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CASE REPORT

A Case Report of a Rare Subset of Meningiomas: Intraosseous Meningioma

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Abstract

Meningiomas are the most common central nervous system neoplasms and occur most frequently along the dural folds and cerebral convexities. While meningiomas are slow growing and typically do not invade brain parenchyma they are of clinical importance as they can impinge surrounding structure causing a variety of signs and symptoms depending on size and location. We report here a rare subgroup of extradural meningiomas that emerge from the calvaria, a primary intraosseous meningioma, found during cadaveric dissection in a graduate anatomy course in the Department of Physician Assistant Studies at Samford University.

Key Words: *Intraosseous meningioma; Skull; Cranial; Tumor*

Introduction

Meningiomas are central nervous system neoplasms arising from arachnoid cap cells. These tumors are typically benign, slow growing, and commonly occurring along dural folds and cerebral convexities [1,2]. These neoplasms are the most common primary brain tumor, accounting for between 12% and 26% of all intracranial tumors, and, according to Ostrom et al. [3], account for 35.6% of all primary CNS tumors, while extradural meningiomas constitute 1% to 2% of all meningiomas [1,4]. Meningioma incidence increases with advanced age, and, owing to progesterone receptor expression, occur more frequently in females [4-6].

Meningiomas generally do not invade the brain parenchyma and are thus asymptomatic, leading frequently to incidental detection, but can in some cases grow to compress neighboring brain structures and give rise to focal neurologic deficits.

Primary Intraosseous meningioma (PIM) is a term used to describe a rare subgroup of extradural meningiomas that emerge from bone and especially the calvaria [5]. Although PIMs can develop at any location of the calvaria, the orbital and frontoparietal regions are the most common locations [7,8]. Clinically, PIMs can be seen on imaging with tissue biopsy performed to confirm the diagnosis. In this report, we present a case of a PIM found during cadaveric dissection in a graduate anatomy course in the Department of Physician Assistant Studies at Samford University and discuss potential clinical implications of this anomaly.

Case Report

An 84-year-old female, Caucasian cadaver, with cause of death listed as “coronary heart disease”, was obtained by Samford University for cadaveric dissection in a physician assistant studies anatomy course. The cadaver had no signs of significant surgical history and presented

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with unremarkable height and weight. To view the brain and interior aspect of the calvaria, the scalp and temporalis muscles were reflected inferiorly, and a chisel was used to scrape the bones of the calvaria clean of any remaining soft tissue. The cadaver was decapitated at the mid-cervical level using a bone saw and chisel. A scalpel was used to make a mid-sagittal line from the mandible to the occipital bone. A bone saw was used to cut along the line created by the scalpel, being careful not to cut the brain. The edge of the chisel was then used to insert into the cut calvaria and twisted to begin hemisecting the head and to separate the dura mater from the bone. The dissection alternated between using the bone saw and chisel until the decapitated head was hemisected (Figure 1). The brain was then removed.

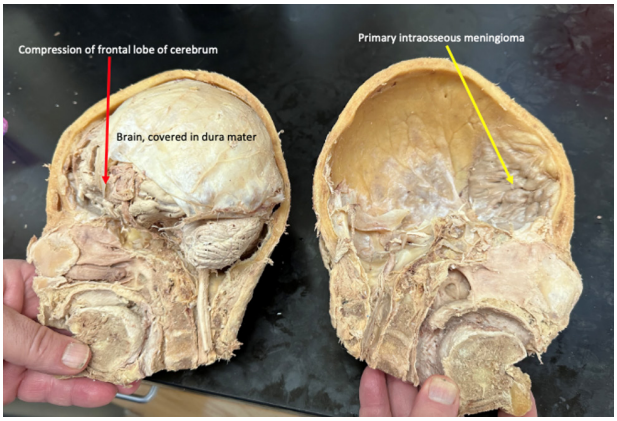

Figure 1) *Hemisected head showing primary intraosseous meningioma and the dura mater covering the brain.*

Visual appraisal of the internal skull revealed a bony anomaly throughout the frontal bone and portions of the sphenoid, temporal, and parietal bones bilaterally (Figures 2 and 3). Upon visual inspection, a meningioma was discovered, which appeared to have been cut in half during the autopsy. The dura was not adhered to the meningioma, and the meningioma grossly involved only the bone, as the dura peeled off the bone easily. Further inspection of this anomaly and discussions with anatomists and pathologists revealed this anomaly was a PIM. The total surface area of the PIM in our subject was 300 square cm and seems to have only extended inwards, compressing the frontal lobe of the cerebrum (Figure 1), with no obvious signs of osteosclerosis through the periosteum to the external frontal bone. Thinning and erosion of the bone cortex, and infiltration growth patterns within the bone, were also observed during the dissection.

