Spinal Cord Infarction after Successful Coil Embolization of Recurrent Basilar Bifurcation Aneurysm: A Case Report

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Objective: We report a case of spinal cord infarction that developed after successful coil embolization of a recurrent basilar bifurcation aneurysm. This complication has been rarely reported in the literature, but may cause severe sequelae following the endovascular embolization.

Case Presentation: During a follow-up examination 6 years after balloon-assisted coil embolization by bilateral vertebral artery (VA) approach to treat an unruptured basilar bifurcation aneurysm, recanalization was noted in a 78-year-old patient. There had been no complications after the original surgery and the postoperative course was uneventful. We planned retreatment by stent-assisted coil embolization via the left VA approach. Immediately after the surgery was successfully completed, severe left-sided hemiparesis appeared, but there were no discernible intracranial ischemic lesions causing the symptom. Cervical MRI revealed an infarction on the left side of the cervical spinal cord between the first and fourth cervical vertebrae. At 6 months' follow-up, she was able to walk with minimal assistance.

Conclusions: The spinal cord infarction seemed to have been caused by wedging of the guiding catheter, which had not occurred during initial treatment. When performing endovascular treatment for posterior circulation disease, wedging of the guiding catheter should be avoided.

Keywords ▶ basilar bifurcation aneurysm, coil embolization, complication, spinal cord infarction

Introduction

Recently, coil embolization of both ruptured and unruptured cerebral aneurysms has been one of the most common treatments owing to its low morbidity and mortality rate.1,2 For posterior circulation aneurysms, which usually present challenges in direct surgery, favorable outcomes of coil embolization have been reported, especially for aneurysms at the bifurcation of the basilar artery.3 When performing coil embolization of aneurysms of the posterior circulation, the vertebral artery (VA) is usually selected as an approach route that supplies the cervical spinal cord. Accordingly, it is theoretically understandable that spinal cord infarction may occur as a procedure-related complication, but few studies have reported such complications.4

In this paper, we report a patient who developed a spinal cord infarction after the second session of coil embolization of a recurrent basilar bifurcation aneurysm. Although she experienced no complications after the first session of coil embolization, she suffered this complication after the second treatment. We discuss the etiology of this rare complication based on the vascular anatomy of the spinal cord by comparing the two sessions in the same patient.

Case Presentation

A 72-year-old woman with a history of hypertension and chronic heart failure complained of dizziness and visited our hospital. MRI of the head revealed an aneurysm at the bifurcation of the basilar artery (maximum diameter: 9.6 mm, neck diameter: 9.5 mm) for which coil embolization...
was selected. Clopidogrel 75 mg was administered from 1 week before surgery as an antiplatelet drug. Under local anesthesia, 6 French (Fr) guiding catheters were placed in the bilateral VAs (tips of both catheters were placed just proximal of the transverse foramen of the axis [V2]). Balloon-assisted double-catheter coil embolization using a Hyperform 4 mm × 7 mm (Medtronic, Minneapolis, MN, USA), Excelsior 1080 (Stryker, Fremont, CA, USA), and SL-10 (Stryker) was performed (Fig. 1). The anterior spinal artery (ASA) was visualized on bilateral VA angiograms (Fig. 1, white arrowheads). There were no changes in the appearance of ASAs after treatment compared with preoperative angiography. Postoperative MRI did not show any new ischemic lesions, and the patient was discharged without any new neurologic deficits. Periodic follow-up by serial MRA was continued at the outpatient clinic after the procedure. MRA obtained a few years after surgery showed recanalization of the aneurysm, and at 6 years after surgery, showed marked enlargement. The recanalization was located on the left anterior side of the previously inserted coils. As repeat cerebral angiography revealed apparent recanalization of the aneurysm, the patient was admitted for additional treatment. There were no neurologic deficits on admission.

