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Case Report

Spinal cord infarction after coil embolization of a basilar tip aneurysm: A case report and literature review

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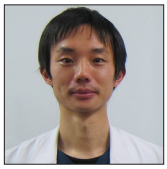

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ABSTRACT

Background: Spinal cord infarction is a rare but serious complication of neurointervention that has been rarely documented. An association between spinal cord infarction and the placement of large bore catheters in the vertebral artery (VA) has been mentioned, but the precise etiology remains unclear.

Case Description: A 72-year-old female presented with the right hemiparesis and left thermal hypoalgesia directly after endovascular coil embolization for an unruptured basilar tip aneurysm. Magnetic resonance imaging demonstrated right-sided upper cervical (C2-3) spinal cord infarction. Conventional bilateral vertebral angiograms revealed no opacification of the anterior spinal arteries. Cone-beam computed tomography showed a watershed area of radiculomedullary arteries that was correlated with the extent of the ischemic lesion. Thus, the spinal cord ischemia may have had multifactorial causes combined with reduced perfusion pressure to the spinal cord, which was caused by the placement of the guiding catheter in the VA and intensive hypotension during general anesthesia.

Conclusion: Spinal cord infarction should be recognized as a serious complication of endovascular treatment involving posterior circulation.

Keywords: Coil embolization, Complication, Intracranial aneurysm, Posterior circulation, Spinal cord infarction

INTRODUCTION

Spinal cord infarction is a rare but serious complication of neurointerventional procedures, but its etiology has been rarely recognized. It is a severe complication that requires awareness because it can significantly impact the functional outcome of patients. To the best of our knowledge, spinal cord infarction has only been described in four previous case reports.[3,4,7,8] In these reports, the placement of a large bore guiding catheter in the vertebral artery may have resulted in spinal cord ischemia because the wedged catheter caused the decreased perfusion pressure of the anterior spinal artery (ASA) and disruption of blood supply in the spinal cord. However, little is currently known about the angiographical features of spinal cord infarction after neurointervention.

Herein, we report a patient with cervical spinal cord infarction after an elective neurointerventional procedure. The patient presented with Brown-Séquard syndrome after the procedure, and acute ischemia was detected on the right side of the spinal cord using magnetic resonance imaging (MRI). To investigate the etiology of the spinal cord infarction, we performed

cone-bean computed tomography (CBCT), which allowed us to precisely identify the angioarchitecture. We discuss the etiology of the spinal cord infarction based on the findings of the CBCT scan and review the literature.

CASE REPORT

A 72-year-old woman with a history of multiple anterior circulation cerebral aneurysm clipping (6 years previously) was referred to our department because of a gradually growing and unruptured basilar tip aneurysm. Her neurological examination was normal and a cerebral angiogram revealed a basilar tip aneurysm with a maximum size of 4.6 mm. Endovascular balloon-assisted coil embolization was performed under general anesthesia. Dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg) was started 2 weeks before the surgery, and cilostazol (200 mg) was orally administered on the day of the treatment. A 6-French (Fr) introducer was placed in the right femoral artery. After systemic heparinization (intravenous injection at 5000 units, followed by continuous intravenous infusion of 1000 units/h), a 6-Fr guiding catheter (Roadmaster STR 90 cm; Goodman, Aichi, Japan) was placed in the right VA. The tip of the guiding catheter was placed in the distal end of the V2 segment and a VA angiogram confirmed no findings of decreasing contrast flow from the arterial to venous phases [Figure 1a]. Balloon-assisted coil embolization using a microcatheter (SL-10; Stryker Neurovascular, Fremont, CA, USA) and compliant balloon (SHOURYU 4 mm × 7 mm; Kaneka Medics, Kanagawa, Japan) was performed. Six coils were inserted into the aneurysm, with preservation of the bilateral posterior cerebral arteries [Figure 1b]. During the procedure, there was no blood regurgitation into the Y-connector and no complications with the continuous infusion of saline with heparin in the guiding system. The duration of the guiding catheter placement in the right VA was approximately 120 min. According to the anesthesia records, mean arterial pressure (MAP) during the procedure ranged from 46 to 76 mmHg. The activating clotting time at the completion of the procedure was 283 s and was reversed with protamine.

