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Case Report

A case of subdural hemorrhage due to ruptured cerebral aneurysm presenting with atypical imaging features ^{☆,☆☆}

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ABSTRACT

Acute subdural hemorrhage caused by ruptured cerebral aneurysms is rare. Herein, we report an atypical case of subdural hemorrhage caused by ruptured anterior communicating artery aneurysm in a 49-year-old woman. Computed tomography revealed subarachnoid, intracerebral, and subdural hemorrhages. After the treatment with endovascular coiling significantly decreased the patient's subdural hemorrhage. However, the subdural hemorrhage revealed and became iso-attenuation compared with the white matter on the 11th day, and hypo-attenuation on the 19th day. On the 33rd day, this subdural hemorrhage completely resolved after discontinuation of dual antiplatelet therapy. Due to rapid changes in the radiologic features of SDH, frequent computed tomography scans at least once a week may be needed especially in patients who receive antiplatelet therapy during the vasospasm phase.

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Introduction

Rupture of intracranial aneurysms usually results in subarachnoid hemorrhage (SAH). Although co-existent subdural hemorrhage (SDH) is observed in about 2%-10% of patients with aneurysmal SAH, isolated SDH due to ruptured intracranial aneurysm has been rarely reported [1]. Herein, we report a case of acute SDH with atypical imaging features due to ruptured anterior communicating artery aneurysm.

Case report

A 49-year-old woman with hypertension visited our hospital for sudden-onset posterior cervical pain in absence of any history of trauma. Computed tomography (CT) showed subarachnoid, intracerebral, and subdural hemorrhages in the frontal area and subdural hemorrhage in the left hemisphere (Fig. 1A). Cerebral angiography revealed an aneurysm of the anterior communicating artery, which was identified as the etiology of hemorrhage (Fig. 1B).

Abbreviations: SDH, subdural hemorrhage; CT, computed tomography; SAH, subarachnoid hemorrhage.

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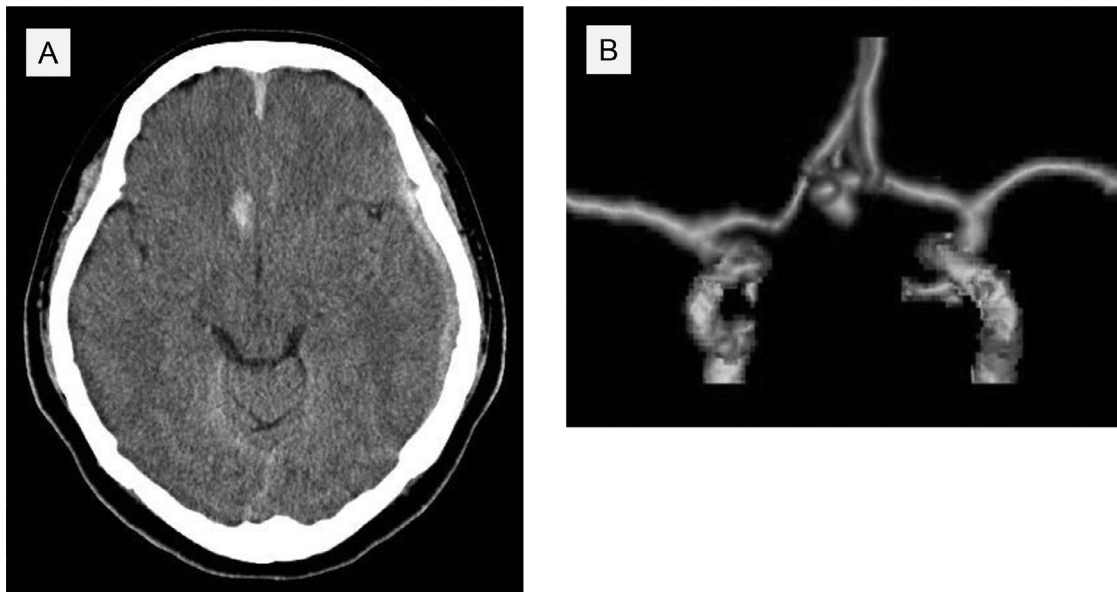


Fig. 1 – Initial CT scan and 3D-CT angiography. (A) CT scan shows an acute subdural hemorrhage over the left hemisphere and intracerebral hemorrhage in the frontal area. (B) 3D-CT angiography shows anterior communicating artery aneurysm.

