Acute Chest Pain and Paraparesis

Silja Räty, MD; Kirsi Rantanen, MD; Sophia Sundararajan, MD, PhD; Daniel Strbian, MD, PhD

Case Description

A 63-year-old man with a history of hypertension, paroxysmal atrial fibrillation, and dyslipidemia had a sudden intense chest pain radiating to his left arm, and he lost control of his legs. He was not on anticoagulant therapy and had no history of back trauma. During transport, he was hypotensive (blood pressure 88/51), his right arm was pale, and he was not able to move his legs. ECG showed inferolateral ST-segment depressions. He was given 250 mg aspirin and morphine for pain. With intravenous fluid-replacement therapy, his blood pressure rose and the activity of his legs improved. Aortic dissection was suspected, and the patient was urgently admitted to the surgical emergency room within the university hospital. In the emergency room, his blood pressure was 108/62. Cardiac and pulmonary auscultation and abdominal palpation were normal, and peripheral pulses were symmetrical. Computed tomography of the aorta revealed a hematoma next to the ascending aorta, raising a suspicion of type A aortic dissection reaching from the ascending aorta to the beginning of the left renal artery. The aortic valve was intact.

During an emergency operation, the ascending aorta and a part of the aortic arch were replaced with a prosthetic graft. The arrest time was 24 minutes, and closure of the aorta took 112 minutes. In the beginning, the patient needed vasoactive support because of hypotension but later on his hemodynamic was stable. However, the next day, the patient had to undergo a resternotomy because of postoperative bleeding. After the operation, he was hypertensive and was treated with antihypertensive medication.

The patient was extubated on the second postoperative day and mobilization was started the next day, when it was noticed that the patient could not stand on his feet and coordination of his lower limbs was impaired. On the fourth postoperative day, a neurologist was consulted. The patient was somnolent but oriented, and denied any back or limb pain. His cranial nerves were intact, and his upper limbs had normal strength and sensation. He could hold his left leg up for 5 and his right leg for 3 s in a prone position. He could not press/push against resistance neither distally nor proximally, the right leg being somewhat weaker, and tonus of his lower limbs was weakened. The patient reported impaired sensation for pain and light touch on his feet. Reflexes were normal, and Babinski sign was negative. There was no impaired sensation in upper body. Diuresis was monitored by means of urinary catheter.

Next, Magnetic Resonance Imaging of the Spinal Cord Was Performed.

What Would You Expect to Find?

Magnetic resonance imaging showed increased signal intensity of the anterior spinal cord between T11 and conus in T2-weighted images with edema and restricted diffusion in diffusion-weighted images. The finding suggested an ischemic lesion.

On the 19th postoperative day, sensory deficits were no longer found. The patient was able to walk with assistance of a walker, but distal and motor weakness was still detected, the right side being weaker. Proprioceptive and temperature sense was normal. Vibration sense was impaired in both upper and lower limbs, probably unrelated to the acute situation. Bowel function was normal but the patient was still catheter-bound, partially because of the postoperative complication (bleeding in the groin area).

A few days later, the patient was transferred to the intensive care unit because of septic shock, esophageal perforation, and gastrointestinal bleeding. Despite recurrent surgical interventions, antibiotics, maximal vasoactive medication, massive blood transfusions, and renal replacement therapy, the patient died on the 27th day after admission.

Discussion

Our patient had a motor paraparesis with mild sensory findings and possible paresis of the urinary bladder, suggesting of anterior spinal cord infarction but preserved dorsal column function. We concluded that aortic dissection had caused the infarction, the probable mechanism being occlusion of the anterior segmental medullary artery supplying lower thoracic and upper lumbar level. The fact that the patient had reported paraparesis preoperatively suggested that the causative factor was the dissection and not the operation. An alternative mechanism is hypoperfusion of the spinal cord because of the documented hypotension. The transient alleviation of symptoms after intravenous fluid therapy suggests that hypotension was at least a contributory factor.

Infarction of the spinal cord is a rare cause of paraparesis. It is commonly caused by atheromas involving the aorta and is a potential complication of thoracoabdominal surgery. Other causes include collagen-vascular disease, syphilitic arteritis,
embolic infarction, pregnancy, sickle cell disease, neurotoxic effects of iodinated contrast agents, compression of the spinal arteries by tumor, systemic arterial hypotension, and decompression sickness.1

Arteries supplying the spinal cord are branches of the vertebral, thyrrocervical, costocervical, intercostal, lumbar, and lateral sacral arteries. Two anterior thirds of the spinal cord are supplied by the anterior spinal artery and the posterior third by 2 posterior arteries, running along the entire length of the cord. They are joined by 6 to 9 large segmental medullary arteries originating from the same arteries except for lateral sacral arteries. In the cervical region, the blood supply is rich in collateral branches. In the thoracic region, the anterior spinal artery is joined by only a few branches from the thoracic aorta, making it more vulnerable to ischemia. Lower thoracic and lumbosacral areas are supplied by the largest medullary artery, the great anterior segmental medullary artery of Adamkiewicz, usually found at level L-1 or L-2 (occasionally as high as T-12 or as low as L-4). Conus and cauda equina are also supplied by the sacral branches ascending from the iliac arteries.

