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# RECURRENT ACUTE SUBDURAL BLEEDING AS A RARE COMPLICATION OF A HEMORRHAGIC NON MALIGNANT MENINGIOMA

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**We report the case of a 66-year-old male patient who presented acute and sub-acute subdural hematomas complicating a grade I meningioma.**
**In the absence of trauma, detection of a subdural hematoma necessitates an etiologic research, in particular the exclusion of a vascular anomaly or a tumour. Subdural bleeding as a complication of a non malignant meningioma is a very rare and threatening situation and requires prompt surgical removal of the tumor.**

**Key-word: Meningioma.**

Acute subdural haemorrhage is a frequent finding after head trauma, resulting from avulsions of bridging veins crossing the subdural space and subsequently bleeding between the dura mater and the arachnoid membrane, or from cortical lacerations. In the absence of trauma, a vascular malformation or a tumoral lesion must be excluded (1).

Meningiomas are one of the most common primary neoplasms of the central nervous system, arising from the arachnoidal cells of the meninges. Meningiomas are benign in the vast majority of the cases,. The World Health Organization (WHO) classification of meningiomas has been revised in 2007 (2) as follows: benign = grade I (90%), atypical = grade II (7%) and anaplastic = grade III (2%). Meningiomas are often highly vascularized and have a tendency to calcify. Main complications of these tumors are due to their mass effect on brain, spinal cord, nerves and plexuses, resulting in progressive neurological deficits (e.g. neurocognitif, motor and sensory dysfunctions), focal seizures, or intracranial hypertension. Meningioma bleeding into the parenchyma or the subdural space is rare. Our purpose is to report a very exceptional case of recurrent meningioma bleeding into the subdural space.

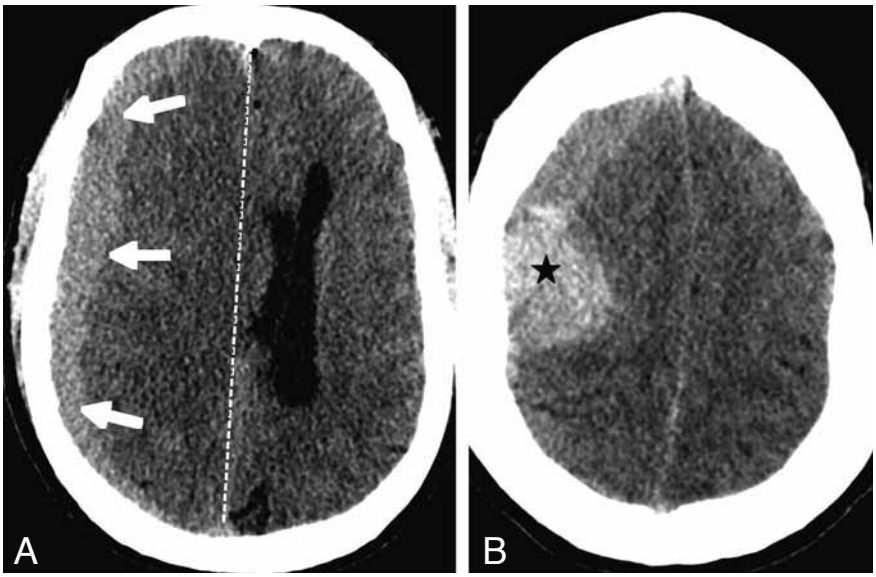


*Fig. 1.* — Two non-enhanced axial MDCT views show a 17mm-thick sub-acute subdural hematoma with a mean density of 43 HU (A – white arrows), associated to a 4 × 4.5 × 2.5 cm hyperdense (hemorrhagic) right frontal convexity dural-based tumor (B – black star). Brain midline shift is observed with sub-falcine herniation (A – mid-line superrimposed in dotted line).

## Case report

A 66-year-old man was admitted to our Emergency Department with a 24-hour history of mental confusion. He had a relevant medical history of treated high blood pressure and atrial fibrillation. He therefore received for a long period an anti-vitamin K treatment he had stopped a few days before admission because of an infection. Clinical examination revealed the presence of a spatiotemporal disorientation, gait disorder, a positive Romberg sign, a left facial paresis grade 3 on the House Brachman grading scale, a left hemiparesis 4+/5 and a right grasping.

