Subarachnoid Hemorrhage in a Patient With a Meningioma and an Unruptured Aneurysm
—Case Report—

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Abstract

Subarachnoid hemorrhage (SAH) is usually elicited by cerebrovascular disease and infrequently by brain tumors. A 64-year-old woman presented with SAH with a left petrous meningioma and an unruptured left internal carotid-posterior communicating artery (IC-PcomA) aneurysm. She suffered sudden onset of headache and nausea followed by consciousness disturbance 7 days after onset. Computed tomography (CT) revealed diffuse SAH and a tumor at the petrous portion. Angiography demonstrated a left IC-PcomA aneurysm. Under a diagnosis of a ruptured aneurysm and a coincidental meningioma, we performed neck clipping of the aneurysm. However, intraoperatively we found that the aneurysm was unruptured and we subsequently performed tumor resection. Intraoperatively we could not find the cause of SAH during resection of the meningioma. The histological diagnosis was transitional meningioma with deposition of fibrin on the surface of the tumor. The findings of initial CT and magnetic resonance imaging, and pathological results could not conclude the definitive etiology of SAH in this case.

Key words: meningioma, subarachnoid hemorrhage, unruptured aneurysm

Introduction

Intracranial hemorrhage from an intracranial tumor is rare compared to cerebrovascular disease. A study of the characteristics of intracranial hemorrhage from tumors reported that 1.3% of meningiomas result in intracranial hemorrhage.234 Intracranial hemorrhages from tumors tend to be intratumoral and subarachnoid hemorrhage (SAH) attributable to meningioma is rare.5–7,9–12,15,17,18,24–26,28,29)

We treated a patient with SAH of unknown origin, coexisting with unruptured left internal carotid-posterior communicating artery (IC-PcomA) aneurysm and left petrous meningioma. We discuss its etiology based on the clinical and histological findings.

Case Report

A 64-year-old woman with no familial history of SAH experienced sudden onset of headache and nausea, and was admitted to another hospital. She developed consciousness disturbance 7 days post-onset and was transferred to our department. On admission, she manifested consciousness disturbance without focal deficits. Computed tomography (CT) revealed diffuse SAH in the cisterna magna and carotid cistern and a left petrous tumor with homogeneous enhancement by contrast medium (Fig. 1A). Cerebral angiography performed 8 days post-onset revealed a left IC-PcomA aneurysm and tumor stain of the left petrous tumor fed from the ascending pharyngeal artery (Fig. 2). There were no other abnormal findings including aneurysms, venous varix around the tumor, or apparent vasospasm. Based on these findings, our diagnosis was SAH due to a ruptured left IC-PcomA aneurysm in the presence of a coincidental left petrous tumor.

She underwent left fronto-temporal craniotomy for aneurysm neck clipping 9 days after the onset. However, at surgery we found that the aneurysm was unruptured (Fig. 3). T1- and T2-weighted magnetic resonance (MR) imaging obtained 12 days after the onset revealed hyper- and hypointense areas, respectively, on the tumor surface (Fig. 1B). These findings were suggestive of subacute hemor-
Fig. 1  A: Computed tomography scans without (left and middle) and with contrast medium (right) acquired 7 days after onset demonstrating diffuse subarachnoid hemorrhage in the carotid cistern, interhemispheric cistern, ambient cistern, sylvian cistern, and cisterna magna. Note the homogeneously-enhanced extra-axial mass lesion in the left petrous portion.  B: T1- (left) and T2-weighted magnetic resonance images (right) acquired 12 days after onset demonstrating hyperintensity and hypointensity, respectively, on the tumor surface (arrows).

Fig. 2  Angiograms obtained on admission 8 days after onset.  A: Left internal carotid angiogram (three-dimensional reconstruction) demonstrating a left internal carotid-posterior communicating artery aneurysm. No severe vasospasm was noted.  B: Left external carotid angiogram showing staining of the petrous tumor.

Fig. 3  Intraoperative photographs at neck clipping of the left internal carotid-posterior communicating artery aneurysm. Microscopic (A) and endoscopic (B) views showing no findings suggesting the rupture point on the anterior or posterior wall.

