Transient Paraplegia Caused by Acute Aortic Dissection  
—Case Report—

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Abstract

A case of acute aortic dissection (AAD) presenting as sudden, transient paraplegia and severe back pain is reported. The patient was a 66-year-old male with a 10-year-history of hypertension. The pain characteristically migrated from the back to the neck and then returned to the back. He showed complete transverse myelopathy at the level of the 9th thoracic cord. Computed tomography disclosed internal displacement of aortic intimal calcifications, without abnormalities in the spinal canal, and myelography showed no spinal canal block or stenosis. Electrocardiography and chest x-ray indicated nonspecific changes of high amplitudes and mild cardiomegaly, respectively. Together, these findings suggested acute aortic dissection with spinal cord ischemia. The initial systolic blood pressure of 220 mmHg was lowered with medication, and the pain was controlled with morphine. He recovered fully and was discharged 80 days after the onset of symptoms, with no neurological deficits. AAD carries a very poor prognosis unless treated immediately. Therefore, it is very important to promptly differentiate this disorder from spinal vascular conditions that also produce back pain and paraparesis.

Key words: acute aortic dissection, paraplegia, myelopathy, spinal cord ischemia, computed tomography

Introduction

Acute aortic dissection (AAD), or dissecting aortic aneurysm, is a catastrophic disease that often causes neurological symptoms. Without intensive treatment, the mortality within 2 weeks of onset is reportedly as high as 80%. If associated with spinal cord ischemia, AAD produces severe back pain and acute paraplegia, as do such spinal vascular diseases as hematomyelia, epidural or subdural hematoma, and simple anterior spinal artery ischemia. Since acute epidural or subdural hematoma in the spinal canal requires prompt surgical treatment, these diseases must be differentiated as soon as possible. We report a case of AAD in which the initial symptoms were severe back pain and transient ischemia of the spinal cord. We also review the literature concerning the differential diagnosis.

Case Report

A 66-year-old male with a 10-year-history of hypertension suddenly developed severe back pain while stretching to wash his back in the bath. Within minutes he was unable to walk, and he was brought to our service approximately 30 minutes after the onset. On admission he was alert, complaining of intolerable low cervical pain. Systolic blood pressure, measured in the right upper arm, was 220 mmHg. Chest auscultation revealed no bruit. In all extremities, the arteries were readily palpable. His abdomen was distended but no pulsating mass was detected by palpation. He was given 20 mg of nifedipine sublingually.

Neurological examination showed complete transverse myelopathy at the level of 9th thoracic spinal cord (Th9). Muscle strength was normal in the upper extremities but a flaccid and complete paraplegia was present in the lower. Superficial abdominal
reflexes, deep tendon reflexes of the lower extremities, and plantar responses were absent. Sensation of touch, including pin prick, and perception of joint position were also absent below Th9, without sacral sparing (Fig. 1).

An electrocardiogram showed nonspecific changes of high amplitudes in precordial leads. A chest x-ray revealed mild cardiomegaly but demonstrated no mediastinal widening. Anteroposterior and lateral views of the thoracolumbar vertebrae indicated no fracture or dislocation. A computed tomographic (CT) scan of the thoracic spinal canal was obtained, but motion artifacts due to the severe pain prevented accurate interpretation. Immediately after CT scanning, a lumbar puncture was performed at L3-4; the cerebrospinal fluid was bloody, but this was thought to be due to trauma during the procedure. Myelography with iotrolan (10 ml at a concentration of 240 mg/ml) showed no block or stenosis in the spinal canal and no filling defect suggestive of abnormal vessels. A repeat CT scan demonstrated internal displacement of some intimal calcifications in the aorta, but showed no abnormalities within the spinal canal (Fig. 2).

The diagnosis was AAD with spinal cord ischemia involving both the anterior and posterior spinal arteries. Arterial blood pressure, monitored with an arterial catheter, was lowered with nitroglycerin and propranolol hydrochloride, and pain relief was achieved with morphine hydrochloride.

During the second CT procedure his low cervical pain returned to his back and he regained some strength in his legs. By the next day he had recovered fully except for residual paralytic ileus. The antihypertensive agents were administered intravenously for 19 days, and orally thereafter. Pre- and postcontrast CT scans obtained 30 days after the onset showed no displacement of intimal calcifications and no evidence of aortic dissection (AD). Aortography performed 59 days after the onset also failed to demonstrate dissection. Apparently, the dissection resolved spontaneously, as occasionally happens in fortunate cases. He recovered fully and was discharged 80 days after the onset.

Discussion

AAD often shows neurological manifestations and
carries a tragic prognosis. In 1972, Anagnostopoulos et al. reviewed 963 cases and found the mortality to be 50.1% within 48 hours of onset and 79.9% within 2 weeks. Many deaths were due to rupture. More recently, however, hospital survival rates have reportedly increased to as high as approximately 80%.6 Obviously, AAD requires very prompt treatment.

