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Osteolytic lesion in primary intracranial meningioma: A case report



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ABSTRACT

Background: Hyperostosis of the skull is one of the characteristics of intracranial meningioma. In contrast, osteolytic lesions are very rarely discovered in meningioma. This study presented a rare instance of intracranial meningioma accompanied by significant osteolytic lesions.

Case presentation: A 70-year-old male patient presented with a chief complaint of a left-sided scalp lump which developed progressively within two years. Seizures and right-sided weakness have been reported. A mass of approximately 8 cm in diameter with a smooth surface was noted during a physical examination. The brain magnetic resonance imaging revealed an

$\pm 10 \times 9.6 \times 8.6$ cm lobulated homogeneous mass on the left temporoparietal region and heterogeneously enhanced after intravenous contrast injection. A localized osteolytic lesion was detected extending from the left temporal region to the left parietal region. A craniectomy was performed to obtain the pathological diagnosis and for complete removal of the mass. Histopathological evaluation revealed the presence of an atypical meningioma.

Conclusion: Bone destruction by meningioma may occur because of either neoplastic infiltration or pressure erosion.

Keywords: complication, intracranial, meningioma, neoplasm, osteolytic.

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INTRODUCTION

Meningiomas account for the most prevalent primary tumors of the central nervous system with a cumulative risk of 0.61. Meningiomas are categorized by the World Health Organization into three grades which include grade I (benign), grade II (atypical), and grade III (anaplastic). They are notably known as benign, slow-growing tumors that arise from mesothelial arachnoid cells (MEC).¹⁻⁴

While the majority of meningiomas are benign, atypical and anaplastic meningiomas have a notably unfavorable prognosis, characterized by a high recurrence rate.^{1,2} Hyperostosis of the skull is one of the distinguishing characteristics of intracranial meningioma noted by the propensity of the tumor to extend into the skull. In contrast, osteolytic lesions are very rarely discovered in meningioma cases.^{5,6} This study presented a rare instance of intracranial meningioma accompanied by

significant osteolytic lesions.

CASE PRESENTATION

A 70-year-old male patient presented with a chief complaint of a left-sided scalp lump. The mass developed progressively over two years and was known to be associated with intermittent headaches that worsened over the last year. Seizures and right-sided weakness have been reported over the past few days. A history of hypertension and diabetes was noted. No trauma was recorded.

A mass of approximately 8 cm in diameter with a smooth surface was noted during a physical examination. The physiology reflexes were decreased in the left extremity and there were not any pathologic reflexes. The brain magnetic resonance imaging (MRI) revealed an $\pm 10 \times 9.6 \times 8.6$ cm lobulated homogeneous mass which filled the left temporal region until the left parietal region (**Figure 1**)

and heterogeneously enhanced after intravenous contrast injection (**Figure 2**). A localized osteolytic lesion was detected extending from the left temporal region to the left parietal region.

A craniectomy was performed to obtain the pathological diagnosis and for complete removal of the mass. An extended pterional incision was made followed by a prepared flap. During surgery, a greyish mass of the tumor was discovered adhering tightly to the dura mater, extending to some parts of the outer and inner dural layers without brain invasion; and the skull, showing signs of bone destruction. The tumor was resected along with a wide margin of surrounding healthy bone (**Figure 2A** and **Figure 2B**) and the calvarial defect was reconstructed using a 10 x 10 titanium mesh (**Figure 2C**). Simpson grade I resection was completed.

Histopathological evaluation revealed the presence of an atypical meningioma

(Figure 3). The patient was discharged from the hospital on the 6th day after surgery with no neurological deficit.

DISCUSSION

Meningiomas are mostly benign tumors originating from MECs with a linear growth rate of 2 – 4 mm/year for asymptomatic meningioma. Meningiomas primarily occur in elderly patients and increase in individuals > 65 years old age.^{1,2,7} The radiological diagnosis of meningioma is typically using a head computed tomography scan and brain MRI evaluation. On brain MRI, meningiomas are usually hypo- to isointense relative to the cerebral cortex on T1-weighted sequences and iso- to hyperintense on T2-weighted sequences. A dural tail sign is seen in up to 72% of meningiomas after intravenous contrast injection.⁸

Meningiomas are more commonly seen in the convexity area (lateral hemisphere) than in the parasagittal and spinal areas. The signs and symptoms are often non-specific but, the location and compression to brain and vascular structures can lead to focal neurological deficit.^{1,9}

The fundamental principles of the surgery are maximum safe resection with low morbidity and preservation of neurological function.¹⁰ In this case, we could reach the goal for the surgery of Simpson grade I without any neurological deficit after the surgery. Grading of meningiomas is based on histopathologic findings. It depends on mitotic rate, brain invasion, or specific histological features. Mitosis count is a significant factor in surgical recurrence and overall survival.¹¹ In this case the histopathological examination of the tumor demonstrated a mass composed of proliferative whorly and solid structure. Four to five mitotic cells per 10 high-power fields were seen.

