Title

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Journal

Veterinary radiology & ultrasound : the official journal of the American College of Veterinary Radiology and the International Veterinary Radiology Association, 64(3)

ISSN

1058-8183

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Publication Date

2023-05-01

DOI

10.1111/vru.13196

Received: 17 June 2022 | Revised: 23 September 2022 | Accepted: 15 October 2022

DOI: 10.1111/vru.13196

IMAGING DIAGNOSIS

WILEY

MRI and CT characteristics of a grade I meningioma with concurrent cribriform plate lysis in a dog

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Abstract

A 6-year-old female spayed German Shepherd mixed-breed dog was presented for treatment of a frontal lobe mass diagnosed on MRI, after an acute onset of generalized seizures and behavior changes. Computed tomography of the head was performed for radiation therapy planning and revealed concurrent cribriform plate lysis without nasal sinus invasion, and focal lysis of the left ventrolateral cranial fossa. Histopathology of the mass obtained via surgical excision was consistent with a grade I fibrous meningioma. The dog had a good outcome following surgery and radiation therapy.

KEYWORDS
brain, canine, osteolysis, tumor

1 | SIGNALMENT, HISTORY, AND CLINICAL FINDINGS

A 6-year-old female spayed German Shepherd mixed-breed dog was presented to a specialty referral hospital for a 2-month history of behavior changes and acute onset of generalized epileptiform seizures one week prior to presentation. The seizures were described as each lasting approximately three minutes. The patient was started on oral levetiracetam (29 mg/kg PO every 8 hours) and had no further seizure activity. General physical examination and neurological examination were unremarkable. Complete blood count and serum biochemistry were within normal limits, with the exception of a mild leukopenia of $4.98 \times 10^3/\mu L$ (ref: 6.00–17.00) and an elevated BUN of 37.6 mg/dl (ref: 9.0–29.0). Given the history of seizures and behavioral changes, the neurolocalization was to the forebrain.

2 | IMAGING, DIAGNOSIS, AND OUTCOME

The patient was anesthetized and magnetic resonance imaging (MRI) of the brain was performed using a 1.5 Tesla magnet (General Electric, unknown model). MRI revealed a broad-based, expansile, well-marginated, multilobular, predominately right-sided extra-axial mass in the region of the olfactory bulb and frontal lobe causing mass effect and leftward deviation of the falx cerebri (Figure 1A–F). The mass was T2-weighted (T2W) hyperintense (Figure 1A and C) and strongly contrast enhancing after administration of intravenous gadobenate dimeglumine (Bracco Diagnostics Inc., Monroe Twp., NJ) on T1-weighted (T1W) images (Figure 1F). On the T2W fluid-attenuated inversion recovery (FLAIR) sequence, there was hyperintensity within the internal capsule and adjacent white matter tracts, indicative of secondary perilesional vasogenic edema (Figure 1D). An additional finding on the MRI consisted of focal dilation of the central canal over the C2-3 intervertebral disc space, suggestive of syringohydromyelia (Figure 1A). This was deemed incidental in this patient. The patient was started on prednisone (0.4 mg/kg by mouth every 12 h) and omeprazole (0.8 mg/kg by mouth every 12 h) and continued on levetiracetam (29 mg/kg by mouth every 8 h) and discharged to the owner's care.

Abbreviations: CT, Computed tomography; FLAIR, Fluid-attenuated inversion recovery; MRI, Magnetic resonance imaging; T1W, T1-weighted; T2W, T2-weighted; WHO, World Health Organization.

