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【 CASE REPORT 】

Cervical Cord Infarction Caused by Dissection of the Intracranial Segment of the Vertebral Artery

Hidefumi Suzuki, Tadashi Kitagawa, Masahiro Gotoh,
Takahiro Mitsueda-Ono and Masaru Matsui

Abstract:
Cervical cord infarction is uncommon but has been increasingly reported as a complication of vertebral artery dissection (VAD). A 54-year-old woman presented with neck pain and neurological deficit following sudden neck movement. Radiological findings suggested cervical cord infarction in the anterior spinal artery territory at the C5-C6 vertebral level and dissection of the intracranial segment of the right vertebral artery. Cervical cord infarction due to VAD is usually caused by dissection of its extracranial segment. The present case indicates that dissection of the intracranial segment of the vertebral artery can also cause cervical cord infarction.

Key words: vertebral artery dissection, cervical cord infarction, anterior spinal artery

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Introduction

Vertebral artery dissection (VAD) is a well-recognized cause of ischemia in the vertebro-basilar artery circulation and presents with occipital headache and/or neck pain (1). Recently, cervical cord infarction, although uncommon, has been increasingly described as a complication of VAD (2). In most cases of cervical cord infarction due to VAD reported to date, dissection of the vertebral artery is detected extracranially.

We herein report a case of cervical cord infarction following dissection of the intracranial segment of the vertebral artery.

Case Report

A 54-year-old woman with a history of untreated hypertension developed severe neck pain immediately after she raised her head to look up on the shelf. This was followed by shooting pain from the shoulders to forearms bilaterally, and she was unable to raise her upper limbs for about 30 minutes, with gradual improvement thereafter. Two days later, she noticed a decreased sensation in the left upper limb, and at the same time, a tingling sensation appeared below the umbilical level. She was referred to our hospital seven days after the clinical onset.

Her blood pressure was 160/80 mmHg, and a general physical examination revealed no other abnormalities. On a neurological examination, no abnormality was found in the cranial nerve territory. No muscle atrophy or weakness was found in the limbs. In addition to the tingling sensation below the umbilical level, her temperature sensation decreased in the left upper limb, corresponding to the C7-Th1 dermatomes, but her vibration and joint position sensation were intact. No apparent decrement in the tactile sensation was found. The deep tendon reflexes were normal in all limbs, and Babinski and Chaddock signs were negative bilaterally. There was no bladder or rectal disturbance.

Blood chemistry was unremarkable except for hyperlipidemia. T2-weighted magnetic resonance imaging (MRI) of the cervical cord revealed an intramedullary hyperintense region at the C5-C6 vertebral level. On axial T2-weighted imaging, the hyperintensity was located mainly in the gray matter, sparing the peripheral part of the spinal cord. Diffusion-weighted image showed hyperintensity in the same area (Fig. 1). Cranial MRI was unremarkable. MR angiography (MRA) revealed aneurysmal dilatation of the in-

Department of Neurology, Otsu Red-Cross Hospital, Japan
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Correspondence to Dr. Hidefumi Suzuki, hidesuzuki13@gmail.com

