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MAN WITH SUDDEN PARALYSIS: INSIDIOUS SPINAL CORD INFARCTION DUE TO A NON-RUPTURED ABDOMINAL AORTIC ANEURYSM

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☐ **Abstract—Background: Acute infarctions of the spinal cord are rare events characterized by sudden paralysis or sensory deficits below the level of injury. Etiologies include spinal cord trauma, vascular injury, arterial dissection, thromboembolic disease, chronic inflammatory conditions, or mass effect on the spinal cord. Case Report: A 63-year-old male presented to the emergency department with sudden-onset bilateral leg numbness and weakness. His physical examination was notable for decreased light touch and temperature sensation and bilateral lower-extremity paresis. Initial magnetic resonance imaging (MRI) of his spine did not show cord injuries. Computed tomography angiography of his chest, abdomen, and pelvis demonstrated a 7.5-cm non-ruptured infrarenal abdominal aortic aneurysm (AAA) extending into bilateral iliac arteries. The patient was diagnosed with clinical spinal cord infarction secondary to a thromboembolic event from his AAA. A repeat MRI 15 h later showed spinal cord infarction from T8 down to the conus. He received an endovascular aortic repair and was ultimately discharged to rehabilitation with slightly improved lower-extremity strength. Why Should an Emergency Physician Be Aware of This?: Atraumatic cord syndrome is exceedingly rare and is associated with dissection or complication of aortic aneurysm repair. There are very few reported cases of thrombotic events leading to ischemic cord syndrome. When presented with a patient with symptoms consistent with cord syndrome in the absence of trauma or mass effect on the spinal cord, providers should work up for vascular etiology. Published by Elsevier Inc.**

☐ **Keywords—ischemic cord syndrome; abdominal aortic aneurysm; stroke; imaging**

INTRODUCTION

Acute infarctions of the spinal cord are rare events characterized by sudden paralysis or sensory deficits below the level of injury. Possible etiologies include spinal cord trauma, vascular injury, arterial dissection, thromboembolic disease, chronic inflammatory conditions, or mass effect on the spinal cord. The manifestation of the cord syndrome depends on the etiology and location of spinal cord insult. We describe a case of anterior cord infarction associated with an unruptured abdominal aortic aneurysm (AAA) in a 63-year-old male patient.

CASE REPORT

A 63-year-old male truck driver presented to the emergency department (ED) with sudden-onset bilateral leg numbness and weakness. After a 4-h drive, he went to use the commode and suddenly noted his legs were numb. He attempted to stand but fell to the side of the toilet and experienced both urinary and bowel incontinence. Review of systems was negative for fever, chills, head trauma, headache, neck pain, diplopia, chest pain,

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shortness of breath, abdominal pain, or illicit drug use. He has never had any surgery and his medical history included tobacco smoking, hypertension, and transient ischemic attacks. Medication included aspirin 81 mg daily.

On arrival to the ED, the patient's vital signs were blood pressure 131/86 mm Hg, pulse rate 78 beats/min, respiratory rate 18 breaths/min, temperature 36.1°C (97°F), and O_2 saturation 95% on room air. On neurologic examination, the patient was alert and oriented and demonstrated a normal cranial nerve examination. His extremity examination was negative for any deformity or joint abnormality, with intact pulses on both arms and legs. He had 5/5 strength in his bilateral deltoid, bicep, tricep, wrist extensors, and grip muscles, but he had 0/5 strength in his bilateral hip flexors/extensors, quadriceps, hamstrings, tibialis anterior, and extensor hallucis longus muscles. The patient was unable to demonstrate gait due to bilateral leg paresis. Special examinations included a negative Hoffman sign, negative clonus, neutral Babinski's, and decreased volitional movement of his anal sphincter on rectal examination, with intact tone and sensation. His reflexes were 2 + throughout his bilateral upper extremities and areflexic in patellar and achilles. Sensation examination was significant for decreased light touch and temperature sensation from his lower abdomen around the T10 dermatome to his lower extremities. His head and neck examinations were notable for a supple neck without adenopathy and moist mucous membranes. His heart, lung, and abdominal examination were normal.

