Plasmacytoma Masquerading as Meningioma

Münibe Büşra Erdem^{1,✉} Pınar Çakmak² Nalan Akyürek² Fikret Dogulu¹

¹Department of Neurosurgery, Gazi University Faculty of Medicine, Ankara, Turkey

²Department of Medical Pathology, Gazi University Faculty of Medicine, Ankara, Turkey

Address for correspondence Münibe Büşra Erdem, MD, Department of Neurosurgery, Gazi University Faculty of Medicine, Emniyet Mah. Mevlana Boulevard, Beşevler, Ankara 06560, Turkey (e-mail: dr.mbusra@gmail.com).

Indian J Neurosurg 2021;10:138–140.

Abstract

Solitary bone plasmacytoma is a rare disease in the skull. We present a 70-year-old patient who presented with a skull deformity due to the left parietooccipital tumor. Neurological examination found no deficit. The systemic scanning revealed no additional lesion. The surgery was planned, and the tumor was removed totally with the marginal bone around it. Histopathological examination confirmed solitary bone plasmacytoma. The patient is being followed-up without recurrence or progression to multiple myeloma. As the calvarial solitary bone plasmacytomas can be easily misdiagnosed preoperatively, the detailed examination of the CT features may be helpful for the planning and course of the surgery.

Keywords

- plasmacytoma
- skull neoplasms
- osteolysis

Introduction

Solitary plasmacytoma is a rare disease composed of colonized monoclonal plasma cells that are found in a single lesion in the body. Generally, there is no plasmacytosis in the bone marrow or only a small amount (< 10%).¹ The lesion may be either in bone or soft tissue, and the disease is classified according to the location: solitary bone plasmacytoma (SBP) and extramedullary (soft tissue) plasmacytoma.² The prevalence of SBP is dominant in males, and the peak incidence is in the 4th to 6th decades. It has a high risk in terms of progression to multiple myeloma than extramedullary plasmacytomas.³ SBPs of the skull have been rarely reported in the literature. Here, we would like to contribute with a case presentation.

Case Presentation

A 70-year-old male patient presented to the outpatient clinic with a complaint of swelling behind the left ear that appeared over the course of a month. Neurological examination found no abnormalities. The cranial CT revealed a crescent-shaped, expansile, 83 × 77 × 41 mm extra axial lesion in the left parietooccipital region (►Fig. 1A). In the bone window of the CT,

the boundaries of the lesion were irregular, and the calvarial bones seemed to be destructed (►Fig. 1D, H). The cranial MRI showed regular contrast enhancement with gadolinium in the mass lesion (►Fig. 1B, C). The skeletal survey, serum and urine protein electrophoresis were performed and revealed no abnormality. For systemic scan, 18-fluorodeoxyglucose positron emission tomography (18F-FDG PET) was performed and any additional lesion or focus was not found in favor of malignancy. Surgical excision of the lesion was planned. During the surgery, the mass was seen after the subgaleal dissection. It was hypervascular, soft, gray-colored, excisable with rongeur, and easily peeled from the dura mater. There was nonpulsatile but abundant bleeding during the excision. The hemorrhage could not be controlled by electrocoagulation or bone wax, since it originated from multiple diploic vessels. When the mass was removed totally and healthy bone margins were seen, the bleeding was brought under control and decreased significantly. The dura mater and subcutaneous tissue were seen to be healthy. The bone defect was repaired by performing cranioplasty with methyl methacrylate (►Fig. 1E, I). In the histopathological examination, the tumor cells were positive with CD 138 antibody, and the tumor was compatible with plasmacytoma (►Fig. 2). The patient underwent the bone marrow

published online
November 19, 2020

DOI <https://doi.org/10.1055/s-0040-1716989>
ISSN 2277-954X.

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Fig. 1 Radiological images of the patient: (A) Left parietooccipital lesion in the preoperative cranial CT. (B) Preoperative T2-weighted cranial MRI. (C) Contrast enhancement of the lesion in the preoperative T1-weighted cranial MRI. (D) Perforated parietooccipital bones seen in bone window of the preoperative cranial CT. (E) Postoperative cranial CT. (F) T2-weighted cranial MRI in the second postoperative month. (G) Postcontrast T1-weighted cranial MRI in the second postoperative month. (H) 3D reconstruction of the preoperative CT shows the perforated skull appearance. (I) 3D reconstruction of the postoperative CT shows the cranioplasty with methyl methacrylate, plate, and screws.

Fig. 2 Histopathological examination: (A) Plasma cells seen in the tumoral tissue. (B) Widespread positiveness with CD138 seen in immunohistochemistry. (C) Monotypic staining seen with Kappa. (D) Negativity with Lambda.

biopsy. Any abnormal blast cells or plasma cell involvement was not found. The patient was discharged on the second postoperative day without any complications. With all the findings, the diagnosis was decided to be solitary bone plasmacytoma, and the follow-up of the patient was started. In the postoperative second month, there was no new mass formation in the cranial MRI (►Fig. 1 F, G). The first postoperative year is over right now, and the patient is being followed-up without recurrence or progression to multiple myeloma.

