

Case report

Reocclusion of the treated vessel due to endothelial injury after mechanical thrombectomy in a patient with acute ischaemic stroke

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SUMMARY

A 92-year-old woman developed sudden consciousness disturbance, global aphasia and right hemiparesis. She had atrial fibrillation and cardioembolic stroke was diagnosed. Tissue plasminogen activator was administered, and endovascular treatment was initiated. The left middle cerebral artery was occluded and complete recanalisation was achieved after direct aspiration first-pass technique. However, MRI immediately after treatment showed reocclusion. Endovascular treatment was repeated and complete recanalisation was achieved. There was no evidence of cerebral artery dissection, but angiography soon after the second procedure revealed early reocclusion. Ozagrel, an antiplatelet agent, was administered intravenously and prevented reocclusion. Endothelial injury was speculated to have occurred during the first mechanical thrombectomy, leading to recurrent occlusion. Though the patient continued to have right hemiparesis, she recovered from her consciousness disturbance and aphasia after re-treatment.

BACKGROUND

A meta-analysis of randomised trials showed that mechanical thrombectomy using specific devices for acute ischaemic stroke is effective and safe and is recommended by the American Heart Association/American Stroke Association guidelines.¹ However, few reports have examined the risk of endothelial injury from thrombectomy.² We present a case of endothelial injury possibly due to mechanical thrombectomy that caused repeated occlusion of the treated vessel.

CASE PRESENTATION

A 92-year-old woman developed sudden consciousness disturbance, aphasia and right hemiparesis. She had taken edoxaban (30 mg/day) for atrial fibrillation but discontinued it because of haemorrhagic cystitis 2 months prior. The onset-to-door time was 102 min. Neurological examination revealed impaired consciousness (Glasgow Coma Scale (GCS) score was 11/15, E4V1M6), global aphasia, right hemiparesis that included the face (manual muscle test score: upper extremity, 1/5 and lower extremity, 1/5) and right side sensory disturbance. The National Institutes of Health Stroke Scale (NIHSS) score was 36.

INVESTIGATIONS

A hypercoagulable state was suspected because of elevated D-dimer and fibrin degradation product levels (6.9 and 13.7 µg/mL, respectively). Electrocardiography revealed atrial fibrillation, and no thrombus was observed on transthoracic echocardiography. Carotid echocardiography revealed decreased end-diastolic flow of her left common carotid artery and suggested occlusion of the left middle cerebral artery (MCA) or internal carotid artery (ICA).³ CT, undertaken 111 min from onset, revealed a slightly low density at the left temporal pole and hyperdense MCA sign on the left side. The Alberta Stroke Programme Early CT score was 9.

TREATMENT

We started intravenous tissue plasminogen activator at 137 min after onset, but her symptoms did not change. Endovascular treatment was initiated, and complete occlusion of the left MCA was confirmed (figure 1A). We placed a 9-Fr Optimo catheter (Tokai Medical Products, Aichi, Japan) at the cervical portion of the left ICA and conducted clot retrieval with direct aspiration first-pass technique (ADAPT).⁴ We placed a 5-MAX ACE Penumbra catheter (Penumbra, CA, USA) at the proximal side of the thrombus and commenced aspiration. However, the catheter slipped into the M2 portion of the left MCA; nevertheless, red thrombus was retrieved and complete recanalisation was achieved (thrombolysis in cerebral infarction (TICI) grade 3; figure 1B). No neurological change was observed after the first mechanical thrombectomy. In our hospital, we conduct MRI/MR angiography (MRA) after mechanical thrombectomy as part of protocol for investigating the stroke volume, intracranial haemorrhage and the treated vessels. MRI just after thrombectomy revealed a minimum high-intensity area on diffusion-weighted images. However, MRA revealed narrowing of the treated vessel (figure 2A). Repeat MRA 10 min later revealed occlusion of the left MCA (figure 2B). A second endovascular treatment was attempted.