Although the awakening from anesthesia was good, the patient presented with right hemiparesis (Manual Muscle Test: 2/5); thus, an emergency brain MRI was performed. Diffusion-weighted imaging revealed very small ischemic lesions involving the pons and bilateral cerebellar hemispheres; however, these lesions were not related to the patient's neurological deficit. Four days after the intervention, the patient noted left thermal hypoalgesia, and cervical MRI revealed spinal cord infarction associated with spinal cord swelling on the right side at the level of the second and third cervical vertebral body [Figures 2a-d]; this seemed likely to have caused the symptoms. Her right-sided hemiparesis rapidly improved but chronic discomfort of the left limb continued. We performed conventional cerebral angiography 27 days after the intervention and confirmed a well-embolized aneurysm without opacification of the

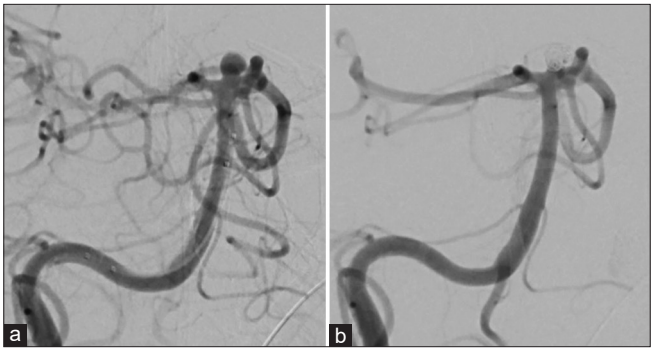

Figure 1: (a) Anteroposterior view of a right vertebral artery (VA) angiogram shows the unruptured basilar tip aneurysm. The tip of the guiding catheter was not wedged, and there was good flow around the guiding catheter. The anterior spinal artery was not visualized in this angiogram throughout the procedure. (b) Anteroposterior view of a right VA angiogram just after coil embolization shows that the aneurysm was successfully embolized, without signs of reduced VA flow.

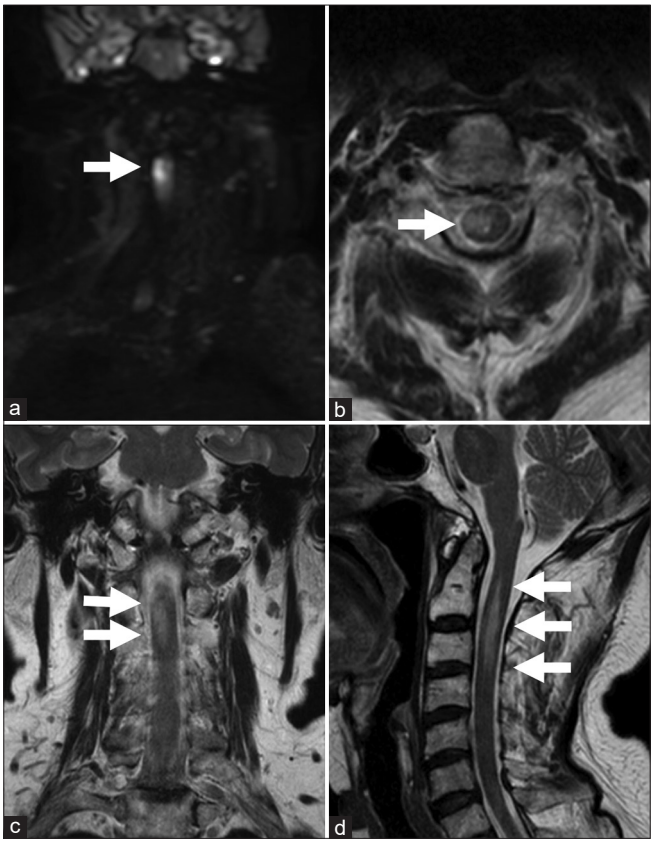

Figure 2: Coronal diffusion-weighted image (a) and axial (b), coronal (c), and sagittal (d) views of T2-weighted images of cervical magnetic resonance imaging on postoperative day 4 show spinal cord infarction at the level of the C2–3 vertebral body (arrows).

