



Case Report

# Meningioma-related subacute subdural hematoma: A case report

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## ABSTRACT

**Background:** Meningiomas are the most frequent benign head tumors, although spontaneous hemorrhage is a rare form of presentation of such lesions. Of all possible bleeding locations associated with them, the subdural space is one of the most uncommon, with very few cases reported worldwide.

**Case Description:** A middle-aged woman presented with progressively worsening left-sided headache, initiated 2 weeks before, with no other complaints, denying any previous head trauma. Head computed tomography revealed a subacute left hemisphere subdural hematoma and left frontal, suggestive of meningioma on magnetic resonance imaging. Surgical treatment was performed with hematoma evacuation and lesion removal. Neuropathology showed a transitional meningioma with signs of hemorrhage. After surgery, no neurological deficits were registered, and headache abated.

**Conclusion:** As we could not identify any other cause for the subacute subdural hematoma, hemorrhage from the meningioma was the most probable cause, and thus, we decided to remove it along with clot evacuation. Based on neuropathological findings, we propose an alternative mechanism for this spontaneous hemorrhage from the meningioma, involving the place where the periphery of the lesion insertion, the dura mater as the origin of the hemorrhage. Knowledge of this association could help define the best treatment in such cases.

**Keywords:** Meningioma hemorrhage, Meningioma, Spontaneous bleeding, Subdural hematoma, Tumor hemorrhage

## INTRODUCTION

The most frequent benign brain tumors, meningiomas, are generally slow-growing lesions that may have a broad clinical presentation depending on its location. Convexity meningiomas comprising 15% of these lesions, most frequently present with headache, followed by seizures, and other site-specific symptoms.<sup>[22,29]</sup> Spontaneous hemorrhages from these lesions are very rare, and its underlying mechanisms are yet to be understood.<sup>[4,8,9]</sup>

## CASE REPORT

A postmenopausal woman presented to our emergency room with an intense left-sided parieto-occipital pulsatile headache, with 2 weeks of evolution and progressive worsening in the previous 2 days and minimal response to acetaminophen. She had no other symptoms or history of head trauma. The neurological examination was normal.

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The patient, hypocoagulated with acenocoumarol due to previous deep vein thrombosis, was also medicated with amlodipine for chronic hypertension.

Head computed tomography (CT) showed a subacute left hemisphere and tentorial subdural hemorrhage with 17 mm of maximum width and signs of acute hemorrhage in the frontal region, as well as a left frontal dural-based lesion, well-demarcated, spontaneously hyperdense, and partially calcified, measuring 15 mm × 18 mm in diameter, causing surrounding edema and mass effect, inducing a 10 mm midline shift [Figure 1]. The bone window revealed hyperostosis overlying the lesion. Blood tests were normal, with no signs of infection.

The patient was then admitted to the neurosurgery ward and head magnetic resonance imaging (MRI) confirmed an extra-axial left frontal lesion with adjacent dural enhancement after intravenous contrast [Figure 2]. A magnetic resonance angiogram excluded cerebral aneurysms or other vascular malformations.

Surgery with clot evacuation and lesion removal was the treatment option.

**Operative procedure**

The patient underwent a small left frontal craniotomy using neuronavigational guidance. The dural opening revealed subacute subdural hemorrhage and a yellowish rounded lesion, very well delimited, that was carefully separated from [Figure 3] the cortical surface, preserving the arachnoid membrane, and removed with the adjacent dural thickening (Simpson I). The subdural hemorrhage was drained, and a soft drain was left in the subdural space. Duraplasty was made with the dural substitute.

Bony hyperostosis was drilled, and the bone flap regularized.

**Histology**

Histology showed a transitional meningioma (the World Health Organization Grade I/III) infiltrating the adjacent dura mater. Along with some areas of fibrosis and necrosis, a few areas of internal hemorrhage were identified. The adjacent dura mater showed signs of fibrovascular proliferation [Figures 4 and 5] and hemosiderin deposits, both intra- and extracellularly.

