

Painless Thoracic Aortic Dissection Presenting as High Paraplegia : A Case Report

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Abstract

Acute aortic dissection is a catastrophic episode that usually presents as a sudden, painful, ripping sensation in the chest or back. Physical findings may include loss of pulses and aortic regurgitation. It is associated with neurologic sequelae in as many as one third of patients. Painless dissection occurs in 5% of patients. We report a case of painless aortic dissection, presenting as acute paraplegia. The patient was a 59-year-old man who presented with paraplegia, with no chest or back pain. On examination, strength was 5/5 in both upper extremities and 0/5 in both lower extremities. Computed tomography of the chest revealed a type A dissecting aneurysm. Painless acute aortic dissection in which paraplegia is the only presenting sign is very rare. However, aortic diseases, including acute aortic dissection, should always be considered as a differential diagnosis of patients with sudden onset, painless paraplegia.

KeyWords : Aneurysm; Dissecting; Aorta; Paraplegia;

Introduction

Aortic dissection is caused by a circumferential tear or less frequently, it is caused by a transverse tear of the intima. It often occurs along the right lateral wall of the ascending aorta where the hydraulic shear stress is high. Acute aortic dissection presents with the sudden onset of pain, which is often described as being very severe with a tearing sensation, and diaphoresis associated with this condition.¹⁾ Aortic regurgitation, bowel ischemia, hematuria, myocardial ischemia, and various neurologic findings due to carotid artery obstruction (hemiplegia and hemianesthesia) or spinal cord ischemia (paraplegia) have all been observed as complications resulting from

the dissection occluding the major arteries.

Physical findings may include loss of pulses and aortic regurgitation. It can be associated with neurologic sequelae in as many as one third of patients. Painless dissection occurs in approximately 5% of patients, and the diagnosis may often be delayed.²⁻³⁾ We report a case of painless aortic dissection that presented as new onset paraplegia.

CASE REPORT

The patient was a 59-year-old right-handed, thin, man with a medical history of hypertension. He had a long history of smoking. On presentation to the emergency department, he complained of sudden onset paraplegia and both leg pain for 1 day.

When questioned, he denied having any chest pain. At local hospital, his blood pressure was 200/80

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mmHg and transferred to our hospital for further evaluation of high blood pressure and paraplegia. He again denied having any chest or back pain. The bilateral lower extremity weakness rapidly progressed, and he had complete loss of sensation and motor control of his lower extremities. He was admitted and diagnosed with hypertension and paraplegia.

On admission, his temperature was 36.3°C, his pulse was 94 beats/min, his respirations were 22 breaths/min, and his blood pressure was 120/80. On physical examination, nearly normal breath sounds were noted in the whole lung field. His heart had a regular rate and rhythm, with a grade I midsystolic murmur heard at the apex and a grade II diastolic murmur heard at the base. Both femoral pulses were symmetric and weak. The patient's abdomen was soft, nontender, nondistended, and there were no bruits or palpable pulsatile masses. He was alert and oriented to person, place, and time. His cranial nerves II - XII were intact. His strength was 5/5 in all muscle groups of both arms. He had no trunk control or volitional movement of any muscle groups in his legs. Muscle tone was normal in both arms, but his legs were flaccid. Deep tendon reflexes were intact in both biceps, triceps, and brachioradialis, but were absent throughout his legs. Sensation to light touch and pinprick was intact through T10 but absent from T10 through S5. The initial diagnostic evaluation included a chest x-ray, which showed mediastinal widening (Fig 1). Electrocardiogram showed no significant abnormalities. (Fig 2) Computed tomography (CT) of the brain showed basal ganglial and cerebellar calcifications that were consistent with age. An x-ray of the entire spine showed osteopenia; an old, healed L1 compression fracture; and degenerative changes of L1 through L5. He had no acute fractures. CT of the thoracic spine was normal. No evidence existed of bony destruction, masses, or bleeding in the spinal canal.

