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Cerebral vasospasm after coil embolization for unruptured anterior communicating artery aneurysm: illustrative case

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BACKGROUND Compared with several reports of cerebral vasospasm after clipping for unruptured cerebral aneurysm, only one study to date has reported cerebral vasospasm after coil embolization. Herein, the authors report a rare case of cerebral vasospasm after coil embolization for unruptured cerebral aneurysm.

OBSERVATIONS A 58-year-old woman with an unruptured anterior communicating artery aneurysm was referred to our department. Stent-assisted coil embolization was performed for the aneurysm, and no obvious adverse events were observed on cerebral angiography obtained immediately after the operation. However, the patient developed mild headache and slight restlessness soon after the operation and new-onset disorientation, left hemispatial neglect, and left hemiplegia the day after the operation. Emergency brain magnetic resonance imaging and cerebral angiography indicated vasospasm in the right middle cerebral artery, and intra-arterial injection of fasudil hydrochloride hydrate was performed to dilate the middle cerebral artery. Blood flow in the middle cerebral artery immediately improved, and she was discharged without neurological deficits 8 days after the operation.

LESSONS Immediate intervention is necessary to prevent cerebral infarction in patients with cerebral vasospasm, which may occur even after coil embolization for unruptured cerebral aneurysm.

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KEYWORDS adverse event; coil embolization; fasudil; unruptured aneurysm; vasospasm

Several studies reported the development of cerebral vasospasm (CV) after clipping for unruptured cerebral aneurysms;[1–12] however, to date, only one study reported CV occurring after coil embolization.[13] Here we report a rare case of symptomatic vasospasm of the middle cerebral artery (MCA) after stent-assisted coil embolization for unruptured anterior communicating artery (AcomA) aneurysm.

Illustrative Case

A 58-year-old, left-handed woman with a history of hypertension was referred to our department because of an unruptured AcomA aneurysm, which was incidentally found during magnetic resonance (MR) imaging (MRI) in another clinic. The patient did not have neurological deficits. Cerebral angiography showed a saccular aneurysm in the AcomA, with a maximum diameter of 4.0 mm, height of 3.1 mm, and neck size of 3.5 mm (Fig. 1A and B). Given the large aneurysm neck size, stent-assisted coil embolization was planned. Antiplatelet therapy with 100 mg/day aspirin and 75 mg/day clopidogrel was initiated 1 week before the operation.

The operation was performed with the patient under general anesthesia. First, a 5-French Fubuki guiding sheath (Asahi Intecc) was inserted into the right femoral artery. After heparinization, the Fubuki guiding sheath was placed in the right internal carotid artery (ICA) using a 0.035-inch Radifocus guidewire (Terumo) and a 125-cm 4-French OK2M (Gadelius) as the coaxial system. Next, a 120-cm Tactics intermediate catheter (Technocrat) was advanced as far as possible into the right ICA. A 200-cm Traxcess14 microguidewire (Terumo) was used to advance a 150-cm Excelsior SL-10 microcatheter (Stryker)

ABBREVIATIONS ACA = anterior cerebral artery; AcomA = anterior communicating artery; CV = cerebral vasospasm; DWI = diffusion-weighted imaging; ICA = internal carotid artery; MCA = middle cerebral artery; MRI = magnetic resonance imaging; SAH = subarachnoid hemorrhage.