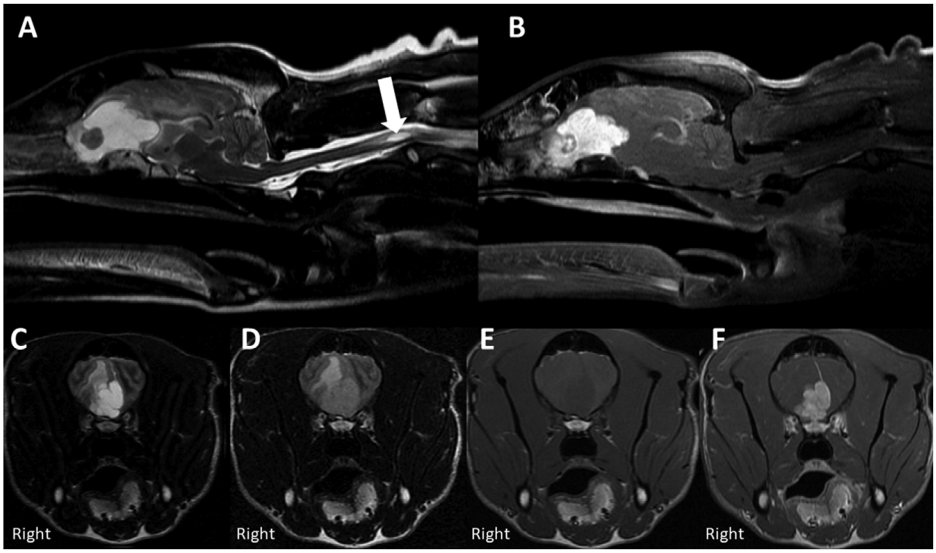

FIGURE 1 MRI images of a predominately right-sided olfactory bulb meningioma. A, Sagittal T2W image. Arrow showing focal region of syringohydromyelia at the level of the C2-3 intervertebral disc space. The angulation at C2-C3 is an artifact due to patient positioning. B, Sagittal post-contrast T1W image depicting the olfactory bulb meningioma to be homogenously contrast enhancing. C, Transverse T2W image showing a well-marginated, right-sided, T2-hyperintensity causing displacement of the falx cerebri indicative of mass effect. D, Tranverse FLAIR image showing marked perilesional edema when compared to post-contrast T1W image. E, Transverse T1W image revealing the mass to be hypointense to the surrounding parenchyma. F, Transverse post-contract T1W image depicting the meningioma to be homogenously contrast enhancing

She improved initially but began to decline as the prednisone was being tapered to 0.4 mg/kg once daily, and her dose was subsequently increased to 0.6 mg/kg once daily.

The following month, the patient was presented to a tertiary referral hospital for further work-up and radiation therapy treatment of the previously diagnosed frontal lobe mass. General physical and neurological examinations did not reveal any abnormalities. Thoracic radiographs were performed and did not reveal any evidence of primary neoplastic or metastatic disease and were otherwise unremarkable. A pre- and post-contrast CT (TSX-101A/LQ Aquilion 16 Slice, Toshiba, Tustin, California, USA) of the head was performed under sedation with intravenous butorphanol (0.1 mg/kg) and intravenous dexmedetomidine (3 mcg/kg) for radiation therapy planning. The CT revealed a patchy contrast enhancing lesion extending from the right olfactory bulb to the right frontal lobe, consistent with the previous MRI findings. Additional findings on CT consisted of lysis of the cribriform plate without invasion of the mass into the nasal cavity, and focal lysis of the left ventrolateral cranial fossa (Figures 2A and B and 3A and B). The aforementioned finding on CT was not reported on MRI initially; however, was noted retrospectively following review of the CT imaging. Three days later, the patient was anesthetized and a transfrontal craniotomy was performed to excise the mass. A large amount of abnormal light gray, friable tissue was removed from the region of the right olfactory bulb and submitted for histopathologic analysis. Histopathology of the debulked tissue was consistent with a grade I meningioma of fibrous subtype. The patient recovered uneventfully from surgery and was discharged with the following oral medications - levetiracetam (29 mg/kg every 8 h), omeprazole (0.9 g/kg every 12 h), gabapentin (13 mg/kg every 8 to 12 h), and prednisone (0.6 mg/kg every 24 h). Approximately three weeks following surgery, the patient was deemed stable with a

