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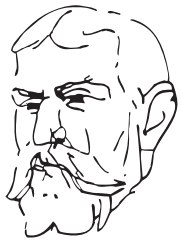

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CASE STUDY

# EXTENSIVE PERITUMORAL BRAIN EDEMA IN A SMALL CLINOIDAL MENINGIOMA: CLINICAL CASE

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**ABSTRACT**

Peritumoral brain edema (PTBE) is seen in 40-78% of all cases of intracranial meningiomas. It may vary in shape and size, occasionally being two to three times larger than the tumor. We present a case of a 62-year-old female patient, suffering from seizure and progressive headache. She was diagnosed with left medial sphenoid wing meningioma and referred for treatment to Uzhhorod Regional Center of Neurosurgery and Neurology. The patient had no major focal neurological deficit and Karnofsky Performance Scale (KPS) of 70 on admission. The preoperative magnetic resonance imaging (MRI) with and without contrast showed a 2.1×2.2×2.5 cm solid mass at the inner third of the left sphenoid wing, with homogenous enhancement and encasement of middle cerebral artery (MCA). In addition, there was a disproportionately extensive PTBE in the left cerebral hemisphere that caused midline shift and mass effect. The patient underwent left pterional craniotomy and gross total resection of the mass. The postoperative course was without complications or new neurological deficit, MRI within 48 hours revealed gross total tumour resection with residual brain edema and the patient was discharged with a KPS of 80 on day 7. Based on several studies, significant correlation between PTBE and tumor volume was observed: larger tumors cause larger PTBE. This particular case had a very large hemispheric PTBE, which was disproportionate to the small size of the meningioma. Most likely, the PTBE in this patient was caused by venous congestion, but this had no influence on surgical outcome. Therefore, the presence of a large PTBE does not necessarily indicate a poor prognosis and isn't always the reason of surgical complications.

**KEY WORDS:** MedialSphenoid Wing Meningioma; Brain Edema: Surgical Outcomes

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## INTRODUCTION

Peritumoral brain edema is seen in 40-78% of intracranial meningiomas. PTBE may vary in shape and size, and sometimes can be two to three times larger than the tumor [1], and in the majority of cases correlates with tumor volume [2]. Several studies have reported that PTBE may raise morbidity and mortality of patients by progressive brain shift and increased intracranial pressure [3-5]. Moreover, PTBE has been associated with a higher riskof postoperative intracranial hematoma [6]. To our knowledge, most studies have shown that PTBE is dependently associated with large tumor size, and the occurrence of PTBE may raise the difficulty of complete resection and increase risk of complication. Herein, we present a patient with a small medial sphenoid wing meningioma (MSWM) associated with sever and extensive brain edema. The patient had a good outcome after surgical resection and within one year of follow-up the tumor was completely cured. The aim was to demonstrate surgical outcome with a rare case of extensive edema in a small clinoidal meningioma.

## CASE REPORT

A 62-year-old female patient, suffering from progressive headaches for 6 months, seizures and blurred vision on the left side for one month prior to admission, was referred to Uzhhorod Regional Center of Neurosurgery and Neurology for treatment. Clinical examination revealed that the patient had no focal neurological deficit and her KPS was 70. Furthermore, the patient had early bilateral papillary edema, which was found on fundoscopy. All laboratory data upon admission were normal. Brain MRI with gadolinium enhancement, demonstrated a 5.8 $cm^3$ solid extra-axial mass with homogenous enhancement at the inner third of the left sphenoid wing with encasement of MCA (Figure 1b) and extensive PTBE.

