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Case Report

# Spinal Cord Infarction after Mechanical Thrombectomy for Acute Basilar Artery Occlusion: A Case Report and Review of the Literature

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**Objective:** We report a case of spinal cord infarction following mechanical thrombectomy for acute basilar artery occlusion, and describe the pathophysiological mechanism of spinal cord infarction and its possible prevention.
**Case Presentation:** A 70-year-old man developed dysarthria and left-sided sensory impairment and was then diagnosed with acute basilar artery occlusion. Mechanical thrombectomy was performed using a 6-Fr guiding sheath via the left vertebral artery (VA). Complete recanalization was achieved within 1.5 hours. However, toward the end of the procedure, the guiding sheath was wedged in the distal portion of the VA. Postoperatively, left-sided flaccid paralysis and right-sided sensory deficit were observed. Cervical magnetic resonance imaging (MRI) demonstrated an acute spinal cord infarction on the left side, at the level of C3. The cause of infarction was suspected to be the wedging of the guiding sheath during the procedure.
**Conclusion:** Spinal cord infarction is a rare but serious complication of mechanical thrombectomy for acute basilar artery occlusion. The selection of appropriate procedure, device, and safe access route are essential for the success of a mechanical thrombectomy for acute basilar artery occlusion.

**Keywords** ▶ basilar artery occlusion, mechanical thrombectomy, spinal cord infarction

## Introduction

Mechanical thrombectomy has been demonstrated to be effective in the management of patients with intracranial large-vessel occlusions of the anterior circulation.[1] Recently, the indication of mechanical thrombectomy has been expanded to the posterior circulation, and its efficacy has been reported.[2] On the other hand, perioperative complications remain unclear. In this study, we report a case of spinal cord infarction after mechanical thrombectomy for acute basilar artery occlusion. We review the pathogenesis and methods of prevention of spinal cord infarction after mechanical thrombectomy in posterior circulation.

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## Case Presentation

The patient was a 70-year-old man brought to our hospital by ambulance. He had developed dysarthria and numbness of the left half of his body the previous night, which persisted till the morning. The time from the onset of his symptoms until arrival at the hospital was 14.5 hours. He had a history of benign prostatic hypertrophy and atrial fibrillation, for which he was being treated with tamsulosin and bisoprolol, respectively. There was no history of hypertension, diabetes mellitus, or dyslipidemia and he was not receiving any antiplatelet drugs or anticoagulant therapy. He smoked a small number of cigarettes in adolescence but has since quit. On examination, his orientation was maintained, but somnolence was noted. Left-sided paresis of both upper and lower limbs (Manual Muscle Test [MMT] score: 3/5) as well as the face was observed, along with diminished sensation on the left side of the body, ataxia of the right upper and lower extremities, and dysarthria. The National Institutes of Health Stroke Scale score was 9. Head computed tomography (CT) revealed a high-density signal at the top of the basilar artery, and a fresh infarction in the region of the right superior cerebellar artery. Head magnetic resonance imaging (MRI) demonstrated the