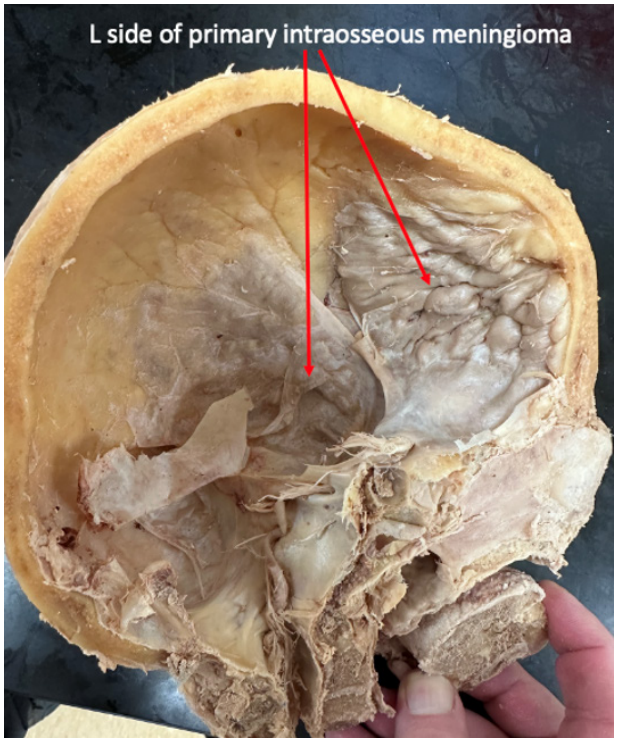

Figure 2) *Internal view of left side of skull showing primary intraosseous meningioma throughout the frontal bone and portions of the sphenoid, temporal, and parietal bones.*

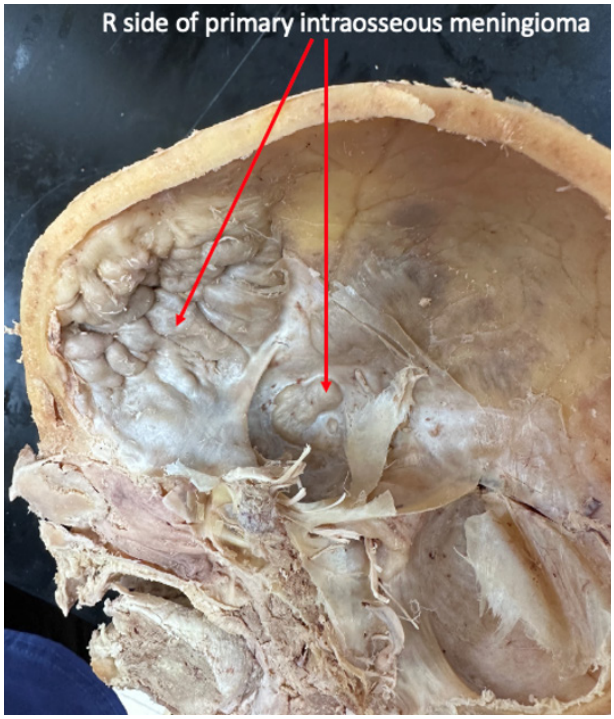

Figure 3) *Internal view of right side of skull showing primary intraosseous meningioma throughout the frontal bone and portions of the sphenoid, temporal, and parietal bones.*

Discussion

Most meningiomas occur in the subdural space, while only 1% to 2% are considered extradural, arising from locations other than the dura mater [9,10]. PIMs account for two thirds of extradural meningiomas and are typically less symptomatic than intradural meningiomas [9,11].

Different theories exist regarding the origin of PIMs [5,9]. Meninges are derived from mesenchymal cells, and, therefore, extradural meningiomas could arise in numerous atypical locations because of aberrant differentiation or misplacement of multipotent mesenchymal cells. Another theory suggests extradural tumors may arise from cells that become misplaced after differentiation into meningocytes or arachnoid cap cells. Cellular dedifferentiation within the skull could also possibly explain the formation of a PIM. Another theory involves small meningoceles containing arachnoid cap cells becoming trapped within skull fractures or sutures because of trauma. Currently, there is little consensus on the risk factors associated with development of CNS cancers in general, and in many cases these tumors are idiopathic in nature. The three meningeal layers have different embryological origins. While the two inner layers called the pia and arachnoid matter arise as derivatives of the neural crests, the outer dura matter is derived from the surrounding mesoderm [12].