Endovascular treatment

Dual-antiplatelet therapy of aspirin 100 mg and clopidogrel 75 mg had been started 1 week before surgery, and cilostazol 200 mg was orally administered on the day of treatment. Under local anesthesia, a 6 Fr long introducer was placed in the right femoral artery. After systemic heparinization (intravenous injection at 5000 units, followed by continuous intravenous infusion of 1000 units/hour), a 6 Fr guiding catheter (Roadmaster STR 90 cm; Goodman Co., Ltd., Aichi, Japan) was placed in the left VA for stent-assisted coil embolization (Fig. 2A and 2B). The tip of the 6 Fr guiding catheter was placed just before the transverse foramen of the axis (V2), but angiography at this position showed stasis of the contrast medium and indicated guiding-catheter wedging. Because the stasis gradually disappeared
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before the latter half of the venous phase, suggesting the preservation of antegrade flow, we decided to continue the procedure. Activated clotting time (ACT) immediately after guiding-catheter placement was 204 seconds. Just after placement of the guiding catheter, the patient complained of cervical pain making it impossible to rest continuously, and sedation by pentazocine administration was required. A stent delivery microcatheter (Prowler Select Plus; Codman & Shurtleff, Johnson & Johnson, Raynham, MA, USA) was guided to the left P2 using a CHIKAI Black 14 200-cm microwire (Asahi Intecc Co., Ltd., Aichi, Japan). A microcatheter (Excelsior SL-10 45°; Stryker) was guided into the site of recanalization. When two microcatheters were inserted, the patient complained of headache and nausea, and metoclopramide and pentazocine were administered. There were no changes in the appearance of the ASA on VA angiograms taken before and after placement of microcatheters. A self-expanding stent (Enterprise 2 VRD 4.5 mm × 23 mm; Codman & Shurtleff) was placed from the left P2 to the basilar artery. The working angle was changed to a right anterior oblique position so that the site of recanalization could be clearly visualized. Three HydroFrame 10 6 mm × 19 cm coils (MicroVention, Inc., Aliso Viejo, CA, USA) were inserted through a jail catheter. Embolization was performed using two HydroFrame 10 4 mm × 8 cm coils, three HydroSoft 10 4 mm × 8 cm coils (MicroVention, Inc.), one HydroSoft 10 4 mm × 10 cm coil, one HydroSoft 10 3 mm × 8 cm coil, two Target 360 Ultra 3 mm × 6 cm coils (Stryker), one Target 360 Ultra 3 mm × 8 cm coil, and one Target 360 Ultra 2.5 mm × 4 cm coil. Using a transcell technique, a Target 360 Ultra 2 mm × 4 cm coil was inserted into the neck region at the left P1 area, and complete obliteration of the aneurysm was confirmed (Fig. 2C and 2D). There were no changes in the visualization of the ASA on VA angiograms before and after coil embolization (Fig. 2, white arrowheads). However, the guiding catheter placed in the left VA remained wedged, and stasis of the contrast medium was observed until the venous phase (Fig. 3). During the procedure, there was no blood regurgitation into the Y-connector or perfusion trouble of the continuous infusion of saline with heparin in the guiding system. The duration of guiding-catheter placement in the left VA was 180 minutes. ACT at the completion of the procedure was 246 seconds, and continuous infusion of heparin was maintained after the surgery. Postoperative CT confirmed the absence of new lesions, and the patient was admitted to the intensive care unit (ICU).

Postoperative course

After admission to the ICU, marked left hemiparesis (Manual Muscle Test [MMT]: 1/5) was noted, and emergency brain MRI was performed. Diffusion-weighted imaging (DWI) revealed fresh, scattered ischemic lesions involving the left medulla oblongata, bilateral cerebellar hemispheres,
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and bilateral occipital lobes. However, we could not identify any lesion that could have caused her hemiparesis. After the procedure, argatroban and edaravone were administered in addition to the oral administration of triple antiplatelet drugs. A second MRI taken the day after surgery did not show any marked enlargement in the ischemic lesions (Fig. 4A), but the left hemiparesis did not improve. Seven days after surgery, cervical MRI revealed spinal cord infarction associated with spinal cord swelling on the left side at the level of the first through fourth cervical vertebrae (Fig. 4B–4D), which seemed likely to have caused the hemiparesis. Cervical MRI 21 days after surgery showed an improvement in spinal cord edema; the final extent of the infarction was well defined (Fig. 5). She continued undergoing rehabilitation for hemiparesis during hospitalization, and had MMT scores of 3/5, 4/5, and 5/5, respectively, for the left shoulder, elbow, and fingers, when transferred to a rehabilitation hospital 42 days after surgery. The mild left hemiparesis (MMT: 4/5), sensory disturbance of the left half of the body, and paresthesia remained for 6 months after surgery, but the patient became able to walk with an assistant cart.

Discussion

In the present case, two sessions of coil embolization were performed on an unruptured basilar bifurcation aneurysm, and a spinal cord infarction occurred after the second session. Because the same angiography suite and head-fixing table were used, the head position might have been the same between the two sessions. For the first session, 6 Fr guiding catheters were placed in the bilateral VAs under the oral administration of a single antiplatelet drug, and coil embolization was completed without wedging of the guiding catheters. Postoperative DWI did not show any high signal intensity areas. When the second coil embolization was performed 6 years later, a single 6 Fr guiding catheter was placed in the left VA under the oral administration of triple antiplatelet drugs. However, spinal cord infarction associated with multiple intracranial ischemic lesions in the posterior cranial fossa was detected after the procedure. The findings of localized ischemic lesion on the left side of the spinal cord suggest that wedging of the guiding catheter may have been involved in this complication.
Because there have been no reports to date regarding the involvement of wedging in patients with similar conditions, we report this complication.