**Postoperative outcome**

The subdural drain was removed 24 h postsurgery. Steroids were slowly weaned postoperatively over the course of days, and seizure prophylaxis was continued for 1 month. Postoperative head CT showed a very good imaging result, with total subdural hematoma evacuation. After surgery,

the patient noticed significant headache relief and needed no analgesics at discharge, maintaining a normal neurologic evaluation.

Postoperative head MRI showed complete lesion removal [Figure 6].

At the last follow-up appointment, 2 years after surgery, the patient remained asymptomatic.

**DISCUSSION**

The most common type of bleeding in meningiomas is subarachnoid hemorrhage, followed by intratumoral and intracerebral hemorrhage. Subdural hematoma, however, is extremely rare.<sup>[2-4,8,17]</sup>

We performed a thorough literature review and found that, in many cases, there was a lack of compelling data establishing a relationship between subdural collections and the presence of a convexity meningioma. This was particularly true in cases of chronic subdural collections in the elderly population and in cases where head trauma led to the diagnosis of both lesions. [Table 1]<sup>[5-7,10-14,16,18,19,21,23,24,26,27]</sup>

**Table 1:** Review of all cases reported in which subdural hematomas could result from meningioma hemorrhage rather than just being coincidentally diagnosed.

Article and references	Patient age	Gender	Meningioma type
Modesti <i>et al.</i> , 1976, <sup>[18]</sup>	69	M	Meningothelial
Kotwica and Zawirski, 1986, <sup>[13]</sup>	32	M	Angioblastic
Tokunaga <i>et al.</i> , 1988, <sup>[26]</sup>	61	F	Transitional
Jones and Blumbergs, 1989, <sup>[10]</sup>	76	F	Meningothelial
Sato <i>et al.</i> , 1989, <sup>[23]</sup>	46	F	Angioblastic
Niikawa <i>et al.</i> , 1990, <sup>[19]</sup>	49	F	Meningothelial
Chaskis <i>et al.</i> , 1992, <sup>[5]</sup>	59	F	Angioblastic
Chen <i>et al.</i> , 1992, <sup>[6]</sup>	79	M	Fibrous
Koumtchev <i>et al.</i> , 1993, <sup>[14]</sup>	26	F	Fibrous
Ueno <i>et al.</i> , 1993, <sup>[27]</sup>	67	M	Meningothelial
Spektor <i>et al.</i> , 1995, <sup>[25]</sup>	73	F	Angioblastic
Shimizu <i>et al.</i> , 1998, <sup>[24]</sup>	67	M	Meningothelial
Lefranc <i>et al.</i> , 2001, <sup>[16]</sup>	59	F	Angioblastic
-	62	M	Meningothelial
Kashimura <i>et al.</i> , 2008, <sup>[11]</sup>	50	M	Lipomatous
Chonan <i>et al.</i> , 2013, <sup>[7]</sup>	67	F	Meningothelial
Kim <i>et al.</i> , 2015, <sup>[12]</sup>	61	F	NS
Ravindran <i>et al.</i> , 2017, <sup>[21]</sup>	36	F	NS
Aloraidi <i>et al.</i> , 2019, <sup>[2]</sup>	49	F	Angioblastic
	49	F	Syncytial

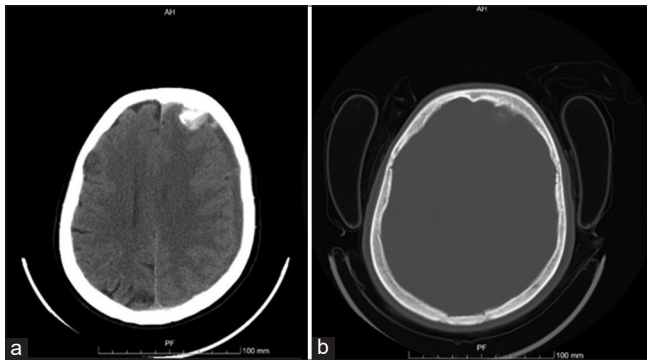
summarizes all cases reported, in which subdural hematomas could result from meningioma hemorrhage rather than just being coincidentally diagnosed.

