

# A Small Meningioma with Extensive Peritumoral Brain Edema: A Case Report

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## What's Known

- Meningioma is the second most common brain tumor worldwide. The extent of peritumoral brain edema (PTBE) is one of the important prognostic factors in patients with meningioma.
- Most studies have reported that the extent of PTBE can be a reason for the poor outcome of surgical resection of meningiomas.

## What's New

- We report the case of a patient with a small meningioma concomitant with a disproportionately large and extensive hemispheric brain edema. The patient had a good prognosis after surgical resection of the mass.

## Abstract

Meningioma is the second most common brain tumor. The extent of peritumoral brain edema (PTBE) is one of the important prognostic factors in patients with meningioma. A 55-year-old female patient suffering from a progressive severe headache and mild left hemiparesis was referred to the Department of Neurosurgery, Rasool Akram Hospital (Tehran, Iran). The preoperative imaging revealed a 2×2 cm solid extra-axial mass with bright enhancement at the outer third of the right sphenoid wing. In addition, there was a disproportionately extensive peritumoral brain edema in the right cerebral hemisphere that even involved the right internal capsule. The patient was operated through the right pterional approach and the mass was totally resected. Twenty-one days after surgery, the brain CT scan surprisingly showed only mild frontal edema and the patient was asymptomatic 1 year after the surgical treatment. According to the literature, the size and extension of the PTBE are correlated with the prognosis of meningioma. A larger edema is associated with a larger tumor, higher grade, and a more invasive meningioma with a higher recurrence rate. Our patient had a very large hemispheric PTBE which was disproportionate to the small size of the meningioma and the tumor had not directly invaded the adjacent brain tissue. We believe that the visible compression of the tumor on major veins of the Sylvian fissure was the reason for the PTBE in our patient. The presence of a large PTBE concomitant with a meningioma does not necessarily indicate a poor prognosis. Hence, we recommend a preoperative venogram to be performed in such patients.

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**Keywords** • Meningioma • Vasogenic brain edema • Prognostic factors

## Introduction

Meningioma is the second most common brain tumor worldwide.<sup>1</sup> In preoperative assessment, the extent of PTBE is one of the important prognostic factors in patients with meningioma.<sup>2,3</sup> Most studies have reported that the extent of PTBE could be a reason for the poor outcome of surgical resection of meningiomas.<sup>4</sup> Herein, we present a patient with a small meningioma concomitant with a disproportionately large and extensive hemispheric brain edema. The patient had a good prognosis after surgical resection of the mass.