The blood supply of the spinal cord is divided into central and peripheral areas. The former is supplied by the ASA and its branch, central artery, and the latter by a pair of posterior spinal arteries (PSAs) and the pial artery plexus. Furthermore, some radicular arteries supplying the dura mater and nerve roots anastomosing with the ASA or PSA, also supply the spinal cord as a radiculomedullary artery (RMA). The ASA comprises vessels branching from bilateral VAs and the RMA as well as branches from bilateral VAs. In both the central and peripheral areas of the cervical cord, blood flow is abundant, so ischemic lesion may not occur even if a supplying vessel is occluded.

Several studies have reported cervical cord infarction related to unilateral VA dissection. One possible reported mechanism of spinal cord infarction is that the occlusion of the predominant-side VA may induce ischemia if the blood supply of the RMA is unilateral. Other studies have reported another mechanism: when the ASA is predominantly supplied by the unilateral VA, the predominant-side VA may be occluded, causing ischemia. Furthermore, the ASA may be partially duplicated at the level of the cervical cord in some cases. The central artery branching from the duplicated level supplies only the unilateral side; therefore, if obliteration occurs, the infarcted focus may involve the unilateral side in some cases.

Matsubara et al. speculated that guiding-catheter wedging in the VA might have led to spinal cord infarction because of the occlusion of RMAs by thrombus formation following hypoperfusion. They concluded that procedures with the guiding-catheter wedging should be avoided.

In the present case, 6 Fr guiding catheters were used for the two sessions at the initial and additional treatments. After the first session (6 Fr guiding catheters were placed in the bilateral VAs), there was no spinal cord infarction or ischemic lesions on brain MRI. On the second session, a 6 Fr guiding catheter was placed in the left VA alone, but the wedged state persisted throughout the procedure (Fig. 3). As a cause of guiding-catheter wedging, the aging-related progression of arteriosclerosis over the prior 6 years may have been involved. During the procedure, contrast medium in the left VA was washed out in the latter phase of the venous phase, and blood flow from the contralateral VA was present; therefore, we did not consider exchanging the
guiding catheter for a thinner one to resolve the wedging. Thus, the continued wedging of the guiding catheter may have resulted in the complication in our case. It was impossible to evaluate the RMA with regard to whether thrombotic or ischemic mechanisms were involved, based on imaging findings. Because the ischemic lesions involved the cervical spinal cord, medulla oblangata, and bilateral cerebellar hemispheres, we cannot rule out the possibility of a wedge-related thromboembolic mechanism. Dissection of the VA could have occurred during placement of the guiding catheter, but neither intraoperative angiography nor postoperative MRA suggested dissection and this possibility was ruled out. Occlusion and recanalization of the ASA could have occurred during the procedure, but the ASA was visualized before and after treatment, and thus we consider it unlikely that it occurred during the procedure. Considering that the spinal cord infarction was localized on the left side (Fig. 4B and 4C), guiding-catheter wedging under left RMA predominance may have led to blood-flow reduction and thrombus formation.

In our patient, hemiparesis, which may have occurred during the procedure, was detected after ICU admission for sedation to treat intraoperative pain. The procedure was performed under local anesthesia, and it may have been possible to evaluate neurologic deficits by conducting neurologic examination immediately after guiding-catheter insertion or for a specific period. Although we spoke to the patient several times during the procedure, spinal ischemia could not be detected. If an ischemic symptom had been detected, the complication could have been avoided by conducting the procedure after exchanging a 6 Fr for a 5 Fr guiding catheter to resolve the wedging. It may also be necessary to evaluate the spinal cord blood flow distribution separate from the issue of guiding-catheter wedging, but because of the complexity of the spinal cord vasculature, as described above, quantitative evaluation may be difficult.

In the present case, we had not recognized the risk of cervical cord infarction as a complication related to coil embolization of a basilar bifurcation aneurysm, and this led to a delay in diagnosis. On final angiography, the ASA was visualized, and we did not consider spinal cord disorder as the etiology of neurologic symptoms after surgery; it was impossible to promptly conduct additional examination. When treating posterior circulation lesions, clinicians should be aware of the possibility of guiding-catheter wedging as a possible cause of spinal cord infarction. Although cervical cord infarction is rare, it is difficult to predict its risk. It may occur during or after endovascular treatment involving the posterior circulation. We reported the present case to raise awareness of this potential complication in readers of this journal, including treating physicians.

## Conclusion

We reported a patient with spinal cord infarction after coil embolization of a recurrent basilar bifurcation aneurysm. The infarction may have been related to guiding-catheter wedging in the VA; accordingly, wedging should be avoided. Spinal cord infarction should be recognized as a serious complication of endovascular treatment involving the posterior circulation.

## Disclosure Statement

There is no conflict of interest regarding this article for the main author or coauthors.

## References