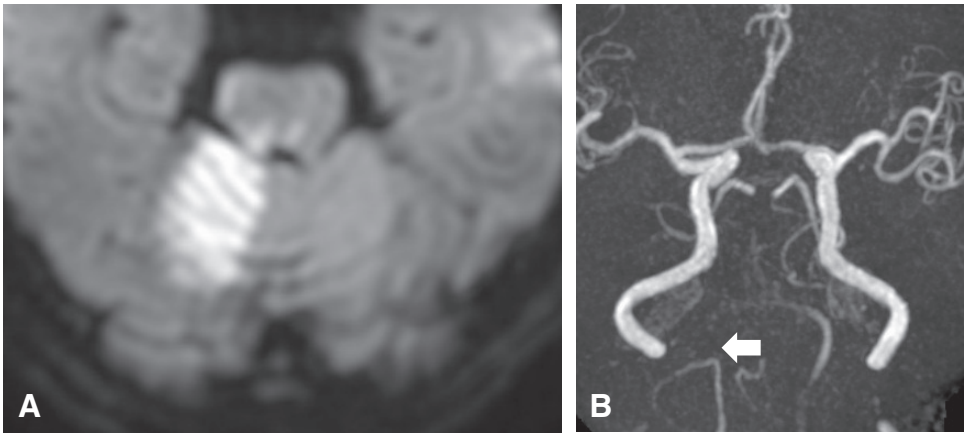


**Fig. 1** MRI at admission. (**A**) Diffusion-weighted imaging showing acute infarction in the right cerebellar hemisphere and dorsal pons. (**B**) MRA of basilar artery occlusion and the hypoplastic right VA terminating in the posterior inferior cerebellar artery (arrow). MRA: magnetic resonance angiography; VA: vertebral artery

findings of acute-phase infarction involving the right cerebellar hemisphere and dorsal pons, related to the occlusion of the basilar artery (**Fig. 1**). The interval from onset to treatment was long; however, there was a clinical-diffusion mismatch, and therefore mechanical thrombectomy was selected for treatment.

### Endovascular treatment

Under local anesthesia, a 4-Fr short sheath was inserted into the right femoral artery. As the patient seemed restless, the patient was sedated with dexmedetomidine. For a guiding system, a 6-Fr Fubuki (Asahi Intecc, Aichi, Japan) guiding sheath was selected considering the use of an aspiration catheter. The 4-Fr short sheath was exchanged for this guiding sheath. After inserting the guiding sheath, heparin was injected intravenously, with 4000 units injected initially, followed by 1000 units/hour to maintain systemic heparinization. Preoperative magnetic resonance angiography (MRA) did not confirm the right vertebral artery (VA) distal to the posterior inferior cerebellar artery (**Fig. 1B**). As the left VA was prominent, an approach via the left VA was selected. The origin of the left VA was slightly tortuous, but there was no stenosis. The Fubuki was navigated to the V2 segment of the left VA at the level of the body of the C4 vertebra. The activated clotting time (ACT) after guiding sheath insertion was 327 seconds. Left vertebral arteriography demonstrated a slight delay in opacification, which was suspected to be related to the basilar artery occlusion. As there was no wedging of the guiding sheath, the procedure was continued. Four sessions of mechanical thrombectomy were performed: (1) thrombectomy with a Solitaire 6 × 40 mm (Medtronic, Minneapolis, MN, USA), (2) a direct aspiration first pass technique (ADAPT)[3] with a Penumbra ACE 60 (Penumbra Inc., Alameda, CA, USA), (3) continuous aspiration prior to intracranial vascular embolectomy (CAPTIVE) technique[4] with a Solitaire 6 × 40 mm and Penumbra ACE 60, and (4) CAPTIVE technique with a Trevo XP 6 × 25 mm (Stryker, Kalamazoo, MI, USA) and Penumbra ACE 60. The result was complete recanalization, achieved 1.5 hours after puncture (19 hours and 47 minutes after onset) (**Fig. 2B**). At the completion of mechanical thrombectomy, the sheath tip reached the level of the 1st vertebral body, beyond the tortuous part of the VA, through the disappearance of the deflection of the guiding sheath associated with procedure-related vascular linearization. Angiography done to confirm recanalization revealed wedging associated with the guiding sheath (**Fig. 2C**). Neither the anterior spinal artery (ASA) nor the radiculomedullary artery (RMA) were visualized on left vertebral arteriography, before or after treatment. Furthermore, dissection of the left VA was not observed on left subclavian arteriography after the completion of treatment (**Fig. 2D**). The ACT after the procedure was 225 seconds. The effects of heparin were reversed with protamine and the guiding sheath was removed.