Aortic dissection begins with an intimal tear whereby blood enters the vessel wall and splays apart the laminar planes of the media to form a false lumen. The dissecting hematoma spreads along the laminar planes and may lead to occlusion of its branches. It may also rupture through the adventitia and cause massive hemorrhage, leading to hypotensive shock. The most common place for spontaneous dissections is the proximal ascending aorta that is susceptible to the greatest sheer stress. Aortic dissections are classified into 2 types. Type A involves the ascending aorta, whereas type B arises after the origin of the great vessels of the aortic arch. The most important risk factor for aortic dissection is hypertension.5

The typical manifestation of aortic dissection is acute intense chest pain that may radiate to back and propagate downward. If it affects the ascending aorta, it may cause cardiac complications such as myocardial infarction, aortic valve insufficiency, or cardiac tamponade. By occluding the arteries originating from the aorta or under general hypotension, dissection can cause hypoperfusion and ischemia of brain, kidneys, limbs, bowel, or spinal cord.

Neurological symptoms of aortic dissection include persistent or transient ischemic stroke, hypoxic encephalopathy, spinal cord ischemia, and ischemic neuropathy.5 Neurological involvement is reported in 17% to 40%.5 Proposed risk factors for neurological complications in type A aortic dissections include advanced age and classic type of dissection.5 At the onset, patients with neurological symptoms are more often pain free than those without (33% versus 5.6%).6 Neurological symptoms often manifest early after dissection and may rapidly resolve. It is speculated that this might be explained by transient arterial occlusion during the propagation of dissection.6 In some studies, neurological complications predicted poor outcome whereas others could not find a correlation.6,9

Spinal cord ischemia is reported in 1% to 9% of type A aortic dissections.4 However, it is more common in distal dissections, ranging ≤10%.7 Spinal cord ischemia in patients with aortic dissection can be caused by occlusion of the intercostal and lumbar arteries, the Adamkiewicz artery, or the thoracic radicular arteries or by hypotension. The most frequent location of infarction is the watershed zone in the middle thoracic spinal cord.1,7

The classic clinical presentation of spinal infarction is the anterior spinal artery syndrome: sudden (primarily plegic) paraplegia, local and radicular pain, loss of pain, and temperature sensation but preserved proprioception and vibration because of sparing of the dorsal columns.1 Importantly, partial syndromes are also seen because of vascular border zones created by anastomosis from penetrating branches of the spinal arteries. If ischemia affects only the motor horns of the spinal cord, pure motor clinical manifestations can be seen.1 It can also manifest as a transverse spinal cord infarction, Brown–Séguard syndrome, progressive myelopathy or transient spinal cord ischemia.4

Surgical treatment is always indicated in type A aortic dissections, whereas uncomplicated type B dissections can be treated conservatively. Medical treatment aims at managing blood pressure to prevent propagation of dissection without compromising adequate perfusion to vital organs. Two approaches are used in surgical treatment: open surgery, and, more recently, endovascular stent graft repair (TEVAR), which is mainly used in type B dissections. In open surgery, the dissected segment of the aorta is replaced with a prosthetic vascular interposition graft during cross-clamping of the aorta, usually during temporary circulatory arrest in type A. In TEVAR, an endovascular stent graft is placed in the true lumen through the femoral artery while avoiding cross-clamping of the aorta. Both techniques require temporary or permanent interruption of arterial collaterals supplying the spinal cord.

The reported frequency of spinal cord ischemia after thoracoabdominal surgery varies widely between 2.6% and 28% in open surgery and between 4% and 7% in endovascular procedures.8,9 Risk factors for postoperative spinal cord ischemia in thoracoabdominal surgery include aneurysm extent, open surgical repair, previous distal aortic operations, cross-clamp duration, the sacrifice of T9–L1 intercostal vessels, emergency operation, severe peripheral vascular disease, perioperative hypotension, and anemia.9,10 Different strategies for spinal cord protection and early detection of ischemia have been used. These include minimizing the surgical time, using hypothermia and pharmacological neuroprotection, protecting spinal cord perfusion by maintaining adequate mean arterial pressure, CSF drainage and by reimplantation of intercostal and lumbar segmental arteries, and using intraoperative monitoring of somatosensory and motor evoked potentials.10

TAKE-HOME POINTS

• Aortic dissection can be the cause of neurological dysfunction and should be considered in patients with chest pain, shock, asymmetrical pulses, or new heart murmur.
• In case of a sudden nontraumatic motor paraparesis, aortic dissection should be ruled out even in absence of chest pain.
• Paraparesis should be recognized as a possible complication of aortic surgery.
• Spinal infarction can be diagnosed with magnetic resonance imaging that should include diffusion-weighted images.
Disclosures

None.

References


Key Words: chest pain ◼ paraparesis ◼ spinal cord
Acute Chest Pain and Paraparesis
Silja Räty, Kirs Rantanen, Sophia Sundararajan and Daniel Strbian

Stroke. 2015;46:e111-e113; originally published online March 24, 2015;
doi: 10.1161/STROKEAHA.115.008635
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2015 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628
The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/46/5/e111

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published
in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office.
Once the online version of the published article for which permission is being requested is located, click
Request Permissions in the middle column of the Web page under Services. Further information about this
process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/