The following laboratory values were abnormally

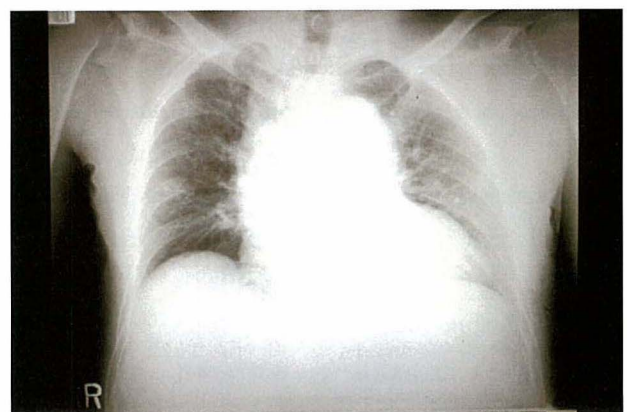
high : initial creatine phosphokinase (CPK) was 3396 with an isoenzyme MB fraction of 31.9. Myoglobin, over 4000 : troponin T, 0.38. The remainder of the laboratory values were normal (sodium, potassium, chloride, carbon dioxide, blood urea nitrogen, creatinine, white blood cells, alanine transaminase, gammaglutamyl transferase).

Because of the elevated CPK level, small and mediastinal widening in chest X-ray, echocardiogram was ordered to rule out cardiac abnormality. Echocardiogram revealed grade III aortic regurgitation. CT of the chest was ordered to rule out an aortic dissection. A type A dissecting aneurysm was found extending from the aortic valve leaflets to the takeoff of both iliac arteries (fig 1) and renal infarct on both kidneys.

During his hospital stay, the patient was followed by physicians of internal medicine, cardiology, neurology, cardiovascular surgery. The surgeons believed he would not survive aorta replacement surgery, so he was not a candidate for surgery. He refused all kinds of invasive procedures.

On his second hospital day, His blood pressure was decreased and creatinine level was increased. After a few hours later, his blood pressure was uncheckable, and his electrocardiogram showed flat. He and his family didn't want any kind of resuscitation procedure. we declared death on him.

Fig 1. Chest PA showed mediastinal widening.



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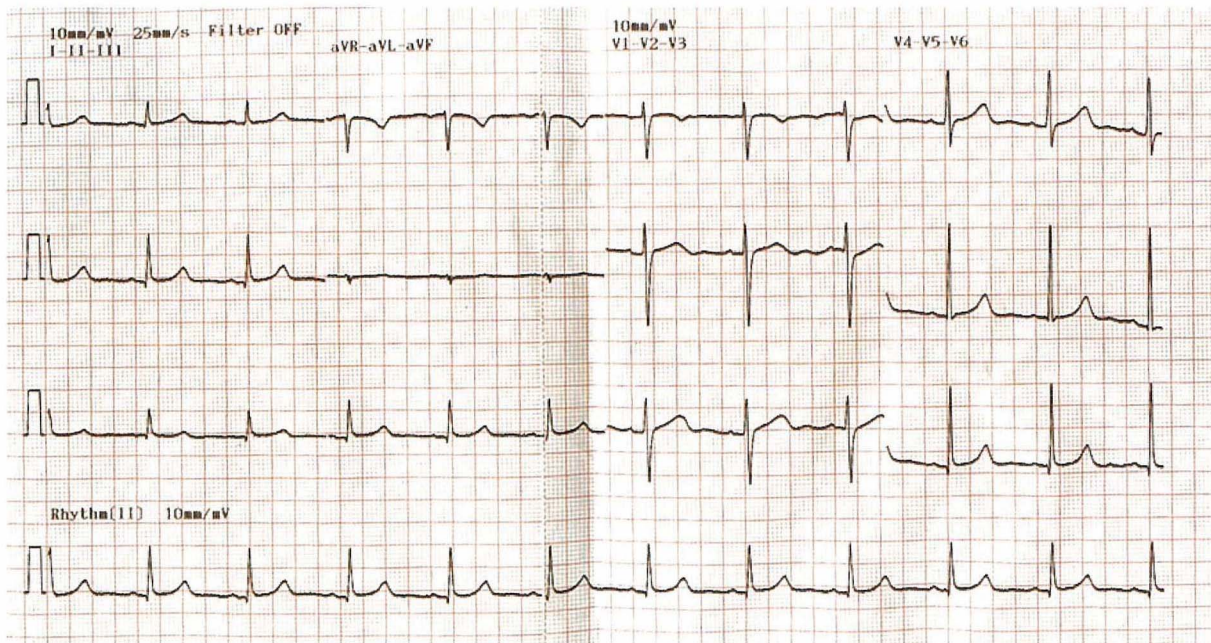