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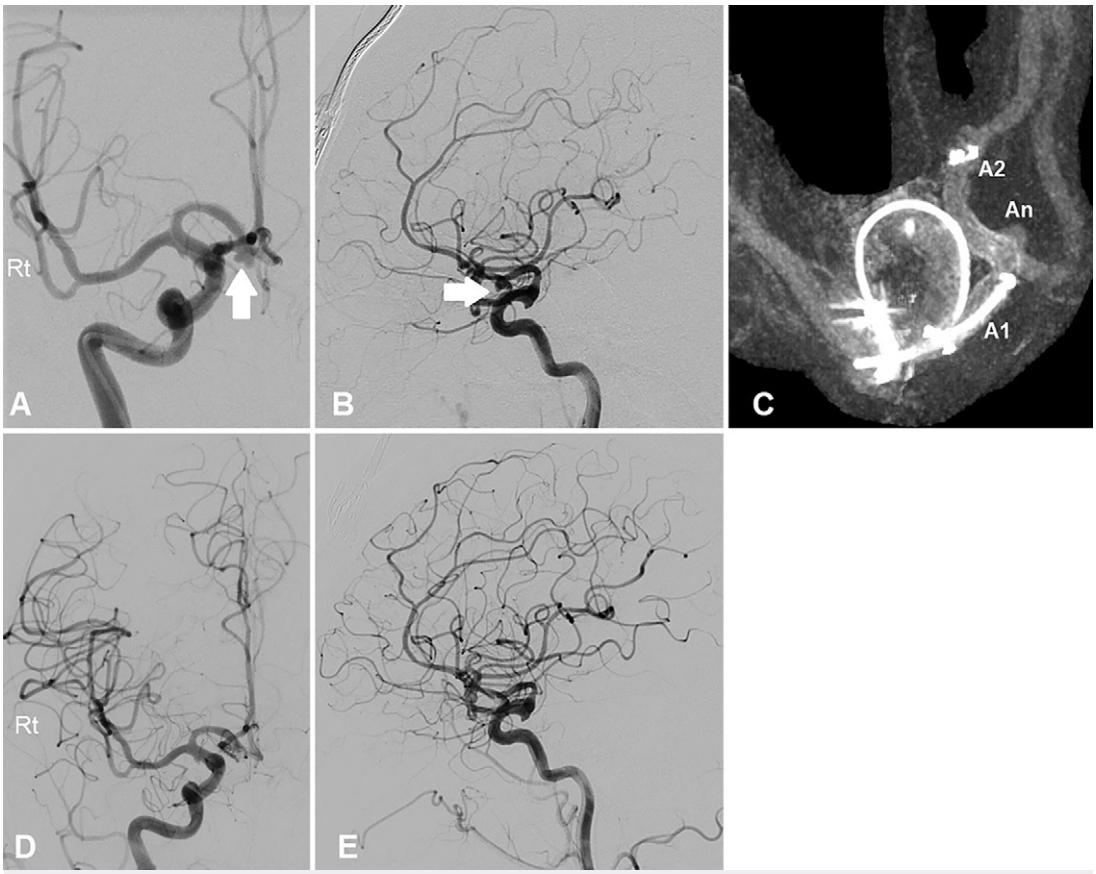

FIG. 1. Stent-assisted coil embolization for unruptured AcomA. Initial right ICA angiography (**A**, anteroposterior view; **B**, lateral view). Placement of the stent (**C**). Final right ICA angiography (**D**, anteroposterior view; **E**, lateral view). *White arrows* show the aneurysm. An = aneurysm; Rt = right.

to the right A2 segment of the anterior cerebral artery (ACA), and a Neuroform Atlas stent (3 × 21 mm) was placed from the right A2 segment of the ACA to the right A1 segment of the ACA (Fig. 1C). After stent placement, an Excelsior SL-10 microcatheter was inserted into the aneurysm through the transcell approach, and Axium PRIME Frame (3 mm × 8 cm; Medtronic), Target 360 NANO (2 mm × 4 cm; Stryker), and i-ED coil silky soft (1.5 mm × 3 cm; Kaneka) were inserted to embolize the aneurysm. Finally, angiography of the right internal cerebral artery indicated good aneurysm embolization, no spastic change, and no embolic complications (Fig. 1D and E). During the operation, the microguidewire entered the right MCA origin for a very short time, as it approached the right ACA, but no other devices within the right MCA were operational. Hemostasis was performed by manual compression.

The patient did not have any focal symptoms after the operation. However, she experienced a minor headache and slight restlessness in the lower right extremity. Abdominal computed tomography revealed a pseudoaneurysm at the puncture site accompanied with marked subcutaneous hematoma, and repair operation for the pseudoaneurysm was necessary. No significant hypotension occurred during the operation. On the day after the repair operation, in addition to the unresolved restlessness, she developed disorientation, left hemispatial neglect, and left hemiplegia. There was no evidence of subarachnoid hemorrhage (SAH) on an emergency brain MRI. However,

