

## Type A Aortic Dissection Presenting with Isolated Paraplegia

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### ABSTRACT

Acute type A thoracic aortic dissections most commonly present with sudden onset of severe chest and/or back pain. We summarize the case of a patient with an acute type A dissection who presented with acute, painless paraplegia caused by malperfusion of the artery of Adamkiewicz. Although an uncommon cause of acute paraplegia, type A dissections should be included in the differential diagnosis.

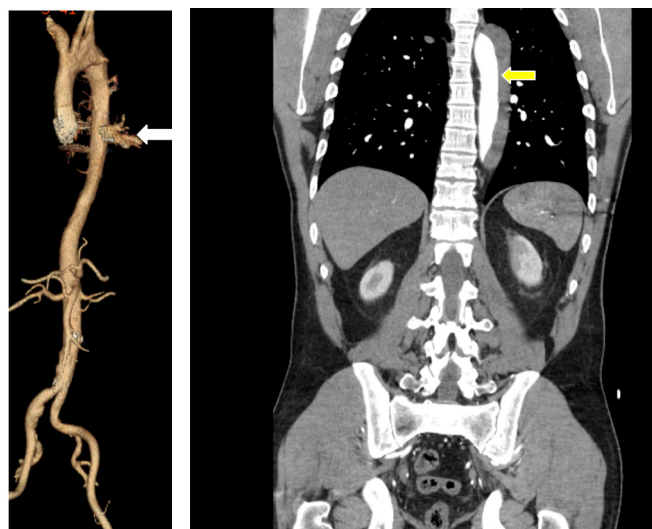
### INTRODUCTION

A delay in the diagnosis and treatment of an acute type A thoracic aortic dissection can significantly increase the associated morbidity and mortality. That can occur when a patient presents with symptoms that are not commonly associated with the pathology of an acute aortic dissection. In this report, we summarize the case of a patient with an acute type A dissection who presented with acute, painless paraplegia. The patient's paraplegia was due to spinal cord malperfusion secondary to an unusually large artery of Adamkiewicz that arose from the thrombosed false lumen.

### CASE REPORT

A 51-year-old man presented with acute onset of paralysis of his bilateral lower extremities. He denied any chest or back pain. His vital signs were normal except for severe hypertension, with a blood pressure of 180/90. His physical examination demonstrated motor and sensory loss below the waist and absent reflexes in both lower extremities. His medical history was significant only for hypertension. Magnetic resonance imaging of his spine demonstrated no bony spinal abnormalities but was suspicious for an aortic dissection. A contrast-enhanced computed tomography scan confirmed the presence of an aortic dissection, with an intimal flap seen in the proximal, mid, and distal ascending aorta, as well as in the aortic

arch and descending aorta, with the dissection terminating at the level of the aortic bifurcation. A large artery of Adamkiewicz arose from the false lumen of the dissected aorta and was thrombosed (Figure). The patient was transferred to our institution for management of his dissection. The patient was normotensive on esmolol and nitroprusside. A neurologic examination demonstrated flaccid paraplegia with an absent position and vibration sense in both lower extremities, and it revealed a sensory level at T10. The patient was emergently taken for repair of a type A aortic dissection. After placement of a spinal drain, a transesophageal echocardiogram confirmed the type A dissection. There was no aortic insufficiency or pericardial effusion. Operative findings included an intimal flap in the ascending aorta with an intimal tear just distal to the ostia of the left coronary artery. The aortic valve leaflets appeared normal. The ascending aorta and hemiarch were replaced, with the latter made by using antegrade selective cerebral perfusion via axillary artery cannulation. The patient had an uneventful postoperative course, although he remained paraplegic. He was discharged to a rehabilitation facility.



Shown are a computed tomography (CT) scan of the thoracic aorta and a 3-dimensional reconstruction. The large artery of Adamkiewicz is indicated (white arrow). It is not evident on the coronal view because the artery comes off the false lumen (yellow arrow).

