Case Report

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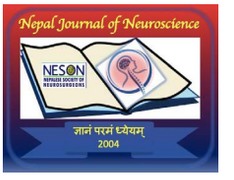
# Spontaneous Subdural Hematoma associated with Meningioma : Two case reports

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## Abstract

Spontaneous subdural hematoma rarely presents with ahyper vascular or malignant tumor but even less frequently in a benign tumor like meningioma. We encountered two cases with spontaneous subdural hematoma associated with benign meningioma.

**Case Description:**A 47-year-old Asian woman presented with severe headache and forgetfulness, initiated 3 months before, with no other complaints, denying any previous head trauma. Head computed tomography revealed a chronic left hemisphere subdural hematoma for this issue she underwent burr hole and evacuation of hematoma 1 month before. However her symptoms was not relieved and her magnetic resonance imaging of brain showed left frontal lesion , suggestive of meningioma. Surgical treatment was performed. The tumor was histologically confirmed as a transitional meningioma grade I.After surgery, no neurological deficits were registered, and headache subsided.

Similarly, second case , a 56-year-old woman presented with complaints of severe headache for 6 hours. There is no history of seizure like activities, any previous history of head trauma. On examination, no neurological deficits noted. Computed Tomography(CT) of head showed a hyper dense extradural lesion with acute subdural lesion on left hemisphere. MRI brain revealed well defined extra axial round to oval lesion measuring 3.1 X 3.5 X 2.5 cm with areas of heterogeneous enhancement of the left parietal parasagittal area .She underwent left parietal craniotomy and en-bloc tumor resection done. Post operatively there was no neurological deficits and she was discharged on 5thpost-operative day.

**Conclusions:**As we could not identify any other cause for the subacute subdural hematoma, hemorrhage from the meningioma was the most probable cause.Although meningiomas are commonly benign according to their histological traits, they can lead to spontaneous bleeding and cause neurologically unstable condition. Therefore, meningiomas need to be considered as a cause of spontaneous subdural hematoma if radiologically suspicious, which should be reflected by proper management for a positive outcome.

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

## Introduction

Of all primary brain tumors, meningioma is the most common one, presenting in 37.6% of the cases. This entity makes up 53.3% of all non-malignant brain or CNS tumors[1]. However, spontaneous bleeding from meningioma is rare and the mechanism is not found yet[2] . Usually, meningioma cases present with typical symptoms like headache, dizziness, seizures or focal neurological deficiencies and are often diagnosed incidentally. The extent of these clinical symptoms shows variability depending on factors like size and location of the tumor, as well as the invasive growth into adjacent vessels or tissue and the obstruction of cerebrospinal fluid pathways[2]. Histopathologically, 80 – 90% of the meningioma cases diagnosed, are classified as benign WHO grade I tumors [1,3]. Even though their risk for bleeding is much lower than that of a malignant tumor, incidences of bleeds ranging from 1.3% - 2.4% have been reported as results of preoperative embolization, radiotherapy or spontaneous events[4,5]. Most commonly, meningioma related bleedings occur as subarachnoid hemorrhage, with intracerebral hemorrhage being the second most frequent [6,7]. Hemorrhages in patients with meningioma generally occur more seldom as subdural hematoma, even though the usual location of the tumor being the subdural space. The incidence of subdural hematoma is reported to be less than a quarter of that of intracranial hemorrhage in meningioma patients [5,8,9]. Here, we report two cases of nontraumatic SDH originating from a small convexity meningioma. We discuss the possible mechanisms of tumor bleeding, as well as the outlines of this condition, with a review of the previously published literature.