Endovascular coiling was performed to treat the aneurysm. After coiling, antiplatelet therapy with sodium ozagrel was initiated to prevent vasospasm and acute thrombosis on the surface of coils. After 5 days, CT showed significant improvement in SDH (Fig. 2A). Cilostazol (200 mg/day) was then administered to prevent vasospasm. Due to concerns of potential aggravation of intracranial hemorrhage that might have been caused by dual antiplatelet therapy, sodium ozagrel was discontinued. On the 11th day, CT showed improvements in intracerebral hemorrhage and worsening of SDH (Fig. 2B). Moreover, SDH appeared as iso-attenuation relative to cerebral white matter. These findings were consistent with a diagnosis of subacute SDH. Due to lack of symptoms caused by SDH, cilostazol was continued for 19 days after coiling to prevent vasospasm. Although CT on the 19th day revealed a slight increase in volume, a decrease in density of SDH was observed (Fig. 2C). Since there were no symptoms or neurologic deficits, we did not perform surgical evacuation. On the 23rd day, the patient was discharged without any neurologic deficits. On the 33rd day, CT showed complete resolution of SDH (Fig. 2D).

Discussion

Herein, we describe a rare case of acute SDH due to ruptured aneurysm, which progressed to chronic SDH. After discontinuation of dual antiplatelet therapy for vasospasm, SDH spontaneously disappeared. We suggest that it is important not only to evaluate the vessels during cerebral vasospasm phase in patients with SAH and SDH, but also evaluate SDH on head CT once a week.

Several mechanisms have been proposed to explain occurrence of acute SDH following aneurysmal hemor-

rhage [2,3]. First, minor bleeding could cause adhesions between aneurysm and arachnoid membrane, resulting in SDH with eventual aneurysmal rupture. Second, blood could directly extravasate into the subdural space through high pressure. Lastly, intracerebral hemorrhage could tear the arachnoid membrane to reach the subdural space. In the present case, the patient had no obvious symptoms of minor bleeding, implicating direct extravasation or tearing of the arachnoid membrane as the likely causes of SDH.

Chronic SDH may have already existed in the present case, causing fresh bleeding from the ruptured aneurysm. Thapa et al. [4] described a case of pure acute-on-chronic SDH due to ruptured posterior communicating artery aneurysm. Moreover, Kim et al. [5] reported that complete resolution after 4 weeks of onset was observed in 23% of patients who underwent surgery for chronic SDH. Similarly, our patient may also have had chronic SDH before her ruptured aneurysm, causing SDH to appear, and be absorbed in a short time.

SDH resolution usually demonstrates decreased density and increased width as hygroma. Lee et al. [6] reported that it took more than 80 days, on average, for an increased hygroma to disappear. In this case, the increased lesion disappeared in 14 days. The influx of subarachnoid hemorrhage into the hygroma might have temporally appeared as SDH on day 1 and day 5. Although, there have been no strong evidence of whether the lesion was hygroma or SDH, it might be unusual for lesion to appear, and disappear very quickly as in this case.

In this case, chronic SDH spontaneously resolved after discontinuation of cilostazol. Wada et al. [7] reported that antiplatelet therapy significantly influenced chronic SDH recurrence. Furthermore, a systematic review by Poon et al. [8] showed that the use of antiplatelet drugs was significantly associated with chronic SDH recurrence. In