The underlying pathophysiologic mechanisms have been subject to several studies, although it remains unclear as to the reason why and which under the circumstances some meningiomas bleed.<sup>[15]</sup> Accepted to date theories suggest that rupture of tumor vessels, tumor growth causing subdural

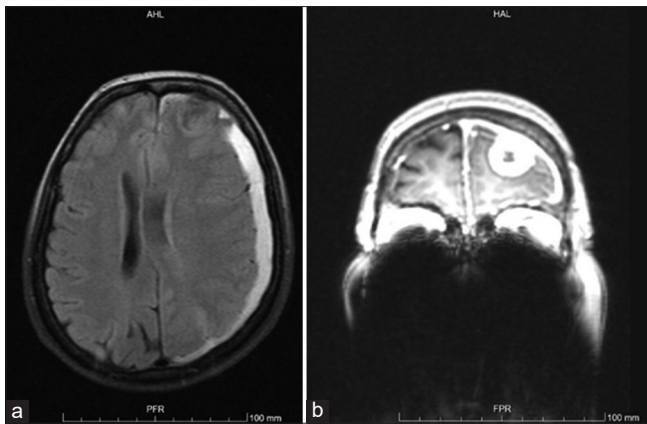
bridging vein rupture, rapidly expanding tumors, tumor cells invading and disrupting blood vessels and tumor-derived vasoactive substances could lead to tumor bleeding.<sup>[1,2,8]</sup> Nevertheless this still does not explain why this is such a rare event, since most meningiomas we see in our daily practice do not follow this course.<sup>[15,28]</sup> It is accepted that several mechanisms might simultaneously be involved in the development of hemorrhage.<sup>[4]</sup>

This patient was under amlodipine, which has a known vasodilator effect, reducing the peripheral resistance that added to the hypocoagulating effect of acenocoumarol which could possibly have influenced the development of the hemorrhage.

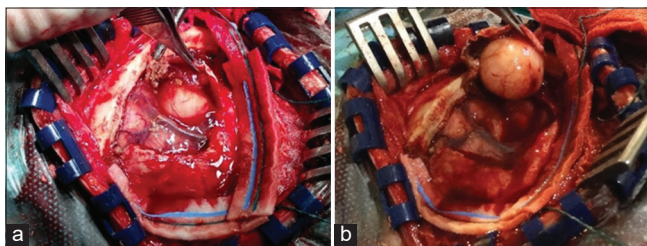
In this particular case, hypertension and hypocoagulation are considered risk factors causing the tumor to be more prone to bleeding when some minor tumor change occurs.<sup>[25]</sup>



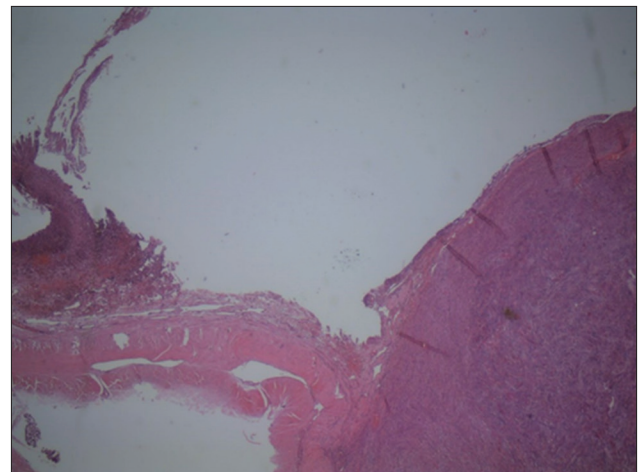
**Figure 1:** (a and b) Head computed tomography in soft tissue and bone window, showing a spontaneously hyperdense left frontal rounded lesion with ipsilateral subdural hemorrhage and the hyperostotic bone reaction.