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### Case Illustrations

### Case 1

A 47-year-old Asian woman presented to our emergency department with the complaints headache for last 3 months. The headache was gradual on site, generalized in nature, throbbing in nature, without any aggravating or relieving factors and progressively worsening over last 2-3 weeks. It was followed by forgetfulness as revealed by her relatives. She also complained of an episode of brief loss of consciousness a month back but denies any history of relevant trauma. Her relatives also stated that she has been drowsy ever since the last syncopal attack. Her medical and social history was unremarkable. She was initially taken to another center where a CT scan of head was performed which revealed left sided chronic SDH (Fig .1) and an emergency burr hole and evacuation was done with an up-to-the mark intra operative and post- operative events. Her symptomatic relief was short lived with re-emergence of the precedent symptoms for which she presented to our center for further evaluation and definitive management. Examination of her vitals at the time of presentation to the casualty was within normal range. and all laboratory tests were within the normal range. After thorough clinical, neurological and laboratory examination, brain magnetic resonance imaging (MRI) was dispatched which demonstrated a heterogeneously enhancing, round-shaped, well defined broad based measuring 2.5 cm × 1.5 cm, extra-axial mass along the convexity of left frontal lobe with diffuse SDH underneath itwith an ensuing midline shift to the right (Fig. 2). Proper counselling to the patient and her relatives was executed explaining the culprit behind her unyielding neurology with recommendation to undergo an upfront craniotomy and excision of the neoplasm. She was admitted to the neurosurgical department and treatment was initiated with intravenous mannitol and high dose steroid to counteract the increased intracranial pressure and mass effect due to perilesional edema. On the next day, she underwent neuronavigation guided craniotomy and gross total resection of the tumor.

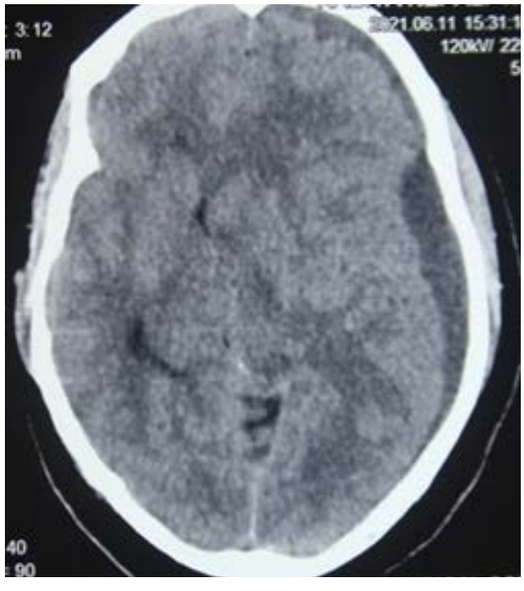


*Figure 1: Preoperative axial computed tomography 1 month back ,showing an chronic subdural hematoma extending to the right fronto-temporo-parietal area*

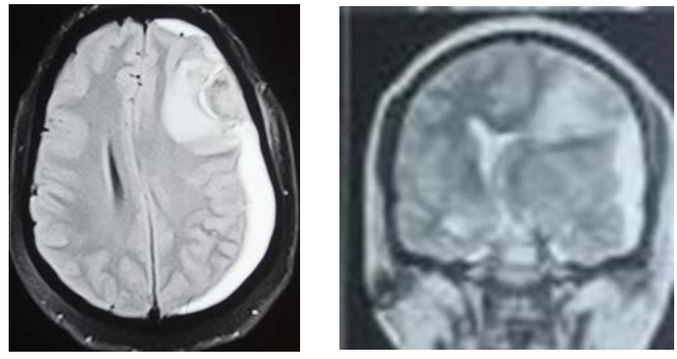

*Figure 2: T2-weighted brain magnetic resonance images demonstrating a heterogeneously enhanced, 2.5 × 1.5 cm sized, extraaxial mass on the left frontal convexity with diffuse subdural hematoma in both axial view (a) and coronal view (b)*

Following neuronavigation guided craniotomy, dura was opened which revealed an acute type SDH. The meningioma as depicted in the MRI was conspicuous as it was concealed within the confines of blood clots. As the hematoma was evacuated from the surgical field, the neoplasm came into picture and a gross total excision was executed. A notable intra operative finding was a small tearing on a cortical vein adhered to the tumor base arachnoid capsule. Adequate hemostasis was maintained. Bleeding points were secured within the tumor domain. A concern about replacing the bone flap was steered clear considering an insignificant brain swelling. Henceforth, the bone flap was grafted back and the wound was closed in anatomical layers.