In our study, the tumor and edema volume were measured using pre-operative contrast-enhanced MRI scans. The sagittal and coronal diameters were determined from the axial images. The coronal images were used to measure the axial diameter. These measurements were employed to estimate the volume of the lesion by applying the formula abc/2, where (a) is the sagittal, (b) is the coronal and (c) is the axial diameter [7,8]. Calculated tumor volume on pre-operative MRI was 5.8 $cm^3$ (Figure 1a). Same formula was used to measure PTBE by identifying high signal intensity changes on T2-weighted images. In this case PTBE volume was 104.8 $cm^3$ (Figure 1b). Edema index (EI) represents the degree of peritumoural edema compared with tumor volume and is calculated by dividing the PTBE volume on the tumor volume. EI of more than 1.0 means that edema is present, and in this particular case it was 18 indicating a severe PTBE.

The patient underwent surgery under general anesthesia with endotracheal tube in supine position.With her head

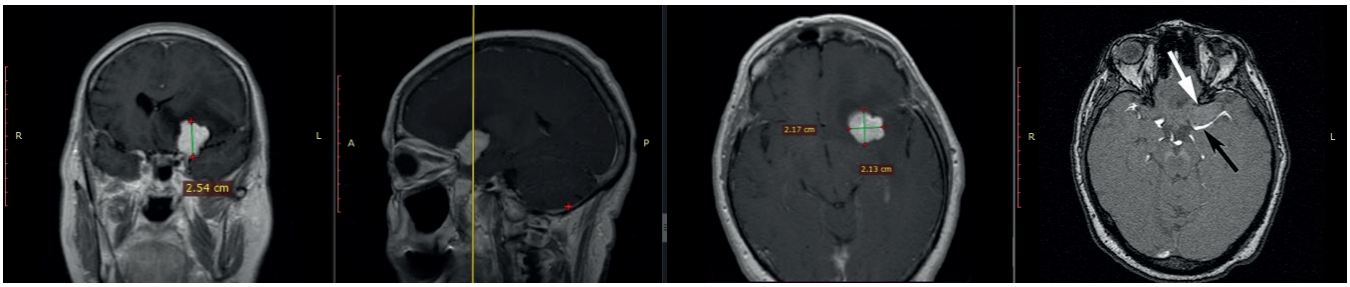


**Fig. 1a.** Preoperative brain magnetic resonance images (MRI) with Gadolinum demonstrated a 2.1×2.2×2.5 cm3, extra-axial enhanced mass in the inner third of sphenoid wing with irregular shape. The mass (white arrow) encases middle cerebral artery (black arrow)

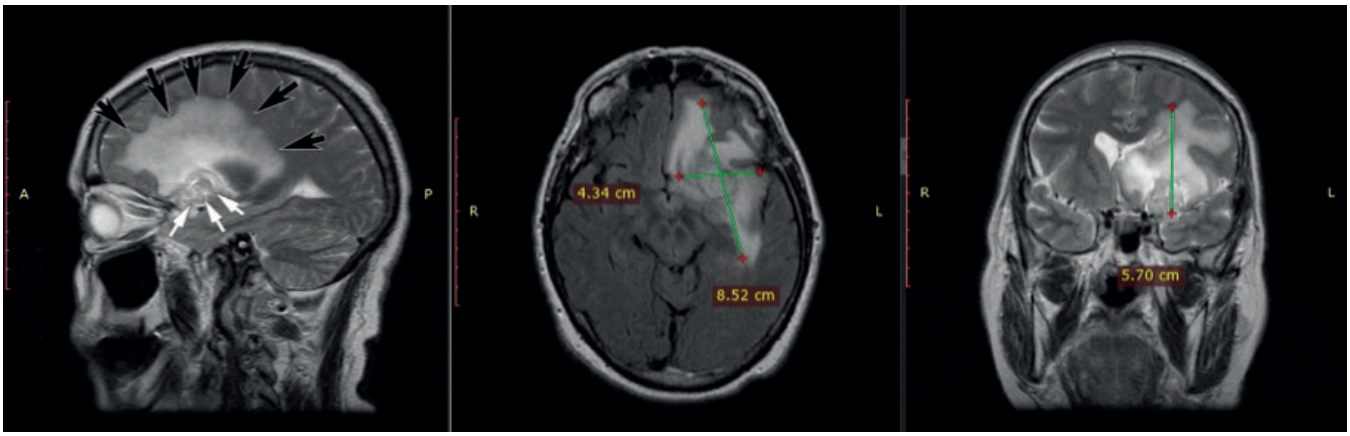


**Fig. 1b.** Preoperative brain T2-weighted MRI showed extensive PTBE, 4.3×8.5×5.7 cm3, causing midline shift and mass effect. The mass (white arrow) is surrounded by massive edema (black arrow).

