Case Report

Magnetic Resonance Imaging of Meningiomas Associated with Transcalvarial Extension through Osteolytic Skull Defects in a Cat and Two Dogs

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Abstract

Here we describe the magnetic resonance (MR) imaging features of three cases of intracranial meningiomas characterized by transcalvarial tumor extension or cerebral herniation through spontaneous osteolytic skull defects overlying the tumors. All tumors appeared as solitary, lobulated, well marginated, extra-axial, and markedly contrast enhancing mass lesions, with inherent MR signal characteristics similar to those described for meningiomas. Histopathological examinations indicated that calvarial osteolysis can occur via tumor invasion of bone or tumor associated bone resorption. Osteolysis of the skull and transcalvarial tumor extension are rare manifestations of Grade I or Grade II canine and feline meningiomas.

INTRODUCTION

Meningiomas are the most common primary intracranial tumor in both dogs and cats [1,2]. Calvarial osteolysis with or without extracranial tumor extension is a rare manifestation of meningiomas in humans and animals [3-6]. The purpose of this case series is to describe the imaging features of meningiomas associated with osteolytic skull defects and extracranial tumor extension in a cat and two dogs, and to review mechanisms of calvarial osteolysis described in humans with transcalvarial extension of meningiomas.

CASE PRESENTATIONS

Signalments, Histories, and Clinical Findings

A 16-year-old, 5 kg, spayed female domestic shorthair cat (Case 1) was evaluated for displaying aggressive types of behaviors for 5 months, such as biting and hissing when the owner attempted to pet the cat’s head. Intermittent circling to the left of approximately 2 weeks duration was also described. A neurological examination revealed abnormalities consistent with a left prosencephalic lesion, including circling to the left, proprioceptive positioning deficits in the right thoracic and pelvic limbs, and an absent menace in the right eye associated with visual tracking deficits and normal pupillary light reflexes.

Case 2 was a 9-year-old, 23 kg, castrated male mixed breed dog that presented for evaluation of three generalized tonic-clonic seizures observed by the owner in the two weeks prior to presentation. Physical examination abnormalities consisted of signs of pain noted when opening the mouth and direct upon palpation of the right tympanic bulla. The interictal neurological examination was unremarkable.

6-year-old, 27 kg, spayed female Boxer dog (Case 3) was evaluated for a 3-month history of seizures with a recent onset of head pressing and intermittent episodes of opisthotonus. Neurological abnormalities included episodic obtundation, ambulatory ataxia of all limbs with absent postural reactions in the right thoracic and pelvic limbs, absent menace responses bilaterally, bilateral nasofacial hypalgesia, and depressed gag
and oculocephalic reflexes. A multifocal or diffuse brain disorder with left forebrain predilection was suspected based on clinical examination.

**Magnetic Resonance (MR) Imaging**

MR imaging examinations of the head were performed under general anesthesia in all cases. Various matrix sizes and fields of view were used. Cases 1 and 2 were imaged with a 0.2T system (Esaote Vet-MR, Genoa, Italy) using the following sequences: spin-echo T1-weighted (T1W-SE) images in the sagittal and transverse planes, spin-echo T2-weighted (T2W-SE) images in the sagittal and transverse planes, spin-echo proton density T2-weighted (T2W-PD) in the dorsal plane, and a gradient-echo T2-weighted (T2W-GRE) images in the transverse plane. Post-contrast T1W-SE images were also obtained in the dorsal, transverse, and sagittal planes. Case 3 was imaged with a 1.5T system (General Electric Signa, Little Chalfont, UK) and the following sequences were obtained: fast spin-echo T1-weighted (T1W-FSE) and T2-weighted (T2W-FSE) in the sagittal and transverse planes, and a post-contrast T1W-FSE in the transverse plane. Post-contrast images were obtained following intravenous injection with 0.1 mmol/kg of gadopentetatedimeglumine (Magnevist, Bayer, Wayne, NJ) in all cases.