The postoperative course was uneventfulwithout any alarming neurological deficits. Her symptoms gradually improved and was discharged on the 5th postoperative day as an independent individual and an up to par neurology with an advice to be on regular follow up. A brain MRI was recommended 2 months later showed complete tumor removal with no residual or hemorrhage (Fig. 3).

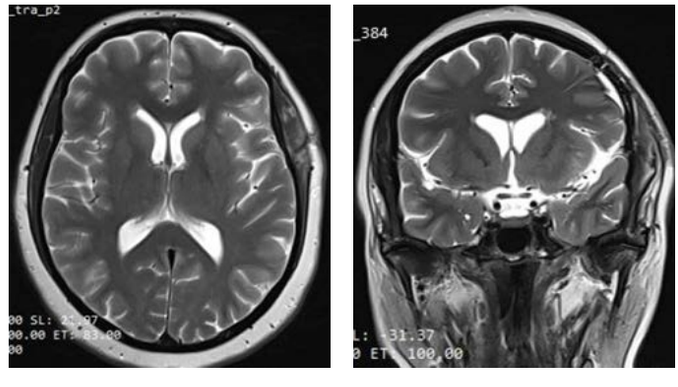

*Figure 3: T2 weighted MRIshowed complete tumor removal with no residual or hemorrhage*

**Histopathology:**

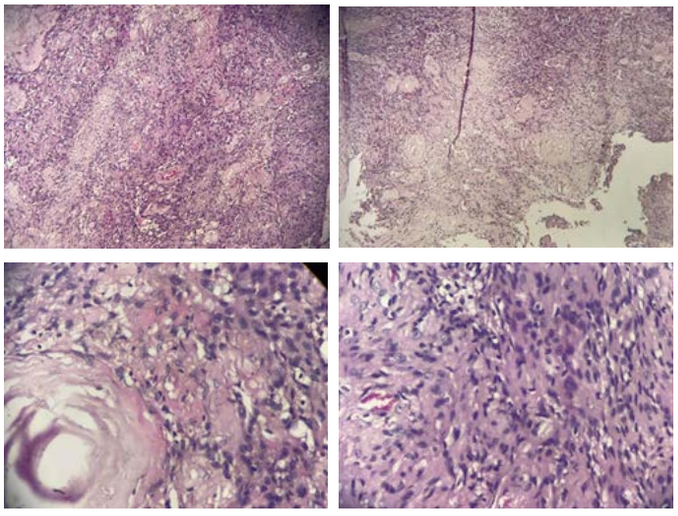

*Figure 4 : showing tumor cells arranged in fascicles and whorl pattern , cells are pleomorphic, The nuclei are round , oval and elongated with peripheral condensation of chromatin , also shows hyalinised and ectatic vessels and psammoma bodies.*

Two months follow-up brain magnetic resonance images revealing no residual meningioma or subdural hematoma

The tissue specimen obtained during surgery was sent for histopathologic analysis which revealed tumor cells arranged in fascicles and whorl pattern (Fig.4). The cells are mildly pleomorphic with round to oval and spindle shaped having moderate to abundant amount of eosinophilic cytoplasm. The nuclei are round to oval and elongated with peripheral condensation of chromatin. It also showed hyalinized and ectatic blood vessels and Psammoma bodies. Necrosis and atypical mitotic figures are not seen.

These picture was suggestive of Transitional Meningioma WHO Grade I.

**Case 2**

A 56-year-old woman presented to our institution with complaints of severe headache for 6 hours. There is no history of seizure like activities, there is no any previous history of head trauma. She had no previous medical history. On examniation, no neurological deficits noted. Computed Tomography(CT) of head showed a hyperdense extradural lesion with acute subdural lesion on left hemisphere (Fig.5 A). MRI brain revealed well defined extra axial round to oval lesion measuring 3.1 x 3.5 x 2.5 cm with areas of heterogeneous enhancement of the left parietal parasagittal area seen closely abutting the adjacent left parietal bone with a small focal defect in the inner table and a small extension into the diploid space (Fig.5 B & C). Calcification with mass effects with displaced underlying parietal lobe and effaced left lateral ventricle and lesion is seen.

