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SUDDEN ONSET PARAPLEGIA AS THE PRESENTATION OF AN ABDOMINAL AORTIC ANEURYSM

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<p>Article Info <i>Received 14/11/2015</i> <i>Revised 22/11/2015</i> <i>Accepted 25/11/2015</i></p> <p>Key words: Aortic aneurysm, Paraplegia, Spinal cord infarction, Adamkiewicz.</p>	<p>ABSTRACT A 56 year old male presented with the sudden onset of paraplegia. An emergency department ultrasound showed an abdominal aortic aneurysm. This was followed by a CT scan which showed it was infrarenal and extended through the iliac vessels with intraluminal thrombus. The case is presented with a brief discussion on acute paraplegia and a review of the spinal cord vascular supply.</p>
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INTRODUCTION

Abdominal aortic aneurysms are always a concern for emergency physicians and a high index of suspicion must always be maintained. The consequences of missing an early presentation of an expanding or leaking aneurysm can be devastating to the patient as well as the emergency physician. Paraplegia as a presenting feature of an aortic aneurysm is uncommon and eight cases have been reported in the literature [1-6]. We present a case of sudden onset paraplegia as the presenting symptom of an expanding abdominal aortic aneurysm. This is the first case of an aortic aneurysm, and not an aortic dissection or a complete aortic occlusion, resulting in paraplegia reported in the emergency medicine literature.

CASE REPORT

A 56 year old white male presented to the emergency department complaining of the sudden inability to walk. He awoke in the middle of the night to have a bowel movement. After which, he claimed to have no pain, no feeling or strength in his legs, and the inability to get off the commode. His significant other lifted him off

the commode as he could not stand unassisted. She activated the 911 system and he was transported to our hospital on a long board by paramedics. Shortly after arrival in the emergency department he developed generalized abdominal pain.

Past medical and surgical history consists of cardiac disease of an unknown nature and peripheral neuropathy unrelated to diabetes. He is on several medications but did not know their names. There are no known allergies. He lives with his girlfriend and denies alcohol or tobacco consumption. Despite being a poor historian, review of systems is negative for trauma, fever, chills, nausea, vomiting, diarrhea, melena, hematochezia, chest pain, or dyspnea. He denied any other gastrointestinal, genitourinary, respiratory, cardiac, or neurologic symptoms.

Physical examination showed him to be awake, alert, oriented, diaphoretic, and in moderate discomfort. Vital signs revealed a blood pressure of 170/120, pulse of 100, temperature of 98.8°F, and respirations of 56 with abdominal breathing. The head examination was



unremarkable. The neck was supple without bruits or jugular venous distention. The lungs were clear to auscultation and percussion. The heart had a regular rhythm without any murmurs, gallops or rubs. The abdomen was obese, soft and non-tender. Hypoactive bowel sounds were noted with no hepatosplenomegaly. Crepitation was felt in the left lateral abdominal wall. Rectal exam revealed no sphincter tone. The stool was negative for occult blood. The genital exam was unremarkable. Pulses were equal and symmetric in all four extremities. The lower extremities were without edema, but were mottled. Blood pressures were obtained in all four extremities and no discrepancies were identified. Neurological examination showed no deficits in cranial nerve function. The upper extremities had normal strength, reflexes, and sensation. The torso was noted to have no sensation below the umbilicus. The lower extremities were areflexic with muscle strength of 0/5. He was unable to identify light touch, pinprick, or vibratory sensation to the lower extremities.

This patient had sudden paraplegia with later developing abdominal pain, abdominal wall crepitation, and mottled lower extremities with equal pulses. These findings suggested an expanding or leaking abdominal aortic aneurysm or an aortic dissection. Our emergency department is equipped with an ultrasound machine and attending physicians certified in ultrasonography. An emergency department ultrasound was performed which showed an abdominal aorta measuring 8 cm in diameter with an intraluminal thrombus. Vascular surgery was contacted, evaluated the patient, and requested an abdominal CT scan.

The CT scan showed an infrarenal abdominal aortic aneurysm (fig. 1) that extended to the aortic bifurcation. The aneurysm had an 8 cm anteroposterior diameter. A thick layer of lining mural thrombus was noted with a residual lumen (figs. 1 & 2). The renal, celiac and superior mesenteric arteries were patent and uninvolved. The inferior mesenteric artery was totally occluded. There was no dissection of the aortic wall. The abdominal organs were unremarkable.

The patient was taken to the operation room immediately after the CT scan. The abdominal aortic aneurysm was repaired without difficulty. It was then noted that the mid-descending and sigmoid colons were infarcted. A total colectomy was performed during which the patient became hypotensive requiring vasopressor support and developed disseminated intravascular coagulation. During the surgery he required 15L lactated ringers, 3L normal saline, 4 units albumin, 2 eight-packs of platelets, 5 units of cell-saver red blood cells, and 6 units of packed red blood cells. He was transferred to the surgical intensive care unit in critical condition. Shortly thereafter, he suffered a cardiac arrest and was unable to be resuscitated.

An autopsy was performed in our hospital. The patient was noted to have three vessel coronary artery disease with occlusion ranging from 10-80%. Evidence of an old myocardial infarction was present in the anterior left ventricle and septum, extending from the base of the heart to the apex. There was no evidence of an acute myocardial infarction. He was also found to have cholelithiasis and hyperlipidemia. The spinal cord showed acute ischemic necrosis from T-10 through L-4.