ASAs [Figures 3a and b]. A coronal CBCT scan of the right VA showed an avascular area consistent with spinal cord infarction [Figure 4a, blank arrow]. Radiculomedullary arteries (RMAs) at the level of C4 and C5 were visible; however, the RMA of the upper cervical spine from the right VA was not visualized. Although an axial CBCT view of the right VA at the level of C3 [Figure 4b] showed faint opacification around the right side of the spinal cord, an axial view of the left VA at the same level [Figure 4c] showed clear opacification of the left side of the spinal cord. On the other hand, an axial CBCT view of the right VA at the level of C4 [Figure 4d] showed clear opacification of the bilateral spinal surface pial vessels. A coronal CBCT scan of the left VA showed clear opacification of the RMA [Figure 4e], arrow) connected to an anterior spinal artery. Cervical MRI 28 days after the intervention showed an improvement in spinal cord edema; the final extent of the infarction was well defined [Figures 5a-c, arrows]. The patient was discharged home and showed improvements in motor function after an intensive rehabilitation program. Despite chronic pain in the left upper and lower limbs, she was able to conduct her daily life without any assistance (modified Rankin Scale: 2) at a 3-month follow-up.

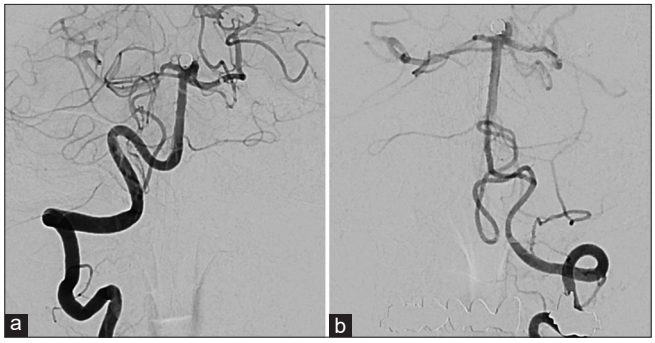

Figure 3: Anteroposterior views of the right (a) and left (b) vertebral artery angiograms obtained 4 weeks after the treatment show no opacification of the anterior spinal artery.

DISCUSSION

The blood supply of the spinal cord can be separated into a central and a peripheral system. The central system, which supplies two-thirds of the spinal cord, is supplied by the ASA and its branch (the central artery). The peripheral system is supplied by a pair of posterior spinal arteries (PSAs) and the pial artery plexus.[6] Furthermore, some radicular arteries that supply the dura mater and nerve roots anastomosing with the ASA or PSA also supply the spinal cord as RMAs. The ASA comprises vessels branching from bilateral

