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Case Report

A case of segmental arterial mediolysis with subarachnoid hemorrhage due to anterior cerebral artery dissection followed by internal carotid artery dissection

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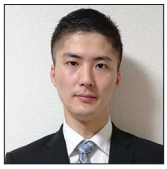

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ABSTRACT

Background: Segmental arterial mediolysis (SAM) causes subarachnoid hemorrhage (SAH) due to intracranial aneurysm rupture and arterial dissection. We encountered a case of SAM-related SAH due to ruptured dissection of the A1 segment of the anterior cerebral artery concomitant with internal carotid artery (ICA) dissection.

Case Description: A 53-year-old man presented with SAH due to a ruptured right A1 dissecting aneurysm. The aneurysm was trapped; however, 7 days after the onset of SAH, he experienced right hemiparesis and aphasia. Angiography showed left ICA dissection; urgent carotid artery stenting was performed, leading to symptom improvement. Abdominal computed tomography angiography showed aneurysms of the celiac and superior mesenteric arteries. He was diagnosed with SAM based on clinical, imaging, and laboratory findings.

Conclusion: In the acute phase of SAM-related SAH, cerebral ischemia could occur due to both cerebral vasospasm and intracranial or cervical artery dissection.

Keywords: Carotid artery stenting, Segmental arterial mediolysis, Subarachnoid hemorrhage

INTRODUCTION

Segmental arterial mediolysis (SAM) is a relatively rare disease characterized by vascular media necrosis without atherosclerotic changes or inflammation. Subarachnoid hemorrhage (SAH) is a rare primary symptom of SAM, and careful management of symptomatic vasospasm and fragile vessels is needed. We report a case of SAM-related SAH due to anterior cerebral artery (ACA) dissection followed by ischemic symptoms due to internal carotid artery (ICA) dissection.

CASE REPORT

A 53-year-old Japanese man was transferred to our hospital with sudden onset of severe headache and disturbance of consciousness. On admission, his consciousness level was E4V5M6 on the Glasgow Coma Scale and he had no neurological deficits. Head computed tomography (CT) revealed a thick SAH with Sylvian hematoma (Fisher Group 4), and CT angiography

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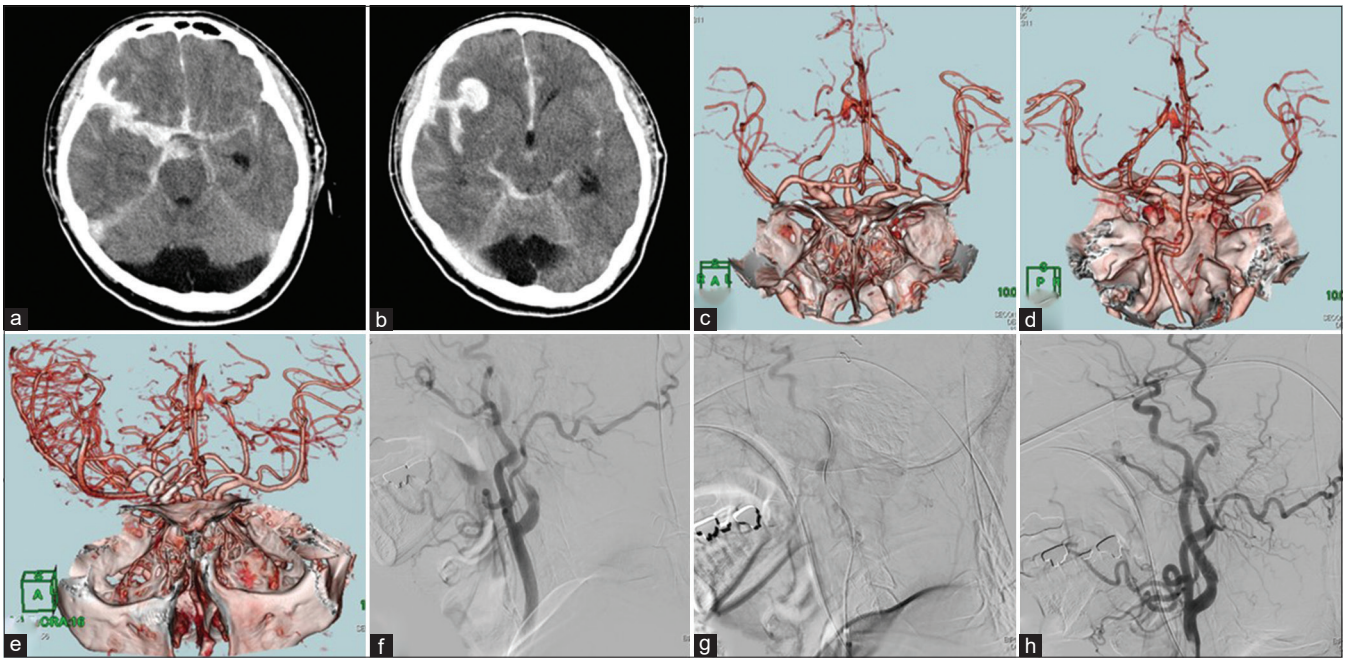

