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Case Report
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Atypical Bleeding Meningioma: A Case Report

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Abstract

Meningiomas are the most common primary CNS tumors in adults, accounting for about one third of all brain lesions. Their clinical presentation is highly variable, ranging from accidental asymptomatic lesions to fatal tumors. The majority of meningiomas are benign, and gross total resection results in very low recurrence rates, with radiotherapy or radiosurgery reserved for recurrence or residual tumor growing on post operating imagery. At the other ends of the spectrum, malignant meningiomas represent only 1% of meningiomas and have a very high recurrence rate. Only 1% has been recognized as having aggressive behavior that ultimately proves lethal, regardless of the extent of resection or the use of adjuvant radiotherapy and chemotherapy. In the last two decades, there has been a pathological recognition of a third type of meningioma, the so-called "atypical" meningioma, whose histological and clinical features lie on the borderline between benign and malignant meningiomas. Here we describe a case of an atypical bleeding meningioma and discuss the management in light of the associated clinical, radiological and surgical challenges of this case.

Keywords: Atypical meningioma; Thrombosis; Tumors; Multidisciplinary management

Introduction

Meningiomas represent 20% to 30% of primary extra-axial tumors in adults. They develop at the expense of arachnoid cells and are benign in most of the cases. Generally, meningiomas are solitary tumors (>90%) and are more commonly diagnosed in women with a mean age of 50 years at onset [1-3].

Meningiomas are sub-classified by location, type, and histological characteristics. The 2016 World Health Organization (WHO) classification describes three histological grades: Grade I (benign), Grade II (Atypical Meningioma (AM), chordoid, and clear cell) and Grade III (anaplastic or malignant) [4].

Atypical meningiomas represent about 7% to 10% of all meningiomas. They show an increased mitotic activity with 4 or more mitoses per 10 High Power Fields (HPF) or brain invasions on histology. In addition, they have at least three of the following characteristics: Sheet-like growth, spontaneous necrosis, increased cellularity, prominent nucleoli and small cells with a high nuclear/cytoplasmic ratio [5].

Optimal management of atypical meningiomas is difficult to establish. Complete surgical excision is considered to be the general treatment strategy for all types of meningioma, the role of postoperative radiotherapy as standard adjuvant treatment remains controversial [6].

We describe a case of an atypical bleeding meningioma and discuss the challenges associated with the management of this case.

Case Presentation

Our patient, 54 years-old women, right-handed, with no history of arterial hypertension, diabetes, or intake of anticoagulants. She reported acute headaches of abrupt onset in the previous 48 h associated with a heaviness of the left hemi-corpus and speech impairment within a context of preserved general condition. Laboratory parameters were normal. Neurological examination showed an aphasic patient with a score of 11 on the Glasgow Coma Scale (GCS), pupils were

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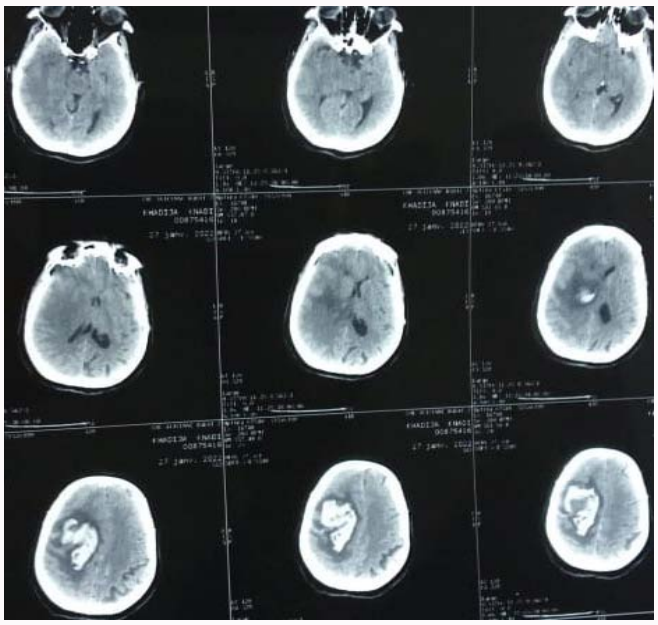

Figure 1: Cerebral CT scan. Axial section, parenchymal window, non-injected; showing a spontaneously hyperdense left frontal lesion associated with perilesional edema making a mass effect on the midline.

