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ACUTE SPINAL CORD INFARCTION PRESENTING WITH CHEST PAIN AND NEUROGENIC SHOCK: A CASE REPORT

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☐ **Abstract—Background: Spinal cord infarction (SCI) is rare, accounting for approximately 1% of strokes. Case Report: We present the case of a 63-year-old male who presented to the emergency department (ED) with chest pain and acute-onset generalized weakness and was ultimately diagnosed with SCI secondary to suspected occlusion of the artery of Adamkiewicz. Why Should an Emergency Physician Be Aware of This?: SCI may present diagnostic challenges, with its predilection for mimicking other major emergency conditions, such as acute aortic dissection, aortic aneurysm rupture, spinal cord compressive myelopathy, or transverse myelitis. Its consequences are often significantly disabling initially, though patients may experience subsequent clinical improvement. It is important to include SCI in the differential for patients with chest or back pain coupled with neurologic symptoms.**

☐ **Keywords—spinal cord; infarction; stroke; Adamkiewicz; neurogenic shock; chest pain**

INTRODUCTION

Acute spontaneous spinal cord infarction (SCI) is rare, accounting for approximately 1% of strokes (1,2). Spinal cord stroke may occur as a postoperative complication of aortic surgery, or in relation to aortic dissection or aneurysm, but can also be idiopathic (3–5). It is frequently a diagnosis of exclusion; alternative aortic emergencies must first be excluded. The consequences of SCI are potentially devastating to the patient, typically involving paresis or plegia. We present the case of a 63-year-old male who presented to the ED with chest pain and acute-onset generalized weakness, who was ultimately diagnosed with SCI secondary to suspected occlusion of the artery of Adamkiewicz.

CASE REPORT

A 63-year-old male with medical history of hypertension and stage IV prostate cancer with metastasis to the spine presented to the ED complaining of severe chest pain and generalized weakness. The pain was described as 24 h of severe, constant, and "achy," across the lower anterior chest. The patient presented to another ED earlier in the day, and was discharged home following evaluation for potential acute coronary syndrome. Later that evening, he suddenly became nauseated, vomited, then acutely experienced full-body generalized weakness and numbness, and collapsed onto his mattress. He denied any back or abdominal pain.

On initial evaluation, he was awake, alert, and oriented to person, place, time, and situation. Initial vitals were as follows: temperature 36.2°C, heart rate 78 beats/min, decreased blood pressure measured at 60/37 mm Hg, respiratory rate 20 breaths/min, and room air oxygenation

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saturation of 95%. Physical examination revealed slightly decreased strength in bilateral upper extremities, and significantly decreased strength in bilateral lower extremities. He could not lift either leg off the stretcher. Sensation to crude touch was decreased diffusely, and significantly more pronounced in the lower extremities. Bilateral lower extremities had no response to pain, and patellar reflexes were absent. Facial examination demonstrated no asymmetry, and speech was fluid. Patient had equal blood pressures in bilateral upper extremities, and distal pulses were equal throughout. Proprioception, vibration, and temperature sensations were not assessed at the initial ED encounter, though subsequent evaluations did note loss of temperature sensation below the T4 level, with intact proprioception in bilateral lower extremities. Otherwise, comprehensive physical examination was normal.

Electrocardiogram was unremarkable for dysrhythmia or acute ischemic changes, and initial troponin level was normal. A computed tomography (CT) angiogram of the chest, abdomen, and pelvis was performed to assess for aortic pathology. The CT demonstrated no evidence of aortic dissection or aneurysm rupture, though multiple metastatic bony lesions of the spine were seen.

Upon re-evaluation of the patient, his blood pressure improved to 97/62 mm Hg following 3 L of crystalloid. Further re-examination revealed plegia and numbness in the bilateral lower extremities and lower abdominal region, with resolution of his upper extremity weakness and numbness. The patient developed urinary retention and rectal tone was absent. Subsequent magnetic resonance imaging (MRI) again demonstrated multiple metastatic lesions in the thoracic spine, without accompanying cord impingement. Neurosurgical consultation was obtained, with concern for possible SCI.

Upon transfer to a tertiary referral center, the patient underwent spinal angiography that demonstrated a right L2 intersegmental artery occlusion, with nonvisualization of the artery of Adamkiewicz. Further review of MRI noted reduced diffusivity within the ventral and central aspects of the spinal cord extending from the T1–T2 down to the T6–T7 levels, compatible with SCI. Noncontrast CT of the brain as well as MR angiography of the head and neck were unremarkable for acute findings. The patient was ultimately diagnosed with anterior spinal artery stroke with associated paraplegia, dysautonomia, and neurogenic bowel and bladder. Management was conservative with aspirin therapy and focused on rehabilitation. The patient remained paraplegic for several months and died after a prolonged hospitalization.