Figure 1: (a and b) Head computed tomography (CT) showing thick subarachnoid hemorrhage (SAH), Sylvian hematoma, and acute hydrocephalus. (c and d) Head CT angiography showing right A1 anterior cerebral artery (ACA) aneurysm with bleb and left vertebral artery saccular aneurysm. (e) Head CT angiography on day 7 after SAH showing trapped right A1 ACA aneurysm. The left middle cerebral artery is visualized less clearly than the right middle cerebral artery. (f) Initial common carotid angiography (CCAG) showing dissection of the cervical portion of the left internal carotid artery with delayed distal flow. (g) Internal carotid angiography from the distal lumen of the dissecting lesion. (h) Postoperative CCAG showing the dissecting lesion after carotid stenting.

(CTA) revealed a fusiform aneurysm of the right A1 segment of the ACA and a saccular aneurysm of the left vertebral artery (VA) [Figure 1]. The right A1 segment of the ACA was identified as the site of rupture based on the distribution of the hematoma, and the patient underwent right frontotemporal craniotomy. Intraoperatively, the right A1 segment of the ACA was dilated, fusiform, and resembled a blood blister-like aneurysm, indicating arterial dissection. The right A1 segment of the ACA aneurysm was trapped with cisternal drainage and external decompression to prevent involvement of the perforators. Postoperatively, the patient had no neurological deficits; therefore, routine maintenance infusion and pain medication were continued. The patient was not on any blood pressure medication, and his systolic blood pressure was <180 mmHg. However, on day 7 after SAH, he experienced right hemiparesis and aphasia. The left intracranial ICA was not clearly visible on CTA. Digital subtraction angiography showed ICA dissection at the cervical portion with delayed distal flow. Urgent carotid artery stenting (CAS) for symptomatic left ICA dissection was performed. Before the procedure, we administered 200 mg aspirin and 300 mg clopidogrel to prevent thrombotic complications. Complete coverage of the dissected segment of the left ICA was achieved using an 8 mm × 60 mm stent (Protégé RX; Covidien, Irvine, CA). Blood flow in the left ICA improved, and it was checked for restenosis and acute in-stent thrombosis. The patient's symptoms disappeared postoperatively. Postoperative magnetic resonance imaging showed left cerebral infarction in the anterior watershed area; however, symptomatic cerebral vasospasm and secondary hydrocephalus did not occur.

Laboratory results showed that the C-reactive protein level was not elevated, and immunological tests for antineutrophil cytoplasmic antibody and antinuclear antibody were negative. Abdominal CTA showed dissecting aneurysms of the celiac and superior mesenteric arteries [Figure 2]. SAM was strongly suspected because of the lack of features characteristic of other diseases and radiographic findings of multiple nonconsecutive dissecting lesions.

The patient underwent cranioplasty, and on day 46 after SAH, he was discharged with no neurological deficits. At present, he undergoes regular follow-up imaging to monitor the left VA, celiac artery, and superior mesenteric artery aneurysms.

DISCUSSION

SAM, first described by Slavin *et al.* in 1976, is a nonatherosclerotic and noninflammatory vascular disease characterized by arterial mediolysis.[4] It is characterized by multiple dissecting lesions that are not adjacent to each other.