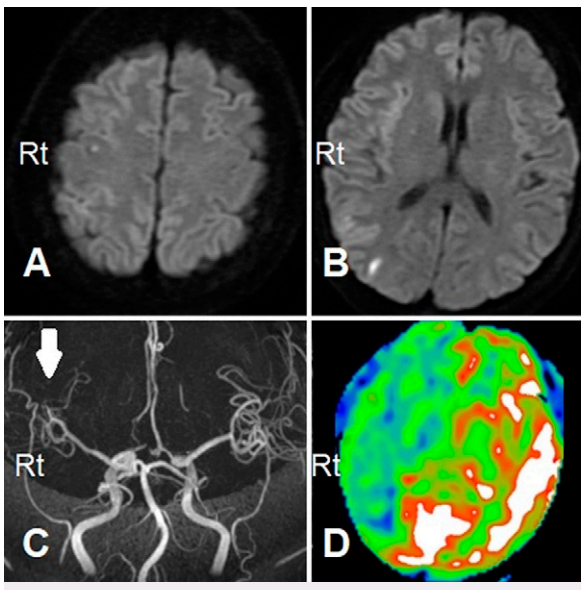

FIG. 2. MRI scans obtained 1 day after the first operation: DWI (**A and B**), MR angiography (**C**), and arterial spin labeling (**D**). *White arrow* shows vasospasm of the distal right MCA.

