

A Case of Posterior Spinal Artery Syndrome in the Cervical Cord: A Review of the Clinicoradiological Literature

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Abstract

We describe a patient with posterior spinal artery (PSA) syndrome due to vertebral artery (VA) dissection. A 63-year-old woman developed neck pain, bilateral shoulder and arm numbness, and paraparesis after prolonged neck extension during a dental procedure. Neurological examination revealed sensory deficits in the legs, paraparesis, cerebellar ataxia, urinary retention and constipation. Magnetic resonance imaging disclosed T2-hyperintense lesions in the posterolateral C4-C7 cord with partial enhancement. T1-hyperintensity and stenosis were found in the right VA at C3-C5. These clinicoradiological findings suggested bilateral PSA syndrome and unilateral VA dissection. This is the fourth report of VA dissection-induced PSA syndrome.

Key words: posterior spinal artery syndrome, cervical cord, vertebral artery dissection, neck extension, MRI

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Introduction

Spinal cord infarction is an infrequent disease with heterogeneous etiologies such as neck trauma, atherosclerosis and vertebral artery (VA) dissection (1). Anterior spinal artery (ASA) syndrome commonly occurs in spinal cord infarction. Posterior spinal artery (PSA) syndrome is very rare (1-4). PSA syndrome exhibits a variety of neurological symptoms and signs due to the lesion topography in the posterior or posterolateral region of the spinal cord, including the posterior column, posterior horn and lateral column (2-19). PSA syndrome has been reported in the thoracolumbar cord (2-7, 11, 13, 14, 16-19). This syndrome is extremely rare in the cervical cord (8-10, 12, 15). We herein describe a patient with bilateral PSA syndrome in the cervical cord triggered by unilateral VA dissection and review previous clinicoradiological literature of cervical PSA syndrome.

Case Report

A 63-year-old woman noticed neck pain, bilateral shoulder and arm numbness, and muscle weakness in the legs im-

mediately after she had maintained neck extension in the sitting position for more than 1 hour during a dental procedure. Because motor and sensory deficits in the lower extremities persisted for 2 weeks, she visited our department. She had a history of hypertensive medication, currently smoked (20 cigarettes/day for 40 years) and drank alcohol (ethanol 45 g/day). Physical examination showed blood pressure of 144/88 mm Hg. On neurological examination, cognitive function and cranial nerves were normal. A mild to moderate degree of weakness existed in the lower extremities (Medical Research Council grade 3-4). Muscle stretch reflexes were increased in the lower extremities, and there were bilateral extensor plantar responses. Paresthesia was found in the left arm and all sensation was decreased below the Th10 cord level. There was marked reduction of vibration and proprioception sensation. Romberg's sign was present. The patient's gait was spastic, wide-based, and ataxic. She had marked urinary retention and constipation. Routine laboratory studies were within normal ranges. Serological studies of the autoimmune system, including anti-aquaporin-4 (AQP4) antibody, were normal. Pathogen tests for bacteria, syphilis and viruses were negative. Chest X-ray, electrocardiography, and carotid ultrasonography were not remarkable. A cerebrospinal fluid study exhibited 35 mg/dL

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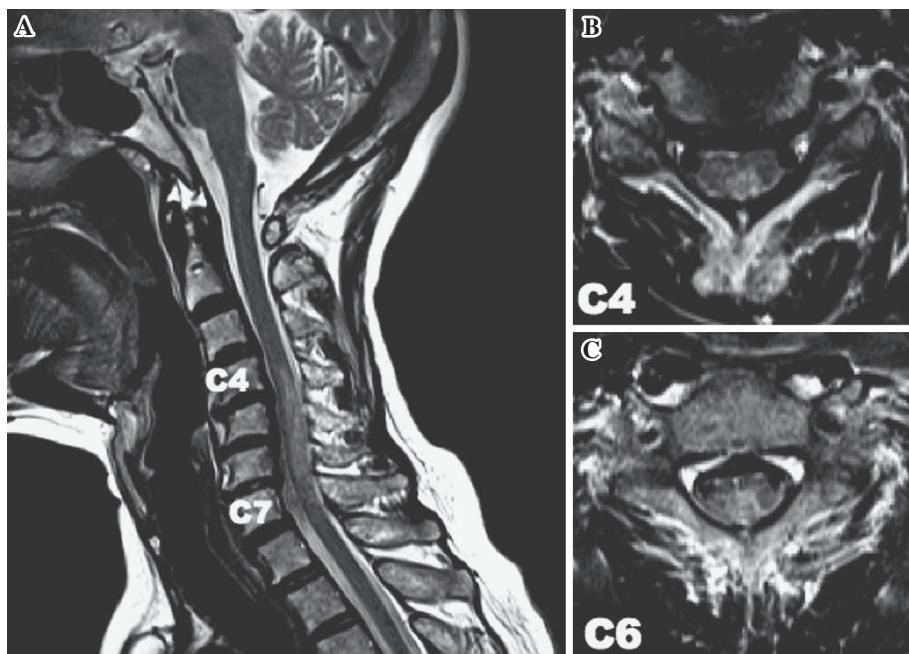


