

Case Report

Endovascular treatment with tirofiban during the acute stage of cervical spinal cord infarction due to vertebral artery dissection

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Context: Cervical spinal cord infarction is a rare and severe complication of vertebral artery dissection (VAD). We report a case of VAD in an acute stage followed by cervical spinal cord infarction that was treated using direct endovascular tirofiban infusion via digital subtraction angiography (DSA) monitoring.

Findings: A 48-year-old man presented with vertigo, neck pain, numbness and weakness in four limbs with subsequent cardiac and respiratory arrest. Neurological examination revealed hypoesthesia below the neck with grade one myodynamia on the right side of the limbs and zero on the left side. The diagnosis of VAD-related cervical spinal infarction was confirmed using DSA imaging and cervical vertebra magnetic resonance imaging (MRI). The patient received timely treatment with endovascular tirofiban infusion and achieved good outcome without any sequelae.

Conclusion: Endovascular treatment with tirofiban may be a selective choice for cervical spinal cord infarction due to VAD in acute stage and warrants further study.

Keywords: Endovascular, Tirofiban, Cervical spinal cord infarction, Vertebral artery dissection

Introduction

The annual incidence of cervical artery dissection (CeAD) is approximately 2.6-3.0 per 100,000. CeAD is classified as carotid artery dissection (CAD) or vertebral artery dissection (VAD).¹ Spinal cord stroke accounts for only 0.3-1% of all strokes.² Therefore, VAD-related cervical spinal infarction is very rare. The mechanism of dissection is associated with intimal tear of arteries followed by hemodynamic-relevant stenosis and secondary thrombosis, which results in acute ischemic stroke and symptom progression.³ Cervical spinal cord infarction may result in severe neurological complications, and the prognosis is closely related to early diagnosis and prompt treatment.⁴ We describe a case of symptomatic VAD treated with an endovascular tirofiban infusion during the acute stage. This treatment ceased progression of the condition, and the patient exhibited a good outcome.

Case report

A 48-year-old man visited a local clinic with complaints of vertigo, accompanied by neck pain at

12:00 AM. His blood pressure was 150/120 mmHg. The physician was able to lower the patient's blood pressure, and the patient was sent home. The patient was admitted to our hospital three hours later due to the reappearance of vertigo accompanied by numbness and weakness of the four limbs as well as difficulty breathing. The patient suddenly developed mental confusion, and cardiac and respiratory arrest that required tracheal intubation after 10 minutes. His heartbeat and breathing resumed after cardiopulmonary resuscitation (CPR). Physical examination revealed hypoesthesia below the neck with and grade one myodynamia on the right side of the limbs and zero on the left side.

Head computed tomography (CT) and computerized tomography angiography (CTA) scanning were normal at admission. However, the patient's condition deteriorated. An immediate DSA was performed approximately 10 hours after onset. Nonselective arteriography revealed that the blood flow of the right carotid artery was from an innominate artery, and blood flow of the left carotid artery was directly from the aortic arch. Blood flow of the right vertebral artery (VA) was from the left subclavian artery, which

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was normal but much faster than that of the left VA. Bilateral internal carotid arteries supplied the middle cerebral arteries and anterior cerebral arteries, and the intracranial right VA supplied the basilar artery. Selective arteriography revealed no visible thrombi but severe stenosis of the left VA at the V2 segment, which slowed the blood flow, and disappearance of the segment of the anterior spinal artery (ASA) that supplied the upper cervical spinal cord had (Fig. 1A). The left VA was treated using an endovascular 10-ml tirofiban infusion via DSA monitoring. Brain and cervical vertebrae MRI scanning were performed approximately 48 hours after symptom onset. Brain MRI was normal. The cervical vertebrae MRI scan revealed high signals on T2-weighted images, which indicated ischemia extending from C4 to C1 (Fig. 2A). Axial MRI revealed the left VA with severe luminal stenosis and hyperintensity of intramural hematoma which indicated dissection (Fig. 2B). Therefore, the patient was diagnosed with VAD-related acute cervical spinal cord infarction. An intravenous drip of tirofiban at 13 ml/h was administered for the first three days. DSA re-examination on the third day revealed a decreased degree of stenosis at the left V2 segment, and obvious improvement in blood flow. The segment of the ASA that supplied the upper cervical spinal cord and the anterior radiculomedullary arteries reappeared, and blood flow recovered (Fig. 1B and C). Treatment was adjusted to anticoagulant therapy.