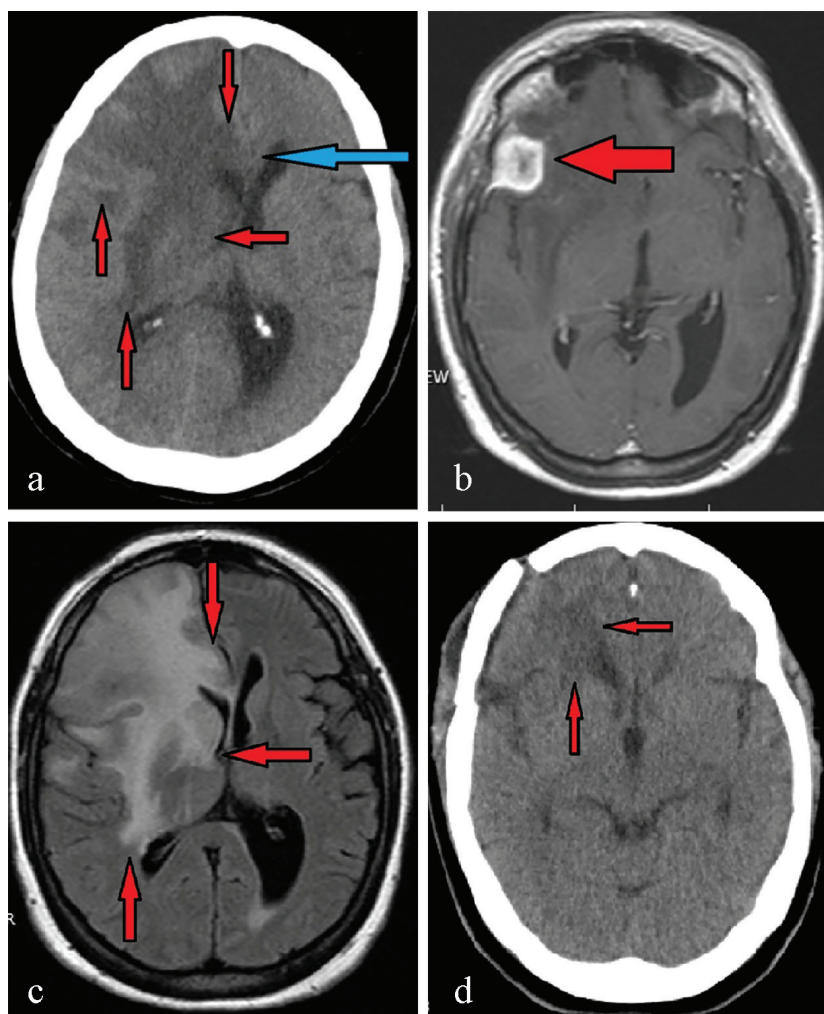
## Case Presentation

A 55-year-old female patient was referred to the Department of

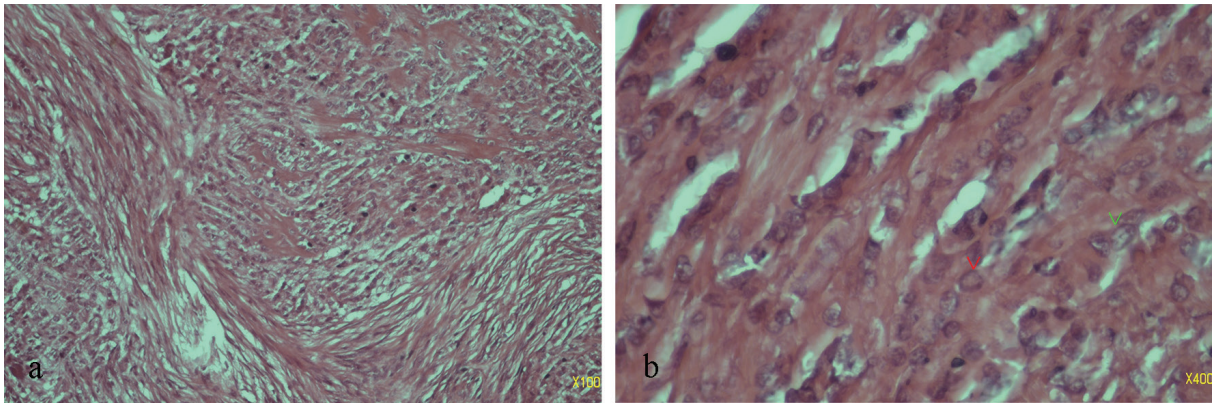
Neurosurgery, Rasool Akram Hospital (Tehran, Iran) suffering from progressively severe headache, vertigo, nausea, and vomiting for 10 days prior to admission. Clinical examination revealed that the patient had a mild left hemiparesis as a positive drift sign. Furthermore, the patient had a positive bilateral Hoffmann's sign and a bilateral papillary edema was found on fundoscopy. Other examinations revealed no pathologic findings and she had no subjective neurological deficits. All laboratory data upon admission were normal. The initial brain computed tomography (CT) scan revealed an extensive right hemispheric brain edema with a concomitant frontal subfalcine herniation (figure 1a). The subsequent brain magnetic resonance imaging (MRI) with and without gadolinium (Gd) showed a 2×2 cm solid extra-axial mass with bright enhancement at the outer third of the right sphenoid wing (figure 1b). In addition, there was an extensive peritumoral brain edema in the

right cerebral hemisphere that even involved the right internal capsule (figure 1c).

The patient was operated through the right pterional approach under microscopic magnification. The mass as well as a 2 cm margin of adjacent normal dura mater, as a safe margin, were totally resected. The adjacent skull was not involved, the mass was completely out of the adjacent brain's pia mater, and there was no invasion of the adjacent brain parenchymal. The patient was discharged 5 days after surgical treatment with normal clinical findings and without a headache or vertigo. On follow-up examination, 21 days after surgery, the brain CT scan showed only mild frontal edema (figure 1d). The patient was asymptomatic and without any neurological deficit. The histopathological report of the mass indicated fibroblastic meningioma grade 1 and, subsequently, we decided to follow-up the patient without any adjuvant treatment (figure 2). At the last follow-up, 14 months after surgery, the



**Figure 1:** The preoperative brain CT scan (a) revealed an extensive PTBE (short red arrows) in the right cerebral hemisphere with frontal subfalcine herniation (long blue arrow). The brain MRI with Gadolinium (b) showed a 2×2 cm extra-axial enhancing mass presenting the dural tail (red arrow). The fluid-attenuated inversion recovery (FLAIR) image sequence of MRI (c) revealed an extensive preoperative right hemispheric edema (red arrows). The postoperative brain CT scan, 21 days after surgical removal (d), demonstrated a mild frontal edema without herniation (red arrows).