OUTCOME AND FOLLOW-UP

Cerebral angiography revealed occlusion of the left MCA at the M1 portion, 4 mm distal to the initial occlusion site (figure 3A). We repeated the initial procedure. A 5-MAX ACE Penumbra catheter was carefully advanced to the proximal side of the



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Figure 1 (A) Left ICA angiography shows occlusion of left MCA. (B) After first mechanical thrombectomy. Left ICA angiography shows favourable recanalisation of left MCA (thrombolysis in cerebral infarction Grade 3). ICA, internal cerebral artery; MCA, middle cerebral artery.

thrombus to prevent device slipping. The thrombus was removed using ADAPT, and complete recanalisation (TICI grade 3) was achieved again (figure 3B). However, cerebral angiography 10 min after the second treatment again revealed narrowing of the left MCA at the M1 portion (figure 3C). No findings indicated dissection. Endothelial injury may have occurred when the catheter slipped into the left M2, leading to repeated thrombus formation. We administered ozagrel 80 mg, aspirin 100 mg and clopidogrel 75 mg. After 13 min, cerebral angiography revealed complete left MCA recanalisation (figure 3D).

We continued ozagrel, aspirin and clopidogrel for 5 days. MRA on hospital day 6 revealed no MCA reocclusion (figure 4). Eventually, edoxaban (30 mg/day) and clopidogrel (75 mg/day) were chosen for secondary prevention. She recovered from her consciousness disturbance and aphasia (GCS score was 15/15, E4V5M6), though she remained right hemiparetic after re-treatment. NIHSS on her 21st day of hospitalisation was 26. She was transferred to a rehabilitation hospital for further treatment.

DISCUSSION

Factors contributing to thrombogenesis in recanalised vessels after mechanical thrombectomy for acute ischaemic stroke include endothelial injury, release of tissue factors, platelet activation, adhesion and aggregation.⁵ In our case, endothelial injury may have occurred during thrombectomy, leading to recurrent occlusion. Abraham *et al* analysed endothelial injury through endovascular treatment using MRI. They assessed six patients' MRI 24 hours after mechanical endovascular treatment with stent type devices. All these patients showed signal enhancement

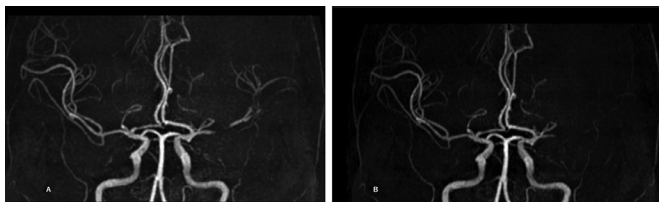


Figure 2 (A) MRA after first endovascular treatment; it reveals poor visualisation of the MCA. (B) 13 min later, MRA shows the reocclusion of left MCA. MCA, middle cerebral artery; MRA, MRA angiography.

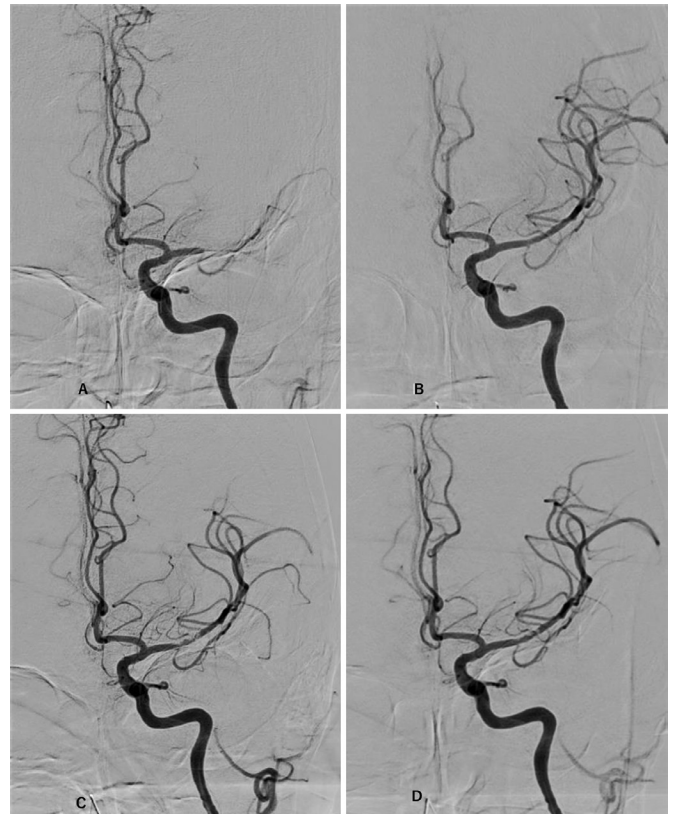


Figure 3 (A) The second mechanical thrombectomy. Left ICA angiography shows occlusion of left MCA at M2 portion. (B) Just after second mechanical thrombectomy. Angiography of the left ICA shows recanalisation of left MCA (thrombolysis in cerebral infarction Grade 3). (C) Angiography after 10 min interval shows mural thrombus. (D) Angiography after administration of antiplatelet drugs shows improvement of the mural thrombus. ICA, internal cerebral artery; MCA, middle cerebral artery.