**Case 1**

A sharply-marginated, multilobular extra-axial mass (Figure 1) was present in the left cerebrum dorsolateral to the left lateral ventricle. The mass was isointense to the cerebral cortical gray matter on T1W-SE (Figure 1A), T2W-SE (Figure 1B), and T2W-PD (Figure 1C) sequences, and homogeneously contrast enhancing (Figure 1D and E). There was increased thickness and enhancement of the perilesional dura, consistent with a dural tail sign. Adjacent to the cerebral mass lesion, there was hyperostosis of the left parietal bone surrounding a focal, well-defined expansile osseous defect. At the margin of the defect, the bone has a circumferentially flared appearance, and there is a linear region of increased signal intensity within the hyperostotic bone on all sequences (Figure 1A, B, and D). The increased signal intensity was attributed to bone edema, cellular infiltrate, or diploic fat. The lateral aspect of the mass was herniated through the osseous defect into the extracranial musculature, while the majority of the mass was intracranial, distorting the left lateral ventricle and resulting in a falcine shift to the right. There was also moderate dilation of the left olfactory recess, and caudal displacement of the cerebellum with herniation of the caudal ventral aspect of the cerebellar vermis through the foramen magnum. A fusiform T2W-SE hyperintense cavity was present the cervical spinal cord in the plane of the central canal, consistent with marked syringomyelia. Bilaterally, the tympanic bullae contained material that was iso- to hypointense on T1W-SE, hyperintense on T2W-SE, and peripherally contrast enhancing (Figure 1E).

**Case 2**

An extra-axial, multilobular mass was present, centered in the region of the right temporal bone (Figure 2). The mass was of mixed signal with the periphery being mildly hyperintense to gray matter and the central core hypointense on T1W-SE.
(Figure 2A) images, and on T2W-SE and T2-GRE it appeared heterogeneously hyperintense, with a central region of marked hyperintensity (Figure 2B). Portions of the mass extended into the ventral and lateral aspects of the temporal bone. The mass demonstrated moderate to marked, heterogeneous enhancement on post-contrast T1W-SE images (Figure 2C and D). Multifocal areas of calvarial osteolysis were noted in the temporal bone in the central part of the mass, and the mass extended through these defects into the soft-tissues external to the calvarium (Figure 2A-D). On post-contrast dorsal T1W-SE images, dural tail signs extended rostrally along the parietal bone and caudally in the occipital region (Figure 2D). There was significant associated mass effect, manifested as compression and deviation of the right lateral ventricle, left falk shift, right unilateral transtentorial herniation, flattening of the caudal aspect of the cerebellum, and caudal ventral extension of the cerebellar vermis through the foramen magnum.

Case 3

A well-defined, broad-based, multilobular extra-axial mass lesion distorted the left cerebrum (Figure 3). The mass was predominantly isointense to gray matter with a central focus of hypointensity on T1W-TSE (Figure 3A), iso- to mildly hyperintense on T2W-TSE (Figure 3C) images, and demonstrated marked and slightly heterogeneous enhancement on post-contrast T1W-TSE (Figure 3B) images. The mass lesion was also surrounded by increased thickness and enhancement of the meninges, consistent with a dural tail sign. At its dorsal aspect, the mass and a portion of the adjacent cerebral cortex were herniated through a well-defined expansile defect in the left calvarium, although the majority of the lesion was intracranial. There was hyperostosis of the left parietal bone adjacent to the defect (Figure 3 A-D). Ventral to the mass at the level of the aforementioned herniation, there was a focal lytic defect in the squamous portion of the temporal bone, with mild ventral herniation of the normal appearing left piriform lobe. There was severe mass effect manifested as marked compression and rightward displacement of the left lateral ventricle, falkine herniation, moderate dilation of the right lateral and third ventricles, left unilateral caudal transtentorial herniation (Figure 3E), concave deformity with loss of cerebrospinal fluid (CSF) signal in the rostral aspect of the cerebellum, and herniation of the cerebellar vermis through the foramen magnum (Figure 3F). There was marked perilesional T1W-FSE hypointensity and T2W-FSE hyperintensity, consistent with white matter edema (Figure 3A-F). Medial to the mass there was a small focal region within the white matter edema that was T1W hypointense, T2W hyperintense, without enhancement, suggesting cavitation, necrosis, or loculated CSF.