Pre- and post-contrast injection cerebral MDCT at admission revealed the presence of right frontal dural based extra-axial tumor of 4 x 4.5 x 2.5 cm in the three orthogonal axes. The tumor was heterogeneous with spontaneously hyperdense foci, suggesting intratumoral bleeding. Mild tumor enhancement was observed after IV contrast material injection. A 17 mm ipsilateral subdural hematoma (SDH) of intermediate density (43 HU), suggestive of a sub-acute SDH, was associated to the tumor resulting in contra-lateral deviation of the midline and mass effect on the right lateral ventricle (Fig. 1).

The patient underwent undelayed surgical decompression with evacuation of the SDH hematoma. Immediate tumor removal was not realized because the procedure was performed during the night and because a second intervention could be scheduled in the following days with a more experienced neurosurgeon. Procedure was uncomplicated. Post-operative follow-up CT demonstrated complete resolution

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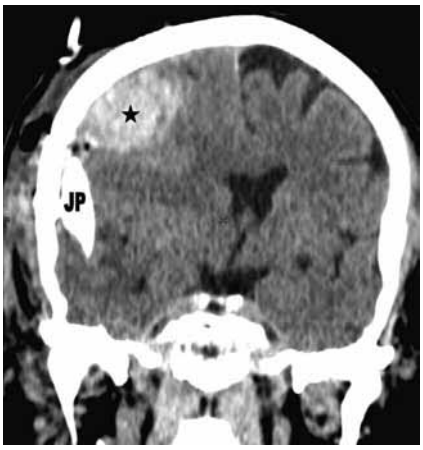


*Fig. 2.* — Reformatted coronal view of follow-up unenhanced MDCT after surgical drainage of the hematoma. SDH and brain herniation have been reduced but underlying heterogeneous meningioma is unchanged (black star). A subdural Jackson-Pratt drain has been put in operative site to complete the evacuation of the SDH (JP).

of the collection but persistence of the dural based extra-axial tumor (Fig. 2). Our patient presented a complete recovery of his cognitives functions but persistent slight left brachio-facial paresis and left hand paraesthesias.

Initial cerebral magnetic resonance (MR) examination was performed five days after admission. Prominent areas of very low-T2 and T2* signal intensity were observed throughout the tumor, confirming intratumoral multifocal hemorrhages (Fig. 3A,B). The alternative diagnosis of intratumoral calcifications was not considered because density on unenhanced CT image was not considered sufficient for this hypothesis. Post-contrast T1-weighted images revealed upper area of enhancement which was restricted to the superior third of the mass (Fig. 3C). These imaging features were evocative of a hemorrhagic meningioma. Radiological differential diagnosis included atypical meningioma or meningeal hemangiopericytomas.

Surgical removal of the meningioma was initially scheduled one week after the drainage, but our patient voluntarily postponed the procedure in order to consult other neurosurgeons.

Follow-up MR examination three months after initial event revealed the recurrence of an acute SDH localized anteriorly to the meningioma with subsequent worsening of the brain midline shift (Fig. 4). After right frontal craniotomy, complete removal of the collection and the extra-axial tumor was performed. Postoperatively, the patient presented a progressive recovery of his left brachio-facial paresis and left hand paraesthesias.

Histologic examination revealed a benign meningioma with large hemorrhagic foci. A mean Ki-67 proliferation index of 5% up to 10% in some tumor areas was observed. This meningioma was classified as grade I according to the "WHO 2007 classification and grading of meningiomas" (2).

## Discussion

On cerebral CT or MRI, meningiomas presented as well-defined lobulated mass.

On CT scan, meningiomas usually appear hyperdense with respect to cerebral grey matter (1). Necrosis, cystic or lipomatous infiltration and old intratumoral hemorrhage appear as low density areas. On the contrary, calcifications and acute hemorrhages appear hyperdense. Thereby, lesions often appear as heterogeneous masses (1, 3). Hyperostosis or bone destruction can be observed in contact with the tumor.

On T1-weighted MRI, most tumors present a nearly isointense signal with the cortical grey matter. Hypointense meningiomas are less frequent, and hyperintense tumors

