

Ischemic Cervical Myelopathy Caused by Vertebral Artery Dissection

The Clinical Utility of a Motor-evoked Potential Study

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Introduction: Vertebral artery dissection (VAD) has been increasingly recognized as one of the important causes of ischemic stroke especially in young and middle-aged population. Rarely, VAD can involve the spinal cord, causing ischemic cervical myelopathy.

Case Report: A 51-year-old man presented with sudden onset of weakness and hypesthesia involving right upper extremity, accompanied by posterior neck pain and headache. He also complained nonwhirling type of dizziness and nausea and he exhibited ataxic gait. Brain magnetic resonance imaging showed acute infarction at the right cerebellar hemisphere and temporo-occipital lobe. Right VAD was identified by MR angiography and confirmed by transfemoral cerebral angiography. Because the selective proximal weakness of the right upper extremity and hypesthesia at C5 and C6 dermatomes was not be fully explained by the brain lesion, electrophysiological studies were performed. The motor-evoked potential studies revealed that the latency and central motor conduction time was prolonged in the right cervical and lumbosacral roots. These studies implied cervical myelopathy involving the right anterior column. Magnetic resonance imaging of the cervical spine demonstrated high signal intensity of the right anterior column at C3-C4 level, which confirmed cervical ischemic myelopathy.

Conclusions: When there is diagnostic ambiguity between the upper and lower motor neuron lesions in VAD, motor-evoked potential study can be helpful to diagnose peripheral neurological complication of VAD.

Key Words: central motor conduction time, motor-evoked potentials, spinal cord ischemia, stroke, vertebral artery dissection

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Vertebral artery dissection (VAD) is a rare cause of ischemic stroke with an estimated incidence at 1 to 1.5 per 100,000.¹ Even though this accounts for only about 2% of all ischemic stroke, it has been considered as one of the important causes of ischemic stroke especially in young and middle-aged population occupying 10% to 25% of such cases.² The VAD usually results in posterior circulation ischemia, which is

closely associated with clinical manifestations. In rare cases, however, peripheral neurological complications can be developed due to VAD, involving the spinal cord or nerve roots, which cause ischemic cervical myelopathy (ICM) or radiculopathy. In the previous study, only 1 case of ICM was reported among 111 patients with VAD.³ Because diagnosis of this disorder is challenged due to low incidence and atypical presentation, it is critical to precede clinical suspicion and establish proper diagnostic plan. Herein, we report a case of ICM caused by VAD with the clinical, electrophysiological, and radiologic features and show the clinical utility of the motor-evoked potentials (MEP).

CASE REPORT

A 51-year-old man presented to our emergency department with a sudden onset of weakness and numbness in the right upper extremity, accompanied by posterior neck pain and headache. He demonstrated an ataxic gait and also complained nonwhirling type dizziness, nausea, and dysarthria. There was weakness of Medical Research Council grade 2 in the right shoulder abductor and flexor, and of grade 3 in the right elbow flexor. Light touch sensation was diminished for right C5 and C6 dermatomes. The deep tendon reflex was decreased for the right biceps brachii. In contrast to the proximal weakness of the right arm, fine movement of the right hand was preserved. Brain magnetic

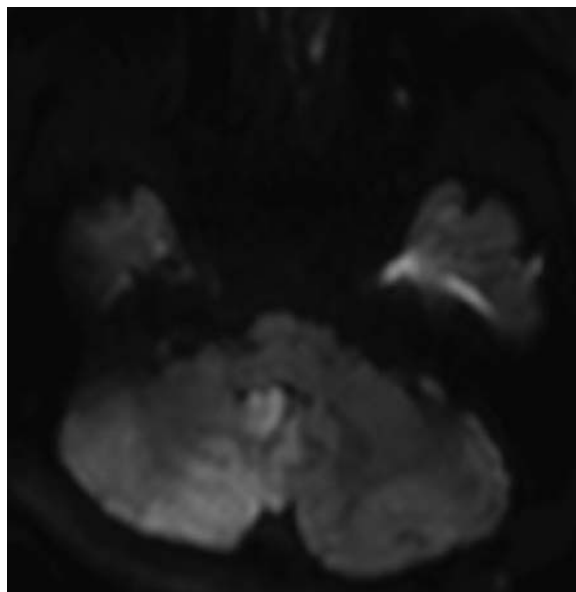


FIGURE 1. High signal intensities in the right cerebellar hemisphere and the temporo-occipital lobe were demonstrated in diffusion-weighted magnetic resonance imaging.