The neurological symptoms of the patient obviously improved 15 days later. Conventional dual antiplatelet therapy was recommended instead of anticoagulant therapy.

At the six-month follow-up, three-dimensional CTA revealed that the left VA dissection at the V2 segment had disappeared, and the vasculature was completely recanalized.

Discussion

Representative cases of spinal cord infarction due to VAD with manifestations of headache or neck pain, and neurological deficits of motor weakness and hypoesthesia have been reported.⁵ Our patient also suffered cardiac and respiratory arrest because the ischemia extended from C4 to C1, which is responsible for respiration.

The segmental spinal arteries supplying the cervical region vary considerably between individuals because these arteries generally originate from the vertebral or subclavian artery.⁶ ASA is formed by the confluence of the bilateral vertebral artery branches and descends to the conus medullaris. ASA is a series of anastomotic

vascular loops, not a single artery, and thus requires a segmental radiculomedullary arterial supply to provide adequate blood flow to the anterior two-thirds of the spinal cord tissue.⁷ Radiculomedullary arteries at the cervical segment often originate from a deep cervical artery or vertebral arteries. The mechanism of dissection is associated with intimal tears of arteries followed by hemodynamic-relevant stenosis and secondary thrombosis, which results in acute ischemic stroke and progressive symptoms.³ The dissection in our patient occurred at the left V2 segment and resulted in stenosis of the lumen and hemodynamic changes. There was insufficient time for contralateral vertebral or subclavian artery compensation; the left radiculomedullary arteries were suddenly disrupted, and the cervical segment of ASA was insufficient. The cervical spinal cord infarction formed over time (Fig. 2A and B). Moreover, the patient presented with a progressing stroke due to secondary microemboli induced by dissection, which preceded the neurological deficit. Timely and effective therapy was necessary.

VAD-related cervical spinal cord infarction should be treated on a case-by-case basis, and treatment efficacy is closely associated with onset time and stroke progression. Several treatment options for VAD are indicated, including thrombolysis, anticoagulation, antiplatelet, surgical and endovascular repair.¹ The safety and efficacy of thrombolysis are not well established.⁸ A randomized trial demonstrated no difference in the efficacy of antiplatelet and anticoagulant drugs in preventing stroke and death in patients with symptomatic carotid or vertebral artery dissection.⁹ Endovascular and surgical repair should be considered when patients are unresponsive to any medical therapy.^{1,4} The diagnosis of our patient was not clear at admission, but his condition deteriorated. We immediately performed a DSA and found severe stenosis of the left V2 segment. Conservative medication took effect slowly, and the time window for thrombolysis passed. No visible thrombus was found to perform thrombectomy, and stent implantation is not a routine indication for dissection. Therefore, direct endovascular tirofiban infusion in the left VA under DSA monitoring was chosen for the first time because it was the most direct and effective treatment. American heart association/American stroke association (ASA/AHA) or Chinese guidelines indicate no definite time window for tirofiban infusion in the treatment of acute cerebral or spinal stroke, and more randomized and controlled trials (RCTs) are required.^{10,11} Tirofiban is a highly selective, nonpeptide glycoprotein IIb/IIIa platelet receptor antagonist that exhibits a short half-life.