of the vessel wall which suggested endothelial injury. So, we are afraid that the incidence of such event may not be rare.²

Gory *et al* found that both stent and aspiration devices might cause endothelial denudation and medial oedema.⁶ However, aspiration devices were less likely to cause endothelial denudation and intramural thrombus formation. They speculated that



Figure 4 MR angiography after second thrombectomy and administration of antiplatelet drugs reveals favourable visualisation of left MCA. MCA, middle cerebral artery.

aspiration devices had a smaller contact area within the vessel and were less likely to cause endothelial injury than stent devices.

Teng *et al* reported that the pattern and severity of endothelial injury differed depending on the endovascular treatment device.⁷ They conducted mechanical thrombectomy using various types of devices for different vessel sizes, and concluded that aspiration devices caused least endothelial injury and that the narrower the vessel, the more severe the endothelial injury.

In our case, endothelial injury probably occurred because the catheter slipped into the M2 portion, which was smaller than the device diameter. We should have carefully placed 5-MAX ACE Penumbra catheter not to slip to the M2 portion.

Ozagrel, a selective thromboxane A₂ synthase inhibitor, was administered intravenously. Therapy with ozagrel, aspirin and clopidogrel was definitely effective for resolving thrombotic occlusion in this case.

Repeated occlusion of a treated vessel in this case was probably due to endothelial injury after mechanical thrombectomy for an acute ischaemic stroke. This is an uncommon but serious adverse event, but thrombosis may occur from endothelial injury after mechanical

thrombectomy regardless of the type of device. Selecting the proper device size to avoid endothelial injury is important.

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REFERENCES

- 1 Powers WJ, Derdeyn CP, Biller J, *et al*. 2015 American Heart Association/American Stroke Association Focused Update of the 2013 Guidelines for the Early Management of Patients with Acute Ischemic Stroke Regarding Endovascular Treatment: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. *Stroke* 2015;46:3020–34.
- 2 Abraham P, Scott Pannell J, Santiago-Dieppa DR, *et al*. Vessel wall signal enhancement on 3-T MRI in acute stroke patients after stent retriever thrombectomy. *Neurosurg Focus* 2017;42:E20.
- 3 Yasaka M, Omae T, Tsuchiya T, *et al*. Ultrasonic evaluation of the site of carotid axis occlusion in patients with acute cardioembolic stroke. *Stroke* 1992;23:420–2.
- 4 Turk AS, Spiotta A, Frei D, *et al*. Initial clinical experience with the ADAPT technique: a direct aspiration first pass technique for stroke thrombectomy. *J Neurointerv Surg* 2014;6:231–7.
- 5 Becker R. Dynamics of coronary thrombolysis and reocclusion. *Clin Cardiol* 1997;20:2–5.
- 6 Gory B, Bresson D, Kessler I, *et al*. Histopathologic evaluation of arterial wall response to 5 neurovascular mechanical thrombectomy devices in a swine model. *AJNR Am J Neuroradiol* 2013;34:2192–8.
- 7 Teng D, Pannell JS, Rennert RC, *et al*. Endothelial trauma from mechanical thrombectomy in acute stroke: in vitro live-cell platform with animal validation. *Stroke* 2015;46:1099–106.

Learning points

- ▶ We report a case of repeated occlusion of a treated vessel after mechanical thrombectomy for acute ischaemic stroke. The endothelial injury caused by thrombectomy may have triggered thrombotic occlusion, which resolved with antiplatelet agents.
- ▶ Mechanical thrombectomy may cause endothelial injury and thrombotic occlusion of a treated vessel.
- ▶ The occurrence do not differ owing to the device type.
- ▶ Antiplatelet agents may be effective in such case.

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