### Postoperative course

Detailed neurological assessment immediately after the procedure was difficult, due to sedation. However, deterioration of the left-sided paralysis was noted (now complete flaccid paralysis). On the right side of the body, active movement of the upper and lower extremities was possible, and there was no obvious paralysis. However, there was no response to painful stimuli, suggesting right-sided

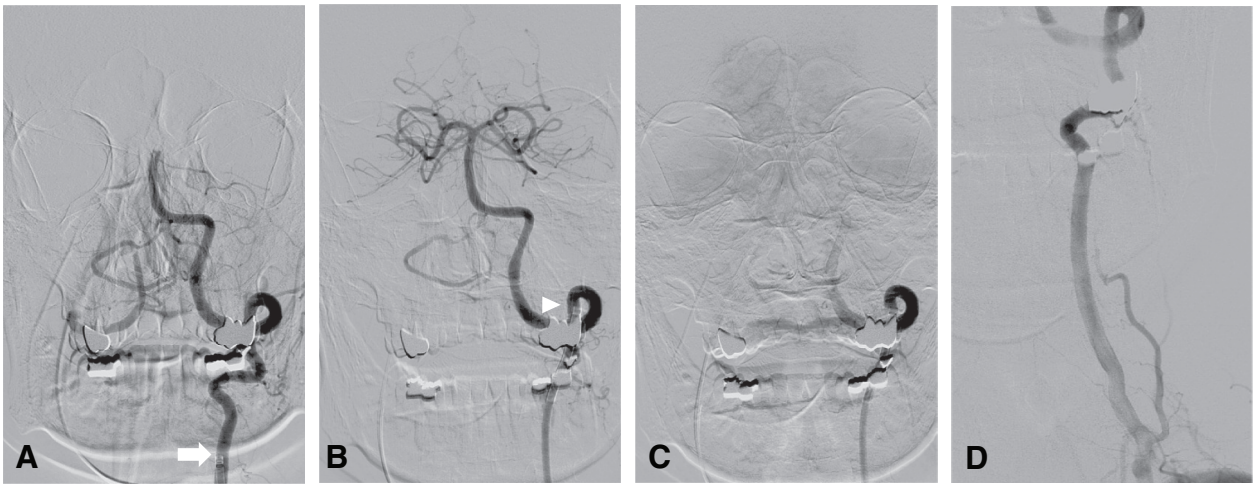


**Fig. 2** Intraoperative angiography. (**A**) The basilar artery is occluded at the upper part before mechanical thrombectomy. A guiding sheath is placed on the V2 segment (arrow). (**B**) Complete recanalization is obtained. The guiding sheath moved to the distal part of V2 (arrowhead) at the end of the procedure. (**C**) Flow stagnation due to wedging of the guiding sheath is seen in the venous phase on left vertebral angiography. (**D**) Iatrogenic VA dissection is not seen on left subclavian angiography. VA: vertebral artery

sensory loss. On the 2nd postoperative day, the patient was started on apixaban at 10 mg/day. On the 3rd postoperative day, his consciousness deteriorated, with a Glasgow Coma Scale (GCS) score of E1V2M3. Head MRI did not reveal reocclusion of the basilar artery (**Fig. 3A**). Due to the swelling at the site of right cerebellar infarction, and due to the obstructive hydrocephalus, external decompression and ventricular drainage were performed. After surgery, consciousness improved promptly, with a GCS score of E2V3M6. Head MRI did not reveal any lesion that could be responsible for the exacerbation of left-sided paralysis or right-sided sensory disturbance. Diffusion- and T2-weighted images of cervical MRI revealed a high-intensity area on the left side of the spinal cord, at the level of the 3rd cervical vertebra, suggesting a spinal cord infarction related to occlusion of the ASA system (**Fig. 3**). Due to disrupted consciousness, it was difficult to evaluate deep sensation and truncal ataxia, but the exacerbation of left-sided paralysis and analgesia of the right upper and lower extremities may have been related to the spinal cord infarction. External decompression was done, and anticoagulant therapy was transiently discontinued, but apixaban (at 10 mg/day) was resumed on the 5th postoperative day. The patient was referred to another hospital for recovery-phase rehabilitation on the 40th postoperative day. At the time of referral, his level of consciousness was almost normal, and the sensory disturbance of the right upper and lower extremities (related to the spinal cord infarction) was almost resolved. However, left-side paralysis (MMT score: 3/5), ataxic dysarthria, left-side dysesthesia, and ataxia of the extremities and trunk (related to pons and cerebellar infarction) remained. The patient was discharged approximately 6 months after mechanical thrombectomy, with a modified Rankin Scale (mRS) score of 3.