She underwent left parietal craniotomy and en-bloc tumor resection done. Grayish white extra axial vascular dural lesion with initial stage of calcification with small subdural hematoma noted. Bone also involved. Post operatively there was no neurological deficits and she was discharged on 5thpost-operative day.

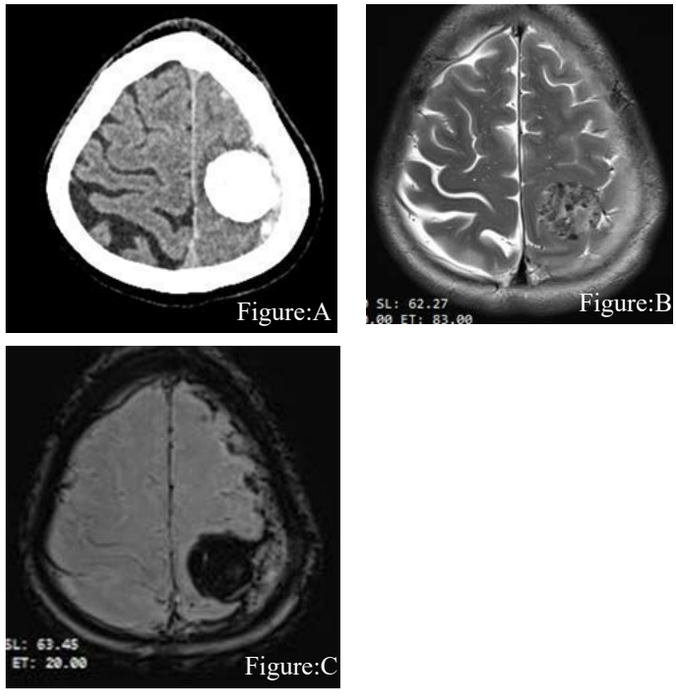


*Figure:5 (A)Preoperative axial computed tomography showingwell defined extra axial round to oval lesion measuring 3.1 x 3.5 x 2.5 cm at left parietal parasagittal area (B) and (c) MRI T2/ SWI showing well defined extra axial round to oval lesion measuring 3.1 x 3.5 x 2.5 cm with areas of heterogeneous enhancement of left parietal parasagittal area seen closely abutting the adjacent left parietal.*

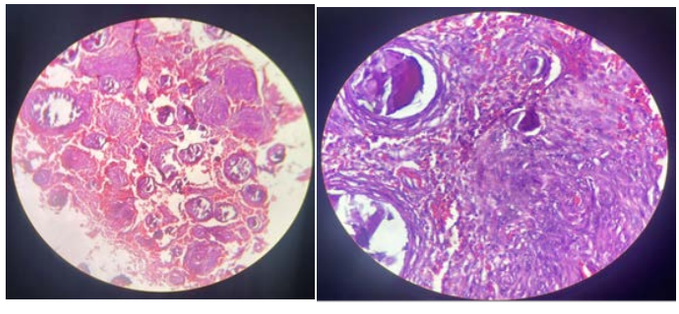

*Figure 6: Histological examination showing tumor cells are arranged in diffuse and whorl pattern with plenty of psammoma bodies , thecells are mildly pleomorphic and round to oval having moderate to abundant amount of eosinophilic cytoplasm.The nuclei are round , oval and elongated with peripheral condensation of chromatin , also shows hyalinised and ectatic vessels and psammoma bodies*

## Discussion

Spontaneous SDH can be defined as any unusual disruption of cranial vessels within the subdural space and an ensuing accumulation of variable amount of hematoma without any preceding traumatic event[10]. Unlike the traumatic SDH, spontaneous SDH is rare and only infrequently reported.The major causes accompanying spontaneous SDH are rupture of cortical arteries, coagulopathy, vascular malformation or

aneurysm, neoplasm, cocaine use, or spontaneous intracranial hypotension[11]. Amidst these etiologies, as much as 5.4% of spontaneous SDH cases are found to correlate with an underlying brain neoplasms, explicitly in middle-aged adults,comparable to our case[11,12]. More so,the metastatic variant of brain tumors or high-grade primary tumors are peculiarly liable to exhibit as an intracranial hemorrhage because of their malignant and invasive histological trait kindred with its innate coagulopathy[13]. Nonetheless, even benign tumors like meningioma or schwannoma as well unveil with the aforementioned[4].

