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## An unusual cause of chest pain

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**Dr. Jennifer Pope:** Today's patient is a 57-year-old man with no significant past medical history who presented to the Emergency Department (ED) with severe abdominal pain. He reported an aching pain that began in the epigastrium and spread in a band-like distribution around his upper abdomen to his back. When the pain was intense, he felt it was difficult to draw a full breath. He called emergency services for assistance. En route to the ED, paramedics administered 2 sprays of sublingual nitroglycerin, which lessened his pain. On review of systems, he reported diaphoresis at home, but no nausea, vomiting, diarrhoea or blood in his stools. He denied chronic medical problems or prior surgeries. He was a former smoker, having quit one year prior to this evaluation.

**Dr. Chris Fischer:** Was this pain exertional? What did your physical exam reveal?

**Dr. Pope:** No, this pain was neither exertional nor relieved by rest. His initial vital signs included a temperature of 36.8°C, a heart rate of 88 beats/min, a blood pressure of 150/88 mmHg, a respiratory rate of 16 breaths/min and an oxygen saturation of 98% on room air. He was alert, oriented and in no apparent distress. He had equal and reactive pupils and anicteric sclerae. His oropharynx was clear. His lungs were clear to auscultation bilaterally. His heart rate

was regular, with no murmurs or adventitious sounds. His abdomen was soft, nontender and nondistended. A rectal exam showed normal tone and brown, guaiac-negative stool. His extremities were warm and well perfused, with no oedema. He had normal strength and sensation to light touch in his extremities. He had no diaphoresis or rash.

**Dr. Shamai Grossman:** Epigastric pain, although usually a symptom of gastrointestinal disease, has a wide differential diagnosis encompassing multiple organ systems. The differential in this case must remain broad, as the physical exam was unrevealing. It is also important to remember that pain relief with nitroglycerin is not specific to cardiac disorders and does not narrow our diagnostic possibilities [1].

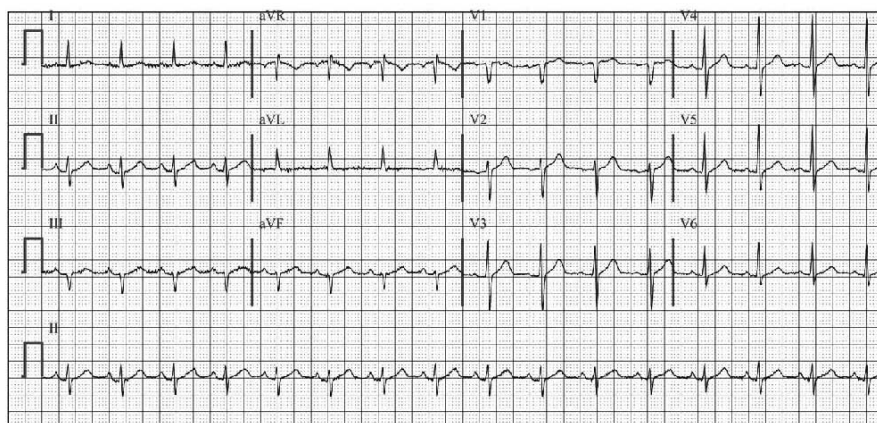
**Dr. Lara Kulchycski:** The band-like nature of his discomfort is noteworthy and raises the possibility of radicular pain. Were there any indications of neurological disease?

**Dr. Pope:** The neurological review of systems and gross motor and sensory exam were normal, so our initial plan was to evaluate for cardiovascular and gastrointestinal pathology. The patient was placed on a cardiac monitor and given supplemental oxygen by nasal cannula. An electrocardiogram, chest radiograph and cardiac enzymes were ordered. Because epigastric pain radiating to the back raises concerns for aortic dissection, a chest computed tomography (CT) scan with intravenous contrast was performed. Although abdominal pathology is less likely in the setting of a benign physical exam, liver function tests and pancreatic enzymes were requested.

**Dr. Fischer:** What was your interpretation of these studies and what was the patient's subsequent clinical course in the ED?

**Dr. Pope:** The electrocardiogram showed normal sinus rhythm at a rate of 86 beats/min with left axis deviation (Fig. 1). Complete blood count, chemistry panel and liver function

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**Fig. 1** The electrocardiogram revealed a normal sinus rhythm with left axis deviation and non-specific T-wave flattening in aVL

tests were within normal limits. Cardiac enzyme testing revealed a creatine kinase (CK) of 206 IU/l, a normal CK-MB isoenzyme fraction and a troponin I of less than 0.03 ng/ml. The chest X-ray was unremarkable (Fig. 2). The chest CT showed aortic atherosclerosis with an associated mural thrombus in the descending thoracic aorta, but no dissection or aneurysm. As the patient remained pain-free, the clinical plan was to admit him for serial cardiac enzymes and an inpatient stress test. While awaiting transfer to the floor, he began to complain of difficulty voiding and lower extremity muscle weakness.