The presence of associated neurological signs and symptoms facilitates the differentiation of AAD from myocardial infarction. The reported incidence of neurological complications ranges from 7.8 to 46.0%,2,4,7,11,12,19,20,23 with paraplegia or paraparesis occurring in 1.2–8.0%.3,4,7,11,15,19,20 However, these may be underestimates, since some critically ill patients cannot be adequately examined.

If AAD produces spinal cord ischemia, the patient experiences severe back pain and acute paraplegia. As these symptoms resemble those in such spinal vascular diseases as hematomyelia, epidural or subdural hematoma, and simple anterior spinal artery ischemia,13,19,23,25 these disorders must be ruled out as soon as possible.

Generally, the symptoms of spontaneous spinal epidural or subdural hematoma develop gradually and progressively worsen,5,17 whereas in myelopathy due to AAD the onset of neurological signs and symptoms is abrupt, as occurred in our case. It should be noted, however, that some authors19,23 have reported gradual, progressive myelopathy with AAD. Also, in hematomyelia, complete transverse myelopathy can develop within minutes,13 as happened to our patient.

There have been several reports of AAD associated with transient ischemic attacks (TIAs) of the spinal cord.6,16,21,24,25 Moersch and Sayre25 addressed the discrepancy between the extensive tearing of the intercostal arteries and the absence of both neurological and pathological findings. According to their hypothesis, the dissecting blood flow stretches, narrows, and completely occludes the intercostal arteries, and further stretching leads to their rupture and reopening of the lumen, which then receives its blood supply from the false lumen of the AD. This hypothesis can also explain the TIAs of the spinal cord in patients with AAD. Suzuki et al.20 suggested that spasm of the intercostal arteries may account for transient myelopathy in patients with AAD. Inoue proposed that an intimal flap of the aortic dissection may temporarily obstruct the orifice of the intercostal arteries in a valve-like fashion.21

The neurological examination must be conducted carefully and the upper level of cord impairment determined. Patients with AAD can develop either anterior spinal artery syndrome16,21,24 or complete transverse myelopathy, depending on the degree of disruption of the spinal circulation.

A thorough physical examination is also essential in detecting AAD associated with acute paraplegia. Zull and Cydulka29 emphasized that AAD should be assumed if a patient with acute paraplegia has one of the following signs or symptoms: chest pain, migratory pain, pulse deficit, aortic insufficiency murmur, cardiac tamponade, stroke, or alteration in mental status. They also mentioned that AAD should be suspected in patients with paraplegia and hypotension, unless the etiology of the hypotension is known to be otherwise. Bruit due to stenosis of a major artery is also important.22 Our case was typical in that the pain first occurred in the back, later migrated to the neck, and finally returned to the back.

On chest x-rays, such findings as mediastinal widening, inward displacement of aortic intimal calcifications by more than 6 mm, a double aortic image, a change in the configuration of the aorta, tracheal deviation, and/or hemothorax suggest the presence of a dissection.14,18,22 However, they are rarely pathognomonic, as not in our case. A definite diagnosis requires CT (or magnetic resonance imaging, if available).

In patients with acute paraplegia, an intraspinal hematoma on precontrast CT scans is diagnostic. If no intraspinal hematoma is present, the following CT findings should suggest AD: internal displacement of aortic intimal calcifications; higher density in the acutely thrombosed false lumen than in the true lumen; enlargement of a long segment of the aorta; and pericardial or mediastinal hemorrhage.10,14,18,22 Of these, internal displacement of the calcified intima on precontrast CT is pathognomonic of AD and is reported in 40% of cases.10 If the above findings are absent, contrast medium should be administered either intravenously or intrathecally, and postcontrast images of patients with AD include the following: the presence of an intimal flap; opacification of only true lumen; and delayed opacification of the false lumen on dynamic scanning.10,14,18,22 The signs, symptoms, and radiological findings of AD are summarized in Table 1.

In the treatment of AAD with spinal cord ischemia, Lindsay and Hurst21 recommended that antihypertensive drugs be avoided. However, when further dissection is suspected, they should be administered. Fortunately, our patient recovered some strength in his legs at the time of the second CT scanning, which revealed AAD, and his neurological deficits disappeared during continuous intravenous

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administration of antihypertensive drugs.

In summary, in some cases neurosurgeons are the first physicians to encounter patients with AAD, as this disease can produce dramatic neurological symptoms. Since AAD is life-threatening and requires immediate treatment, neurosurgeons should be familiar with the signs and symptoms that suggest AAD and with the radiological findings that are diagnostic.

**References**


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1976


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