Diagnoses and Outcomes

Case 1 was anesthetized and a left lateral rostrotentorial craniectomy performed to remove the mass. Upon elevation and retraction of the temporalis muscle, the extracranial portion of the mass appeared as a bulbous expansion of the frontoparietal aspect of the calvarium (Figure 1F). Visual inspection and palpation the dorsal portion of the extracranial extent of mass be covered with bone, while the central and ventral portions of the mass were covered with a thin layer of periosteum, with several punctate areas through which a tan granular mass were protruding through full thickness skull defects. The craniectomy defect was enlarged circumferentially around the extracranial portion of the mass to facilitate en bloc excision of the extra- and intracranial portions of the mass and removal of the hyperostotic portion of the calvarium. The cat recovered from surgery uneventfully and was discharged 2 days after surgery. A neurologic examination performed 12 days after surgery was normal. The final histopathologic diagnosis was a Grade I meningothelial meningioma. Submitted portions of the hyperostotic calvarium demonstrated multifocal areas of tumor invasion into the dura and skull with adjacent osteonecrosis. The cat was euthanized 22 months following the diagnosis of intracranial meningioma because of progressive, proteinuric chronic kidney disease.
Surgical treatment was declined by the owner of Case 2, and percutaneous biopsies of the extracranial portion of the mass were obtained immediately following the MR examination using a 14-gauge biopsy needle (Tru-cut, Medline, Mundelein, IL). Levetiracetam, prednisone, and tramadol were prescribed, and the dog discharged following anesthetic recovery. Approximately 6 weeks later, the dog was euthanized after developing cluster seizures. Histopathological examination of biopsy specimens revealed dense connective tissue, skeletal muscle, and a neoplastic mass with no natural borders. The neoplastic cells consisted of a heterogeneous population of hyperchromatic spindle cells arranged in highly cellular and largely patternless sheets. Several areas of the neoplasm contained regions of necrosis and 4-6 mitotic figures per ten 400x fields. There was infiltration of the neoplasm into the connective tissue and myofibers (Figure 2E). The final diagnosis was a Grade II, atypical meningioma.

Case 3 was euthanized at the owner’s request while under anesthesia following completion of the MR examination and a necropsy was performed. Necropsy examination revealed the large extra-axial mass in the left cerebral cortex, with secondary complications associated with mass effect including hemispheric peritumoral vasogenic white matter edema, transcalvarial herniation of the dorsal aspect of the mass and overlying cerebral cortex (Figure 3D and G), and falcine, left unilateral transtentorial, and foramen magnum herniations. The histologic appearance of the mass was consistent with a Grade I transitional meningioma. Sections from the peripheral portion of the herniated mass contained discontinuous fragments of thin and irregular calvarial bone attached to the overlying dura (Figure 3C). Robust cellular reaction surrounded the bone fragments, most of which was osteoclastic. No tumor invasion of the bone was observed, although the dura was infiltrated by tumor in multiple regions.

**DISCUSSION**

Transcalvarial extension of meningiomas and other primary brain tumors through spontaneous osteolytic skull defects has been a rarely described phenomenon in humans. 3 Meningiomas are the most common primary brain tumor in dogs and cats [1,2]. However, extracranial extension of meningiomas has also been rarely reported in cats, and, to our knowledge, has not been previously reported in dogs [1,2,4-6]. The most common osseous abnormality associated with meningiomas in both cats and dogs is calvarial hyperostosis, which can be observed in up to 73% of feline and 23% of canine intracranial meningiomas [1,7]. Concurrent calvarial hyperostosis and osteolysis were present in 2/2 of the previous published feline meningiomas cases for which imaging was available, as well as in 2/3 of the cases reported here [4,5].