Fig 2. Electrocardiogram showed no significant changes.

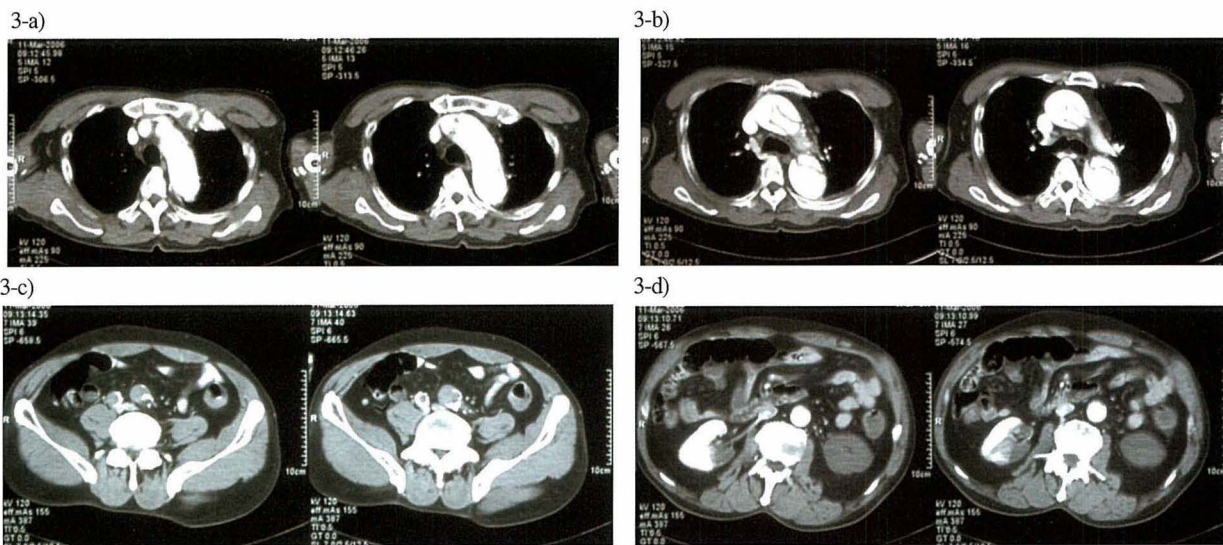


Fig 3. a) Computed tomography of thorax showing dissection in the aortic root (top arrow) and the descending aorta (bottom arrow). b) Computed tomography showing dissection of aortic arch c) Computed tomography of abdomen showing dissection and both renal infarct d) Computed tomography of lower extremity showing continued dissection to both femoral arteries

Discussion

Aortic dissection is uncommon; it accounts for approximately 1 in 10,000 hospital admissions.⁴⁾ Without intensive treatment, the mortality rate is as

high as 80%.²⁾ The precipitating event in aortic dissection is a circumferential or transverse tear of the intima, usually beginning in areas where hydraulic shear stress is high; eg, tears often begin along the right lateral wall of the ascending aorta. The intimal

tear in type A dissections, which our patient had, most commonly begins just above the aortic valve cusps.⁴⁾ Hypertension is a predisposing condition, and exists in 70% of patients.^{1,4)} More commonly in males (at a 2 : 1 ratio) and in persons in the 5th through 7th decade, aortic dissection is a major cause of morbidity in patients with Marfan syndrome and is seen in patients with congenital aortic valve anomalies, coarctation of the aorta, and in women during the third trimester of pregnancy.^{1,4)}