Atypical (WHO grade II) and anaplastic (WHO grade III) have a significantly poor prognosis with a high rate of recurrence. The 5-year recurrence rate of WHO Grade I, II, and III meningioma following Simpson grade I gross total resection are 7-23%, 50-55%, and 72-78%, respectively. Metastases are relatively a rare condition, estimated to occur in 0.1%, most of which are WHO grade III meningioma.¹

Primary and adjuvant radiotherapy

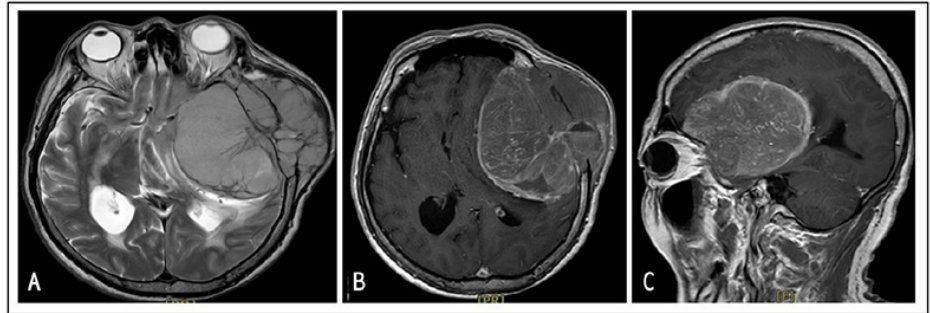


Figure 1. A: Axial T2-weighted brain magnetic resonance; B: Axial T1-weighted brain magnetic resonance imaging with intravenous contrast; and C: Sagittal T1-weighted brain magnetic resonance imaging with intravenous contrast showed a 10 x 9.6 x 8.6 cm lobulated homogenous mass at the left temporal region which obliterated the calvaria and destroyed the bones.

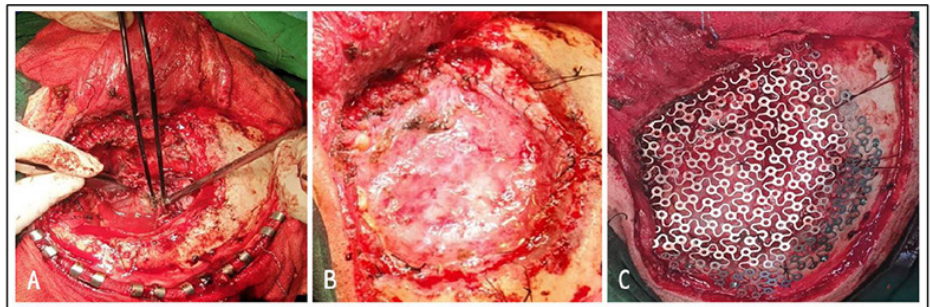


Figure 2. A: The piecemeal tumor debulking was performed until the tumor bed was seen. B: Duraplasty was done using autologous fascia. C: Cranioplasty with a 10 x 10 cm titanium mesh and seven miniscrew was performed.

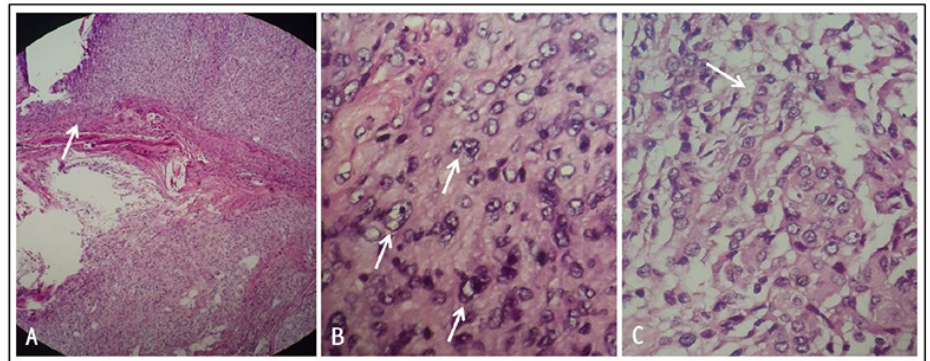


Figure 3. A: The tumor mass invades the adjacent structure (white arrow). B: Atypical meningothelial cell with enlarged nucleus (white arrow). C: Pathognomonic whorls of meningioma (white arrow).

are often considered when total resection is impossible.¹² Long-term follow-up is needed including molecular genetic examination, immunohistochemical, and radiology evaluation to examine the recurrence of the meningioma. It has been documented that a Ki67 proliferation index over 4% has been correlated with increased recurrence risk and it is most commonly used as an adjunct to standard WHO grading.¹³⁻¹⁵

The pathogenesis and molecular mechanism underlying the bone invasion in meningioma are poorly understood. Bone destruction (lysis) by meningioma may occur because of either neoplastic infiltration or pressure erosion.¹⁶ Tumor cells can secrete parathyroid hormone-related protein (PTHrP), which stimulates osteoblasts to produce a mimic receptor to activate osteoclast. Activated osteoclasts degrade bone matrix-releasing embedded

growth factors, stimulating tumor cells to produce more PTHrP.⁶

CONCLUSION

This was a case of atypical meningioma accompanied by skull invasion and subsequent necrosis. The tumor extended to the skull and invoked an osteolytic cascade based on the brain MRI and histopathology examination following the surgery. To our knowledge, this was the first case reported in Indonesia. Due to the rarity of this complication, this study was made in the hope of serving as a reference for future cases.

ACKNOWLEDGMENTS

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CONFLICT OF INTEREST

We disclose no conflict of interest in this report.

FUNDING

Not applicable

ETHICAL CLEARANCE

Since this work is classified as care-report-intending to improve and provide a better understanding of the reported case, our view toward this technical issue is none of ethics approval was required to deliver this case to the forum; even though we also believe the patient's consent is unquestionably essential to be confirmed,

as elaborated in the next section. The informed consent was obtained from the patient before any procedure was carried out. Accordingly, the patient (the 70-year-old Indonesian man in this study) also consented to the documentation, case discussion, and possible publication by clinical science development intention.

AUTHOR'S CONTRIBUTION

AD contributed to concepts, design, definition of intellectual content, literature search, clinical studies, data acquisition, data analysis, manuscript preparation, manuscript editing, and manuscript review. ST contributed to concepts, definition of intellectual content, clinical studies, data acquisition, manuscript review, and guarantor. CA contributed to concepts, design, definition of intellectual content, literature search, clinical studies, data analysis, manuscript editing, and manuscript review.

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