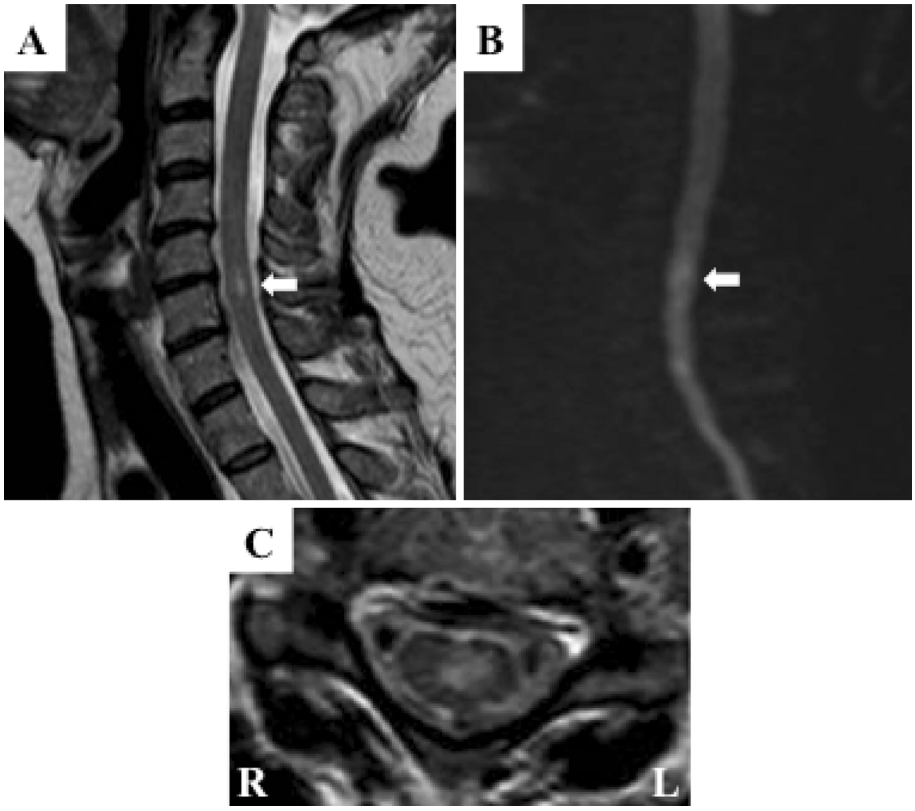

Figure 1. **Magnetic resonance imaging (MRI) of the cervical cord. Sagittal T2-weighted image 14 days after the clinical onset showed intramedullary hyperintensity at the C5-C6 vertebral level (arrow) (A). Axial T2-weighted imaging at the C6 vertebral level showed intramedullary asymmetric hyperintensity, mainly located in the gray matter and sparing the peripheral part of the spinal cord. The involved area corresponded to the anterior spinal artery (ASA) territory (C). The lesion identified in the panel A showed hyperintensity (arrow) on diffusion-weighted imaging at 15 days after the clinical onset, a finding compatible with acute spinal cord infarction (B).**

tracranial segment of the right vertebral artery. No abnormality was found in the extracranial portion of the vertebral artery (Fig. 2).

The patient was not treated with antithrombotic agents in order to prevent possible subarachnoid hemorrhaging due to rupture of the dissected arterial wall. Six months later, her sensory disturbance had almost completely recovered, and a cranial MRA study showed that the aneurysmal dilatation of the right vertebral artery remained unchanged.

Discussion

In the present case, neck pain and symptoms of the upper limbs were induced by sudden neck movement, suggesting VAD as a probable cause of cervical cord infarction. Based on the clinical presentation and the neurological and radiological findings, a diagnosis of inferior cervical cord infarction in the anterior spinal artery (ASA) territory due to right VAD was made.

Cervical cord infarction is uncommon but has been increasingly described as a complication of VAD and likewise ischemia in the vertebro-basilar artery territory (2). To our knowledge, 21 cases presenting with cervical cord infarction due to VAD have been reported in the literature (2-6). The ASA territory was affected in 15 cases, while the posterior spinal artery (PSA) territory was involved in 6 cases. With regard to the part of the vertebral artery affected by the dissection, the extracranial vertebral artery was involved in most cases. The present case is rare because only one reported case manifested “man-in-the-barrel syndrome” as a result of the ASA infarction due to dissection of the intracranial segment (6).

Regarding the axial MRI findings primarily affecting the gray matter, the ASA infarction in the present case is postulated to have been caused by hemodynamic pathogenesis. The intrinsic blood supply of the spinal cord is divided into two arterial systems: the central and the peripheral circulations (7). The central circulation, composed of the sulcal arteries originating from the ASA, supplies most of the gray matter and the adjacent white matter, whereas the peripheral circulation of the penetrating branches from the pial arterial plexus supplies the white matter. There exists a watershed area between the two arterial systems in consequence of their insufficient anastomoses. Hypoperfusion of the ASA territory results in heterogeneous patterns of infarction involving the gray matter and the watershed area. It is hy-