Initial laboratory results were significant for estimated glomerular filtration rate 51 U (reference range > 60 U), high-sensitivity troponin 20 ng/L (reference range < 19 ng/L), total cholesterol 294 mg/dL (reference range 150–250 mg/dL), and white blood cells 14 B/L (reference range 4.0–11.0 B/L). The remainder of his metabolic, serologic, and urinary studies were normal. His chest and abdominal one-view anterior–posterior plain films demonstrated background emphysema and mildly distended air-filled loops of large and small bowel, consistent with generalized ileus. Magnetic resonance imaging (MRI) without gadolinium of his cervical, thoracic, and lumbar spines revealed a subtle T2 signal abnormality within the distal cord, but there was no evidence of central stenosis, cord compression, swelling, or associated restricted diffusion (Figure 1). Upon returning to the ED, the patient underwent a computed tomography angiography of his chest, abdomen, and pelvis, which demonstrated a 7.5-cm non-ruptured infrarenal AAA with an opacified 2.5-cm true lumen that extended into the bilateral iliac arteries, as well as a left common carotid artery dissection with subsequent occlusion and recanalization (Figure 2). His brain CT was negative for intracranial hemorrhage or mass effect.

The patient was diagnosed with clinical spinal cord infarction secondary to his AAA from a likely thromboembolic event, along with a concomitant left carotid artery dissection. He received 81 mg salicylate in the ED, but he did not receive systemic anticoagulation after discussion with the vascular surgery service due to potential emergent surgical interventions. His systolic blood pressure goals were set between 120 and 150 mm Hg due to the high clinical suspicion of a devastating spinal cord infarction.

Shortly after hospitalization, a repeat MRI performed just 15 h after the initial MRI showed extensive infarction of the spinal cord from the level of T8 down to the conus (Figure 3). His hospital course was pertinent for an endovascular aortic repair, complicated by persistent abdominal pain and *Staphylococcus aureus* bacteremia secondary to cellulitis, for which he was given 2 weeks of i.v. vancomycin. Our patient was discharged on hospital day 20 to an out-of-state rehabilitation center with a slightly improved neurologic examination, including 1/5 strength in bilateral hip flexors and 3/5 ankle and toe muscle strength with greater overall mobility in the right leg. His sensation examination was unchanged at the time of discharge.

DISCUSSION

Spinal cord syndrome is divided into five main categories: anterior cord syndrome, posterior cord syndrome, central cord syndrome, Brown-Sequard syndrome, and Cauda equina syndrome. Patients with anterior cord syndrome experience flaccid paralysis, areflexia, autonomic dysfunction, and loss of pain and temperature sensation below the level of the injury. Interestingly, as the posterior columns supplied by the posterior spinal arteries are not

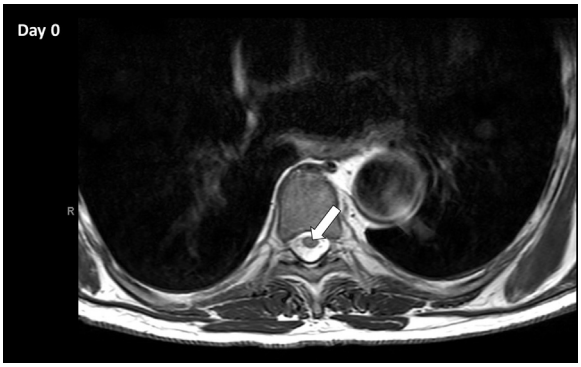

Figure 1. T2 weighted magnetic resonance imaging (axial view) of the thoracic spine on day 0 demonstrating no signs of spinal cord ischemia (arrow) or compression.

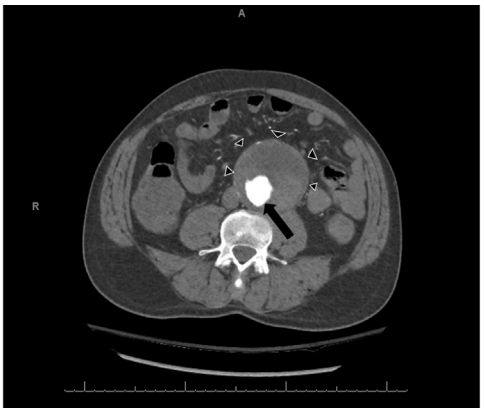

Figure 2. Computed tomography angiography of the abdomen and pelvis with i.v. contrast (axial view) demonstrating a 7.5-cm infrarenal aortic aneurysm (arrowhead) with 2.5-cm true lumen (arrow).

affected, vibration and proprioception are preserved in anterior cord syndrome.

