Subclavian Artery Dissection and Triple Infarction of the Nervous System

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Background: Subclavian artery dissection is a rare entity. It is usually associated with anomalous aortic vasculature. Only with trauma or catheterization procedures is subclavian artery dissection with normal aortic vasculature reported.

Patient: We describe a patient with intrascapular pain, an occipital headache, and 3 distinct infarctions in the nervous system. He had spontaneous subclavian artery dissection with normal aortic vasculature.

Conclusion: Subclavian artery dissections should be suspected in patients with intrascapular pain, occipital or cervical pain, and symptoms within the posterior circulation.

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SUBCLAVIAN ARTERY DISSECTION (SAD) is rare. When present, SAD is usually associated with anomalies of the aortic arch.1,2 Only with trauma or catheterization procedures is SAD reported with normal aortic vasculature.3,4 The main clinical manifestations of SAD are chest and back pain.1,2,5 In vertebral artery dissection, the most telling symptoms are headache and neck pain, which often precede ischemic symptoms.6,7 Pain and weakness in the arm are also presenting symptoms in either type of dissection. We describe a patient with spontaneous SAD that resulted in infarctions to 3 distinct parts of the nervous system.

REPORT OF A CASE

A 54-year-old man with controlled hypertension for 1 year suddenly developed vertigo and crushing intrascapular pain while at a grocery store. He vomited on his way home. At home, he was unable to lift his arms over his head, so he proceeded to a local hospital. In the emergency department, he complained of left-sided facial numbness, a left-sided occipital headache, left-sided neck pain, and weakness and pain in his upper limbs. There was no history of trauma or neck manipulation. His family history was significant for an abdominal aortic aneurysm in his father. A left-sided cerebellar hypodensity was identified on computed tomography.

On transfer to our institution, he was alert and coherent, although his speech was mildly hypophonic. A cranial nerve examination revealed decreased perception of a pinprick over the left lower jaw. The left-sided deltoid, supraspinatus, and triceps muscles were weak (Medical Research Council classification 3−/5). Muscle stretch reflexes demonstrated depressed left-sided biceps’ and triceps’ reflexes and pendular knee jerks. Plantar responses were extensor. The left upper limb was ataxic. Moderate dysmetria was found in both upper extremities, along with a severely ataxic gait. No bruits were auscultated over the neck or supraclavicular spaces. His radial arteries were symmetric.

During the initial days of hospitalization, the patient complained of paresthesias in both hands and a severe left hemicranium headache. Diffusion-weighted imaging confirmed a large left cerebellar hemispheric infarct and showed a small infarct in the right cerebellar hemisphere (Figure, A). A computed tomographic scan with contrast of the chest demonstrated a subintimal hematoma in the left subclavian artery (Figure, B). A magnetic resonance angiogram of the chest revealed a dissection of the proximal left subclavian artery (Figure, C). An arteriogram of the aortic arch and great vessels showed the dissection of the left subclavian artery distal to the thyrocervical trunk (Figure, D), and demonstrated a subintimal thrombus and occlusion of the left vertebral ar-

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tery. No evidence of fibromuscular dysplasia was noted in any of the vessels. Magnetic resonance imaging of the cervical region showed an intramural thrombus in the left vertebral artery (Figure, E) and a faint hyperintensity in the C4 to C5 segments of the spinal cord (Figure, F).

The results for serum homocysteine level, erythrocyte sedimentation rate, C-reactive protein level, antinuclear antibody titer, and a coagulation panel were within reference ranges. The result of a lipid panel was normal, except for a total cholesterol level of 201 mg/dL (5.20 mmol/L). Rapid plasma reagin was nonreactive. The result of magnetic resonance angiography of the renal arteries and abdominal aorta was normal.

The patient underwent brief anticoagulation with heparin sodium and subsequently received aspirin. Over 1 week, his gait returned to normal. Mild weakness of the left-sided deltoid, triceps, and infraspinatus muscles progressively improved; this symptom was fully resolved at 6 months. The paresthesias of his hands stopped, and all reflexes returned to normal.

Magnetic resonance angiography at 1 month showed normal filling in the left vertebral artery. The left-sided subclavian subintimal thrombus was much smaller. Electromyography 10 weeks after onset disclosed increased insertional activity and positive waves in the left middle to lower cervical muscles; the left-sided triceps, deltoid, infraspinatus, and rhomboid muscles; and the pronator teres, indicating a cervical polyradiculopathy. The results of sensory and motor nerve conduction studies of the median, ulnar, musculocutaneous, and radial nerves were normal.

**COMMENT**

This case is unique because of the spontaneous dissection of a normal subclavian artery and the consequent ischemic effect on 3 parts of the nervous system.

In most reports of SAD in the literature, coexisting vascular anomalies of the aortic arch or other great vessels were found. In one report, proximal SAD and normal aortic vasculature without vertebral artery involvement occurred at the end of a golf game. No neurological symptoms were reported in this patient, and the result of a computed tomographic scan of the brain was normal. Similarly, an elderly patient with hypertension was recently described; this patient presented with subacute thoracic pain and bilateral upper extremity pain. A spontaneous, aberrant, right-sided SAD was found, but no neurological symptoms occurred. In our case report, the subclavian artery dissected into the vertebral artery, resulting in multiple ischemic infarctions. Evidence for this sequence of events is supported by the initial symptom of intrascapular pain and the subsequent neuroimaging test and angiographic findings. Also, the dissection in our patient was spontaneous. There was no evidence of a connective tissue disorder, systemic arteriopathy, or other contributing factors. As with most cases of vertebral artery dissection, our patient had a good outcome.

Three separate parts of the nervous system were involved by the SAD. Intracranially, there was radiological evidence of asymmetric cerebellar infarcts present-

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**Figure.** Diffusion-weighted imaging demonstrates bilateral cerebellar infarcts (arrows) (A); a computed tomographic scan of the chest with contrast demonstrates an intramural hematoma of the left subclavian artery (arrow) (B); a magnetic resonance angiogram of the chest shows a left subclavian artery dissection (arrow) (C); angiography depicts the left subclavian artery dissection (arrow) (D); and using cervical spine magnetic resonance imaging, a T1-weighted axial image shows an intramural hematoma of the left vertebral artery (arrow) (E) and a T2-weighted sagittal image discloses a hyperintensity in the C4 to C5 segment of the spinal cord (arrow) (F). In panel A, the bar denotes 5 cm. L indicates left.
ing with gait ataxia and limb dysmetria. Bilateral abduction paresis and paresthesias in the hands were associated with a high signal intensity in the fourth and fifth cervical segments of the spinal cord. Interestingly, there have been few reports of spinal cord infarcts caused by vertebral artery dissection.\(^9,10\) Last, an ischemic insult occurred to the cervical roots, as evidenced by focal weakness in the left arm and findings of denervation by an electrophysiological study. This multiple rootlet injury was likely the result of ischemia from involvement of radicular arteries by the affected vertebral artery.\(^11,12\)

Patients who present with back or thoracic pain followed by neurological symptoms should be examined for a SAD. Also, in cases of vertebral artery dissection without a clear cause, it may be worthwhile to obtain imaging studies of the subclavian artery. We propose that a triad of intrascapular pain, occipital or cervical pain, and symptoms within the posterior circulation heralds a spontaneous SAD.

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