The clinical presentation of PIMs depends on the site and size of the pathology. Since PIMs are a slow growing and painless benign growth of the skull, they are often asymptomatic unless the PIM reaches a significant size [7,8]. Indeed, 50% of meningiomas observed in one epidemiological study were discovered upon autopsy, reinforcing the slow-growing and frequently asymptomatic nature of these tumors [13].

Meningioma growth often expands outward, and a resulting visible and painless external scalp mass is the most common clinical presentation of PIMs [9,11]. Patients with PIMs often notice slowly growing cranial swelling without sign and symptoms, as the presentation of PIMs are usually less aggressive than intradural meningiomas [8]. The PIM described in this current care report expanded internally only, resulting in gross focal compression of the frontal lobe. In cases in which the neoplasm expands into the cranial space, most typical symptoms include generalized headaches due to increased intracranial pressure, seizures, or signs and symptoms specific to the region of brain compression. The PIM in this case was a large mass involving the frontal, sphenoid, temporal and parietal lobes, with visible gross compression of the frontal lobes bilaterally.

Without medical records we cannot definitively link this neoplasm finding on autopsy to any specific symptoms, however general symptoms associated with damage to the frontotemporal regions could include communication deficits, emotional and behavioral changes, movement, and memory deficits. Of note, symptoms of frontal lobe damage that may have occurred with this meningioma may mimic frontotemporal dementia or subdural hemorrhage, with all three diagnoses occurring with higher frequency with advanced age. For this reason, it may be reasonable that a meningioma such as the one described here may have been misdiagnosed as dementia or cognitive/motor changes often occurring with advancing age.

The location of the PIM will ultimately affect the patient presentation. Depending on the location, signs and symptoms can include seizures, nystagmus, cranial nerve defects, headaches, dizziness, proptosis, hearing loss, tinnitus, visual disturbances, and vague sensations in the head [8-10]. Due to the vast range of signs and symptoms, patients with PIMs are often misdiagnosed [5]. Treatment for PIM depends on size, location, and clinical presentation. The best treatment option in many cases of PIMs is surgical excision, which is potentially curative [8]. After surgical excision, there is a reported recurrence rate of 12.6% to 22% [5,10].

Conclusion

PIMs are rare tumors that originate in the skull. These lesions are often asymptomatic

but can cause a variety of signs and symptoms depending on the size and location of these lesions and should be considered in the differential diagnosis of patients presenting with increased intracranial pressure. The treatment of choice for PIM is surgical excision, which is potentially curative.

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Authors' contributions

RMC and NBW were responsible for ideal conceptualization. KA, DDB, RMC, WMS, and NBW were responsible for writing and approving manuscript prior to submitting.

References

1. Goodman CC, Boissonnault WG, Fuller KS. Pathology: Implications for the Physical Therapist. (2ndedn), Saunders, USA. 2003.
2. Warren P. Neurooncology. In: Amthor FR, Theibert AB, Standaert DG, Roberson ED, eds. Essentials of Modern Neuroscience. McGraw Hill; 2020.
3. Ostrom QT, Gittleman H, Liao P, et al. CBTRUS statistical report: primary brain and central nervous system tumors diagnosed in the United States in 2007-2011. Neuro Oncol. 2014;16:1-63.
4. Marosi C, Hassler M, Roessler K, et al. Meningioma. Crit Rev Oncol Hematol. 2008;67:153-71.
5. Zou M, Yang R, Tang Z, et al. Primary intraosseous meningioma with subcutaneous and dural invasion: a case report and literature review. Front Surg. 2022.
6. Agopiantz M, Carnot M, Denis C, et al. Hormone receptor expression in meningiomas: a systematic review. Cancers (Basel). 2023;15:980.
7. Chen TC. Primary intraosseous meningioma. Neurosurg Clin N Am. 2016;27:189-93.
8. Elwatidy S, Alanazi A, Alanazi RF, et al. Intraosseous meningioma, a rare presentation of a common brain tumor: Illustrative case. J Neurosurg Case Lessons. 2022;4.
9. Elder JB, Atkinson R, Zee CS, et al. Primary intraosseous meningioma. Neurosurg Focus. 2007;23:E13.
10. Omofoye OA, Huynh T, Jhun R, et al. Primary intraosseous meningioma of the calvarium: a systematic review. Clin Neurol Neurosurg. 2020; 199:106283.
11. Butscheidt S, Ernst M, Rolvien T, et al. Primary intraosseous meningioma: Clinical, histological, and differential diagnostic aspects. J Neurosurg. 2019; 21:1-10.
12. Hegazy A. Clinical embryology for medical students and postgraduate doctors. Lap Lambert Academic Publishing; 2014.
13. Chamoun R, Krisht KM, Couldwell WT. Incidental meningiomas. Neurosurg Focus. 2011;31:E19.