A Patient with Delayed Cerebral Vasospasm after Mechanical Thrombectomy

Toshiaki Goda, Junya Kobayashi, Akihiro Watanabe, and Daisuke Takahashi

Objective: We report a patient in whom symptomatic delayed cerebral vasospasm was suspected after mechanical thrombectomy, and medical treatment led to an improvement.

Case Presentation: A 52-year-old female. To treat embolic cerebral infarction related to occlusion of the left internal carotid artery (ICA), mechanical thrombectomy was performed, and the recanalization of the occluded blood vessel was achieved. Immediately after treatment, findings suggestive of vasospasm of the left middle cerebral artery (MCA) were noted, but subsided 2 days after admission. Transient right hemiplegia occurred 5 days after admission, and restenosis at the same site was observed. Medical treatment resulted in the disappearance of stenosis 6 days after admission.

Conclusion: Considering the risk of delayed cerebral vasospasm after mechanical thrombectomy, follow-up should be carefully conducted.

Keywords ➤ mechanical thrombectomy, stent retriever, cerebral vasospasm

Introduction

Cerebral vasospasm is relatively frequent as a complication related to mechanical thrombectomy, but is temporarily observed during surgery to immediately after surgery in most cases. No study has reported delayed cerebral vasospasm. In this study, we present a patient with delayed cerebral vasospasm a few days after mechanical thrombectomy, and review the literature.

Case Presentation

Case: A 52-year-old female.
Complaints: Consciousness disorder, aphasia, and right hemiplegia.

Department of Vascular Neurology, National Hospital Organization Osaka Minami Medical Center, Kawachinagano, Osaka, Japan

Received: May 19, 2017; Accepted: October 30, 2017
Corresponding author: Toshiaki Goda. Department of Vascular Neurology, National Hospital Organization Osaka Minami Medical Center, 2-1 Kidohigashi-machi, Kawachinagano, Osaka 586-8521, Japan
Email: t-5da@ommc-hp.jp

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives International License.
©2018 The Japanese Society for Neuroendovascular Therapy

Medical history: Uterine myoma.
Lifestyle: She had smoked 20 cigarettes/day for 30 years.
Present illness: The last known time without symptom was 23:00 the day before onset. Her family detected her falling in front of the toilet at 6:00 in the morning. She was brought to our hospital by ambulance.

Physical examination on arrival: Concerning the consciousness level, the Japan Coma Scale score was 30. The blood pressure and pulse were 124/50 mmHg and 70 beats/min, respectively. Left conjugate deviation, total aphasia, right unilateral spatial neglect, and right hemiplegia were observed. The National Institutes of Health Stroke Scale (NIHSS) score was 26.

Laboratory data: A blood test showed that the D-dimer level was 0.8 µg/mL. There were no other abnormal findings. Electrocardiography showed sinus rhythm. Brain MRI revealed diffuse high-signal-intensity areas in the left middle cerebral artery (MCA) region on diffusion-weighted images. The Alberta Stroke Program Early CT Score was 6 points (Fig. 1). On brain MRA, movement made detailed assessment difficult, but intracranial occlusion of the left internal carotid artery (ICA) was suspected.