The unifying and unique features of the MR examinations presented in this case series was the transcalvarial herniation of the meningiomas or cerebrum through spontaneous geographic osteolytic skull defects, which were similar to appearance on MR as to what has been reported in humans and a cat with extracranial extension of meningiomas [5,8]. Other unusual imaging findings were observed in individual cases. For example, in Case 1, a region of increased signal intensity was identified in the hyperostotic portion of the parietal bone, which was histologically confirmed to represent bone invasion by the meningioma. In Case 2, tumor invasion of the temporal bone was also suspected based on the intraosseous signal changes present, but this suspicion could not be confirmed on biopsy. Case 3 was also atypical in that 4 brain herniations were simultaneously present in the patient, including caudal transtentorial, falcine, foramen magnum, and transcalvarial types.

These cases illustrate the potential difficulties encountered when attempting to classify meningiomas that involve extracranial structures. The literature contains numerous descriptors for those rare meningiomas that occur in extradural locations, such as cutaneous, ectopic, extradural, intraosseous, and secondary meningiomas [9-11]. Extradural meningiomas are hypothesized to arise from either extension of primary intracranial meningiomas or ectopic arachnoid cap cells that
were displaced during development [10]. Based on the available evidence, we postulate that the cases presented here are most representative of primary intracranial (neuraxial) meningiomas that extended into extradural locations, principally because of the degree of intracranial involvement present as well as pathological evidence of dural invasion present in 2/3 cases. Although controversial, dural invasion is a criteria used by some authors to preclude classification of a meningioma as extradural in origin [10,11]. However, we cannot totally exclude the possibility that these cases could be advanced stage examples of primary intraosseous meningiomas of the osteolytic or mixed osteoblastic-osteolytic types [10,11].

With the previously noted exceptions, several of imaging features and intrinsic signal characteristics of these tumors are similar to those reported for canine and feline meningiomas [1,2]. All of the meningiomas in this case series were well marginated, extra-axial, and displayed marked contrast enhancement with dural tail signs which are hallmark features of this tumor type [1,2,12]. Based on the collective imaging findings, the differential diagnoses considered for these cases included meningioma, histiocytic sarcoma, lymphoma, or locally invasive secondary neoplasms [1,2,13,14]. A mixed or pure granular cell tumor could also be considered, especially for Case 2, in which the tumor demonstrated mild hyperintensity on pre-contrast T1W images, although approximately 20% of canine intracranial meningiomas have been demonstrated to be hyperintense on pre-contrast T1W imaging sequences [2,15]. Osteolysis of the skull with secondary transcalvarial tumor or brain extension into extradural soft tissues are rare features of human, canine, and feline primary brain tumors, including meningiomas [3-6]. In humans, mechanisms of osteolysis associated with meningiomas can include pressure induced bone erosion, invasion of the calvarium by neoplastic cells, or degradation of bone and the extracellular matrix by metalloproteinases or serine proteases elaborated by tumor cells [3,4,8]. We hypothesize that multifactorial mechanisms of bone destruction are also likely present in dogs and cats with osteolytic meningiomas, identified calvarial invasion by neoplastic cells as a cause of osteolysis in the Case 1, and suspected tumor invasion into the skull in Case 2 based on the MR findings. Additional studies are required to document other potential etiologies of bone resorption or degradation in dogs and cats with osteolytic meningiomas. Similar to humans and previously reported feline cases, the presence of meningioma-associated calvarial osteolysis and extracranial tumor extension does not necessarily imply the presence of an atypical or malignant histopathological phenotype [5,6,16].

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REFERENCES