



Case of a Patient Who Developed Basilar Artery Occlusion after Treatment of Traumatic Vertebral Artery Dissection with Proximal Coil Occlusion

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Objective: To examine a case of basilar artery (BA) occlusion caused by traumatic vertebral artery (VA) dissection after incomplete endovascular therapy.

Case Presentation: A 32-year-old man who got caught sustained injuries in a truck accident wherein he was stuck between two trucks was transported to a nearby hospital. Stenting was performed for the left common carotid artery (CCA). For left VA dissection, coil embolization of the VA was performed to prevent thromboembolic infarction because floating thrombus was found at the V3 segment. On postoperative day 1, he exhibited conscious disturbance. MRA revealed BA occlusion. Upon transfer to our hospital, thrombectomy was performed. After revascularization, the patient was alert.

Conclusion: Although optimal treatment of traumatic VA injury is still debated, proximal coil occlusion is regarded as an effective preventive treatment for thromboembolic stroke. In case of VA dissection with floating thrombus, proximal VA occlusion is insufficient to prevent thromboembolic stroke, and thrombus capture should be considered.

Keywords ► traumatic vertebral artery dissection, basilar artery occlusion, proximal coil occlusion, revascularization

Introduction

When a thrombus from traumatic vertebral artery (VA) dissection causes basilar artery (BA) occlusion, serious symptoms develop, and the prognosis is poor.¹⁻⁴⁾ Favorable outcomes achieved with coil embolization and stent placement in dissected VA regions to prevent distal embolic complication have been occasionally reported⁵⁻⁷⁾; however,

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Received: February 21, 2019; Accepted: August 6, 2019

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there have been no reported cases of a thrombus present distal to the dissected region or that of complication caused by BA occlusion despite coil embolization being performed for the dissected region. Even though VA dissection is present with a thrombus distal to the dissected region, when there is almost no distance between the dissected region and thrombus, thrombus migration may be prevented by performing coil embolization of the dissected region. However, when dissection occurs in the proximal side of the VA and the thrombus is present at a site distal to the V3 segment, the treatment decision can be challenging. We encountered a patient who developed embolic BA occlusion after coil embolization of traumatic VA dissection at the origin of the artery with a floating thrombus in the V3 segment, and it was treated with thrombectomy.

Case Presentation

Patient: 32-year-old male patient.

Past medical history: Unremarkable.

History of present illness: The patient was caught between two trucks and injured while guiding a 10-ton truck and

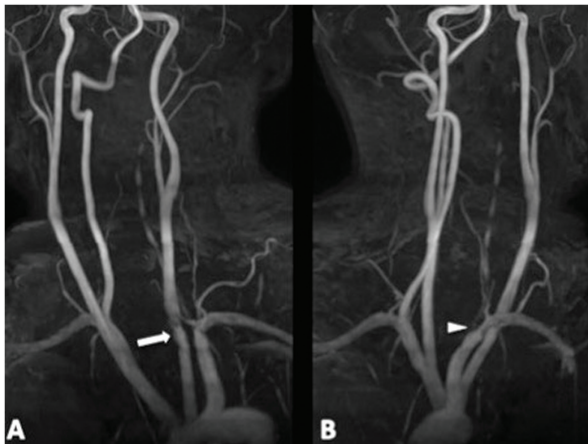


Fig. 1 MRA reveals left common carotid artery dissection (arrow) and left VA dissection (arrowhead). (A) Left anterior oblique view 30° and (B) right anterior oblique view 50°. VA: vertebral artery

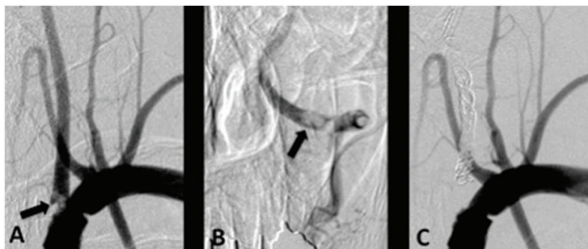


Fig. 2 Angiography of the left subclavian artery shows complete occlusion of the left VA after coil embolization. (A) Before the embolization, (B) during the surgery, floating thrombus at the V3 segment, and (C) after the embolization. VA: vertebral artery

was transported to a hospital. He lost consciousness for about 3 minutes at the time of injury; however, consciousness was clear at the time of arrival, and no symptoms of nerve loss were observed. Left second and third rib fractures and hemothorax were noted on chest CT; left common carotid artery (CCA) and left VA dissections were noted on MRA of the head (**Fig. 1**). Stenting of the CCA and parent blood vessel occlusion of the VA with coils were urgently performed. A floating thrombus was noted in the V3 segment during treatment of VA dissection (**Fig. 2**), and it was judged that if blood flow is blocked at the origin of the VA, dissemination is unlikely. The consciousness level decreased on the day after the treatment. No infarct lesion was noted in the vertebral-basilar arterial system region on MRI, but BA occlusion was observed on MRA (**Fig. 3**). Hemothorax was aggravated following intravenous heparin administration; therefore, the patient was transferred to our hospital after drain placement in the left thoracic cavity. At the time of arrival, 6 hours and 36 minutes