Figure 1. T2-weighted imaging at 14 days after clinical onset. (A) Sagittal view showed widespread hyperintense lesions at the C4-C7 cord level. (B, C) Axial view showed hyperintense lesions in bilateral posterior C4 cord and posterolateral C6 cord.

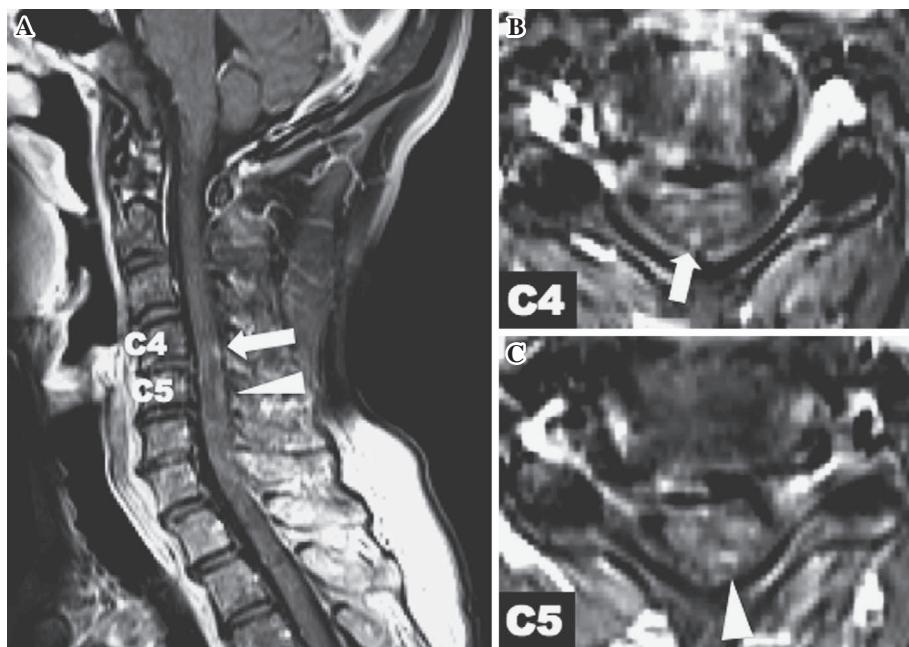


Figure 2. Gadolinium-enhanced T1-weighted imaging. (A) Sagittal and (B, C) axial views. Partial enhancement was found in the posterior regions of the C4 (arrows) and C5 cord (arrowheads).

of protein, 2 mononuclear cells/mm³ and normal cytology. Myelin basic protein and oligoclonal immunoglobulin G band were not detected. Motor and sensory nerve conduction studies were normal in the four extremities. P100 latencies were normal on visual evoked potential examination. On sensory evoked potential testing using peroneal nerve stimulation, N12 latencies were normal, but P38 latencies were prolonged on both sides. At 14 days from clinical onset, T2-weighted imaging revealed hyperintense lesion in bi-

lateral posterior or posterolateral regions from the C4-C7 cord (Fig. 1). Gadolinium-enhanced T1-weighted imaging displayed partial enhancement in the posterior regions of the C4-C5 cord (Fig. 2). Axial T1-weighted imaging revealed hyperintense wall and stenotic changes in the right VA at the C3-C5 cord level (Fig. 3). Brain and thoracolumbar cord magnetic resonance images (MRI) were normal. Brain and neck magnetic resonance angiography showed thin and tortuous changes in the right VA. She refused to undergo con-



Figure 3. T1-weighted imaging showed hyperintense wall (arrowheads) and stenosis in the right VA at the C3-C5 cord level.