**Figure 2:** Histopathological examination of the tumor (fibroblastic meningioma) using a microscopic magnification power of  $\times 100$  (a) and  $\times 400$  (b). The images revealed the fibroblastic tissue (a,  $\times 100$ ) composed of spindle cells with small and medium-size vesicular nuclei without the clear border of cytoplasm. In addition, there were round, spindle shape, and coffee bean nuclei (nuclear groove, green arrowhead,  $\times 400$ ) without prominent mitotic features. Intra-nuclear inclusion (red arrow head,  $\times 400$ ) was also observed. There was no evidence of necrotic change and brain invasion.

patient was asymptomatic and the brain CT scan showed complete resolution of the brain edema.

A written informed consent was obtained from the patient for the publication of her images and clinical data for scientific purposes. Additionally, confidentiality of the information was explained to the patient.

## Discussion

Meningioma is a benign extra-axial brain mass originated from arachnoid cap cells. Due to compression or even invasion into the adjacent normal brain, this type of tumor can produce intracranial pressure (ICP) with signs and symptoms such as headaches, nausea and vomiting, vertigo, hemiparesis, generalized tonic-clonic seizure, and even loss of consciousness. The cause of these manifestations can only be a direct compression or other causes such as the presence of PTBE.<sup>1</sup>

Extensive PTBE usually occurs in cases of malignant brain tumors or benign masses with invasion into adjacent normal brain tissue. It has been proposed that brain edema in meningiomas is associated with many factors including the size, location, and histological features of the tumor; the secretory activity of meningioma cells, meningioma with positive sex hormone receptors, venous channel compression, and occlusion by the tumor.<sup>2, 5-7</sup> However, recent research has suggested that PTBE is the result of a damage to the brain cortical structure caused by the tumor.<sup>5</sup> The size and extent of the PTBE correlate with the prognosis of meningioma and a larger edema is associated with a larger tumor, higher grade, and a more invasive meningioma with a higher recurrence rate.<sup>2, 5, 8</sup> Both the frontal

convexity and frontobasal regions have been associated with edema more frequently than the anterior parasagittal, where meningioma is located.<sup>5</sup> Most researchers have not found any relation between the site of this tumor and the larger edema.<sup>9</sup> In meningioma cases, we only found a few studies that correlated the tumor at the sphenoid ridge with the larger edema.<sup>9-11</sup> Based on histopathological findings, the transitional, meningotheliomatous, angioblastic, and malignant meningioma result in edema more than other histological subtypes.<sup>5</sup>

Our patient had a very large hemispheric PTBE which was disproportionate to the small size of the meningioma. Additionally, the absence of direct invasion to adjacent brain tissues, its benign nature, and the histopathological report revealing a low-grade tumor (grade 1) were atypical symptoms. In our patient, based on the findings of other studies, the site of the tumor (sphenoid ridge) and the histopathological report (meningothelial) were indicative of a larger concomitant edema. However, we considered these factors to be insufficient as a full explanation. Some researchers have described other possible causes of PTBE, such as obstruction of venous outflow drainage of the tumor and secretion of some fine substances by the tumor in the adjacent tissue to overexpress the aquaporin-4 (AQP-4).<sup>12, 13</sup> Considering the very small size and non-invasive nature of the meningioma in our patient, again we did not regard this as the definite cause.

In our opinion, the particularities in our patient were the site of the tumor and its visible compression on the major veins of the Sylvian fissure. Extensive PTBE and its subsequent early clearance, 3 weeks after the surgical



removal of the tumor, could only be explained by its benign nature and the fact that the process was reversible. The presence of such a large and extensive edema can be due to the disturbed venous drainage of the adjacent normal brain tissue, as in our patient, and cannot always be a predictor of poor prognosis. Although we did not have a preoperative venogram, the extent of PTBE and its location were proportional to the brain drained by Sylvian's veins. We believe that the obstruction of venous outflow of the adjacent normal brain tissue was the main reason for PTBE in our patient. In all meningiomas, this can also be a plausible factor for the occurrence of PTBE. We propose that the presence of PTBE in patients with meningioma is neither necessarily a predictor of tumor invasion nor the reason for a patients' poor prognosis, and does not always correlate with the size of a meningioma. It is recommended to design and perform a study to evaluate the role of a preoperative venogram, angiographic, or magnetic resonance venography (MRV) in all cases of a meningioma with PTBE. Such studies would allow a better diagnosis of this condition.

### Conclusion

The prognosis in our patient was good despite a disproportionately large and extensive hemispheric PTBE. In our patient, we did not observe any adjacent brain parenchymal invasion. We believe that the cause of extensive PTBE was a long-term compression of the tumor on the major veins of the Sylvian fissure which obstructed the normal venous drainage. This case demonstrated that the presence of a large PTBE concomitant with a meningioma does not necessarily predict poor prognosis. A preoperative venogram of such patients can assist in diagnosing this condition, but this requires more research and evaluation of patients' records.

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**Conflict of Interest:** None declared.

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