## Discussion

In the endovascular approach for treatment of posterior circulation, the VA is selected in most cases. Only seven reports (13 cases) of spinal cord infarction after VA-related endovascular treatment have been published.[5–11] No study has reported spinal cord infarction related to mechanical thrombectomy for the basilar artery (**Table 1**), which was observed in our case. In most cases, spinal cord infarction is a complication associated with the parent artery occlusion in VA dissection.[5,7,10,11] Four types of pathogeneses have been reported: (1) direct device-related occlusion of the RMA,[5] (2) occlusion of the ASA due to vascular stump thrombosis of the VA postoperatively,[5,7] (3) occlusion of the posterior spinal artery (PSA) due to intentional flow arrest during the procedure by a balloon guiding catheter,[10] and (4) occlusion of the PSA due to flow arrest caused by wedging of the guiding catheter.[10,11] With regard to procedures other than parent artery occlusion in VA dissection, several studies have suggested that flow arrest due to wedging of the guiding catheter, as described in the pathogenesis types (3) and (4) above, induced spinal cord infarction.[6,9] In these cases, coil embolization of ruptured or unruptured cerebral aneurysms was performed using 6-Fr or 7-Fr guiding catheters, and wedging occurred on insertion

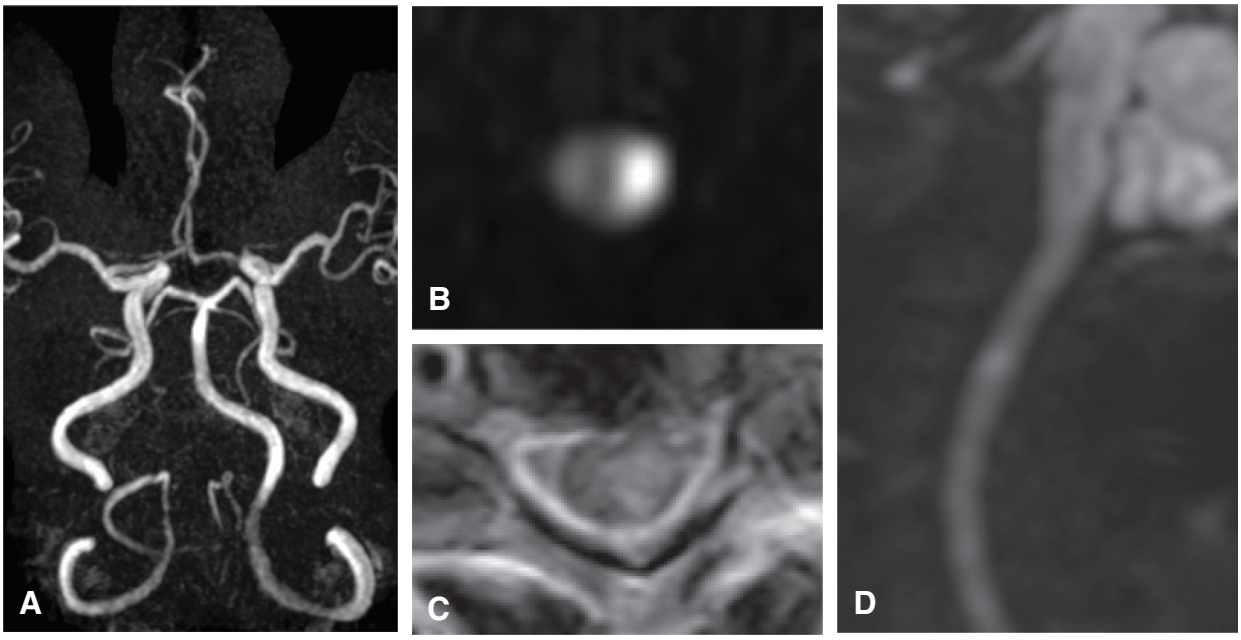


**Fig. 3** (**A**) MRA performed 2 days after mechanical thrombectomy shows no reocclusion of the basilar artery and the hypoplastic right VA at the distal part of the posterior inferior cerebellar artery. (**B, C, D**) Cervical MRI shows a high-intensity area on the left side of the spinal cord at the C3 level on axial DWI (**B**), axial T2-weighted image (**C**), and sagittal DWI (**D**). DWI: diffusion-weighted imaging; MRA: magnetic resonance angiography; VA: vertebral artery