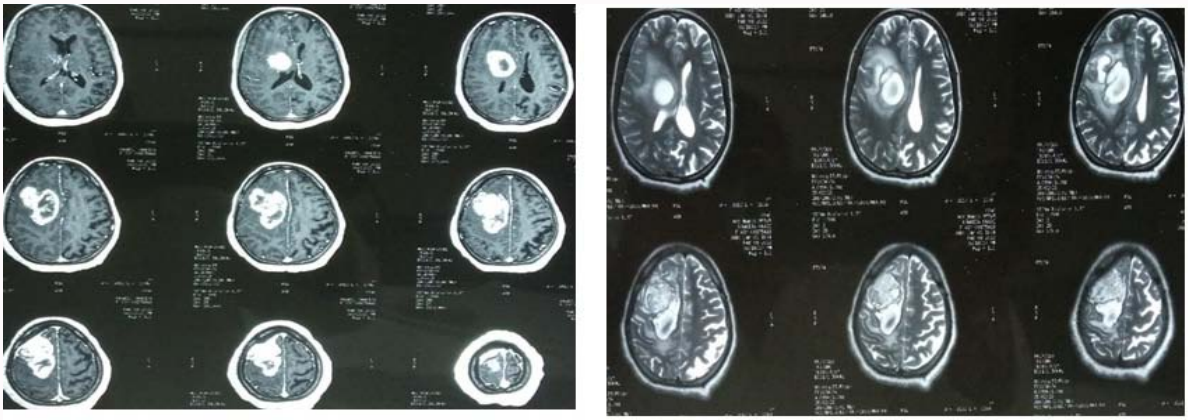

Figure 2: Cerebral MRI. Axial section, T1 (left) and T2 (right)-weighted showing a hyperintense right frontal lesion with a meningeal enhancement and a perilesional edema.

equal and reactive, a blood pressure of 110/60 mmHg and a heavy hemiparesis. The neurological examination did not present any other particularities. A cerebral CT scan and an MRI were performed (Figure 1, 2). The radiological assessment found a spontaneously hyperdense right frontal lesion on the cerebral CT scan with significant perilesional edema exerting a mass effect on the midline and on the ipsilateral ventricle. The T1-weighted MRI showed a hyperintense lesion in the periphery and a hypointense signal lesion in the center, strongly enhancing after injection of gadolinium. We concluded to a stroke, an Arteriovenous Malformation (AVM) or an expansive intracranial process with bleeding.

Within 72 h from admission to our department, the patient developed a swelling in the whole left leg with loss of calf movement and a rapid neurological deterioration with GCS at 9. A Doppler ultrasound of the lower limbs was carried-out and showed the presence of a venous thrombosis in the left lower limb that required the placement of a vena cava filter. The total exercise of the cerebral lesion was performed during the same intervention using a direct right front-lateral approach (Figure 3). Total removal of the tumour was confirmed by a brain CT scan.

The vena cava filter was removed on the 11th day after surgery. The patient was treated with heparin therapy at a curative dose, followed by oral Xarelto at a dose of 15 mg twice daily. No complications occurred during follow-up. The histopathological results revealed an atypical meningioma grade 2.

Discussion

Atypical meningiomas vary considerably in their histological characteristics and consequently in their behavior. Some are more aggressive and are more likely to invade the surrounding parenchyma and metastasize [4]. In general, atypical meningiomas are more probably to recur in the presence of brain invasion [5]. This behavior has been partially explained by mitotic and MIB-1/Ki-67 indices and by further examination of the cytogenetic profile of meningiomas in general [6]. A single meningioma may show a combination of meningothelial, transitional and fibrous histological patterns.

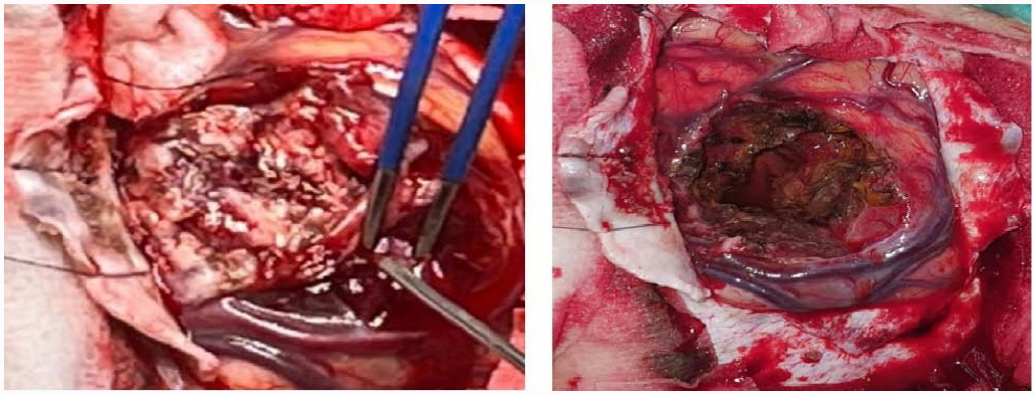

Figure 3: Intraoperative views. On the left, frontal flap 1 cm from superior longitudinal sinus: Discovery of an infiltrated Dura Mater (DM); Arciform opening of the DM; Discovery of a process adherent to the inner lining of the DM, pearly white in places with large old blackish blood clots and multiple fibrin deposits. On the right, total removal by splitting the process with the infiltrated DM while respecting the cleavage planes.

Other variants of classical meningiomas, although having other architectural and cytological features, are usually combined with the three prototypical histological patterns [7].

Benign intracranial tumors are very rarely associated with hemorrhage except in cases of pituitary adenoma [8-10]. Meningiomas are benign, slow-growing, and highly vascularized tumors; it is extremely uncommon that their onset is apoplectiform, mimicking cerebrovascular incidents; therefore affecting diagnostic workup, treatment, and outcome [8,9]. The risk factors for intratumor hemorrhage are history of radiosurgery and atypical tumor histology. Cerebral edema with venous obstruction can cause tumor infarction leading to rupture of tumor vessels and hemorrhage. On the other hand, enlarged feeder arteries become tortuous and less resistant to changes in blood pressure, increasing the risk of rupture [8-10].