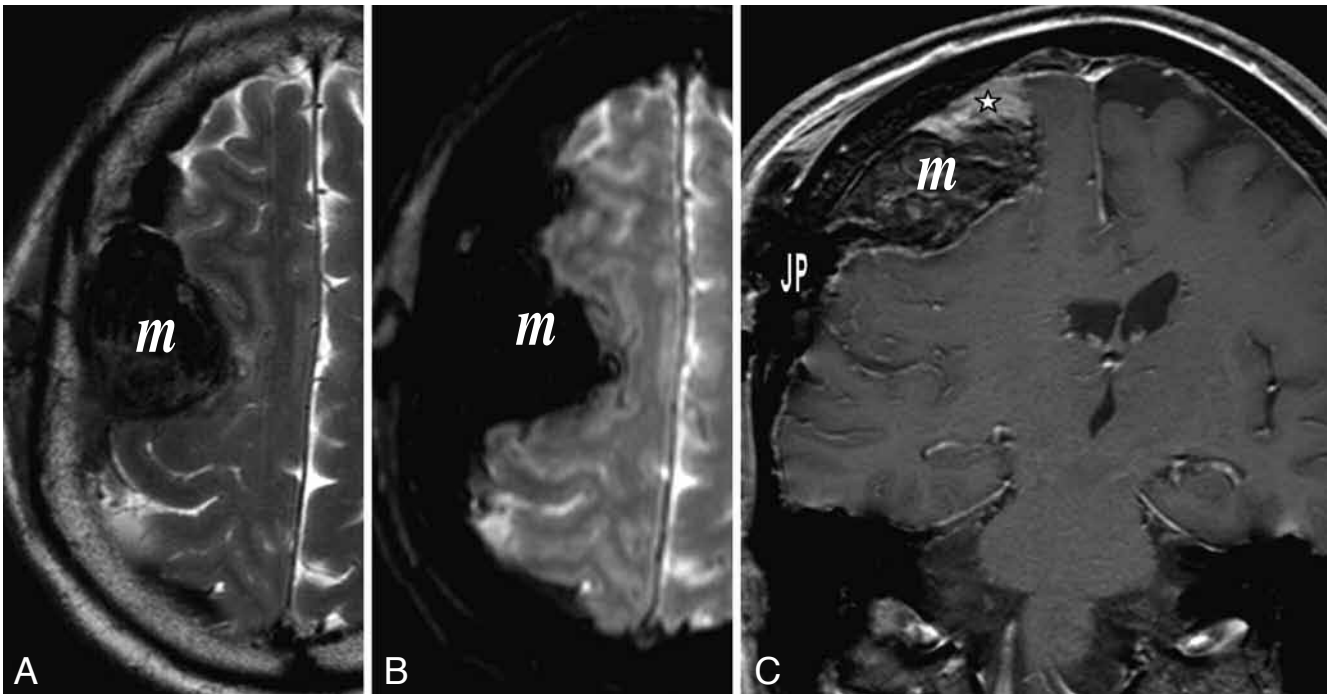


*Fig. 3.* — Axial T2 (A) and T*2-weighted (B) and frontal post-contrast T1-weighted images (C) MR views after surgical drainage of the subdural hematoma. Very low hypointense T2 and T*2-weighted signal (together with susceptibility artifacts) confirmed intratumoral hemorrhage (m – meningioma). Heterogeneous enhancement is observed after gadolinium injection, restricted to the upper third of the tumor (C – white star). Subdural JP drain is seen as an artifacted low signal area (C – "JP").

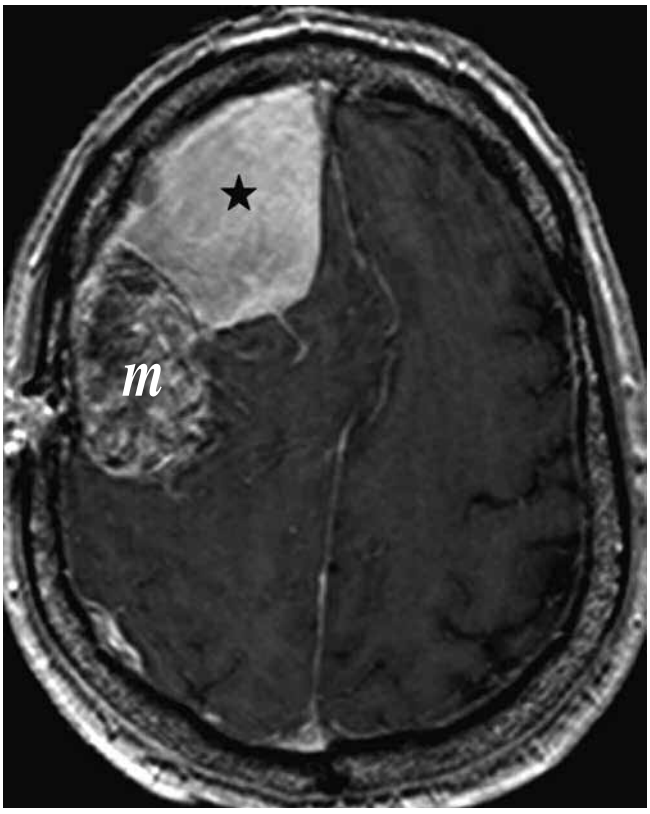


*Fig. 4.* — Axial post-contrast T1-weighted MR view 3 months after initial imaging work-up. Recurrence of an acute localized subdural hematoma (black star) in front of the meningioma (m).

are very rare (4). On T2-weighted MRI, about half of meningiomas remain isointense with the brain cortex, hyperintense masses are secondly more frequent than hypointense tumors (4).