Review of the literature disclosed that approximately 1.3–2.4% of all meningiomas manifest in the form of intracranial hemorrhage[4,5]. Moreover, the bleeding tendency of meningioma is shown to be increased in convexity and intraventricular locations, as well as in fibroblastic, malignant, or angioblastic subtypes[8,9,14,15]. Matsuoka et al. in his review study found that nine cases of repeated bleeding from benign meningiomas and demonstrated that its related mortality of 28–50% is not negligible16. On that account, an immediate diagnosis and prompt surgical intervention by evacuation of hematoma and resection of the underlying neoplasm is warranted.

SDH is a rare type of hemorrhage related to meningioma, particularly in cases with benign histology. Nonetheless, tumor location and the apoplectic event is a matter of debate while some authority still hold contend that the whereabouts of the tumor has no concurrence with hemorrhagic events[21]. Furthermore, with regards to the histological subtypes of meningioma, scrutiny of the reported cases portray that the risk of spontaneous SDH is highest among the meningothelial type, followed by the transitional subtype[18,19,28].

Angiomatous meningioma ranks among the rarest variant of meningioma accounting for approximately 2.1% of all meningiomas. The attribute of these tumors are stipulated by the presence of highly vascularized tissues intervening in the background areas of the syncytial pattern of typical meningioma cells[29,30]. Its vascular channels mostly consist of small- or medium-sized, markedly hyalinized walls[31,32]. The inherent tendency of this variant to bleed is ascribed to its numerous vascular layout and the often supplemented peri tumoral edema at the outset of disease process, which in all likelihood may be attributed to the significant role of the angiogenic protein namely, vascular endothelial growth factor. Hua et al. in his study revealed that peri tumoral edema was encountered in 47.1–100.0% of angiomatous meningioma[32]. An abrupt deterioration in neurological status of patient harboring meningioma, henceforth, may be attributed to progression of cerebral edema and an ensuing mass effect. Fortunately, the clinical aftermath of this tumor is innocuous considering its histologically benign genre.

A discrete conjecture in the genesis of SDH kindred to benign meningioma is yet to be formulated. Nonetheless, several authorities have proposed several presumptionshinged on their intraoperative findings and pathologic examinations They include (1) rupture atthe weakest point of a vessel wall in the distended feeding artery with tumor growth; (2) disruption of an abnormally developed vessels within the tumor, with consequent intratumoral hemorrhage or necrosis; (3) change in histochemical dynamics by vasoactive substances released by the tumor cells, leading to neovascularization or forming a neomembrane to the same degree as in the aetiopathogenesis of chronic SDH; and (4) probablevulnerability of the bridging vessels or subdural veins stretched by the tumor mass and consequent susceptibility to minor trauma[6,7,15].

With reference to our case, there were no evidence of cellular necrosis within the tumor locus. However, a myriad of hypervascular structures and related bleeding were noted. Surprisingly, a small tearing on the wall of cortical vein beneath the tumor margin was noted, presumably,implying a mechanical stretch and distortion by inertial impact of the neoplastic bulk. Not to mention, preceding minor traumatic events caused by vigorous coughing or a Valsalva maneuver has also been reported to be a culprit in the inception of vascular injury[12,15,33]. Nonetheless, controversy still persist,whilst most of the bleeding meningiomas were indecipherable to the numerous hypothesis behind the emergence of hemorrhagic event, on that account, rather an amalgamation of diverse possible factors could take root instead.

## Conclusions

Meningioma exhibiting with a spontaneous SDH is an exceptional event. While no distinct pathophysiology behind this occurrence is postulated, the benign convexity meningiomas are among those thatpresent frequently as an SDH and their clinical progress can be worse than expected. While and infrequent occurrence, a meningioma should be fully suspected as the cause of a spontaneous SDH in cases with no vascular abnormalities or coagulopathies. Furthermore, an early diagnosis and prompt management with hematoma evacuation and tumor excision is the assurance to alleviate neurologic symptoms.

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