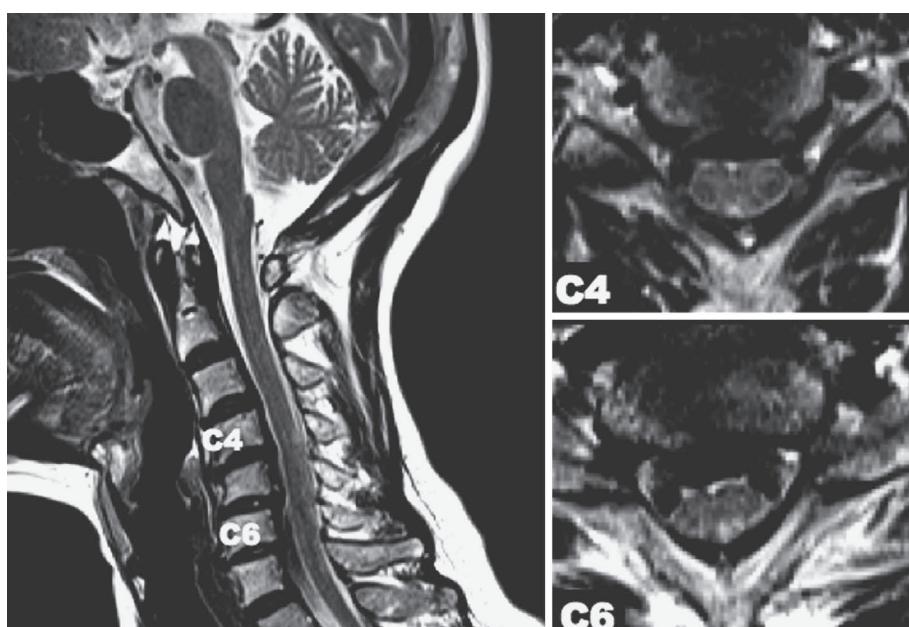


Figure 4. T2-weighted imaging at 3 months after clinical onset. Hyperintense lesions were markedly attenuated in the posterior regions of the C4-C6 cord.

ventional cervical angiography. We diagnosed her with bilateral PSA syndrome due to right VA dissection. She was treated with clopidogrel (75 mg/day, po). Three months later, T2-hypersintense lesions were markedly attenuated and limited in the posterior regions of the C4-C6 cord (Fig. 4). Abnormal enhancement disappeared. Her leg weakness, cerebellar ataxia and autonomic dysfunction were moderately ameliorated. Her spastic gait and deep sensory deficits persisted at 4 months after clinical onset.

Discussion

We reported the clinicoradiological hallmarks in a patient with posterior C4-C7 infarction caused by unilateral VA dissection.

Previous reports of cervical PSA syndrome are summarized in Table 1. Based on anatomic connection between the PSA and VA, VA dissection or occlusion could play a crucial role in the pathogenesis of posterior cervical cord infarction. Cervical manuature, neck exercise and trauma are reported as possible etiologies of VA dissection and occlusion. A large study of cervical artery dissection reported that

the frequency of cervical trauma in the previous month was approximately 40% in patients with VA dissection (20). The present and two previous cases of cervical PSA syndrome suggested that sustainment of neck stress and abnormal neck movements were common etiological factors for VA dissection-related PSA syndrome (8, 10).

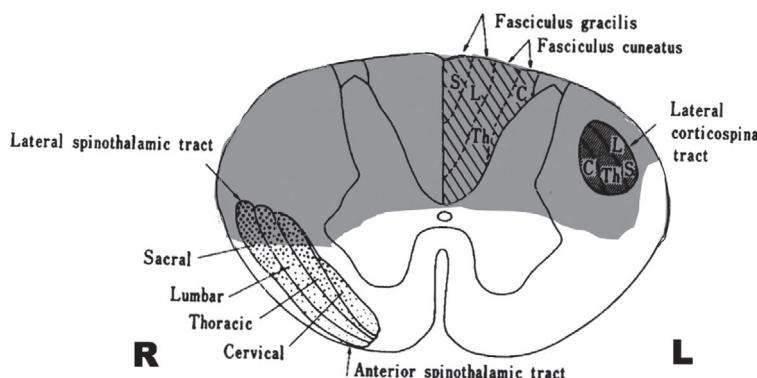
The blood circulation of the spinal cord is supplied from ASA and PSA. ASA supplies the anterior two-thirds of the spinal cord, and paired PSAs supply the posterior third. There are rich anastomotic networks of direct penetrating vessels and a plexus of pial vessels fed by both PSAs (21). These arterial structures might contribute to low frequency of PSA syndrome compared with ASA syndrome. PSA originates as branches and the radiculopial arteries from the proximal intradural VA. This artery supplies the posterior column, dorsal horn, lateral column and a part of anterior column where the corticospinal, spinocerebellar and lateral spinothalamic tracts are localized. This vascular anatomy accounts for profound proprioceptive deficits and variable degrees of motor deficits, superficial sensory loss and cerebellar ataxia in patients with PSA syndrome. In fact, in terms of the neurological features of cervical PSA syndrome, py-