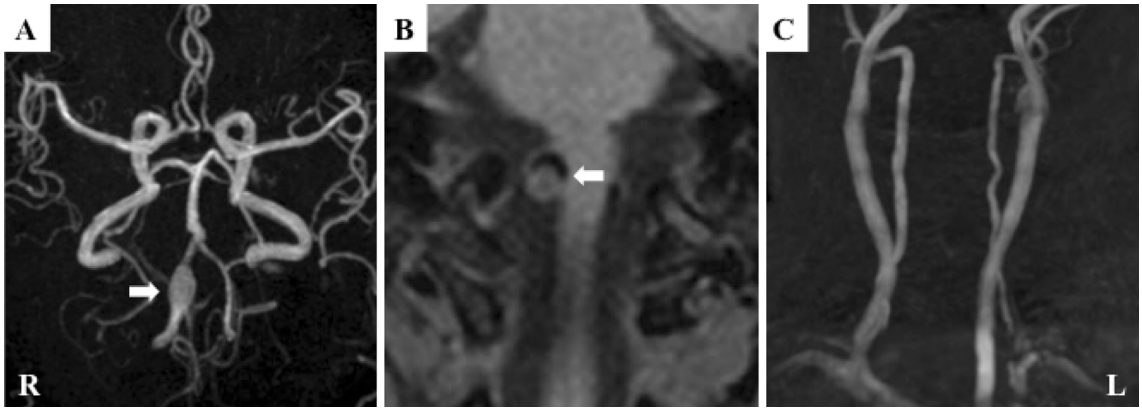

Figure 2. **Cranial and cervical magnetic resonance angiography (MRA) and MRI. Cranial MRA 15 days after the clinical onset revealed aneurysmal dilatation of the intracranial segment of the right vertebral artery (arrow) (A). Intramural hematoma was detected in the wall of the affected artery (arrow) on T1-weighted imaging 35 days after the clinical onset (B). These findings suggest vertebral artery dissection. On cervical MRA 35 days after the clinical onset, no abnormality was found in the extracranial portion of the vertebral artery (C).**

pothesized that the gray matter is affected because of its selective vulnerability to ischemia, while the watershed area is affected because of its intrinsic vulnerability to low blood perfusion (8). In some cases of hemodynamically induced infarction in the ASA territory, characteristic MRI findings of a "snake-eyes" appearance are demonstrated on axial T2-weighted imaging, reflective of the vulnerability of the bilateral anterior horns located in the watershed area (9, 10).

The ASA, originating from the intracranial vertebral arteries, descends in the median anterior fissure receiving the blood supply from radicular arteries. Several different arteries forming anastomosis to each other participate in the vascularization of the cervical cord (11). Generally, the superior cervical segments are supplied by the intracranial vertebral arteries, and the middle and the inferior cervical segments are supplied by the radicular arteries arising from the extracranial vertebral arteries and the costocervical trunks, respectively. In addition, the occipital, deep cervical and ascending cervical arteries are also the feeders of the cervical cord. Considering the anatomical situation, the intracranial VAD is expected to be less likely to cause inferior cervical cord infarction. Although the intracranial vertebral arteries rarely participate in perfusion below the 4th cervical segment (11), the radicular arteries vary in the size, number and location among individuals (7). It is plausible to postulate that, in the present case, the blood supply from the extracranial vertebral arteries and the costocervical trunks via radicular arteries might have been insufficient, causing the intracranial vertebral arteries to supply the inferior cervical segments.

The transient weakness of the upper limbs following neck pain might have been caused by transient ischemic attack in the anterior horns of the middle cervical cord. The sensory deficit of the left upper limb and tingling sensation below the umbilical level, which occurred two days later, are postulated to have been caused by an ischemic lesion in the left posterior horn and bilateral spinothalamic tracts, respectively, at the inferior cervical level.

In conclusion, the intracranial vertebral artery predominantly supplies the superior cervical segments but it may also supply the inferior cervical segments in some cases.

The authors state that they have no Conflict of Interest (COI).

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