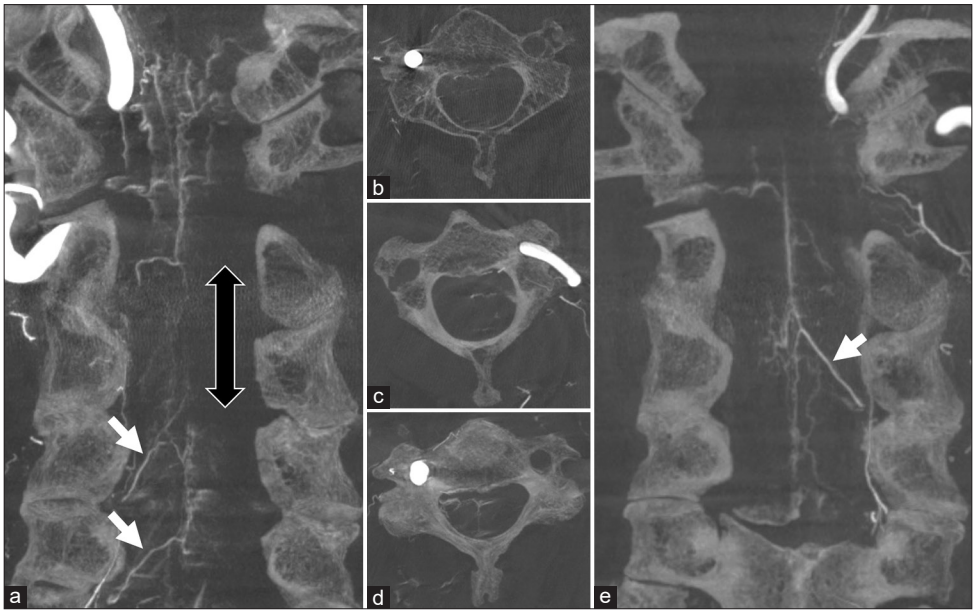

Figure 4: Reconstructed cone-beam computed tomography (CBCT) scans obtained 4 weeks after coil embolization. A coronal view of the right (a) vertebral artery (VA) shows an avascular area consistent with spinal cord infarction (blank arrow). Arrows indicate radiculomedullary arteries (RMAs) at the level of C4 and C5. An axial CBCT view of the right VA at the level of C3 (b) shows faint opacification around the spinal cord; however, an axial view of the left VA at the same level [Figure 4c] showed clear opacification of the left side of the spinal cord. On the other hand, an axial CBCT view of the right VA at the level of C4 [Figure 4d] showed clear opacification of the bilateral spinal surface pial vessels. A coronal CBCT scan of the left VA showed clear opacification of the RMA [Figure 4e], arrow) connected to an anterior spinal artery.

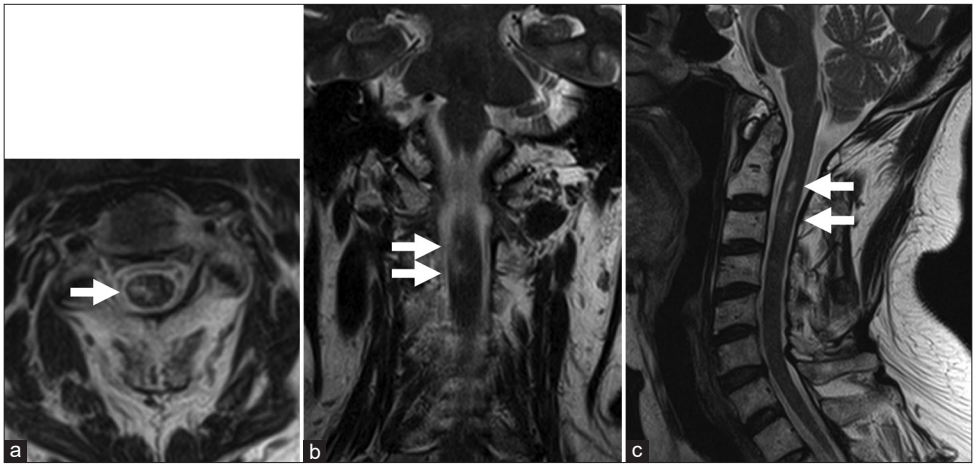

Figure 5: Follow-up axial (a), coronal (b), and sagittal (c) views of T2-weighted cervical magnetic resonance images 4 weeks after intervention show the final extent of the cervical spinal infarction (arrows).