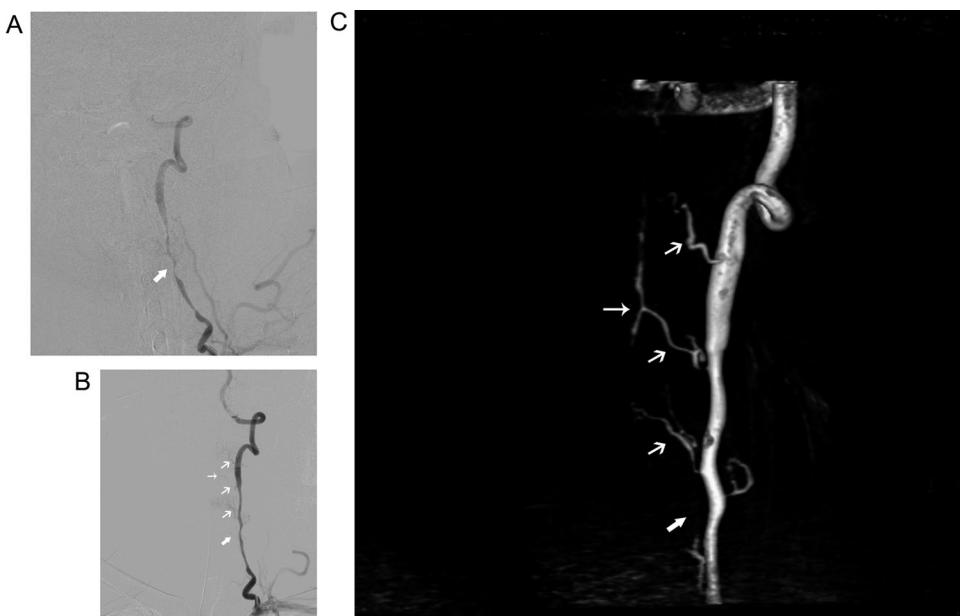


Fig. 1. (A) DSA revealed severe stenosis of the left VA in the V2 segment. The anterior spinal artery and anterior radiculomedullary arteries had disappeared (thick arrow). (B and C) DSA imaging reexamination revealed that the degree of left VA stenosis in the V2 segment (thick arrow) was approximately 50%, resulting in the reappearance of anterior radiculomedullary arteries (blunt arrows) and ASA (thin arrow), which supplied the upper cervical spinal cord.

Rapid recovery of prolonged bleeding time is observed after discontinuation, and its safety was preliminary demonstrated in the SaTIS trial.¹² Intravenous tirofiban infusion was used previously in coronary and carotid dissection.^{13,14} Low-dose intra-arterial tirofiban was confirmed to improve functional outcome in acute ischemic stroke with endovascular treatment.¹⁵ It may have directly acted on new microemboli of the ASA from intimal tears of the left V2 segment in our patient, and avoided an “artery to artery” embolization

of the basilar occlusion to quickly prevent progression to stroke.^{16,17}

Spinal cord infarction patients experience more significant improvement and exhibit lower long-term mortality than do cerebral infarction patients.² The segment of the ASA that supplied the upper cervical spinal cord from C4 to C1 reappeared after tirofiban treatment because the left radiculomedullary arteries from the left VA recovered their blood flow (Fig. 1B and C). Our patient achieved a good outcome without any sequelae and exhibited no

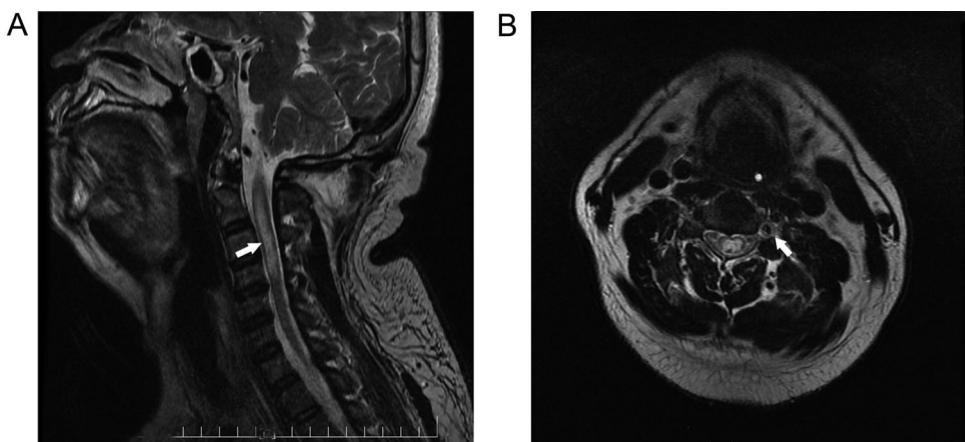


Fig. 2. (A) T2-weighted images on sagittal MRI revealed a high signal, which indicated that the ischemia extended from C4 to C1. These signs were compatible with infarction of the cervical medulla. (B) Axial T2-weighted images revealed severe luminal stenosis and a hyperintense area of smooth vascular wall thickening, which indicated intramural hematoma of the left VA in the V2 segment. These signs were compatible with dissection of the left V2 segment.

recurrence of cervical spinal infarction despite tirofiban infusion approximately 10 hours after symptom onset.

Conclusion

Spinal cord infarction is a rare complication of VAD that exhibits atypical manifestations of headache or neck pain and neurological deficits of motor weakness and hypoesthesia. Spinal infarction should be considered if these acute symptoms occur, and the cerebral image is normal. Endovascular tirofiban infusion may be a good choice for treatment during the acute stage in select cases of VAD-related infarction, but further study is warranted.

Disclaimer statements

Contributors None

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Conflicts of interest The authors declare that they have no conflicts of interest to disclose.

Ethics approval None.

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