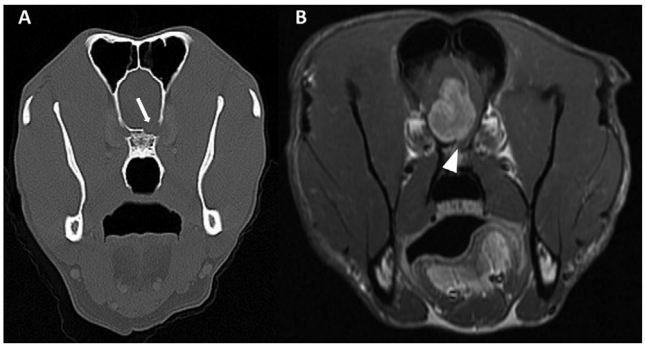

FIGURE 2 Transverse images of the head at the level of the frontal sinus. A, CT transverse, 3 mm slices, bone window. Transverse post-contrast image showing focal region of lysis of the left ventrolateral cranial fossa (arrow). B, Transverse T1W post-contrast MR image showing extension of tumor and parenchyma through bone defect (arrowhead). The osteolysis is presumed to be due to pressure necrosis from focal tumor expansion, resulting in subsequent herniation of brain parenchyma

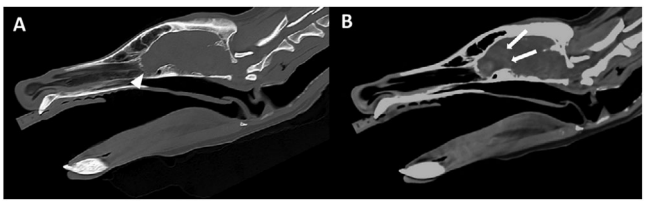

FIGURE 3 CT of the head, 3 mm sliced images. A, Bone window. Focal region of cribriform lysis depicted by the arrowhead. B, Soft tissue window. Sagittal post-contrast image revealing a patchy, contrast enhancing lesion extending from the olfactory bulb to the frontal lobe (arrows)

normal neurologic exam and behavior and underwent 18 fractions of 2.5 Gy of radiation therapy via linear accelerator (Elekta Precise SLi 15, Crawley, West Sussex, UK). At the time of manuscript preparation, the patient is still alive 14 months post-surgery.

3 | DISCUSSION

This report is the first published description of MRI and CT findings for a histologically confirmed, frontal lobe, grade I meningioma with concurrent cribriform plate lysis in a dog. Meningiomas are generally slow-growing, extra-axial, well-defined primary tumors that arise from the arachnoid cap cells of the leptomeninges.[1] Meningiomas are the most common primary intracranial tumor in dogs and are most frequently observed arising from the olfactory bulbs and frontal lobes, consistent with the findings of our case.[2] The most common osseous abnormality described with meningiomas in dogs and cats is calvarial hyperostosis, which can be observed in up to 73% of feline and 23% of canine intracranial meningiomas.[3] This feature was not apparent in our case. Histologically confirmed meningiomas with transcalvarial extension through osteolytic skull defects have been described in a cat and two dogs.[3] Meningiomas with concurrent osteolytic lesions are a well described phenomenon in humans, with a reported incidence of 15–20% amongst olfactory groove meningiomas extending into the ethmoid sinuses and nasal cavity.[4] Osteolytic behavior, such as cribriform plate destruction, is rarely described in veterinary species but was described in a recent case report of a cat with a histologically confirmed transitional meningioma.[5] In this paper, a 9-year-old male neutered domestic longhair cat had a large extra-axial meningioma with cribriform plate destruction and extensive nasal invasion on MRI.[5] Cribriform plate erosion has been suggested in one dog with an olfactory meningioma in a case series of 13 dogs with cranial vault meningiomas.[6] This finding was based on MRI depicting potential extension of contrast enhancement rostral to the cribriform plate, suggestive of cribriform plate erosion.[6] However, this erosion was presumptive and not confirmed.[6]

Pathologic disruption of the cribriform plate is well-documented in other canine diseases. Fungal diseases commonly display a mass effect and have a propensity to manifest with osteolysis. Cribriform plate lysis is well-described in sinonasal aspergillosis, with a reported incidence of up to 22%.[7] Although more common in cats, nasal cryptococcosis is another fungal organism known to cause cribriform plate disruption in dogs infected with this pathogen.[8] In a retrospective study looking at the association of MRI and histologic diagnosis in dogs with nasal disease, cribriform erosion was significantly associated with neoplastic diseases over fungal and inflammatory (lymphoplasmacytic) rhinitis.[9] Nasal adenocarcinoma was the most commonly reported neoplasm concurrent with cribriform lysis in this study.[9] Olfactory neuroblastomas are also known to invade the cribriform plate, cranial vault and brain, but these are considered to be rare tumors in dogs.[10] This imaging finding can help clinicians rank differential diagnoses; however, this report outlines the possibility of meningiomas also being concurrent with such osteolytic changes.