DISCUSSION

Spontaneous SCI is a rare condition, thus relatively few large clinical investigations have been reported, with most investigating spinal cord ischemia as a complication of aortic or general surgical procedures, or prolonged arterial hypotension. The incidence of SCI is unknown, but it is estimated to account for 0.3–1.2% of all strokes (1,2). Recent literature suggests underdiagnosis of spontaneous SCI (6,7).

Spinal cord stroke is caused by acute disruption of the spinal cord vascular supply, resulting in ischemia, infarction, and acute spinal cord dysfunction with associated neurologic deficits (1,4,5). The clinical deficit varies, depending on level of ischemia, which corresponds to a vascular territory in the cord (3,8,9). Our patient presented with anterior cord syndrome, also known as anterior spinal artery syndrome (ASAS). Anterior cord syndrome is most commonly due to an interrupted supply of the anterior spinal artery or the artery of Adamkiewicz (9). The spinal cord receives its vascular supply from three major vessels arising from the vertebral arteries in the neck: one anterior spinal artery (ASA), which supplies the anterior two-thirds of the spinal cord, and a pair of posterior spinal arteries (PSAs), which supplies the posterior one-third of the spinal cord (9–11). The ASA supply is reinforced by anterior segmental medullary vessels from the descending aorta, the largest of which is the artery of Adamkiewicz (9,11).

SCI is largely a clinical diagnosis of exclusion. MRI may be helpful in excluding compressive myelopathy or to confirm diagnosis. However, as with any evolving stroke syndrome, its sensitivity can be limited in the acute phase (12–14). Vascular imaging, such as CT or MR angiography, may exclude aortic pathology (3,15). Dedicated spinal angiography is the gold standard diagnostic modality (15,16). A lumbar puncture with cerebral spinal fluid analysis can be considered to exclude infectious or inflammatory myelopathies, such as Guillain-Barré syndrome or transverse myelitis (17). There are a few studies that suggest vertebral body infarct on MRI as a sign of associated spinal cord ischemia (17–19). Several studies suggest consideration of diffusion-weighted MR imaging (DWI) or echo-planar DWI in the diagnosis of SCI in the acute phase (12,14,17,20–22).

A confounding aspect of this case included chest pain as the presenting complaint. Reports by Silwa et al. and Barrera et al. note that ASAS can present with sudden-onset and rapidly progressive pain that can be described as sharp or dull, and may be girdle-like in distribution (11,23). Cheshire et al. noted that transient radicular or back pain can herald the neurologic symptoms of ASAS (4). Acute-onset back pain in SCI has been documented in several studies (14,24). It is prudent for emergency clinicians to remain aware that chest or back pain could be one of the presenting symptoms of SCI.

To date, management of spontaneous SCI in the acute phase remains largely supportive, as no clear guidelines

for treatment exist (2,5,17). Similar to cerebral infarction patients, acute management of blood pressure and glucose is imperative (1,14). Adequate blood pressure is crucial to maintain spinal perfusion (14). Care should also be taken to manage autonomic dysreflexia, and bowel and bladder dysfunction (3). The patient in this case presented with neurogenic shock that was responsive to i.v. fluids. We suspect that the patient initially experienced full-body numbness and weakness due to global hypoperfusion because he was able to better isolate his symptoms after blood pressure improvement. He also required urinary catheterization for urinary retention. It is important to note that non-visualization of the artery of Adamkiewicz during angiography is not definitively diagnostic of occlusion, as anatomic variants exist, including absence of the artery in 15.4% of the population, as reported in a recent large meta-analysis (25). Global spinal hypoperfusion is an alternative mechanism that could precipitate ASAS. Disposition should be made to a tertiary care hospital with available diagnostic capabilities and neurology and neurosurgery specialists.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Though rare, SCI presents acutely and is often associated with potentially devastating sequelae. Its presentation can be similar to aortic dissection, aortic aneurysm rupture, and spinal cord compression. The diagnosis is generally made on clinical grounds following vascular imaging to exclude alternative conditions. It is a vascular emergency that can present with acute-onset chest or back pain coupled with neurologic deficits in the emergency care setting.

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