**Dr. Grossman:** This is a concerning development that radically alters the differential diagnosis. How had his physical exam changed? How did this new information alter your clinical care?



**Fig. 2** Chest radiography showed no acute cardiopulmonary pathology

**Dr. Pope:** His exam was now notable for 4 out of 5 muscle weakness and 3+ hyperreflexia in the lower extremities. He had decreased sensation to pinprick below the level of T6 and diminished rectal tone. Bladder catheterisation revealed a post-void residual of 600 ml of urine. Proprioception and cranial nerve function were preserved. We requested an emergent neurological consultation and a magnetic resonance imaging (MRI) scan of the thoracic spine, which revealed an intramedullary lesion at T6 consistent with infarction in the distribution of the anterior spinal artery (Fig. 3).

**Dr. Jonathan Edlow:** It is not unusual for patients with spinal cord infarction to present with isolated pain. The onset of obvious neurological symptoms, such as weakness and sensory changes, can lag behind the pain by several hours [2]. However, MRI signal changes can also represent transverse myelitis or demyelinating disease. Were these alternate diagnoses considered?

**Dr. Pope:** MRI is the most sensitive test to detect spinal cord pathology, but it has two important limitations [3, 4]. First, signal abnormalities are not always pathognomonic for ischaemia; demyelinated plaques, small neoplasms and myelitis may have a similar appearance [5, 6]. Second, an MRI early in the course of spinal cord infarction may be normal even in the presence of significant neurological deficits [7]. To evaluate for competing diagnoses such as transverse myelitis or demyelinating disease, additional testing was performed. MRI scans of the spine and brain revealed no additional lesions. Examination of the cerebral spinal fluid showed no infection, pleocytosis or oligoclonal banding. Although a normal lumbar puncture (LP) does not rule out myelitis, the clinical picture, coupled with imaging abnormalities in a vascular distribution, was most consistent with spinal stroke [5]. The patient was admitted to the neurology service with a diagnosis of anterior spinal artery syndrome (ASAS).

**Dr. Edlow:** Was embolism from the aortic mural thrombus the presumed aetiology of ASAS in this patient? What was his functional outcome?



**Fig. 3** Thoracic spine magnetic resonance imaging revealed an intramedullary lesion at T6 with enhancement after contrast administration

**Dr. Pope:** Atheroembolic disease remained the most likely explanation for his diagnosis. He was started on aspirin and statin therapy. At hospital discharge he could ambulate without assistance, but had no temperature or pain sensation below the umbilicus, and required intermittent urinary catheterisation. Two months after discharge, he had regained bladder control, but continued to report sensory loss and episodic faecal incontinence.

**Dr. Kulchycki:** Can you review for us the diagnosis and natural history of ASAS?

**Dr. Pope:** When ASAS was first described by Spiller in 1909, the most frequent aetiology was syphilitic arteritis [8]. Today, the list of potential causes is extensive and includes atheroembolic disease, complications from aortic aneurysm repair, aortic dissection, vasculitis, infection, prolonged hypotension and trauma. Even after thorough investigation, up to one-third of ASAS cases remain idiopathic [7].

The ventral thoracic cord is a watershed region that is particularly vulnerable to ischaemia. The spinal cord is supplied by a single anterior spinal artery and paired posterior

spinal arteries. The anterior spinal artery, which has few arterial feeders and minimal redundancy, supplies the ventral two-thirds of the spinal cord. Anterior spinal artery occlusion damages the corticospinal and spinothalamic tracts, resulting in flaccid paralysis, sphincter dysfunction, and loss of pain and temperature sensation. Posterior column function, including proprioception and vibratory sense, is preserved in ASAS as the dorsal spinal cord is supplied by the posterior spinal arteries [9].

The work-up and treatment for ASAS may vary for each patient. An emergent MRI of the spine is imperative in most cases to rule out haemorrhage or operative lesions. LP may be indicated to evaluate for competing diagnoses, such as transverse myelitis. Additional laboratory studies for vasculitides or hypercoagulable states should be utilised when appropriate. For patients with ischaemic spinal stroke, aspirin therapy is often initiated. No clinical trials support the use of thrombolytics; the creation of such a study would be difficult given the paucity of cases and the frequent delays in diagnosis. Clinicians should initiate pharmacological therapy to ameliorate vascular risk factors such as untreated diabetes, hypertension or hyperlipidaemia.

There is little published data on functional outcomes after spinal cord infarction. Twenty-two percent of ASAS patients die during their initial hospitalisation [7]. Small studies have shown that only 18%–41% of survivors can ambulate independently at hospital discharge, while 20%–57% will remain wheelchair-bound. Functional recovery is best predicted by the initial degree of motor dysfunction [7–10].

ASAS remains a diagnostic challenge, as afflicted patients may present with isolated pain and may not reach maximal neurological deficits for over 24 h [11]. Clinicians must remember the value of serial physical examinations and ensure rapid access to MRI to rule out operable lesions. Multi-centre trials may be useful to develop evidence-based guidelines for therapeutic intervention.

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