The present case illustrates how low grade meningiomas can result in subsequent osteolysis. Based on the World Health Organization (WHO) histologic classification scheme, canine meningiomas are broadly characterized into two groups: slow growing, generally benign neoplasms (which consist of a number of subtypes: meningothelial, fibrous, transitional, psammomatous, angiomatous, myxoid, granular, and papillary) and anaplastic tumors which have more malignant cytologic features.[1,11] In a study looking at 112 dogs with histologic confirmation of intracranial meningiomas, there did not appear to be a correlation between MRI findings and histologic subtype or tumor grade.[12] One of the 112 dogs had a histologic grade consistent with malignant meningioma, but a considerable amount of cases had atypical classifications (48/112 dogs).[12] None of these tumors were reported to have concurrent osteolytic lesions. Osteolytic lesions such as meningiomas with concurrent calvarial osteolysis or extracranial tumor extension do not necessarily imply the presence of an atypical or malignant histopathologic phenotype.[3]

The pathophysiology regarding osteogenic changes with meningiomas is not fully understood and is likely multifactorial. A human study looking into the mechanisms of meningioma-related hyperostosis, concluded that the expression of certain proteins involved in bone remodeling such as osteoprotegerin (OPG) and insulin-like growth factor 1 (IGF-1) was associated with the development of hyperostosis.[13] Proposed mechanisms for osteolysis concurrent with meningiomas may include pressure induced bone erosion, invasion of the calvarium by neoplastic cells, or degradation of the bone and the extracellular matrix by metalloproteinases.[3,14] Given the expansile nature of this mass and its proximity to the cribriform plate, we suspect pressure induced bone erosion to be the main mechanism of cribriform plate lysis in this case. However, the other proposed mechanisms of osteolysis also cannot be excluded. In the absence of pre-tumor imaging of the skull, a congenital defect cannot be completely ruled out.

In conclusion, cribriform lysis was initially missed with MRI and more definitively documented using CT in this dog with histologically-confirmed, frontal lobe, grade I meningioma. Clinical and imaging findings supported a prioritized differential diagnosis of intracranial neoplasms such as a high-grade meningioma or an extensive nasal adenocarcinoma due to the presence of osteolysis and a more guarded prognosis. However, this mass was found to be a low-grade meningioma and the dog had a good outcome following surgery and radiation therapy. Findings suggested that osteolytic lesions may be independent of tumor grade. Although cribriform plate lysis is more common in sinonasal fungal diseases and other neoplastic diseases such as nasal adenocarcinoma, low grade meningiomas should also be considered as a differential when a mass effect with cribriform plate lysis is detected.

LIST OF AUTHOR CONTRIBUTIONS

Category 1

a. Conception and Design: Petit, Chen, Roberts
b. Acquisition of Data: Petit, Chen, Valerio

c. Analysis and Interpretation of Data: Petit, Chen, Valerio, Roberts, Murthy

Category 2

a. Drafting the Article: Petit
b. Revising Article for Intellectual Content: Chen, Valerio, Roberts, Murthy

Category 3

a. Final Approval of the Completed Article: Petit, Chen, Valerio, Roberts, Murthy

Category 4

a. Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: Petit, Chen, Valerio, Roberts, Murthy

CONFLICT OF INTEREST

The authors have declared no conflict of interest.

PREVIOUS PUBLICATION OR PRESENTATION DISCLOSURE

None

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How to cite this article: Petit CV, Chen AV, Murthy VD, Roberts GD, Valerio-López C. MRI and CT characteristics of a grade I meningioma with concurrent cribriform plate lysis in a dog. *Vet Radiol Ultrasound*. 2022;1-4. https://doi.org/10.1111/vru.13196