In a high number of patients with hemorrhagic meningiomas, some neurological symptoms and signs that suggest an irritative expansive lesion rather than cerebrovascular syndrome prior to the final hemorrhagic manifestation are present [10-12]. Rebleeding(s) or deterioration after the first hemorrhagic manifestation secondary to the unsuspected meningiomas remains unpredictable. It is most probably to occur in the first hours or day(s) after the major hemorrhagic even [13,14]. There are, however, well-documented reports of unrecognized bleeding sources months and even years before a fatal outcome [12-15].

In this report, we describe the case of a patient with an atypical bleeding meningioma complicated by a venous thrombosis in the left lower limb. The management of this case was challenging due to its rapid evolution, the appearance of the venous thrombosis in the limb (secondary complication of the bleeding meningioma). Different options for managing this case were discussed with a multidisciplinary team including a vascular surgeons, intensivists/anesthesiologists and neurosurgeons. These options were: first treating the thrombosis with anticoagulant medication to avoid pulmonary embolism before proceeding with the surgical removal of the tumor and hematoma. This option was discarded due to the hemorrhagic cerebral lesion and the associated delay in treatment may carry an increased risk of complications especially given the observed rapid clinical evolution; another option was to place a vena cava filter to be able to remove the hemorrhagic cerebral lesion. The latter option was chosen and 2 procedures were performed simultaneously. The filter was removed afterwards, and the patient treated with anticoagulants. No complications were seen postoperatively and the patient evolved favorably.

In conclusion, an awareness of atypical clinical features of hemorrhagic tumors is critical for early recognition of the true nature of bleeding and will affect the diagnostic workup, treatment and prognosis.

References

1. Modha A, Gutin PH. Diagnosis and treatment of atypical and anaplastic meningiomas: A review. Neurosurgery. 2005;57(3):538-50.
2. Maier H, Ofner D, Hittmair A, Kitz K, Budka H. Classic, atypical, and anaplastic meningioma: Three histopathological subtypes of clinical relevance. J Neurosurg. 1992;77(4):616-23.
3. Stessin AM, Schwartz A, Judanin G, Pannullo SC, Boockvar JA, Schwartz TH, et al. Does adjuvant external-beam radiotherapy improve outcomes for nonbenign meningiomas? A Surveillance, Epidemiology, and End Results (SEER)-based analysis. J Neurosurg. 2012;117(4):669-75.
4. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK, Burger PC, Jouvet A, et al. The 2007 WHO classification of tumors of the central nervous system. Acta Neuropathol. 2007;114(2):97-109.
5. Perry A, Scheithauer BW, Stafford SL, Lohse CM, Wollan PC. Malignancy in meningiomas: A clinicopathologic study of 116 patients, with grading implications. Cancer. 1999;85(9):2046-56.
6. Ko KW, Nam DH, Kong DS, Lee JI, Park K, Kim JH. Relationship between malignant subtypes of meningioma and clinical outcome. J Clin Neurosci. 2007;14(8):747-53.
7. Ellison D, Love S, Chimelli L. Meningiomas. In: Neuropathology: A reference text of Abry E, Thomassen IØ, Salvesen ØO, Torp SH. The significance of Ki-67/MIB1 labeling index in human meningiomas: A literature study. Pathol Res Pract. 2010;206:810-5.
8. Asari S, Katayama S, Itoh T, Tsuchida S, Ohmoto T. Neurinomas presenting as spontaneous intratumoral hemorrhage. Neurosurgery. 1992;31(3):406-12.
9. Brady AP, Stack JP. Case report: Magnetic resonance demonstration of haemorrhagic acoustic neuroma. Clin Radiol. 1994;49(1):61-3.
10. Kohli CM, Crouch RL. Meningioma with intracerebral hematoma. Neurosurgery. 1984;15(2):237-40.
11. Lieu AS, Howng SL. Intracranial meningioma with hemorrhage. Kaohsiung J Med Sci. 1999;15:69-74.
12. Martinez-Lage JF, Poza M, Martinez M, Esteban JA, Antunez MC, Sola J. Meningiomas with haemorrhagic onset. Acta Neurochir. 1991;110(3-

4):129-32.

13. Chaskis C, Raftopoulos C, Noterman J, Flament-Durand J, Brotchi J. Meningioma associated with subdural haematoma: Report of two cases and review of the literature. Clin Neurol Neurosurg. 1992;94(3):269-74.

14. Kim DG, Park CK, Paek SH, Choe GY, Gwak HS, Yoo H, et al. Meningioma manifesting intracerebral haemorrhage: A possible mechanism of haemorrhage. Acta Neurochir. 2000;142(2):165-8.

15. Latchaw JP Jr, Dohn DF, Hahn JF, von der Luft E. Subarachnoid hemorrhage from an intracranial meningioma. Neurosurgery. 1981;9(4):433-5.