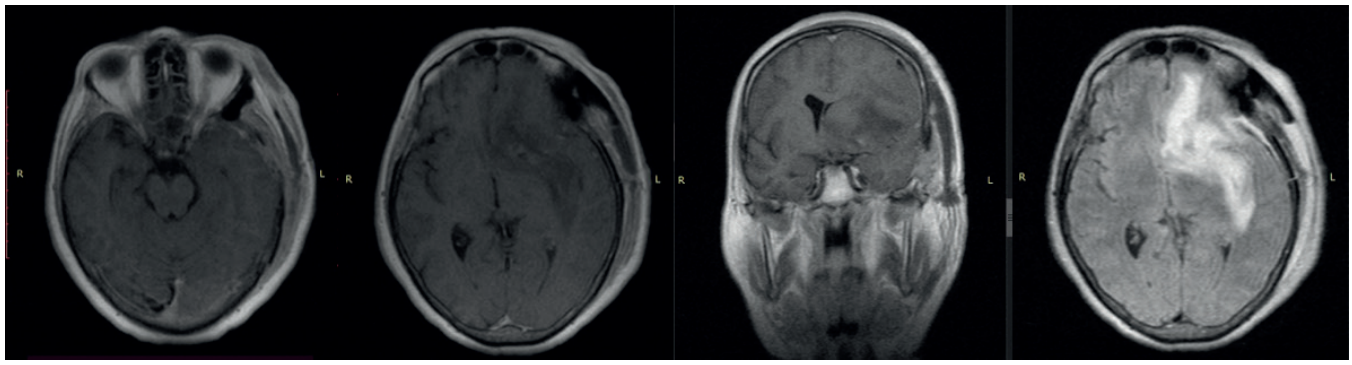

**Fig. 2.** Two days postoperative follow-up MRI revealed brain edema without residual mass.

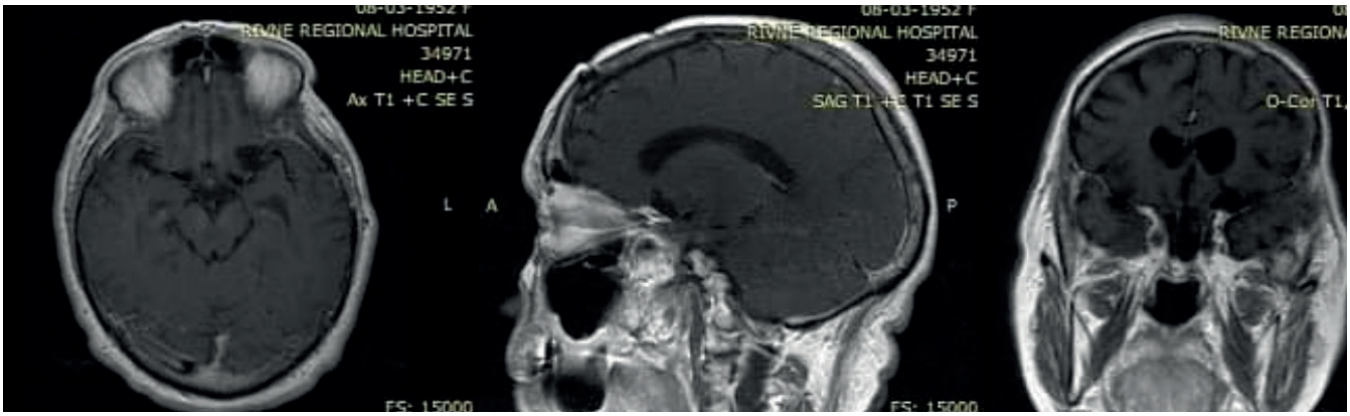


**Fig. 3.** Last follow-up MRI three years after surgery without any sign of recurrence.

turned contralaterally at a 30-degree angle and fixed with three pin head clamp, a standard pterional craniotomy was performed. After curvilinear durotomy, Sylvian fissure was dissected proximally under microscopic magnification until tumor was visualised. Gentle debulking of the tumor following cautery of tumor's dural attachment was