Table 1. Previous Clinicoradiological Literature of PSA Syndrome in the Cervical Cord

Authors (*) reported years	Age/sex	Etiology	Trigger factor	Lesion topography	Time interval**	Gd-enhancement	Neurological deficits
Pryse-Phillips (8) 1989	32 years/woman	Left VA dissection	Neck rotation	Left upper cervical cord, left lower medulla oblongata	No MRI		Sensorimotor
Gutowski et al (9) 1992	35 years/man	Left VA dissection suspect	Sneeze	Left C1-C3 cord	8 days	Peripheral	Sensorimotor, bladder
Fukuda et al (10) 1994	62 years/man	Right VA occlusion	Neck flexion	Right C1-C2 cord, right cerebellar tonsil	7 days	Slight	Sensorimotor, ataxia, Horner's sign
Bergqvist et al (12) 1997	19 years/man	Right VA dissection	Neck extension	Bilateral C2-C3 cord	12 days	Marked	Sensorimotor, ataxia
Masson et al (15) 2002	48 years/man	Atherosclerotic occlusion		Left C2-C3 cord	9 months	Absence	Sensorimotor
Present case	63 years/woman	Right VA dissection	Neck extension	Bilateral C4-C7 cord	14 days	Partial	Sensorimotor, ataxia, bladder, bowel

PSA: posterior spinal artery; VA: vertebral artery; MRI: magnetic resonance imaging; Gd: gadolinium.

*Reference number. **Time intervals between clinical onset and the first MRI.

**Figure 5.** A possible mechanism for neurological deficits and somatic topography in the present patient. Cervical cord cross-section illustrating the posterior fasciculus, spinothalamic and corticospinal tracts. The gray zone indicates infarctive and ischemic territories at the C6 cord level.

ramidal tract sign, cerebellar ataxia or superficial hypesthesia were always combined with deep hypesthesia in the present and previous cases (8-10, 12, 15).

The present patient may have defective development of the vascular network at the posterior C4-C7 cord level, in addition to occlusion of the single dominant PSA branching from the right VA wall dissection and stenosis. The variant vasculature of cervical PSA and the surrounding anastomosis could contribute to bilateral PSA syndrome due to the right VA dissection in the present patient. Moreover, somatic topography of sensory and motor deficits was found below the thoracic segment rather than the cervical cord segment. One possible mechanism for the distribution of sensorimotor deficits found in the present patient is as follows (Fig. 5). MRI lesions were present in the right-dominant posterior column and dorsal lateral column of the C6 cord. The anterior horn was preserved. The cervical segment of the spinothalamic tract was localized in the anteromedial region whereas the thoracolumbar segments were distributed in the lateral region. Therefore, the anatomic topography seemed to have caused hypesthesia below the Th10 cord level and left arm paresthesia without weakness in the present patient. The distinct anatomic background may lead to the misdiagnosis or delayed diagnosis of cervical PSA syndrome. With respect to differential diagnoses of posterior spinal cord infarction, acute demyelinating diseases, myelitis and tumors should have been considered in the present patient. Follow-

up MRI denied spinal cord tumors. Longitudinal widespread spinal lesions are detected on MRI in patients with multiple sclerosis, neuromyelitis optica and acute myelitis. Brain MRI and P100 latencies were normal and serum anti-AQP4 antibody was negative. Infectious and autoimmune myelitis was excluded by negative herpes virus, syphilis, and autoimmune antibody results in the present patient. The sudden onset after prolonged neck extension, cardiovascular risk profile, and radiological findings of VA dissection strongly supported the diagnosis of posterior cervical cord infarction.

In conclusion, we highlighted the clinicoradiological findings in a patient with VA dissection-related PSA syndrome. The present patient indicated variable neurological symptoms and signs, including pyramidal weakness, autonomic dysfunction and superficial hypesthesia at the thoracolumbar cord levels, in addition to remarkable deep hypesthesia. Thus, physicians should pay close attention to the diverse neurological features in this syndrome.

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

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