Endovascular treatment: The interval from the final known time without symptom until arrival was about 8 hours, and alteplase was not administered. Under local anesthesia,
a 9 Fr long sheath was inserted into the right femoral artery. After the intravenous injection of heparin at 4000 units, a 9 Fr Optimo (Tokai Medical Products, Aichi, Japan) was inserted into the left ICA. Using a CHIKAI 14 (Asahi Intecc Co., Ltd., Aichi), a Rebar18 microcatheter (Medtronic, Minneapolis, MN, USA) was guided to the distal horizontal segment (M1) of the MCA, and occlusion involving the ICA top to M1 middle portion was confirmed using cerebral angiography (Fig. 2A and 2B). A Trevo XP (Stryker, Kalamazoo, MI, USA) measuring 4 × 20 mm was guided to the thrombotic site, and a stent was developed at an area involving the M1 distal portion to ICA top (Fig. 2C). As recanalization was not achieved through one pass, the second pass was conducted, and the stent was developed at the same site. As a result, several white thrombi were collected, and Thrombolysis in Cerebral Infarction grade 2b recanalization was achieved. Stenosis was observed at the M1 middle portion (portion at which the distal side of the stent was developed), and peripheral blood flow was delayed (Fig. 3A and 3B). However, angiography after 5 minutes showed an improvement in peripheral blood flow (Fig. 3C and 3D). Furthermore, 3D angiography did not suggest dissection (Fig. 3E). Therefore, we considered the possibility of endovascular treatment-related cerebral vasospasm although we could not rule out the possibility that vascular dissection or atherosclerotic lesions may have been present.
Postoperative course: Brain CT after endovascular treatment did not reveal intracranial hemorrhage. Mild aphasia and higher brain dysfunction (left-and-right disorientation, anarhthmia) remained 2 days after endovascular treatment (day 2), but the NIHSS score was 1. On brain MRI, there was no enlargement of the infarcted focus in comparison with its size on arrival. MRA 24 hours after recanalization showed the complete disappearance of stenosis at the left M1 middle portion (Fig. 4A). Therefore, the mechanism of cerebral infarction may have involved embolism, but not atherosclerotic lesion-related thrombosis. As a disease type, cardiogenic cerebral embolism, including paradoxical cerebral embolism, or cerebral embolism related to other etiological factors (congenital thrombotic predispositions, collagen disease, and Trousseau syndrome) was suggested. To investigate the etiology, carotid artery ultrasonography, transesophageal echocardiography, and lower limb vein ultrasonography were performed, but there was no abnormal finding. Furthermore, a bedside electrocardiographic monitor was attached to the patient for 14 days after admission, but there was no paroxysmal atrial fibrillation. Hematology did not suggest any disease that causes juvenile cerebral infarction, such as coagulation disorder or collagen disease. The continuous intravenous injection of heparin at 10000 units/day was conducted from day 2. MRA on day 4 revealed slight stenosis at the left M1 middle portion (Fig. 4B). Simultaneously, the patient complained of mild headache, but there was no new, abnormal neurologic finding, and follow-up was continued. Atonic seizures of the right upper/lower limbs, persisting for a few minutes during rehabilitation training, occurred three times on day 5. MRA confirmed marked stenosis at the left M1 middle portion (Fig. 4C). On transcranial Doppler (TCD) ultrasonography, the mean blood flow velocity at the stenotic site was markedly increased to 146 cm/s. On T1-weighted images taken using a 3.0 tesla MRI system, there was no high-signal intensity suggestive of a mural hematoma related to vascular dissection, suggesting restenosis related to cerebral vasospasm. To improve cerebral circulation, the circulating blood volume was maintained by administering extracellular fluid-replenishing solution or low-molecular-weight dextran, and the continuous intravenous injection of dobutamine was performed.
Furthermore, the hemoglobin (Hb) level was 7.7 g/dL, being lower than on admission (10.5 g/dL); the progression of anemia was noted. Two units of red-blood concentrate were transfused. As antithrombotic therapy, the continuous intravenous injection of heparin was continued, and the continuous intravenous injection of ozagrel sodium (80 mg/day) and oral administration of cilostazol at 100 mg/day were started. Subsequently, there was no recurrent symptom, and stenosis at the left M1 middle portion had reduced on day 6 (Fig. 4D). Transcranial Doppler ultrasonography showed that the mean blood flow velocity at the left M1 middle portion was normalized (71 cm/s). Dobutamine administration was discontinued on day 8, and ozagrel sodium administration on day 11. On day 14, the oral administration of Warfarin was started, and combined with heparin for 7 days. Subsequently, heparin was discontinued. On day 17, cerebral angiography was performed. The wall of the left M1 middle portion was slightly irregular, but there was no stenosis (Fig. 5). The subsequent course was favorable, and the patient was discharged on day 21. On discharge, only mild motor aphasia was observed as a neurologic symptom, and the NIHSS score was 1. The modified Rankin Scale score was 1. Since discharge, the oral administration of Warfarin and cilostazol has been continued. There has been no recurrent symptom. Although the etiology of cerebral infarction is unclear, the involvement of uterine myoma in its onset cannot be ruled out. Therefore, extirpation is scheduled.

**Discussion**

Cerebral vasospasm is relatively frequent as a complication related to mechanical thrombectomy for acute-phase cerebral infarction. Its incidence is reportedly 20%–26%.1,2) Furthermore, patients with restenosis in the chronic phase after mechanical thrombectomy have also been reported.1,3) However, to our knowledge, there is no case report of symptomatic cerebral vasospasm occurring a few days after surgery and an improvement achieved by medical treatment. Such cases may be rare.

In the present case, cerebral angiography immediately after treatment showed stenosis at the M1 middle portion. An improvement was transiently achieved 2 days after treatment, but restenosis was noted 4 days after treatment. As an etiological factor for stenosis, we could not completely rule out the possibility of cerebral artery dissection. However, neither intraoperative 3D angiography nor brain MRI at the time of recurrence suggested dissection, and an improvement in stenosis/additional...
cilostazol administration reduced the incidence of cerebral vasospasm, improving the clinical outcome. In the present case, the agent was administered to obtain vasodilative actions. As other systemic drug therapies, the administration of fasudil or calcium antagonists is also useful. However, in the present case, the pretreatment blood pressure was approximately 90/50 mmHg, and administration was avoided. Concerning blood transfusion, symptomatic cerebral vasospasm occurred, requiring a prompt improvement in cerebral circulation; therefore, blood transfusion was conducted. On the other hand, a study reported that a blood-transfusion-related increase in blood viscosity induced cerebral vasospasm; the transfusion volume was minimized to a requirement for improving cerebral circulation. As a result, the patient responded to these medical treatments, leading to a prompt reduction in cerebral vasospasm.

On discharge, there was no restenosis at the lesion site, but recurrent cerebral vasospasm or stenosis/occlusion at the lesion site may occur in the future. Strict follow-up by regular diagnostic imaging or monitoring of clinical symptoms may be necessary.

Conclusion

We reported a patient in whom symptomatic delayed cerebral vasospasm was suspected after mechanical thrombectomy, and medical treatment led to an improvement. Considering that such a condition may occur in the acute phase after thrombectomy, follow-up should be carefully performed.

Disclosure Statement

There is no conflict of interest for the first author and coauthors.

References