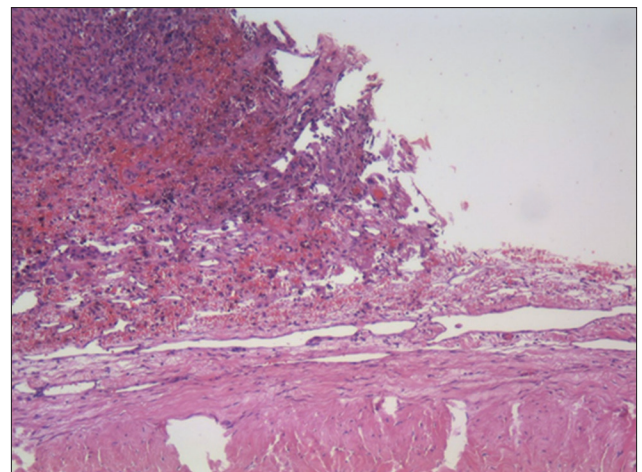
**Figure 2:** (a and b) Axial and coronal T2-weighted head magnetic resonance imaging confirming the presence of the left frontal lesion with adjacent subdural hemorrhage causing important mass effect and right midline shift.



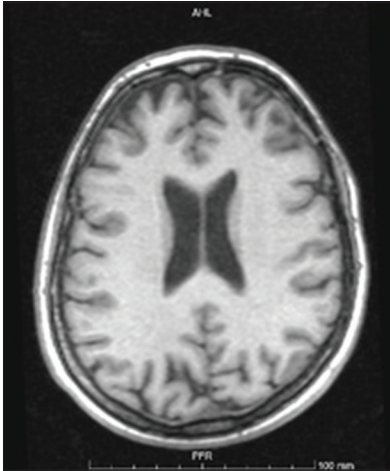
**Figure 3:** (a and b) Intraoperative images of meningioma exposure after major subdural hemorrhage evacuation.



**Figure 4:** H and E  $\times 25$ , in the left corner, we see the subdural hematoma membrane detached from the meningioma periphery. At the right corner, the transitional meningioma is shown.



**Figure 5:** H and E  $\times 100$ , in the left upper corner: subdural hematoma membrane (superior) and the dura-mater (inferior) infiltrated by erythrocytes.



**Figure 6:** Axial T1-weighted head magnetic resonance imaging 6-month postoperatively showing no signs of the previous lesions.

Other risk factors postulated in the review studies are age >70 or <30; location of meningioma being intraventricular or convexity; histopathology consistent with being malignant, fibrous, or angioblastic; and traumatic brain injury.<sup>[4,20]</sup>

Notwithstanding hypertension and hypocoagulatory states being common in the Portuguese population and thus present in many patients that harbor meningiomas, we found no other cases reported in our population, meaning those factors are probably not by themselves essential to this clinical presentation.

Considering neuropathological examination, the proximity of meningioma periphery and contiguous dura mater infiltrated by erythrocytes to the subdural hematoma membrane make this the probable origin of the hemorrhage. The rupture of a vessel in this transition zone could explain this phenomenon. Furthermore, the fact that the meningioma had minor intrinsic hemorrhages that seem to have no contact with the exterior layers of the lesion further supports this theory.

As we considered the bleeding of the meningioma as the probable cause for the acute subdural hemorrhage, we opted to remove both the hematoma and the lesion simultaneously in the same craniotomy.

## CONCLUSION

Spontaneous subdural hemorrhage is an extremely rare presentation for convexity meningioma, but the possibility of an underlying lesion should always be entertained in cases of spontaneous intracranial hemorrhage, even those occurring in the subdural space.

Thorough characterization of possible causality between lesions is necessary, as it will greatly impact on therapeutic decisions.

## Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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