Fig. 4  A: Photomicrograph of a tumor specimen showing the tumor consisting of spindle cells without marked atypia arranged in whorl formation. Note the fibrous deposit on the surface of the meningioma. Hematoxylin and eosin stain, original magnification ×100.  B: Photomicrograph showing deep blue staining on the surface of the meningioma suggesting that the deposit contained fibrin. Phosphotungstic acid hematoxylin stain, original magnification ×100.

Discussion

The radiological, intraoperative, and pathological findings could not conclude the definitive etiology in this case. One explanation is that SAH of unknown etiology conjoined with both the aneurysm and meningioma. In a recent study, the etiology of the SAH could not be verified by petrous bone. Unfortunately, we could not identify the bleeding site on the tumor. Postoperatively her neurological condition improved and she was discharged free of symptoms.

Histological examination of the tumor specimen showed that the tumor consisted of spindle cells without marked atypia, arranged in whorl formations (Fig. 4A). No hemosiderin-laden macrophages, increased mitotic activity, high cellularity, prominent nucleoli, sheet-like growth, microvascular proliferation, or geographical necrosis were identified. The diagnosis was transitional meningioma. Phosphotungstic acid hematoxylin staining was performed to identify the SAH origin. The surface of the meningioma was stained deep blue, indicating fibrin deposits on the tumor surface, suggestive of prior hemorrhage around the tumor (Fig. 4B).
repeat angiography or postmortem examination in as many as 8.4% of patients with SAH. Two thirds of patients with SAH demonstrated a “perimesencephalic pattern” with good prognosis. In contrast, patients with diffuse clot of unknown etiology had risk of rebleeding. In these patients, most hemorrhage is considered to originate from angiographically occult aneurysm. Our case had diffuse SAH, rather than perimesencephalic SAH, at initial presentation. Although follow-up MR imaging and MR angiography revealed no new findings, this case could be categorized into the latter group of SAH of unknown etiology.

Alternatively, SAH can originate from a meningioma or rupture of vessels around a meningioma. Intracranial hemorrhage from meningiomas is very unusual, and the mechanisms for SAH have not yet been understood. However, a few cases demonstrated the source of SAH to be the stretched cortical or tumor draining vein, invasion into middle cerebral artery or saccular aneurysm, and necrosis formed by rapid proliferation of the tumors. Pathologically, a malignant, angiomatous, and fibrous meningioma has a tendency to bleed. In the present case, there were no findings to suggest the definitive source of SAH, but clot was identified on the tumor surface on MR images obtained 12 days after the onset, and fibrin deposit on the specimens obtained from tumor resection 19 days after resection. Review of the clinicopathological features of 145 meningiomas with spontaneous intracranial bleeding found that all 6 tumors arising in the posterior fossa caused SAH, and none of them presented with intratumoral hemorrhage. This finding suggests that SAH could be caused by rupture of the vessels on the tumor surface, rather than the inner structure, including the meningioma or necrosis, in “the posterior fossa” meningioma. Although no cases are known of SAH attributable to meningioma in a patient with coexisting intracranial aneurysm, we presume that the absence of acute or chronic hemorrhage or hemosiderin-laden macrophages in the tumor did not exclude the relationship between SAH and petrous meningioma.

No systematic studies have analyzed the difference in clinical findings between SAH due to rupture of aneurysm and tumors. However, patients with SAH due to meningioma have minor symptoms without disturbance of consciousness at onset compared to the symptoms of rupture of aneurysm. Instead, a significant proportion of patients with SAH due to meningioma developed rebleeding. Few patients have suffered severe vasospasm after SAH from meningioma. In contrast to meningiomas, pituitary apoplexy with and without SAH frequently causes vasospasm due to hypothalamic damage or vasoactive substance from adenoma, other than the SAH. In this way, SAH due to ruptured aneurysm and meningioma have both common and different clinical features, and part of the clinical characteristics of SAH from a meningioma, including minor symptoms and vasospasms, were compatible to those found in the present case.

The present case, of SAH of unknown origin was found coexisting with both unruptured left IC-PcomA aneurysm and left petrous meningioma.

Conflicts of Interest Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices in the article. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

References


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