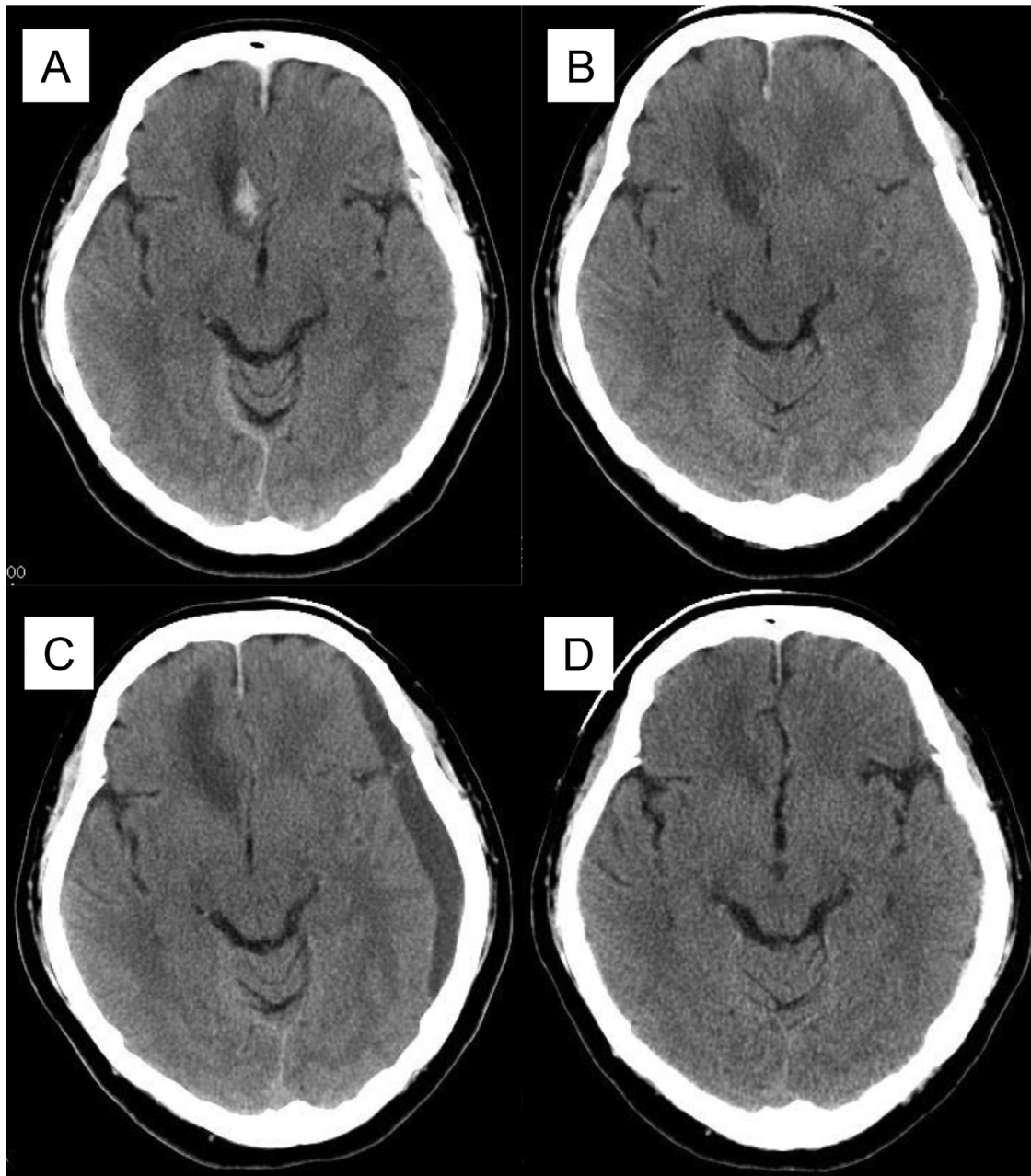


Fig. 2 – CT scans at each time point. (A) CT scan on day 5 shows decreases in SDH. (B) CT scan on day 11 shows decreases in intracerebral hemorrhage and development of SDH. (C) CT scan on day 19 shows increases in volume but decreases in density of SDH. (D) CT scan on day 33 shows disappearance of SDH.

our case, antiplatelet drugs administered to prevent vasospasm may have affected development, and progression of SDH.

Ommaya et al. [9] reported that tearing of bridging veins led to accumulation of subdural hemorrhage regardless of trauma. In this case, laceration of bridging veins accelerated development of acute SDH. Since subarachnoid hemorrhage was minimal and cerebrospinal fluid regulation was normal, the hemorrhage was temporarily cleared. However, bleeding from bridging veins exceeded the capacity of clearance, resulting in gradual accumulation of

blood in the subdural space. Moreover, bleeding from bridging veins was gradually absorbed after discontinuation of cilostazol.

Acute SDH due to ruptured aneurysm is a life-threatening condition, which requires early surgical evacuation with decompressive craniotomy [10]. However, some reported cases of ruptured aneurysms were treated with endovascular techniques [11]. With increases in the number of endovascular treatments, cases of SDH due to ruptured aneurysms may consequently increase. Frequent follow-ups with imaging are necessary to detect any changes in volume, espe-

cially in patients who receive antiplatelet therapy to prevent vasospasm.

Gong et al. [12] reported that internal carotid artery aneurysm was the most commonly associated with isolated SDH. They also reported that anterior communicating artery aneurysms as in the present case were reported to be the third most common. Furthermore, Schuss et al. [1] reported a similar ratio for acute SDH caused by ruptured aneurysms, and there may be a common mechanism for development of isolated SDH and acute SDH.

We report an atypical case of SDH caused by a ruptured cerebral aneurysm. We emphasize that frequent imaging studies are necessary. Furthermore, we also suggest that it is important not only to evaluate the vessels during cerebral vasospasm phase in patients with SAH and SDH, but also evaluate SDH on head CT once a week.

Patient consent

Written, informed consent was obtained from the patient featured in this case report.

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