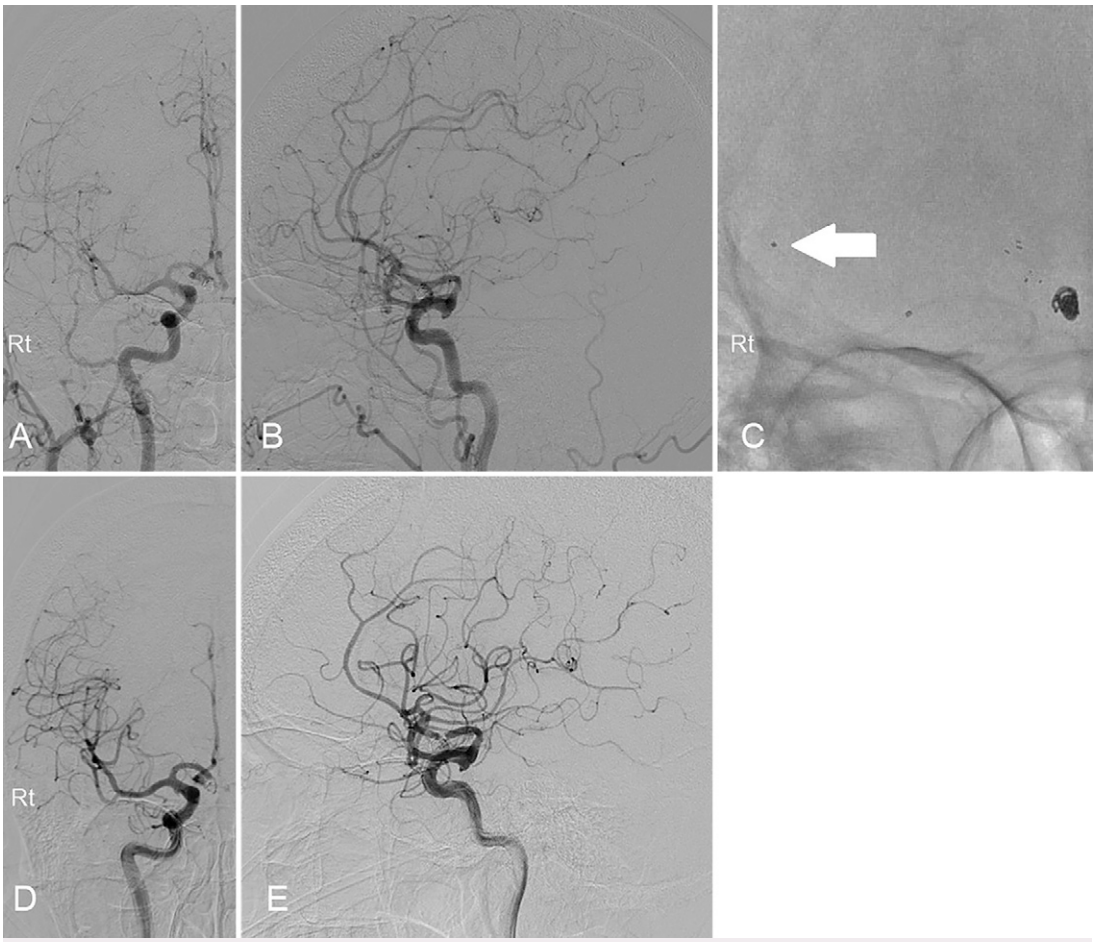

FIG. 3. Angioplasty for the delivery of fasudil hydrochloride hydrate for the vasospasm of the distal MCA. Initial right common carotid artery angiography (**A**, anteroposterior view; **B**, lateral view). Placement of the tip of the microcatheter in the right M2 segment (**C**). Final right common carotid artery angiography (**D**, anteroposterior view; **E**, lateral view). *White arrow* shows the tip of the microcatheter.

diffusion-weighted imaging (DWI) indicated a heterogeneously hyperintense area in the right hemisphere (Fig. 2A and B); magnetic resonance angiography showed spastic changes in the right MCA (Fig. 2C), which did not exhibit obvious vascular changes other than the site of stent placement. Arterial spin labeling suggested a widespread decrease in blood flow in the right brain compared with the left brain (Fig. 2D). Altogether, these findings suggested symptomatic vasospasm in the right MCA; thus, emergency cerebrovascular angiography was performed while maintaining blood pressure with sufficient intravenous infusion. Angiography of the right ICA revealed no issues in the ACA; however, spastic changes were observed in the distal MCA, and an operation was planned.

The operation was performed with the patient under local anesthesia. Vasospasm in the distal MCA was confirmed by right common carotid artery angiography (Fig. 3A and B); therefore, a 4-French Fubuki guiding sheath was inserted into the right brachial artery. After heparinization, the Fubuki guiding sheath was placed in the right ICA using a 0.035-inch Radifocus guidewire and a 120-cm 4-French SY3 guiding catheter (Gadelius) as the coaxial system. Next, a 200-cm-long CHIKAI-14 guidewire (Asahi Intecc) was used to advance a 150-cm-long Phenom17 microcatheter (Medtronic) into the right M2 segment of the MCA (Fig. 3C), and 30 mg fasudil hydrochloride hydrate was administered via continuous infusion over 10 minutes through the Phenom17 microcatheter. Finally, right internal cerebral angiography confirmed the improvement of vasospasm in the distal MCA (Fig. 3D and E).

The day after the second operation, the patient's consciousness improved, and her headache was resolved; however, she had persistent left hemiplegia. Follow-up brain MRI revealed the expansion of the hyperintense area on DWI (Fig. 4A); however, MR angiography and arterial spin labeling showed good blood flow in the right MCA (Fig. 4B) and the resolution of laterality (Fig. 4C), respectively. Blood tests showed a decrease in hemoglobin from 13.5 g/dL on admission to 7.9 g/dL after the second operation, and red blood cell transfusion was performed. Furthermore, edaravone, rosuvastatin, cilostazol, and low-molecular-weight dextran were initiated for treating cerebral infarction and the prevention of CV recurrence. As predicted, brain MRI performed 5 days after the second operation showed the absence of recurrent vasospasm (Fig. 4D) and no spread of the cerebral infarction. Her left hemiplegia gradually

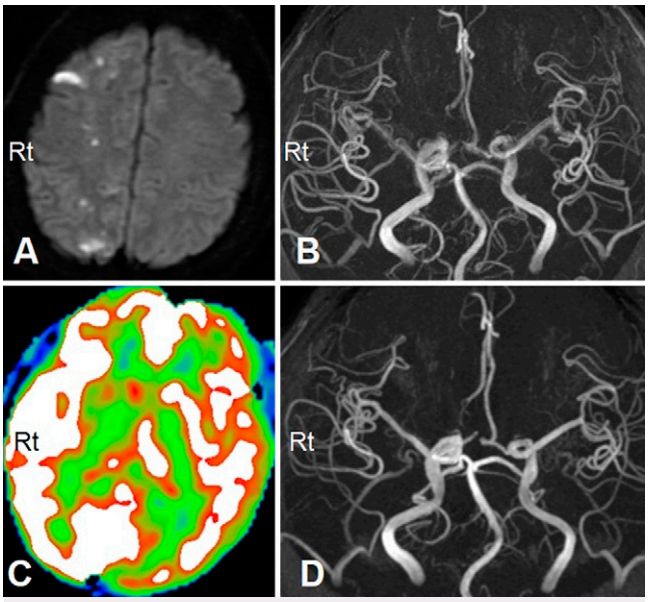

FIG. 4. MRI was performed 1 day after the second operation: DWI (**A**), MR angiography (**B**), and arterial spin labeling (**C**). MR angiography performed 5 days after the second operation (**D**).