Discussion

The cranial SBP cases in the literature could be divided into two groups: the cases located at the skull base, which is a rather complex anatomical structure (sphenopetroclival lesions), and the cases located at the cranial vault.⁴ We investigated the SBPs reported as being located in the calvarium by searching in the PubMed database. To date, there are 45 reported SBPs in the calvarium. (We were able to evaluate the reports whose abstract or full text are available in English.) The radiological appearance of these cases varied according to the involvement of the subcutaneous tissue, outer tabula, inner tabula, or the dura mater. They resembled an epidural hematoma due to the biconvex appearance in the CT,⁵ and a meningioma due to the contrast enhancement and proximity to the bone-dura border in the MRI.⁶ The clinical history is generally discordant with epidural hematoma; additionally, there is a destruction in the calvarial bones. Although the lesion seems similar to a meningioma with its solid structure and contrast involvement in the MRI, meningiomas often constitute hyperostosis instead of osteolytic lesions in the skull.⁷ In differential diagnosis, eosinophilic granuloma is also thought of, as it causes lytic lesions in the skull. However, in eosinophilic granulomas, the lesions are relatively regular-bordered, “punched-out” shaped with no reactive sclerosis, and there may be more than one lesion.⁸ Additionally, 80% of the eosinophilic granulomas occur in childhood and adolescence. In SBPs which invade both the inner and outer table but out of the dura mater, there may be a “soap bubble” appearance formed by expansile, lytic, bubbly, and septated lesion, as detected in our patient^{9–11} (►Fig. 1 H). It is noteworthy that the lesions did not cause reactive sclerosis. We liken this radiological appearance to a “perforated skull.”

Another underlined point in the literature is the bleeding tendency of SBPs. As a result of being a hematologic malignancy and residing in the diploe, the hemorrhage that arises at the surgery may be of a significant amount. The preoperative angiographies could reveal the vascularization of the tumor in detail¹² and embolization by superselective catheterization of superficial temporal,¹² occipital,¹³ middle meningeal,¹⁴ and accessory meningeal arteries¹⁵ could be performed. In our present case, the initial diagnosis was meningioma, and we did not perform preoperative embolization regrettably.

As the disease was localized and the mass was totally removed, our patient did not undergo radiotherapy. However, the close follow-up is still in progress. As SBPs have a risk of progression to multiple myeloma with 10% in 3 years and 65 to 84% in 10 years.^{1,3} Even though the total resection is performed, the close follow-up should be maintained in SBPs in terms of recurrence or progression to multiple myeloma.¹⁶

Conclusion

In this report, we tried to highlight the points of SBPs that should be kept in mind in neurosurgical practice. Although the CT images resemble extra-axial hematomas, they are often not confused due to incompatible clinical history. When the surgery is planned with only examining the MRI, much more intraoperative bleeding than a meningioma may occur. Therefore, bone changes in the CT should be examined in detail, because they may lead us to performing whole body scanning in terms of systemic disease and taking measures such as embolization to reduce the amount of intraoperative bleeding as well as.

Funding

None.

Conflict of Interest

None declared.

References

- 1 Caers J, Paiva B, Zamagni E, et al. Diagnosis, treatment, and response assessment in solitary plasmacytoma: updated recommendations from a European Expert Panel. *J Hematol Oncol* 2018;11(1):10
- 2 Swerdlow SH, Campo E, Pileri SA, et al. The 2016 revision of the World Health Organization classification of lymphoid neoplasms. *Blood* 2016;127(20):2375–2390
- 3 Tsang RW, Campbell BA, Goda JS, et al. Radiation therapy for solitary plasmacytoma and multiple myeloma: Guidelines from the International Lymphoma Radiation Oncology Group. *Int J Radiat Oncol Biol Phys* 2018;101(4):794–808
- 4 Gomez CK, Schiffman SR, Bhatt AA. Radiological review of skull lesions. *Insights Imaging* 2018;9(5):857–882
- 5 Rutherford SA, Leach PA, Richardson PL. Solitary skull vault plasmacytoma masquerading as an extradural haematoma. *Acta Neurochir (Wien)* 2004;146(8):863–864
- 6 Anoop TM, Jain N, Nair SG. Intracranial plasmacytoma mimicking meningioma. *J Neurosci Rural Pract* 2014;5(1):87–87
- 7 Heick A, Mosdal C, Jørgensen K, Klinken L. Localized cranial hyperostosis of meningiomas: a result of neoplastic enzymatic activity? *Acta Neurol Scand* 1993;87(3):243–247
- 8 Angelini A, Mavrogenis AF, Rimondi E, Rossi G, Ruggieri P. Current concepts for the diagnosis and management of eosinophilic granuloma of bone. *J Orthop Traumatol* 2017;18(2):83–90
- 9 Bakar B, Tekkok IH. Plasmacytoma of the skull vault. *Turk Neurosurg* 2012;22(1):95–98
- 10 Munjal S, Srivastava A, Jain S, Mehta VS. Skull vault plasmacytoma mimicking parasagittal meningioma: “mini-brain appearance.” *Asian J Neurosurg* 2019;14(1):231–233
- 11 Wilson P, Chumas P, van der Walt JD. Solitary plasmacytoma of the frontal bone. *Clin Radiol* 1990;42(4):289–290
- 12 Naganuma H, Sakatsume S, Sugita M, Satoh E, Asahara T, Nukui H. Solitary plasmacytoma of the skull: immunohistochemical study of angiogenic factors and syndecan-1—two case reports. *Neurol Med Chir (Tokyo)* 2004;44(4):195–200
- 13 Kuo Y-H, Huang W-C, Wu J-C. Surgical Treatment for a Giant Solitary Plasmacytoma with Skull Erosion. *Cureus* 2018;10(11):e3535
- 14 Pallud J, Page P, Varlet P, Roux F-X. Highly vascular solitary plasmacytoma of the calvarium. *Br J Haematol* 2006;133(1):2–2
- 15 Matsuda M, Nakazawa T, Kizuki H, Matsumura K, Nakasu S, Handa J. Solitary plasmacytoma of the skull vault—case report. *Neurol Med Chir (Tokyo)* 1996;36(6):388–392
- 16 Dimopoulos MA, Terpos E. Solitary bone plasmacytomas need to flow. *Blood* 2014;124(8):1209–1210