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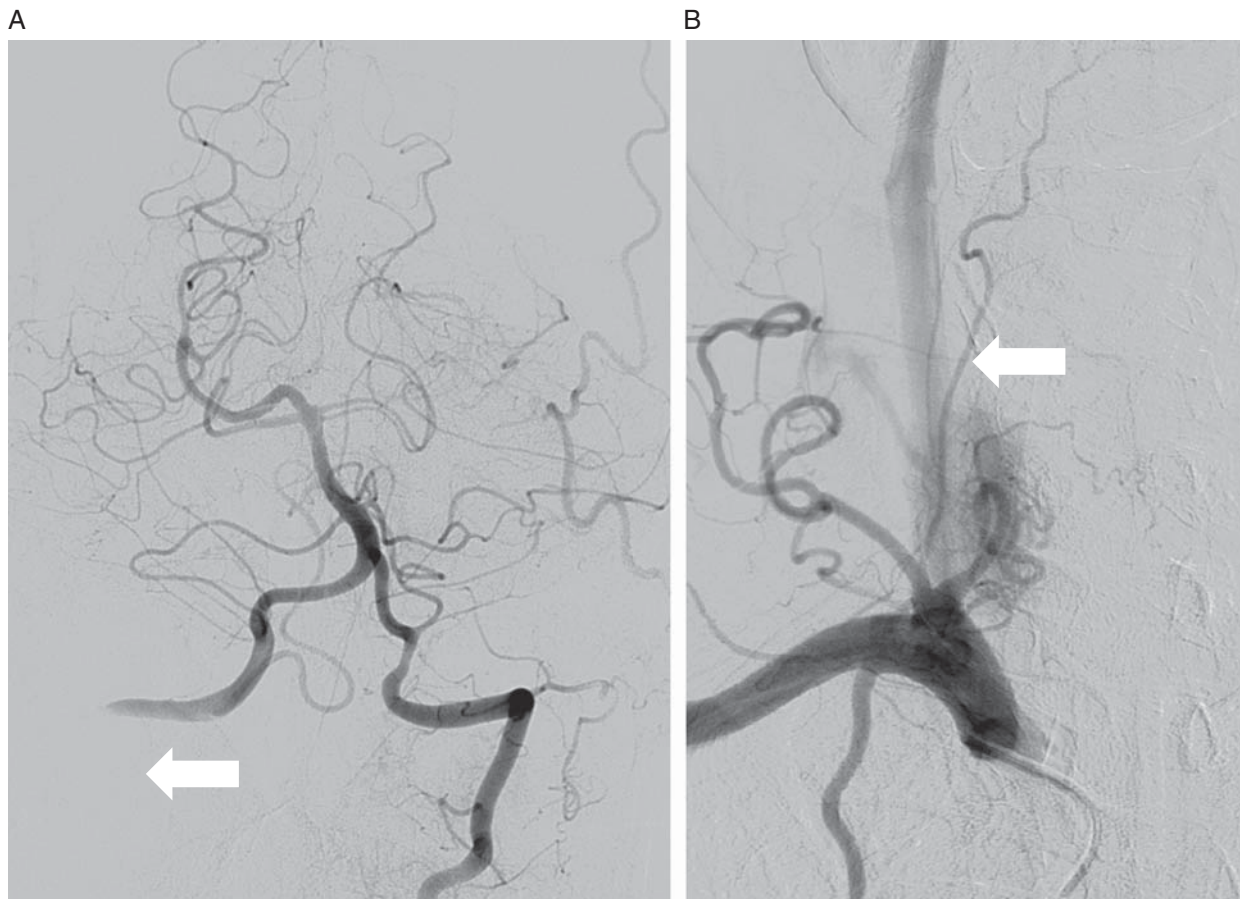


FIGURE 2. Segmental occlusion from the right vertebral artery in transfemoral cerebral angiography. A, White arrow indicated that tapered occlusion, flow stagnation, and delayed contrast clearing in the right V3-V4 segment which is visualized by retrograde flow from the left vertebral artery. B, No visualization (white arrow) of the right vertebral artery implies the occlusion from the orifice in the right subclavian arteriography.

resonance imaging (MRI) on the day of presentation demonstrated evidence of acute infarction in the right cerebellar hemisphere and temporo-occipital lobe (Fig. 1). The right vertebral artery was not visualized on brain MR angiography. Transfemoral cerebral angiography confirmed segmental occlusion from the right vertebral artery orifice to the right proximal V3 (Figs. 2A, B).

Two weeks after the onset, the Brunnstrom stage was 3 for the right arm, in contrast to 6 for the right hand and lower extremity. The Fugl-Meyer motor scale of the right upper extremity was measured as 59 of 66 from the proximal weakness. This selective proximal weakness of the right upper extremity and hypesthesia at C5 and C6 dermatomes could not be fully explained by the brain lesion. Therefore,

we performed electrophysiological studies, including nerve conduction study, needle electromyography, MEP, and somatosensory-evoked potential (SEP) studies to differentiate cervical radiculopathy, myelopathy, and brachial plexopathy. Nerve conduction study showed decreased amplitudes of compound muscle action potential in the right axillary nerve (5.7 mV compared with 11.8 mV in the left axillary nerve). However, there was no abnormal spontaneous activity in the muscles innervated by the right C5 and C6 nerve roots. The MEP studies revealed that the latency and central motor conduction time (CMCT) was increased in the right cervical and lumbosacral roots without increased SEP latency in the right side (Table 1). These results implicated cervical myelopathy involving right anterior column with

TABLE 1. Characteristics of Central Motor Conduction Time Which is Calculated Using Latencies in Motor-evoked Potential, F-Wave, and Compound Muscle Action Potential in the Abductor Pollicis Brevis and Abductor Hallucis

Sites	MEP Latency (ms)	F-Wave Latency (ms)	CMAP Latency (ms)	CMCT (ms)
APB				
Right	26.10	26.40	3.4	11.70
Left	21.75	25.40	3.05	8.03
AH				
Right	43.90	51.00	3.9	16.95
Left	39.50	52.95	4.15	11.45

AH indicates abductor hallucis; APB, abductor pollicis brevis; CMAP, compound muscle action potential; CMCT, central motor conduction time; MEP, motor-evoked potential.