After intravenous contrast agent injection, markedly homogenous diffuse enhancement or, on the contrary, strongly heterogeneous enhancement can be observed. Some meningiomas present an atypical ring enhancement and may be confused with a necrotic primary or metastatic neoplasia, or even an abscess (3).

Typical localization concern the cerebral convexity, the parasagittal region or the sphenoid wings, but meningiomas may equally originate in less frequent sites like the orbit, paranasal sinus, ventricles or the diploic space of the calvaria (intraosseous meningiomas) (3). Rare locations at distance of the neuraxis have been reported, including the mediastinum, lung and adrenal glands (3).

Tumor bleeding most often occurs within the tumor itself. Intracerebral and subarachnoid spaces (including ventricles) are the next frequent locations of tumor-associated bleeding. Subdural locations are very uncommon (5).

Intratumoral bleeding is observed in approximately 5% of intracranial tumors, commonly seen within pituitary adenomas, malignant gliomas and metastatic tumors (most often from melanoma, bronchogenic carcinoma, choriocarcinoma and hypernephroma) (5, 6).

Although meningiomas have often a high vascular density, spontaneous bleeding rarely occurs. However, small intratumoral bleedings may probably be overlooked because of the infraclinical symptoms (6). Meningiomas are most commonly associated with subarachnoid hemorrhage (6). Bleeding from meningioma within subdural is rare and usually co-exists with other hemorrhagic locations e.g intratumoral bleeding (6,7). SDH was not reported in the largest autopsic series of intracranial meningiomas reported by Cushing and Eisenhardt (1938) or Hoessly and Olivecrona (1955).

Although intracranial meningiomas are usually considered as benign tumors having a slow and indolent course (8), clinical presentation of the rare cases of HSD associated with meningioma is frequently sudden, with quick impairment of consciousness and motor disorders. Fatal issue is observed in approximately 50% of those cases (6). In a review of 20 cases of SDH caused by meningioma, Chaskis et al. (6) reported that most of the operated patients had a full or good postoperative neurological recovery. Consequently, these patients should have rapid tumor surgical removal to get the maximum benefit (6, 7).

Sunada et al. (9) reported a case of fibrous-type meningioma with an elevated Ki-67 index at 6.7 associated with acute SDH and suggested that a high index of cell proliferation may act as a risk factor for hemorrhage. But tissue necrosis and/or hemorrhage may provoke by itself a Ki-67 raise that could be therefore a consequence rather than a causal factor of tumor hemorrhage.

Chaskis et al (6) suggested that malignant histological sub-types of meningiomas could be associated to an increased risk of tumour-associated bleeding, but they included into their series "angioblastic meningiomas" which are now classified as meningeal hemangiopericytomas and were removed in the 2007 WHO classification of meningiomas group. Kashimura et al. (10) adhered to this hypothesis, but for Worm et al. (11) histological grading of the tumors failed to reveal significant relationship with hemorrhagic events.

Anticoagulant therapy or blood dyscrasias have never been implicated in meningiomas associated hemorrhage in the literature until now, but in our patient, this could be an associated factor. Head trauma could act as a precipitating factor.

Several hypotheses have been proposed to explain spontaneous hemorrhage associated with intracranial meningioma (10, 11), e.g. rupture of abnormal tumor vessels, vaso-active substances released by meningiomas, venous thrombosis leading to tumor necrosis or rapid tumor growth leading to stretching of subdural bringing veins and their rupture after minor trauma. In some cases, SDH could be secondary to intra-meningioma hemorrhage breaking through the tumor to the subdural spaces. The localization of the tumor at the cerebral convexity could be a risk factor for hemorrhage (6, 8, 11).

In conclusion, many different factors may be synergistically involved

into meningioma-related SDH and the combination of several causal factors is probably necessary.

## Conclusion

Subdural hematoma associated with meningioma is a very uncommon condition. We have reported a case of recurrent SDH in another location due to a meningioma left in place at the first surgery. Physiopathological mechanisms of this association may be multiple. Clinical presentation is frequently sudden and severe. Mortality is high, but patients highly benefit from rapid surgical management including SDH drainage and removal of the tumor.

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