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LETTER TO THE EDITOR

Combined Cervical Cord and Bilateral Cerebellar Infarction Report

The spinal cord is supplied by one anterior and two posterior spinal arteries, and by the radicular arteries that enter the canal along with the nerve roots.[1] The possible causes of spinal cord infarction (SCI) include many possible factors such as aortic aneurysm repair, traumatic aortic rupture, arteriovenous malformation, transient ischemic attack, and arteritis secondary to diabetes mellitus, or cryptogenic.[2] Radicular arterial infarctions result in bilateral anterior or posterior spinal artery infarctions and unilateral infarctions. By contrast, spinal cord hypoperfusion causes transverse or central cord infarctions in the thoracolumbar region.[3] The lesion of infarction can be seen on magnetic resonance imaging (MRI), while diffusion weighted (DW) imaging is needed for the early detection of SCI.[4] Combined cerebellar and spinal ischemic stroke is extremely rare. We report a case with combined cervical spinal cord and bilateral cerebellar infarction, which was proved by DW-MRI.

A 49-year-old man with history of congenital paramyotonia, traumatic intracerebral hemorrhage with left hemiparesis and diabetes mellitus was admitted because of sudden onset of quadriparesis and urinary retention with preceding back pain after lifting heavy objects. Neurological examinations revealed asymmetric quadriparesis (muscle strength: right side, 3–4/5; left side, 2–3/5), hypoesthesia below the C8 level with impaired proprioception, which was worse on the left, and hyporeflexia with up-going Babinski's sign on the left, which might be related to previous brain

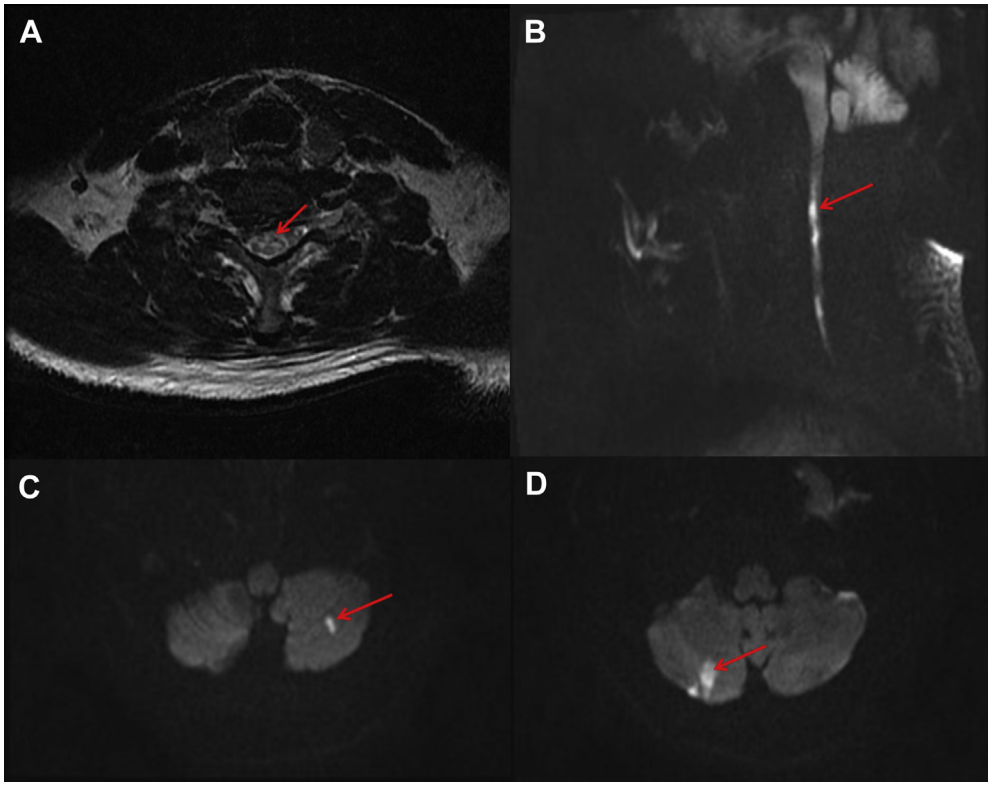

Figure 1 Axial and sagittal findings of magnetic resonance imaging (MRI). (A) Axial MRI of the cervical spine shows the "owl's eyes" sign on T2-weighted image which is compatible with anterior spinal artery infarctions (arrow). (B) Sagittal diffusion weighted (DW)-MRI of the cervical spine shows hyperintense signals at C3–C7, suggesting recent infarction (arrow). (C) Axial DW-MRI of the posterior fossa shows hyperintense signals at the left cerebellar hemisphere, suggesting recent infarction (arrow). (D) Axial DW-MRI of the posterior fossa shows hyperintense signals at the right cerebellar hemisphere, suggesting recent infarction (arrow).