Patients typically present complaining of a severely painful tearing or ripping sensation. The pain is usually substernal, intrascapular, or in the mid-back. Pain may be absent or intermittent in as many as 5% to 10% of patients.^{1,2,5)} Other signs and symptoms include congestive heart failure, angina, dyspnea, syncope, and a 'shocky' appearance (hypotension; tachycardia; oliguria; and cool, mottled extremities).^{1,3,4)} Presentation of aortic dissection with neurological manifestation is not uncommon and the incidence varies from 18% - 29% in different studies.^{6,7,8)} Neurological abnormalities as a result of aortic dissection can be accounted for by three factors, ischaemia of the brain, ischaemia of the spinal cord and ischaemia of the peripheral nerves. It is most likely that our patient had ischaemia to the spinal cord, as there was no radiological evidence for extension of the dissection into the carotids or into the femoral arteries. Although in most patients, neurological deficits from aortic dissection are permanent,⁶⁻⁹⁾ there are two previously reported cases of complete recovery of paraparesis. Waltimo and Karli¹⁰⁾ reported a case of aortic dissection, in which complete recovery of the paraparesis occurred two days later.

The blood supply to the spinal cord must be intact for neurologic function to be normal. The two posterior spinal arteries supply the posterior one third of the spinal cord, including the posterior columns.

The anterior spinal artery supplies the anterior two thirds of the spinal cord, including the motor neurons. In the cervical region, the vertebral arteries form the anterior spinal artery, with or without contribution from a deep cervical artery and the costocervical trunk. In the lower thoracic and lumbar region, the artery of Adamkiewicz (the great anterior radicular artery) provides the blood supply to the inferior two thirds of the spinal cord. The origin of the artery of Adamkiewicz is variable (T6 - T12 or L1 - L3), and it arises more frequently on the left from an inferior intercostal or a superior lumbar artery. It makes a large contribution to the anterior spinal artery. The artery of Adamkiewicz branches from the posterior aspect of the aorta ultimately to supply the anterior aspect of the spinal cord. Note that the aortic dissection in the present case occurred along the posterior aspect of the aorta. Most areas of the spinal cord receive additional blood flow from collateral flow. However, in the thoracic cord, there is a 'watershed' area (T4 - T6) where the collateral blood flow is limited, making this area especially prone to ischemia.^{2,4,11,12)} A literature review revealed few reports of painless aortic dissection presenting with neurologic sequelae. Gerber et al⁵⁾ reported 3 patients with painless aortic dissection, 2 of whom presented with cerebral ischemia and 1 of whom presented with ischemic neuropathy.

Rosen reported a patient who had a transient paraplegia following painless acute aortic dissection. Our patient presented with acute paraplegia with no history or evidence of trauma. Because of absence of history of chest pain, the possibility of neurological disease was initially considered.³⁾

In addition to a diagnosis of aortic dissection (which in this case was not initially considered because of the painless presentation), consideration should also be given to thrombosis or embolism of the artery of Adamkiewicz, any shock causing hypoperfusion of the

cord, epidural or subdural hemorrhage of the spinal cord, hematomyelia secondary to a vascular malformation of the spinal cord, or hemorrhage into an epidural tumor mass. The differential diagnosis of paraplegia also includes spinal cord injury, postinfectious transverse myelitis, tumor, infection or abscess, disc herniation, decompression illness, electrical injury, Guillain-Barre´ syndrome, multiple sclerosis, poliomyelitis, and spondylolisthesis.

CONCLUSION

We report a case of painless aortic dissection that presented as new-onset paraplegia. Although this phenomenon is rare, it should be considered in the differential diagnosis of new-onset paraplegia, particularly in a patient with predisposing risk factors.

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