VAs and the RMA as well those branching from bilateral VAs.[4] Symptoms of spinal cord infarction — which generally appear within minutes or a few hours of the infarction — may include hemiparesis or tetraparesis, sensory disturbance, and incontinence. Spinal cord infarction results from the obstruction of the arteries that supply the spinal cord and is usually caused by arteriosclerosis, aortic disease (such as aortic aneurysm and dissection), or spinal vascular disease.[7] Several studies have reported cervical cord infarction, which is mainly related to unilateral VA dissection. One possible reported mechanism of spinal cord infarction is that occlusion of the predominant VA may induce ischemia if the blood supply of the RMA is unilateral.[4] Other studies have reported that when the ASA is predominantly supplied by the unilateral VA, the predominant VA may be occluded, causing ischemia.[2,5]

The incidence of spinal cord infarction as a complication of neurointerventional procedures is very rare and has only been described in five case reports.[3,4,7,8] A summary of the reported cases, including our own, is listed in [Table 1]. In all reports (including our own), infarction occurred in the cervical region during neurointerventional surgery that targeted posterior circulation territories. One case of unilateral posterior cervical spinal cord infarction following vertebral angioplasty was attributed to flow reversal in the VA caused by a persistent stenosis in this artery, which resulted in hypoperfusion or thromboembolism in the ipsilateral PSA.[3] In three cases of anterior cervical spinal cord infarction following the embolization of basilar tip aneurysms, 6-or 7-Fr guiding catheters were used. The authors of these reports suggested that wedging of a relatively large bore catheter in the VA may have resulted in the thromboembolic occlusion or flow restriction of the RMA supplying the cervical spinal cord.[4,7] One case of anterior cervical spinal cord infarction following the embolization of a cerebellar arteriovenous malformation using Onyx involved the use of a 5-Fr guiding sheath, without any angiographic evidence of wedging during the procedure. The authors of this report concluded that a reduction in perfusion pressure to the ASA and RMA may have occurred when the 5-Fr guiding sheath was placed in the VA — even without catheter wedging or flow arrest — resulting in the spinal cord infarction.[8] To avoid this complication, these authors recommended downsizing the guiding catheter to 5 Fr as a possible solution, and also suggested shortening the procedural time and providing adequate antithrombotic therapy.

In our case, a 6-Fr guiding catheter was not wedged, and there was no flow stagnation in the VA throughout the procedure. As reported in the previous literature, a reduction of VA flow during the procedure is an important risk factor that should be avoided; however, because our patient did not have these factors, we should consider other etiologies. In the present report, we were able to demonstrate the precise angioarchitecture at the subacute stage of spinal cord infarction using CBCT; this has never before been reported. Our CBCT findings revealed that the area of the spinal cord infarction was correlated with a hypovascular watershed area on right VA injection. Although an RMA from the left VA provided blood supply to the ASA at the level of C3, a reconstructed coronal view showed no opacification of the right side of the spinal cord. We speculate that, if a patient has the specific characteristics that we have reported here, spinal cord infarction can occur even in the absence of a catheter wedge or flow reduction.

As previously reported, the cause of the spinal cord infarction will be related to thrombosis of the feeding artery of the spinal cord, adequate antithrombotic therapy was

Table 1: Summary of reported cases of spinal cord infarction related to neurointerventional procedure.

Author, (year)	Age/Sex	Diagnosis	Intervention	Site/Size of GC or GS	Wedge of guide catheter	Origin of ASA	Origin of RMA feeding infarcted lesion	Laterality/ level of SCI	Outcome at discharge
Matsubara *et al*., 2013[3]	66 year/ male	Unruptured BA-tip AN	Coil embolization	Left VA/7 Fr GC	+	From left VA	Left VA	Left anterior/ C4–7	mRS4
	69 year/ male	Ruptured BA-tip AN	Coil embolization	Left VA/7 Fr GC	+	Faintly from left VA	None	Bilateral anterior/ C3–5	mRS4
Iwahashi *et al*., 2017[4]	72 year/ female	Unruptured BA-tip AN	Coil embolization	Left VA/6 Fr GC	+	From left VA	None	Left anterior/ C1–4	mRS3
Elzamly *et al*., 2018[2]	70 year/ male	Left VA stenosis	VA angioplasty	NA	-	NA	NA	Left posterior/ C1	mRS2
Moazeni *et al*., 2021[1]	69 year/ female	Right cerebellar AVM	Onyx embolization	Left VA/5 Fr GS	-	From left VA	Left VA	Bilateral anterior/ C2–7	mRS4
Present case	72 year/ female	Unruptured BA-tip AN	Coil embolization	Right VA/6 Fr GC	-	Unable to detect	None	Right anterior/ C2–3	mRS2

