Anterior spinal artery syndrome presenting as cervical myelopathy in a patient with subclavian steal syndrome

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The anterior spinal artery syndrome (ASAS), described by Preobraschenski in 1908,¹ is characterized by bilateral spastic paraparesis and dissociated sensory deficits involving bilateral loss of temperature and pain sensation but preserved proprioception and touch. The ASAS typically develops acutely, over minutes to hours. While initially linked to infections, in particular syphilis, many other etiologies are now recognized, including aortic atheromatous disease and thromboembolic phenomenon. We report a case of ASAS in a patient with a steal phenomenon secondary to a right-sided aortic arch with interruption of the left subclavian artery (SCA).

A 57-year-old man presented with acute bilateral lower extremity weakness without urinary incontinence. His medical history was notable for diabetes mellitus, hyperlipidemia, syphilis treated 25 years prior, and a ventricular septal defect, repaired during childhood. He reported heavy alcohol and cocaine consumption, last used 10 days before presentation. On admission, paraparesis and decreased sensation to pinprick were noted in both lower extremities, with preserved vibration and proprioception. In a matter of hours, his examination worsened to a dense paraparesis. Osteotendinous reflexes were brisk and symmetric. Babinski sign, crossed adductors reflex, and Hoffman sign were present bilaterally.

MRI was precluded by the presence of metal fragments in one eye. CT myelography showed no cord compression or spinal canal compromise. Laboratory results including vitamin B₁₂ and methylmalonic acid levels, antinuclear antibodies, anti Ro/La, and serologic testing for Lyme disease, human T-cell lymphotropic virus, and HIV were unremarkable. Rapid plasma reagin titer was undetectable, while a positive fluorescent Treponemal antibody–absorption was consistent with a history of prior syphilis infection. CSF was unremarkable (protein 51 mg/dL, glucose 97 mg/dL, immunoglobulin G index 0.5, no oligoclonal bands, and negative Venereal

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Figure Anterior spinal artery steal syndrome concomitant with left subclavian steal syndrome

(A) DSA, right vertebral artery injection, anteroposterior view. The artery of the cervical enlargement (white arrow), coming from the right vertebral artery, is connected to a spinal branch of the left costocervical trunk (black arrow) through an enlarged segment of the anterior spinal axis (ASA) (black arrowhead). The rest of the ASA is opacified faintly and with delay (white arrowheads). (B) DSA, right vertebral artery injection, anteroposterior view, immediately after endovascular treatment. The spinal branch of the left costocervical trunk has been occluded with detachable microcoils (black arrow), and no longer participates in the supply of the left subclavian artery. The sudden decrease in flow velocity in the artery of the cervical enlargement after occlusion of its outflow toward the left subclavian artery further increases the opacification delay within the ASA (white arrowheads). This immediate hemodynamic response was anticipated, and the patient placed under prophylactic antiplatelet therapy using a combination of aspirin and abciximab. (C) DSA, right vertebral artery injection, anteroposterior view, 48 hours after endovascular treatment. The ASA is now more visible, both cranially and caudally to its enlarged segment, and the flow velocity considerably improved (compare the timing of retrograde opacification of the left vertebral artery between C and B).

Disease Research Laboratory). Nerve conduction studies and EMG were normal. A vascular etiology was suspected, and spinal digital subtraction angiography (SpDSA) performed. It revealed a right-sided aortic arch with isolation of the left SCA, a rare anatomic variant resulting in a right-to-left subclavian steal syndrome.² In addition to classic collateral pathways involving the vertebral arteries, the steal phenomenon implicated an enlarged artery of cervical enlargement (ACE), originating from the right vertebral artery. The ACE connected through the anterior spinal axis (ASA), with a prominent anterior spinal contributor from the left costocervical trunk (figure, A). The latter opacified retrogradely to supply the left SCA, while the flow within the ASA itself appeared sluggish, suggesting a steal phenomenon detrimental to the spinal cord. Considering the rapid clinical deterioration and in order to eliminate the steal phenomenon from the spinal cord, the decision was made to occlude the spinal branch arising from the left costocervical trunk. Using a left brachial artery approach, we endovascularly coiled this branch, interrupting the retrograde flow toward the left SCA (figure, B). Postembolization angiography demonstrated improved flow toward the ASA (figure, C). Neurologic examination performed 2 days later showed improved lower extremity strength, with 4+/5 hip flexion and extension, 4-/5 knee flexion and extension, 5/5 plantar and dorsiflexion, downgoing plantar responses, and a normal sensory examination.

DISCUSSION

Our patient presented with a classic ASAS, i.e., dense paraparesis with dissociated sensory loss. The acute onset of symptoms and subsequent stepwise progression was consistent with a vascular etiology. Cord compression was ruled out by CT myelography. Autoimmune, inflammatory, and infectious etiologies appeared unlikely in view of normal laboratory results. Normal immunoglobulin G index and the absence of oligoclonal bands in the CSF argued against a demyelinating process, and the acute worsening observed during hospitalization appeared inconsistent with a contribution from cocaine abuse.

SpDSA revealed a subclavian steal phenomenon, which can occasionally result in neurologic deficits,³ but our patient was not showing typical symptoms of vertebrobasilar insufficiency, such as vertigo, dizziness, and diplopia. SpDSA also documented an unusual collateral pathway

involving the ASA. Combined with the clinical picture of ASAS, this finding strongly suggested an ASA steal phenomenon, as previously documented in the setting of aortic coarctation,^{4,5} and suspected in one case of brachial diplegia without angiographic confirmation.⁶ The dramatic improvement noted after endovascular interruption of the steal phenomenon appears to confirm the role played by this rare collateral pathway in our patient's symptomatology.

We report a case of acute cervical myelopathy secondary to an ASA steal phenomenon, and its improvement after endovascular therapy. While the inability to use MRI in our patient made the diagnostic evaluation challenging, it may have paradoxically led to a more timely angiographic diagnosis and treatment. Our observation underscores the role of SpDSA in the evaluation of patients with unexplained, acute myelopathy.

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