Fig. 3 MRA on the day after the first treatment shows BA occlusion. BA: basilar artery

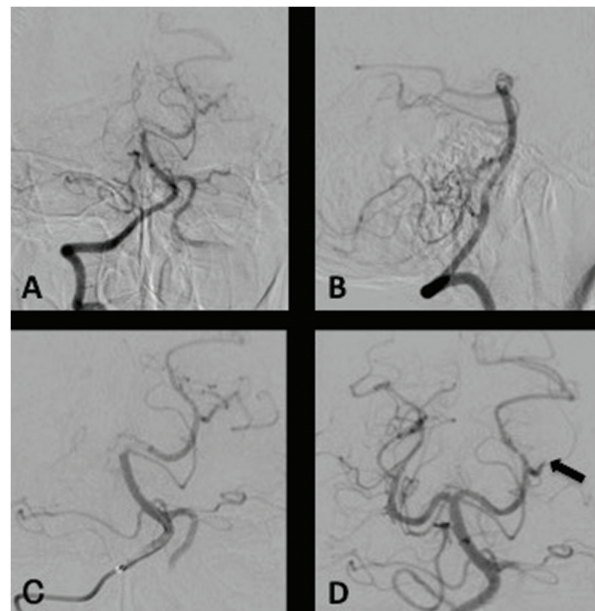


Fig. 4 Right VA angiogram before the thrombectomy. (A) Frontal view, (B) lateral view. Angiography shows thrombus at the top of BA and right PCA occlusion. (C) Preoperative right vertebral angiogram shows BA occlusion. (D) After thrombus removal, the angiogram shows left calcarine artery occlusion (arrow) and recanalization of TICI 2B. BA: basilar artery; PCA: posterior cerebral artery; TICI: thrombolysis in cerebral infarction; VA: vertebral artery

had passed after the appearance of consciousness disturbance. The patient was immediately moved to an angiography room and confirmed to be in a state of BA apex occlusion, based on an angiography of the right VA (**Fig. 4A** and **4B**). The thrombus detected in the left V3 segment by a previous physician was not noted, it was judged that no new thrombus was formed but the thrombus scattered to the

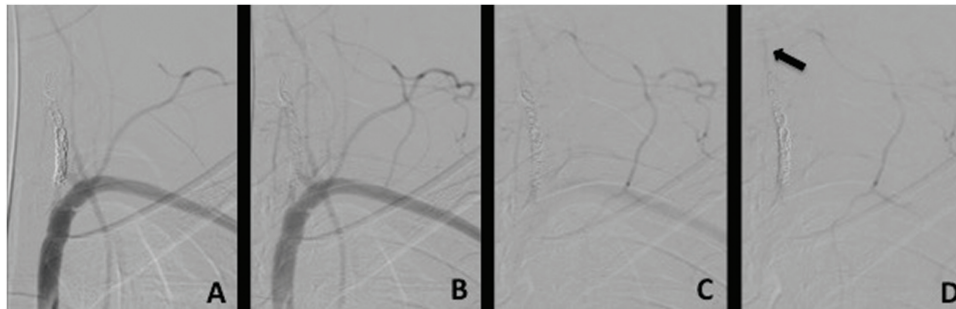


Fig. 5 Angiography of the left subclavian artery after the thrombectomy shows left VA recanalization. (A) Early artery phase, (B) artery phase, (C) early venous phase, and (D) left VA recanalization (arrow) in venous phase. VA: vertebral artery

BA. Using Trevo Pro 18 Microcatheter (Stryker, Kalamazoo, MI, USA) for the inner catheter, Penumbra 5Max Ace (Penumbra, Alameda, CA, USA) was guided to the proximal region of the thrombus, and thrombectomy was performed employing a direct aspiration first Pass (ADAPT) technique. The right posterior cerebral artery was completely recanalized (**Fig. 4C**), but the left posterior cerebral artery was occluded in P3. Using Penumbra 5Max Ace and 3Max coaxially, 5Max Ace was guided to the BA apex, followed by guiding 3Max to the left posterior cerebral artery (P3), and the thrombus was aspirated from this region. Thrombolysis in cerebral infarction (TICI) 2B recanalization was acquired 7 hours after the onset (**Fig. 4D**). The distal left VA was slightly visualized through the muscular branch of the deep cervical artery on angiography of the left subclavian artery (**Fig. 5**). The removed thrombus was histologically a fresh thrombus mixed with neutrophils and fibrin. After treatment, his consciousness became clear. CT performed on the day after the treatment showed no hemorrhage; however, a low-density region was detected in the calcarine artery region that could not be re-perfused (**Fig. 6**). On postoperative day 14, patency of the BA and bilateral posterior cerebral arteries was confirmed on MRA. Medication with an oral antiplatelet agent (Bayaspirin 100 mg/day) was continued after treatment completion. Diplopia, right visual field defect, swallowing disorder, and higher brain dysfunction were observed, and the patient was transferred to another hospital for continued rehabilitation 26 days after the injury. After rehabilitation, diplopia remained but his condition recovered to a level that made him capable of home life, and the patient was discharged to home at a modified Rankin Scale (mRS) of 1. The patient was followed up in the outpatient department. Reduction in the visual field defect and ocular motility disorder were observed at the 6-month follow-up after



