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ACUTE AORTIC DISSECTION PRESENTING AS BILATERAL LOWER EXTREMITY PARALYSIS: A CASE REPORT

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☐ **Abstract—Background: First described by Morgagni in 1761, aortic dissection (AD) is an acute life-threatening and time-sensitive disease process with an increasing mortality approaching 1% for every 1-hour delay in diagnosis within the first 48 hours. Despite continued surgical advancement, overall in-hospital mortality remains significant (27.4%). Case Report: A 56-year-old woman presented to an outlying emergency department with a complaint of isolated lumbar pain associated with right lower extremity paresthesia and paralysis that progressed to the left. Her medical history and a review of symptoms were significant for chronic obstructive pulmonary disease and tobacco abuse. The initial evaluation in the emergency department included laboratory values and a computed tomography scan of the lumbar spine that revealed minimal disease. After transfer to our tertiary care center for an emergent magnetic resonance imaging scan of the lumbar spine, her vital signs were as follows: blood pressure, 176/84 mm Hg; heart rate, 76 beats/min; respiratory rate, 24 breaths/min; afebrile; and oxygen saturation 98% on room air. A repeat examination revealed cold extremities with mottling, bilateral symmetric lower extremity flaccid paralysis, and a loss of pulses and reflexes. She was insensate below the T10 dermatome. Her upper extremities and cranial nerves were normal. She underwent computed tomography angiography, revealing an extensive Stanford type A AD with interim thrombus formation. After successful endograft stenting, she died 24 hours later. Why Should an Emergency Physician Be Aware of This?: Comprising <2% of all ADs, the pathophysiology of paraplegia as the initial presentation of AD is caused by compression of the anterior spinal artery, resulting in ischemia of the spinal cord. Acute AD is a life-threatening medical emergency that requires a high clinical level of suspicion because of its often variable presentation and high incidence of mortality.**

☐ **Keywords—aortic dissection; back pain; lower extremity paralysis; paresthesias**

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INTRODUCTION

We present a case of a 56-year-old woman with a Stanford type A aortic dissection (AD) presenting as bilateral lower extremity paralysis. The patient initially presented to an outlying community emergency department (ED) with a chief complaint of isolated lumbar pain associated with bilateral lower extremity paresthesia and paralysis. A presumed diagnosis of acute spinal cord compression was made and the patient was transferred to a tertiary care ED for definitive imaging of the lumbar spine via magnetic resonance imaging (MRI) and a neurosurgical evaluation. Upon presentation to the tertiary care ED, further history and examination revealed suspicion for AD and a subsequent emergent computed tomography angiography (CTA) scan identified an extensive Stanford type A AD. The patient was taken to the operating suite where endovascular stenting was performed.

First described by Morgagni, AD is an acute life-threatening and time-sensitive disease process with an increasing mortality approaching 1% per 1-hour delay

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in diagnosis during the first 48 hours (1,2). Comprising <2% of all ADs, paraplegia as the initial presentation of AD results from the development of an anterior spinal artery syndrome (1). Our patient's initial presentation of a spinal cord compression syndrome coupled with the interim development of an aortic thrombus resulting in ischemia of the lower extremities has not been previously described in the literature. Acute AD is a life-threatening medical emergency that requires a high clinical level of suspicion because of its often variable presentation and high incidence of mortality.

CASE REPORT

A 56-year-old woman presented to a rural ED with a chief complaint of lumbar back pain associated with bilateral lower extremity paresthesia and paralysis. The patient described walking to her mailbox without difficulty approximately 90 minutes before the onset of her symptoms. The patient reported that her symptoms began with an altered sensation of the right lower extremity while taking a bath that rapidly progressed over several minutes to frank paralysis of her right lower extremity. The patient's right-sided symptoms were quickly followed by subsequent paresthesia of the left lower extremity, progressing to paralysis. The patient stated that she was unable to stand from the bathtub and that she had to crawl from her bathroom to the nearest phone to notify emergency medical services. Her medical history was significant for only chronic obstructive pulmonary disease, and she denied any previous surgeries. Her social history was significant for a 40-pack a year history of tobacco use. On questioning, she denied any history of intravenous drug abuse, any recent trauma, or fever. Upon arrival to the primary ED, the patient's vital signs were unremarkable with the exception of a mildly elevated blood pressure (154/96 mm Hg) and a respiratory rate of 24 breaths/min. Her initial evaluation in the ED included laboratory testing and a noncontrast commuted tomography (CT) scan of the lumbar spine. Her laboratory findings were significant for hypokalemia (2.9 mEq/L), a serum glucose level of 202 mg/dL, and an elevated white blood cell count of 14.2 K/μL. The patient's initial CT scan revealed mild L5/S1 central canal stenosis caused by a mild nucleus pulposus herniation. Her lumbar pain improved after the intravenous administration of morphine sulfate 4 mg and hydromorphone 1 mg. In addition, she received intravenous levofloxacin 750 mg for suspected urinary tract infection and of oral potassium 20 mEq. The neurosurgeon on call for the tertiary care center was consulted and arrangements were made for transfer to the accepting ED; upon arrival, the patient was to undergo an emergent magnetic resonance imaging (MRI) scan of the lumbar spine to exclude epidural abscess or other acute process resulting in spinal cord compression.

Upon arrival to the referral center's ED, the patient's recorded vitals included a blood pressure of 176/84 mm Hg, a heart rate of 76 beats/min, and oxygen saturation of 98% on room air. The patient complained of mild pain in her lower back, and a repeat physical examination was performed by the emergency physician before transfer to the MRI suite.

On examination, the patient was noted to have symmetric flaccid paralysis of her lower extremities bilaterally that were also noted to be cold to palpation. On further examination, the patient was found to have absent dorsalis pedis, posterior tibial, popliteal, and femoral pulses bilaterally. In addition, the patient was found to be insensate from the T10 dermatome distally with absent patellar deep tendon reflexes in the lower extremities. The neurologic examination of the upper extremities and cranial nerves were grossly normal and symmetric bilaterally. In addition, the patient's skin was noted to be mottled from the umbilicus inferiorly. Bedside Doppler ultrasonography was performed and failed to detect dorsalis pedis or femoral pulses bilaterally. The patient was taken emergently for a CTA scan that revealed an extensive Stanford type A AD originating from the aortic valve proximally and involving the innominate, right common carotid, subclavian, and axillary arteries extending to the celiac trunk distally (Figure 1).

In addition, the abdominal aorta was noted to be occluded 2.5 cm proximal to the iliac bifurcation with thrombus (Figure 2). The patient was started on sodium nitroprusside and labetalol infusions for blood pressure management while cardiovascular surgery consultation was obtained. The patient was subsequently taken to the operating suite emergently for surgical repair, where she underwent successful endograft stenting without

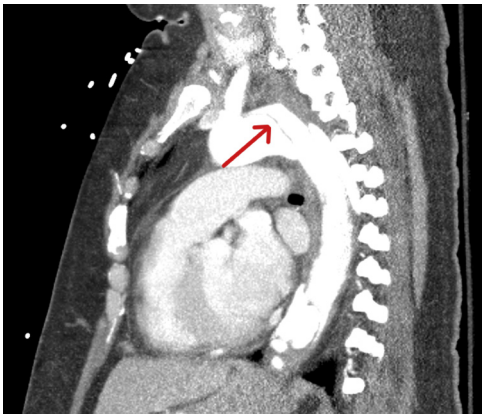

Figure 1. Sagittal computed tomography angiography scan showing aortic dissection originating from the proximal aorta and extending distally. Arrow indicates origin of dissection.