ASA: Anterior spinal artery, GC: Guiding catheter, GS: Guiding sheath, mRS: Modified Rankin scale, NA: Not applicable, RMA: Radiculomeningeal artery, SCI: Spinal cord infarction, VA: Vertebral artery

recommended. Routine use of dual antiplatelet therapy and systemic heparization during procedure was mandatory. In cases of coil embolization without stenting, we have used neutralized heparinization with protamine and performed manual compression hemostasis at the femoral puncture site. Considering these cases, the use of femoral vascular closure devices without using protamine should be considered.

Another possible factor related to spinal cord infarction that should be considered is that intraoperative intensive hypotension under general anesthesia may cause a reduction in perfusion pressure to the ASA and RMA. Intensive hypotension also plays an important role in the development of stroke, and a MAP decrease of more than 30% from the baseline has been reported as significantly associated with the occurrence of postoperative stroke.[1] In the present case, intraoperative MAP ranged from 46 to 76 mmHg during the procedure, although the patient's normal MAP was approximately 100 mmHg. Thus, the rate of MAP decrease was calculated as 24–54%; this may also have created complications. Because cervical spinal cord infarction is a rare complication of neurointerventional procedure, its risk is hard to predict and it is difficult to avoid. Clinicians should be aware that cervical spinal cord infarction may occur during or after neurointerventional procedures, especially those involving posterior circulation, and it should be taken into consideration if neurological deficits without any intracranial lesions are observed.

CONCLUSION

We reported a patient with cervical spinal cord infarction after the coil embolization of a basilar tip aneurysm and reviewed the recent literature. Our patient's spinal cord ischemia may have had multifactorial causes combined with reduced perfusion pressure to the spinal cord, which was caused by the placement of the guiding catheter in the VA and intensive hypotension during anesthesia. Spinal cord infarction should be recognized as a serious complication of endovascular treatment involving posterior circulation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Bijker JB, Persoon S, Peelen LM, Moons KG, Kalkman CJ, Kappelle LJ, *et al*. Intraoperative hypotension and perioperative

ischemic stroke after general surgery: A nested case-control study. Anesthesiology 2012;116:658-64.
2. Crum B, Mokri B, Fulgham J. Spinal manifestations of vertebral artery dissection. Neurology 2000;55:304-6.
3. Elzamly K, Nobleza C, Parker E, Sugg R. Unilateral upper cervical posterior spinal cord infarction after a neuroendovascular intervention: A case report. Case Rep Neurol Med 2018;2018:5070712.
4. Iwahashi H, Fujita A, Tanaka H, Ikeda M, Morikawa M, Kohmura E. Spinal cord infarction after successful coil embolization of recurrent basilar bifurcation aneurysm: A case report. J Neuroendovasc Ther 2018;12:398-403.
5. Machnowska M, Moien-Afshari F, Voll C, Wiebe S. Partial anterior cervical cord infarction following vertebral artery dissection. Can J Neurol Sci 2008;35:674-7.
6. Martirosyan NL, Feuerstein JS, Theodore N, Cavalcanti DD, Spetzler RF, Preul MC. Blood supply and vascular reactivity of the spinal cord under normal and pathological conditions: A review. J Neurosurg Spine 2011;15:238-51.
7. Matsubara N, Miyachi S, Okamoto T, Izumi T, Asai T, Yamanouchi T, *et al*. Spinal cord infarction is an unusual complication of intracranial neuroendovascular intervention. Interv Neuroradiol 2013;19:500-5.
8. Moazeni Y, Cantrell DR, Clark JR, Abdalla RN, Batra A, Hurley MC, *et al*. Case report: Anterior spinal cord ischemia following embolization of cerebellar arteriovenous malformation: An illustrative case and review of spinal cord vascular anatomy. Front Neurol 2021;12:725065.

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