of the guiding catheters. In these cases, the ASA was visualized on angiography and the operative time was between 3 and 5 hours. In our case, there was no wedging of the guiding sheath at the start of the procedure. In addition to the use of a guiding system of large diameter, procedure-related vascular linearization led to the disappearance of guiding-sheath deflection, resulting in the unintentional migration of the guiding sheath to the distal left VA, and development of guiding-sheath wedging at the end of the procedure. Furthermore, the unintentional migration of the guiding sheath to the distal VA may have induced VA dissection. After surgery, cervical MRA was not performed, and assessment of the VA at the site of insertion of the guiding sheath was insufficient. However, the likelihood of dissection was considered to be low, based on the left subclavian arteriography findings at completion of the mechanical thrombectomy. The contralateral VA exhibited hypoplasia, and neither the ASA nor the RMA was visualized on ipsilateral vertebral arteriography. Therefore, hemodynamic ischemia, thrombus formation, or both may have developed, despite a shorter procedure time than in previous studies.

Spinal cord blood supply is divided by central and peripheral regions. The center is nourished by the ASA, and the periphery by the paired PSAs and the peri-spinal pial arterial plexus anastomosing with the ASA and the PSAs. The central sulcal artery arises from the ASA and has two branches: left and right. These branches unilaterally nourish the anterior horn of the spinal cord and anterior lateral funiculus, anastomosing with the ipsilateral upper and lower central sulcal arteries. Some of the radicular arteries (responsible for blood supply to the dura mater and nerve roots) anastomose with the ASA or PSA, nourishing the spinal cord as RMAs. Thus, the spinal cord has abundant anastomoses. Cervical cord blood supply is especially abundant, and infarction is rare, even if a feeding vessel is occluded.[12] In our case, neither the ASA nor RMAs had been visualized on angiography, making it difficult to identify the occluded blood vessel. Furthermore, it was difficult to evaluate deep sensation and truncal ataxia, due to impaired consciousness, even though left-sided paralysis and sensory disturbance of the right upper and lower extremities developed. The symptoms in our case were suggestive of pyramidal and spinothalamic tract involvement, suggesting partial Brown-Sequard syndrome—which is characterized by occlusion of the central sulcal artery causing motor paralysis on the ipsilateral side, and contralateral sensory disturbance, leading to partial Brown-Sequard syndrome.[13] The infarcted area seen on imaging was also consistent with the area supplied by the central sulcal artery. In our case, unilateral central sulcal artery occlusion may have been primarily responsible for the infarct.