The etiology of anterior spinal cord syndrome is vast and includes direct trauma or mass effect to the anterior spinal cord; vascular occlusion from a thrombus/embolus or dissection; or inflammatory conditions, such as vasculitis. The spinal cord is supplied by one anterior spinal artery, which supplies the anterior two-thirds of the spinal cord, and two posterior spinal arteries. The anterior spinal artery arises from thoracic intercostal branches and lumbar radicular arteries. In approximately 75% of patients, the greater radicular artery of Adamkiewicz arises from a left intercostal artery between T9 and T12 and feeds the anterior spinal artery (1). Thus, an occlusion of the anterior spinal artery or the artery of Adamkiewicz can lead to ischemic anterior cord syndrome.

Many documented ischemic spinal cord syndrome cases are caused by complications of aortic aneurysm or dissection repair surgery (2–5). The mechanisms of injury from surgery include ischemia from prolonged hypotension, injury of arteries that feed the anterior spinal artery, injury from prolonged cross-clamping of the aorta, or thromboembolism of the artery that feeds the anterior spinal artery (5). Ischemic spinal cord syndrome after surgical repair, however, is a rare complication with a 1.2% incidence (6). As a result, additional workup may include angiography to further elucidate the arteries that feed the spinal cord (7,8).

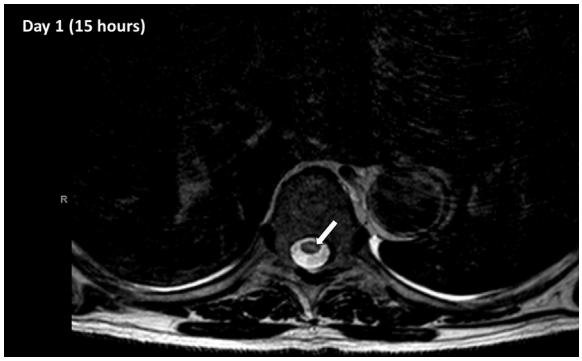

Figure 3. T2 weighted magnetic resonance imaging (axial view) of the thoracic spine obtained 15 h after initial imaging demonstrating increased T2 signal (arrow) within the thoracic spinal cord from the T8 vertebral level compatible with a spinal cord infarct, which has evolved compared to the prior study.

A thromboembolic event leading to ischemic cord syndrome is notably rare. If aortic occlusion is identified, anticoagulation should be initiated and early revascularization should be attempted via aortic reconstruction, bypass, thrombolysis, or thromboembolectomy (9). There are a few cases of thromboembolic events leading to cauda equina syndrome (9,10). However, in the absence of trauma or dissection or surgical manipulation of vasculature with known thrombus or dissection, the authors have not found documented cases in the literature of anterior cord syndrome in a patient with an otherwise dormant AAA. However, there has been a similar case of anterior cord syndrome in a patient with a thoracic aortic aneurysm (11).

In the case of our patient, the sudden onset of symptoms is likely indicative of an embolic event originating from a mural thrombus contained in his AAA. In the setting of non-traumatic acute anterior cord syndrome with no structural explanation (e.g., herniated disc, neoplasm, or epidural hematoma/abscess with mass effect), providers should look to uncover primary vascular injury or occlusion as the etiology of the acute cord syndrome.

The treatment of anterior cord syndrome depends on the etiology and the risk–benefit analysis of instituting a particular therapy. However, for the emergency physician who first identifies the patient with atraumatic acute cord syndrome that can be attributed to a vascular occlusion, the patient should be started on anticoagulation and the vascular surgeon should be alerted to discuss possible revascularization (9). The emergency physician should also avoid hypotension to prevent further ischemia. Although there is limited research on the efficacy of blood pressure management in acute anterior cord syndrome after postoperative aortic repair, there is no clear benefit for steroid treatment in a non-postoperative cord ischemia situation. The initiation of vasopressors should be a shared decision in consultation with neurology and neurosurgery. Finally, while there is no proven benefit for steroids or thrombolysis in reversing cord ischemia, the ultimate management and approach to revascularization (i.e., thrombectomy vs thrombolysis vs aortic repair) will depend on the vascular surgeon (9,10).

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Though this is a rare presentation of acute anterior cord syndrome, when faced with a patient with atraumatic neurologic deficits that indicate a cord syndrome, it may be critical to consider vascular etiologies of cord ischemia. Spinal cord syndrome is rare, and atraumatic cord syndrome is exceedingly rare. Not all cord syndrome is associated with pain or injury. Though more commonly associated with dissection or complication of aortic aneurysm repair, there have been very few reported cases of thrombotic events leading to ischemic cord syndrome. When approached with a patient with symptoms consistent with cord syndrome in the absence of trauma or mass effect on the spinal cord, providers may consider working up for vascular etiology.

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