done. Peripheral dissection of the tumor from the brain parenchyma was performed, following bipolar cautery of the feeding vessels. Finally, delicate tumor dissection from middle cerebral artery was done following the arachnoidal plane. After hemostasis and dural closure, bone and wound were closed in multiple layers.

After the surgery, the patient was transferred to intensive care unit for close observation.In the first postoperative day after neurological exam which was normal, the patient was transferred to the regular patients' ward. In the second postoperative day the patient's MRI revealed brain edema without residual tumor (Figure 2). The patient was discharged 5 days after surgical treatment with normal clinical findings and improvement of vision and headache, with a KPS of 80. The histopathological report of the mass indicated WHO Grade I (meningiothelial subtype) meningioma.

At three year follow-up, no signs of tumor on MRI were noted (Figure 3), patient is not on antiepileptic drugs and is currently symptom-free with a KPS of 100.Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Despite primarily extra-axial locations, slow progression rates, and usually benign histological characteristics, meningiomas are frequently associated with PTBE [9]. Brain edema is defined as an expansion of brain volume due to increased water and sodium content.[10] However, different theories of PTBE pathogenesis were described and suggested, one of which is the venous compression theory, reported by Bitzer M et al. [11]. 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 size, location, histological features of the tumor, the secretory activity of meningioma cells, positive sex hormone receptors, venous channel compression and occlusion by the tumor [2,5,6]. Moreover, the size and extent of the PTBE correlates with the prognosis of meningioma. The extent of edema is associated with larger size of tumor, higher grade and a more invasive meningioma with a higher recurrence rate [12-14].

This patient had a very extensive PTBE (EI – 18), which was disproportionate to the small size of the meningioma. Despite the absence of direct invasion to adjacent brain tissue, the tumor had irregular margins and was hyperintense on T2-weighted images. Histopathological report revealed WHO Grade I meningioma (meningiothelial subtype). Nakano et al. reported that the hyperintense signal on T2WI is considered a multifactorial process and is correlated with tumor consistency and vascularity, indicating higher water content. Furthermore, the more water content tumors have, the easier edema fluid can diffuse to the surrounding brain tissue according to the water pressure gradient [15]. Simis et al. noted that the extent of PTBE in meningiomas had a positive correlation with the presence of irregular margins of the tumor [12]. Based on histopathological findings, the transitional, meningothelial, angioblastic and malignant meningiomas lead to edema more frequently than other histological subtypes [13].

However, several studies showed no significant correlation between histological subtypes of meningiomas and PTBE [2, 16]. Despite the small volume of the tumor, our patient had progression of symptoms and signs one month prior to admission, most likely due to extensive PTBE. Several studies indicated higher risk of intraoperative complications in the presence PTBE, like loss of dissection plane at brain-tumour interface and surgical difficulty during resection; pre and post-operative seizures; post-operative neurological deficit; postoperative intracranial haematoma and subsequent intracranial hypertension [3, 15, 17].

## CONCLUSIONS

In this particular case, our patient had good surgical outcome immediately and after one year of follow-up. Despite the extensive PTBE, gross total resection was achieved without intraoperative complications. We suggest that the extent of PTBE in our patient was caused by venous compression and advise preoperative CT or MR venography in such cases. Our experience demonstrates that extensive PTBE in patients with meningioma does not necessarily predict poor prognosis and isn't always the reason of complications. However, it does not mean that PTBE shouldn't be taken into account.

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