Fig. 6 CT image on the following day reveals left occipital artery infarction.

treatment, and he resumed his social life. Medication with an oral antiplatelet agent was continued until 1 year after treatment, considering that a new thrombus may develop because the right VA was not completely occluded and a stent was placed in the left CCA. The medication was completed after confirming the absence of a novel infarction on MRI, favorable patency of the right VA over the BA on MRA, and the absence of visualization of the left VA from the origin.

Discussion

There is no standardized treatment strategy for traumatic VA injury, and the indication and timing of treatment

including endovascular therapy vary among medical institutions and physicians. Achievement of favorable outcomes via careful course observation, antiplatelet therapy, heparin administration, and tissue plasminogen activator (t-PA) administration has been reported,^{8–10} but patients complicated by BA occlusion^{11,12} and severe hemorrhagic complication associated with antithrombotic therapy¹³ have also been reported. Complication caused by BA occlusion induces serious symptoms, and the prognosis is poor.^{1–4} In order to prevent complication on the distal side, coil occlusion of the parent blood vessel and stent placement in the dissected region may be selected.^{11,14,6,7} In the present patient, a thrombus was present at a site distal (V3 segment) to the dissected region (origin of the VA) on diagnostic imaging performed by the previous physician. No physician with a specialization in endovascular treatment of the cranial nerves was present in the previous hospital; therefore, it was difficult to perform intracranial thrombectomy, and prevention of distal embolism by occluding the proximal region was selected. Proximal occlusion of the injured VA has been occasionally reported and is considered an effective treatment. However, in these reports, the thrombus was not already present at a site distal to the dissected region at the time of treatment, and no treatment strategy has been mentioned for such cases. In the present patient, complete blockage of blood flow from the collateral circulation was not possible, and the thrombus moved to the BA and caused occlusion, showing the presence of cases for which proximal occlusion alone is insufficient. It is necessary to investigate a treatment strategy for patients in whom a thrombus is already present in the periphery at the time of VA dissection diagnosis. There are collateral circulations to the VA from the anterior, posterior, and lateral spinal arteries, dentate artery, and ascending and deep cervical arteries. By occluding the proximal region, overall thrombosing can be expected, but blood flow from the collateral circulation to the VA is abundant, and complete blockage of blood flow is difficult. In the present patient, blood flow in the occluded region was completely blocked on angiography performed at our hospital; however, the periphery was visualized through the muscular branch of the deep cervical artery. When thrombectomy is applicable at the time of diagnosis, to ensure prevention of thromboembolism, both treatments of the dissected region and thrombectomy are necessary. When the contralateral VA is hypoplastic and there is high risk for blockage of the dissected VA, a stent is placed in the dissected region, but in the present case, the thrombus may be moved to the

periphery by an increase in the vertebral arterial blood flow, for which treatment of the dissected region after thrombectomy through the ipsilateral or contralateral approach is necessary. When blood flow from the contralateral side is secured and the dissected region can be occluded, such as in our patient, it may be better to precede the treatment of the dissected region to reduce the risk of thrombus migration. When the floating thrombus still remains in the VA, the approach from the contralateral side may be less likely to move the thrombus toward the periphery. For the treatment of our patient, ADAPT was selected, considering that the thrombus may be moved toward the periphery when a micro guide wire or microcatheter was passed through. For facilities wherein thrombectomy is difficult to perform, it may be safe to rapidly transfer the patient to a facility with a physician who specializes in endovascular treatment at the time of diagnosis.

It is unclear whether antiplatelet agents or anticoagulants are more suitable for VA dissection.¹⁵ An antiplatelet agent was selected, considering the fact that a stent had already been placed in the left CCA by the previous physician, but investigation in individual cases is necessary because multiple traumas may be present. Further reports are awaited.

Conclusion

We encountered a patient who developed BA occlusion due to thrombus migration after parent artery embolization of traumatic VA dissection accompanied by a floating thrombus on the peripheral side. Blood flow from the collateral circulation to the VA could not be blocked by the parent blood vessel occlusion unlike that in the internal carotid artery, and complete prevention of distal embolization is not possible. For floating thrombus, direct treatment at an early stage is necessary.

Disclosure Statement

There is no conflict of interest to be disclosed in this report.

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