**Table 1** Literature review for cases of spinal cord infarction after neuroendovascular treatment

| | Diagnosis | Procedure | Location of SCI | Guiding system | Contralateral VA | ASA | RMA | Cause | Procedure time |
|---|---|---|---|---|---|---|---|---|---|
| Iwai et al. 2000[5] | Unruptured Rt.VAD (partially thrombosed) | PAO | Medulla oblongata C1 | N/A | Equivalence | Rt.VA | N/A | Delayed occlusion of ASA | N/A |
| | Unruptured Rt.VAD | PAO | Medulla oblongata C1 | N/A | Equivalence | Not detected | Occlusion site | Direct obliteration of RMA | N/A |
| | Ruptured Rt.VAD | PAO | Medulla oblongata C1 | N/A | Equivalence | Not detected | Not detected | Suspected direct obliteration of RMA | N/A |
| Matsubara et al. 2013[6] | Unruptured basilar-tip aneurysm | Coil embolization | Multiple Lt. C4-C7 | Lt.VA: 7Fr GC | Equivalence | Lt.VA | Lt. VA C6 level | Wedge of GC | 3.5 hours |
| | Ruptured basilar-tip aneurysm | Coil embolization | Multiple Bilateral C2-3 | Lt.VA: 7Fr GC Rt.VA: 4Fr catheter | Equivalence | Lt.VA | Lt.: not detected Rt.: lower VA | Wedge of GC | 5 hours |
| Kashiwazaki et al. 2013[7] | VAD | PAO | N/A | N/A | N/A | N/A | N/A | Occlusion of ASA | N/A |
| | VAD | PAO | N/A | N/A | N/A | N/A | N/A | Occlusion of ASA | N/A |
| Elzamly et al. 2018[8] | Lt. VA stenosis | Angioplasty | Single Posterior C1 | N/A | Equivalence | N/A | N/A | Delayed occlusion of PSA | N/A |
| Iwahashi et al. 2018[9] | Unruptured basilar-tip aneurysm | Coil embolization | Lt.C1-4 | Lt.VA: 6Fr GC | Equivalence | Bilateral VA | N/A | Wedge of GC | 3 hours |
| Tsuruta et al. 2018[10] | Unruptured Lt.VAD | SACE | Lt.C2 | 6 or 7Fr GC | N/A | N/A | N/A | Wedge of GC | N/A |
| | Unruptured Lt.VAD | Proximal occlusion | Lt.C2 | 6Fr BGC | Equivalence | N/A | N/A | Induced flow arrest | N/A |
| | Unruptured Rt.VAD | Proximal occlusion | Rt.C1 | 6Fr BGC | Equivalence | N/A | N/A | Induced flow arrest | N/A |
| Sarto et al. 2020[11] | Unruptured Lt.VAD | PAO | Rt.posterior C3-4 | Lt.VA: 0.088 inch GC | Equivalence | Rt.VA | Not detected | Wedge of GC | N/A |
| Present case | BA occlusion | Mechanical thrombectomy | Single Lt.C3 | Lt.VA: 6Fr GS | Hypoplasia | Not detected | Not detected | Wedge of GS | 1.5 hours |

ASA: anterior spinal artery; BA: basilar artery; BGC: balloon-guide catheter; Fr: French gauge; GC: guiding catheter; GS: guiding sheath; Lt.: left; N/A: not available; PAO: parent artery occlusion; PSA: posterior spinal artery; RMA: radiculomedullary artery; Rt.: right; SACE: stent-assisted coil embolization; SCI: spinal cord infarction; VA: vertebral artery; VAD: vertebral artery dissection

With regard to mechanical thrombectomy, emergency treatment is required in the absence of preoperative information. The importance of evaluating an approach for endovascular treatment is well known, and methods of evaluating the aortic arch include CT of the body[14] and MRA. Furthermore, prompt evaluation based on intraoperative findings is required. If there is wedging while navigating a guiding catheter, the size of the guiding catheter should be reduced or the guiding system changed. A switch should be made to an 80-cm guiding catheter, which is shorter than a standard guiding catheter, or a specifically shaped guiding catheter such as the NEURO-EBU (Hanaco Medical, Saitama, Japan) should be used. These should be inserted into the origin of the VA or into the subclavian artery. The use of an aspiration catheter may prevent guiding-system wedging and spinal cord infarction. In our case, unintentional migration of the guiding sheath intraoperatively induced wedging. During the procedure, the operator will be focused on the site of the lesion, but the team must pay attention to promptly detect changes involving the guiding system in sites other than that of the lesion.

This case demonstrated spinal cord infarction as a complication related to posterior circulation endovascular treatment, specifically mechanical thrombectomy, and the importance of preoperative and intraoperative risk assessment of approaches. In this case, unintentional guiding-sheath migration induced wedging, leading to spinal cord infarction. If preoperative information regarding the vascular course is obtained, a guiding system can be sufficiently chosen; however, it is necessary to select an appropriate device and procedure during mechanical thrombectomy. To prevent spinal cord infarction, it is important to pay attention to details such as the site of guiding-system insertion and guiding-catheter motions.

## Conclusion

We report a case of spinal cord infarction after mechanical thrombectomy done for basilar artery occlusion. This was an emergency treatment performed in the absence of preoperative information, such as presence of a tortuous artery. In addition to the prompt peri-operative assessment of approaches, the selection of appropriate devices and procedures may be important to the success of the treatment.

## Disclosure Statement

The authors declare no conflict of interest.

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