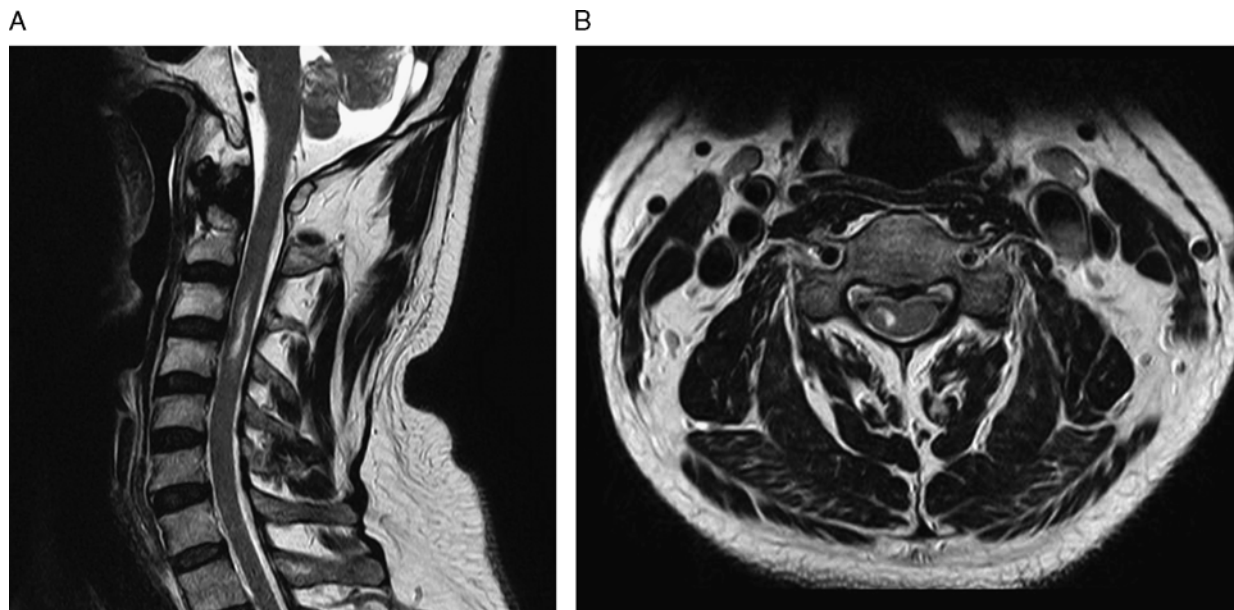


FIGURE 3. High signal intensity of the right anterior column at C3-C4 level confirmed cervical ischemic myelopathy on sagittal (A) and axial images (B) in T2-weighted magnetic resonance imaging.

the preservation of the dorsal column-medial lemniscal pathway. MRI of the cervical spine after the electrophysiological studies demonstrated high signal intensity of the right anterior column at C3-C4 level on T2-weighted images, which confirmed ICM (Figs. 3A, B).

At 3 months, the motor strength of the patient partially improved to Medical Research Council grade 3 in the right shoulder abductor and flexor, and to grade 4 in the elbow flexor.

DISCUSSION

The accurate diagnosis of stroke is essential to manage the patient and make the strategy for rehabilitation. Particularly, ICM caused by VAD, which is uncommon and atypical neurological presentation, deserves more attention from clinicians. In this case, the patient had symptoms which cannot be fully explained by a posterior circulation territory lesion. Accompanied neurological complications such as upper (cervical myelopathy) or lower motor neuron lesion (cervical radiculopathy and brachial plexopathy) should be considered as differential diagnoses.⁴ The ICM was diagnosed by MEP study with prolongation of the latency and CMCT at the affected side compared with the unaffected side, which indicated the impaired central motor conduction in corticospinal tract.

The MEP and SEP studies reflect functional integrity of corticospinal pathway and that of dorsal column-medial lemniscal pathway, respectively.^{5,6} Especially, CMCT, calculated by subtracting the peripheral conduction time from the latency of MEP, can be prolonged in myelopathy and is a valuable parameter to diagnose and localize spinal cord lesions of various causes.⁷ Moreover, CMCT in MEP study is known to be more sensitive than signal change in MRI for identifying subtle abnormality in the corticospinal tract.⁸ In addition, MEP

is even more responsive to the spinal cord ischemia than SEP, because the MEP can detect the lesion of gray matter of spinal cord, which is more vulnerable to ischemic damage.⁶

In conclusion, when peripheral neurological complication of VAD is clinically suspected along with the presence of diagnostic ambiguity between the upper and lower motor neuron lesions, MEP study including CMCT is suggested as a valuable diagnostic tool.

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