recovered, and she was discharged 8 days after the second operation without any neurological deficits.

Discussion

Observations

CV occurs in approximately two-thirds of patients with SAH and is more likely to develop 3–12 days after the bleeding.[14] One study examining the mechanism of post-SAH CV in an experimental animal model suggested that oxyhemoglobin released from the hematoma leads to the activation of various signaling pathways and downstream inflammatory response.[15] In addition, mechanical stress to normal arteries and the brain during clipping has been suggested to lead to CV.[16,17] Therefore, normal arteries and the brain might be exposed to mechanical stress during clipping even in patients with unruptured aneurysms, and SAH might occur even with minor vascular injury; hence, it is possible for CV to occur.

Symptomatic CV has been reported in 30% of patients undergoing coil embolization of ruptured aneurysms;[18] however, only one asymptomatic case of CV after operation for an unruptured aneurysm has been reported.[13] Compared with clipping, coil embolization generates less mechanical stress on normal blood vessels and the brain. Moreover, in unruptured cases, SAH, which causes CV, does not occur unless arterial perforation occurs.

Other causes of CV after treatment for unruptured cerebral aneurysms include metal allergies,[4] hypothalamic disorders,[6,9,10,13,19,20] and trigeminocerebrovascular system stimulation.[4,6,8,9] Ogata et al.[13] reported a patient with an unruptured aneurysm who underwent balloon-assisted coil embolization and developed hypothalamic ischemia, which was considered the cause of subsequent CV. Knight et al.[21] reported a case of unruptured cerebral aneurysm with CV on the contralateral side of the approach side after clipping; although unconfirmed, a combination of the above-mentioned factors was considered the cause of CV.

Because the only device operation within the MCA in this case was to operate the microguidewire for a very short time at the MCA origin, direct mechanical stress to the MCA was not considered a cause of the CV. If the CV was caused by mechanical stress, it should have occurred intraoperatively. In addition, SAH did not occur during the operation, and technical factors were not considered triggers of the CV. Furthermore, the patient did not have a history of metal allergies. Nonetheless, metal allergies would not explain the CV that occurred only in the arteries that did not contain the stent and the coils. Furthermore, it is unlikely that trigeminocerebrovascular system stimulation will develop during endovascular treatment that does not directly stimulate the dura or the adventitia. Hypothalamic disorders were not considered as a potential cause of CV; however, the insertion of catheters into the right ICA might have resulted in decreased blood flow to the hypothalamus, which could have led to the CV. Another possible cause of CV is reversible cerebral vasoconstriction syndrome, which can randomly develop after the operation, although the cause is unknown. In summary, the cause of CV could not be determined in the present case; however, prompt intervention led to improvement and a favorable neurological course and outcome.

Lessons

CV may occur even after coil embolization for unruptured cerebral aneurysms. The development of CV should also be considered in patients who develop neurological deficits, headache, or restlessness after treatment. Although the cause of CV remains unclear, immediate intervention is critical to prevent cerebral infarction.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Fukutome, Aketa, Tei, Shin. Acquisition of data: Fukutome, Aketa, Fukumori, Mitsui, Nakajima, Hayami, Tei. Analysis and interpretation of data: Fukutome, Aketa, Motoyama. Drafting the article: Fukutome, Aketa. Critically revising the article: Fukutome, Aketa, Tei. Reviewed submitted version of manuscript: Fukutome, Mitsui, Nakajima, Hayami, Matsuoka, Tei. Approved the final version of the manuscript on behalf of all authors: Fukutome. Administrative/technical/material support: Fukutome, Fukumori. Study supervision: Fukutome, Shin, Motoyama.

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