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## DISCUSSION

The precipitating event in an aortic dissection is a tear of the intima. It is more common in males and generally presents in the fifth to seventh decades of life [Collins 2004; Raghupathy 2008]. The most common presentation is sudden onset of severe, sharp chest pain that can be knife-like or ripping. Patients can also have pain radiating to their back [Collins 2004; Raghupathy 2008]. Death can occur from rupture of the aorta into the pericardium, thorax, or retroperitoneum. When the dissection involves the aortic root, there can be hemodynamic consequences, which can be caused by heart failure due to acute aortic insufficiency from a loss of the structural integrity of one or more of the aortic valve commissures. Additionally, if there is a large pericardial effusion, the patient can develop cardiac tamponade. If the dissection extends into the coronary arteries, coronary malperfusion and myocardial ischemia may result. Finally, if there is a hematoma of the aortic root with compression of the atrioventricular conduction tissue, heart block can ensue [Collins 2004; Raghupathy 2008]. Patients can also exhibit signs of peripheral malperfusion leading to acidosis and hemodynamic instability [Collins 2004; Raghupathy 2008]. For example, if blood flow to the mesenteric arteries is limited because of one or more of the arteries originating from the false lumen or because of other complex flow patterns of entry/reentry that decrease flow to the mesentery, mesenteric ischemia can result, causing abdominal pain, bloody diarrhea, and/or acidosis. If cerebral perfusion is inadequate because one or more arch vessels are involved in the dissection, patients can experience neurologic deficits and/or strokes. Suboptimal flow into the renal arteries can cause acute renal failure. Obstruction of the common iliac arteries or a more distal artery to the extremity can cause acute lower-extremity ischemia. Finally, obstruction of the intercostals or lumbar arteries can cause paraplegia [Zull 1988; Aktas 2008]. Whether such an obstruction leads to transient or permanent paraplegia largely depends on the time from the onset of symptoms until surgical correction of the aortic dissection. It is therefore critical to make the diagnosis and proceed with surgical intervention expeditiously. Malperfusion of the artery of Adamkiewicz causes severe inadequacy of spinal cord perfusion, leading to acute paraplegia. The midthoracic segment of the spinal cord is the most susceptible to ischemic injury because it is in the watershed zone, located between the blood supplies of the upper and lower spinal cord. The patient presented in this case report had an unusually large artery of Adamkiewicz that arose from the thrombosed false lumen of the dissected aorta. Given the associated large territory of the spinal cord perfused by this artery, significant cord ischemia resulted.

The differential diagnosis of paraplegia depends on whether it is acute or subacute/chronic. Acute paraplegia suggests a vascular etiology with a differential diagnosis that includes embolism of the anterior spinal artery, hypotensive shock with compromised blood flow to the spinal cord watershed area, epidural or subdural hemorrhage, hemorrhage into a spinal cord tumor, and aortic dissection [Zull 1988]. The differential diagnosis for chronic paraplegia includes Guillain-Barré syndrome, multiple sclerosis, poliomyelitis, epidural abscess, epidural tumor, postinfectious transverse myelitis, cervical disk, and spondylolisthesis [Zull 1988]. Therefore, determining whether the onset was acute or chronic is critically important when evaluating a patient with paraplegia. Acute paraplegia should lead one to consider and rule out a vascular insult. If the patient is not receiving anticoagulation therapy and has no history of a bleeding diathesis or cancer, then an acute aortic dissection should strongly be considered. If acute paraplegia is accompanied by chest pain, a pulse deficit, cardiac murmur consistent with aortic insufficiency, and/or signs/symptoms of peripheral organ malperfusion, acute aortic dissection is likely. Furthermore, if acute paraplegia is accompanied by hypotension, the presumptive diagnosis should be an acute aortic dissection.

## CONCLUSION

An acute ascending aortic dissection should be considered in the differential diagnosis of a patient presenting with acute, painless paraplegia. Paraplegia can be caused by malperfusion of one or more of the arteries that originate directly from the thoracic aorta, such as the artery of Adamkiewicz. Like any other patient presenting with a type A aortic dissection, these patients should proceed emergently to the operating room, although the paraplegia is likely to be permanent.

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