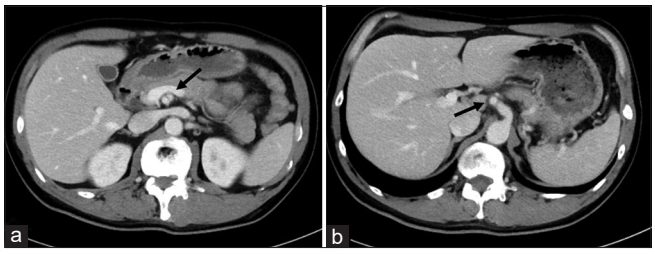

Figure 2: (a and b) Abdominal computed tomography angiography showing dissecting aneurysms of the celiac artery (a, arrow) and superior mesenteric artery (b, arrow).

A definitive diagnosis of SAM requires arterial biopsy to evaluate pathological findings such as intimal disruption and medial necrosis; therefore, the importance of pathological examination is widely recognized. However, biopsy is not routinely performed because of the location of the affected vessels, and because physicians tend to perform fewer direct surgeries in such cases due to the increase in endovascular treatment. Therefore, noninvasive diagnosis based on clinical and radiographic features has been advocated recently.[2,3] Abdominal CTA should be used for the diagnosis of SAM in patients with multiple intracranial dissections. In this case, abdominal CTA showed aneurysms of multiple vessels (celiac and superior mesenteric arteries) that were not adjacent to the lesion. The patient had no preexisting diseases or abnormal laboratory results; therefore, he was clinically diagnosed with SAM.

The indications for surgery in patients with SAM have not been determined. If the patient is hemodynamically stable, medical management of pain, hypertension, and vasoconstriction is preferred over surgical treatment.[3] Endovascular procedures should be performed carefully, especially in the acute phase of SAM, because of the risk of injury to other vessels. Kalva *et al*. reported that catheter manipulation and balloon dilatation may result in progression or new development of arterial dissection.[2] In this case, CAS was performed for the left ICA dissection concomitant with SAH. Because ICA dissection occurred with ischemic symptoms during the crucial phase in which cerebral vasospasm might worsen, normalization of cerebral blood flow was needed; this might not have been possible using medical treatment alone. Postoperatively, although antiplatelet therapy was started to avoid in-stent thrombosis, there were no hemorrhagic adverse events.

No specific strategy for preventing cerebral vasospasm in patients with SAM-related SAH exists. Excessive antihypertensive therapy may lead to cerebral ischemia due to decreased blood flow, and vasodilator or antithrombotic agents increase the risk of hemorrhagic complications. The risk of medial necrosis could be high and the quantity of norepinephrine released is important in determining the extent of mediolysis.[5] SAM-related SAH could lead to extension of arterial dissection in the acute phase due to released norepinephrine. There are several reported cases of SAH with intraperitoneal hemorrhage in the acute phase of SAM, and the importance of the management of SAM-related SAH is gradually being recognized.[1]

In this case, systolic blood pressure was maintained below 180 mmHg, hemodynamics and neurological status were strictly monitored, and imaging was regularly performed. Consequently, symptomatic vasospasm did not occur, and no lesion except the left ICA dissection required surgery. If symptomatic vasospasm of a major intracranial trunk occurs in a patient with SAM-related SAH, vasodilator infusion might be safer than percutaneous angioplasty with balloon inflation or stent placement as it avoids mechanical stimulation of the intima or media. However, as seen in this case, decreased cerebral blood flow with vascular lumen narrowing can also be caused by intracranial or cervical artery dissection; therefore, the cause of cerebral ischemia should be carefully identified.

CONCLUSION

We encountered a case of SAM-related SAH due to ruptured A1 segment of the ACA aneurysm and ICA dissection. In the acute phase of SAH, cerebral ischemia could be caused by both cerebral vasospasm and intracranial or cervical artery dissection. Early recognition of SAM is important in patients with multiple arterial dissections, and prompt diagnosis and careful management can lead to a good prognosis. Furthermore, if surgical intervention is needed, it should be performed in a way that avoids injury to other vessels.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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