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insult. Cervical spine MRI revealed longitudinal high signal intensity at C3–C7 with minor herniated intervertebral disc at C3–4, C4–5, C5–6 and "owl's eyes" sign on T2-weighted image (Figure 1A), suggesting a spinal cord infarction. Brain and cervical DW-MRI for further survey of possible lesion showed hyperintense signals at C3–C7 (Figure 1B) and bilateral cerebellar hemisphere (Figure 1C and D) which were compatible with recent infarction. The cerebrospinal fluid study showed mild elevated protein (68 mg/dL) and glucose (169 mg/dL, compared with serum: 364 mg/dL) or pleocytosis. Extracranial carotid duplex sonography revealed no abnormal findings and the 24-hour Holter electrocardiogram reported no significant arrhythmia. Echocardiography showed no evidence of intramural thrombus.

The patient received aspirin for secondary stroke prevention. His blood sugar was controlled below 200 mg/dL. During hospitalization, hypotension and mild chest discomfort with excessive sweating had been noted. There was no laboratory or electrocardiogram evidence of myocardial ischemia. Computed tomography angiography showed no vascular abnormalities. Reduced R-R interval variability and absent sympathetic skin response supported the evidence of autonomic dysfunction associated with spinal cord infarction. With continuous daily rehabilitation, the patient had improved bilateral muscle strength and became able to stand with the support of a four-limb walker although poor balance was noted 2 months later. The Foley catheter was not removed considering the poor sensation and function of his urinary bladder. There was no definite cause determined in this patient, although the diabetes mellitus with poor control, minor intervertebral disc herniation around the affected level of spinal cord, and preceding heavy lifting were possible precipitating factors for this event. SCI caused by occlusion of spinal arteries and veins by fibrocartilaginous emboli may develop after spine movement.[1] Stenoses of vertebral artery on the ipsilateral side of infarctions were revealed in the cases of combined cerebellar and spinal cord infarction which were reported previously.[5] We performed computed tomography angiography as an alternative examination, which revealed no vascular abnormalities. Clinically, our patient presented with the most common anterior spinal artery syndrome, including muscle weakness, back pain, areflexia, spinothalamic sensory loss, and autonomic dysfunction.[2]

Our MRI results showed "owl's eyes" sign, which is commonly seen in anterior spinal artery infarctions, suggesting involvement of the anterior gray matter.[3] The lesions of the spinal cord in our patient could be confirmed as acute infarction based on the simultaneous involvement of bilateral cerebellum on DW-MRI. The short-term outcome depends on the degree of the initial neurological deficits, especially motor deficits.[1] Therefore, better prognosis could be expected in our case because of the relatively preserved muscle strength in the beginning. In addition to spinothalamic sensory deficit, proprioceptive impairment is usually explained by involvement of the most inner part of the dorsal columns that belongs to the central territory of the anterior spinal artery.[6]

References

1. Kumral E, Polat F, Güllüoglu H, Uzunköprü C, Tuncel R, Alpaydın S. Spinal ischaemic stroke: clinical and radiological findings and short-term outcome. *Eur J Neurol* 2011;**18**:232–9.
2. Millichap JJ, Sy BT, Leacock RO. Spinal cord infarction with multiple etiologic factors. *Soc Gen Intern Med* 2007;**22**:151–4.
3. Cheng MY, Lyu RK, Chang YJ, Chen RS, Huang CC, Wu T, Lee TH, et al. Spinal cord infarction in Chinese patients. *Cerebrovasc Dis* 2008;**26**:502–8.
4. Thurnher MM, Bammer R. Diffusion-weighted MR imaging (DWI) in spinal cord ischemia. *Neuroradiology* 2006;**48**:795–801.
5. Reich P, Müller-Schunk S, Liebetrau M, Scheuerer W, Brückmann H, Hamann GF. Combined cerebellar and bilateral cervical posterior spinal artery stroke demonstrated on MRI. *Cerebrovasc Dis* 2003;**15**:143–7.
6. Chen NC, Yeh CH, Hui CT, Lin RH. Exercise related spinal cord infarction: a case report. *Acta Neurol Taiwan* 2010;**19**:194–8.

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