Cruz, CA

[z] S100 antibody; Dakota, Carpenteria, CA

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