Fig 1. Longitudinal section of a CT scan of the Abdomen demonstrating an infrarenal aortic aneurysm extending into the iliac vessels.

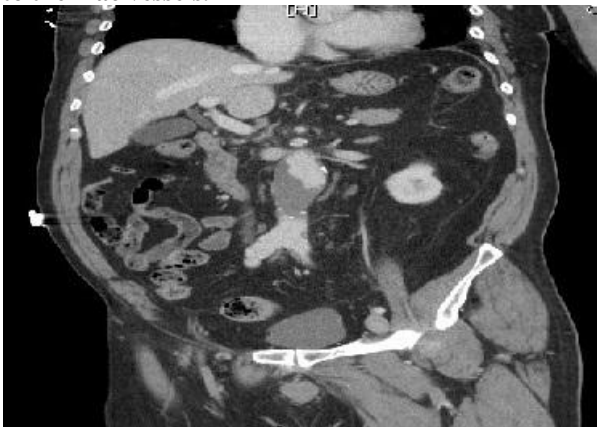


Fig 2. Cross section of a CT scan of the Abdomen demonstrating an infrarenal aortic aneurysm and intraluminal clot.



DISCUSSION

The presentation of an abdominal aortic aneurysm can vary significantly [7]. It may be an incidental finding on physical examination, ultrasonography, or CT scan. Patients may complain of the sudden onset of abdominal or back pain. Syncope can be the presenting symptom if an

acute rupture occurs. The pain associated with aneurysmal expansion can be mistaken for cholelithiasis, myocardial infarction, pancreatitis or renal colic. Physical examination may reveal a pulsatile mass or it may be normal.

The etiology of acute and sudden paraplegia is usually due to spinal cord ischemia, spinal cord infarction,



or trauma to the spinal cord [8]. This can occur from arterial occlusion, hypotension, spinal cord transection, intervertebral disc herniation compressing the spinal cord or more commonly its arterial supply, an acute epidural bleed, epidural anesthetic injection, embolic phenomena or thrombosis [3, 9-13]. It is also commonly seen in association with aortic dissections and complete aortic occlusion [14-19].

Aortic dissection causing acute paraplegia usually presents with other symptoms [20]; although isolated neurological events have been reported [17, 19]. The neurological deficits are thought to be due to shearing and/or thrombosis of the artery of Adamkiewicz [21, 22]. Acute paraplegia associated with spinal cord infarction is a frequently seen complication in 2-8% of people with aortic dissections [23]. Spinal cord infarction has also been reported after traumatic rib fractures causing intercostal artery thrombosis [9].

We presented a case of acute paraplegia associated with an expanding abdominal aortic aneurysm. Acute paraplegia is a known complication of aortic aneurysm repair or other operations that cause stasis of aortic blood flow [24-26]. The paraplegia is often permanent; although recovery may be seen in rare cases [27]. To understand the pathogenesis of spinal cord infarction, the vascular supply of the spinal cord will be reviewed.

In the human, there are thirty-one pairs of segmental arteries. In the thorax, the segmental arteries are also known as intercostal arteries. In the abdomen, the segmental arteries are known as lumbar and sacral arteries. The segmental arteries have dorsal branches that supply the back, vertebral column and spinal cord. Radicular branches from the dorsal branches of the segmental nerve traverse the neural foramina to enter the spinal canal. Once inside the neural foramina, the radicular arteries divide into anterior and posterior rami. The anterior rami may anastomose with the anterior spinal artery but primarily supply the vertebral bodies and meninges. The posterior rami may anastomose with the posterior spinal artery and also supply the posterior bony vertebra. Of the thirty-one pairs of segmental arteries, seven or eight will contribute to the spinal cord blood supply [28].

The arterial supply to the spinal cord is from the anterior spinal artery and paired posterior spinal arteries. The anterior spinal artery arises from the vertebral arteries and terminates in the conus medullaris. It supplies the anterior two-thirds of the spinal cord. Instead of being one continuous artery, there are several areas of atresia or non-communication. The anterior spinal artery thus relies on collateral supply from the anterior rami of the radicular arteries for its blood supply. This makes the spinal cord particularly vulnerable if its blood supply is interrupted. The paired posterior spinal arteries are patent the entire length of the spinal cord and supply the posterior one-third of the spinal cord. It receives collateral supply from the

posterior rami of the radicular arteries but is not dependent on any individual collateral artery for its supply.

The artery of Adamkiewicz, also known as arteria radicularis magna, is a radicular artery of particular importance [29]. This vessel anastomoses with the anterior and posterior spinal arteries to supply the lower thoracic and lumbar spinal cord as well as the conus medullaris. This vessel can originate from an intercostal or lumbar artery (30). Its origin can be traced from T-8 to T-12 in 80% of people and from L-1 to L-4 in 15% of people [28,31]. It enters the spinal canal from the left side in over 80% of people [30-32]. When the artery of Adamkiewicz arises in the thoracic area, it supplies the thoracic spinal cord while lumbar collateral branches supply the distal spinal cord [28]. When the artery of Adamkiewicz arises in the lumbar area, it supplies the distal spinal cord while the thoracic spinal cord receives its blood supply from the collaterals of intercostal arteries [28]. If blood flow is obstructed to the artery of Adamkiewicz, spinal cord ischemia may occur and progress to infarction if not recognized.

Our patient had presented with isolated paraplegia. We hypothesize that this paraplegia is due to thrombotic obstruction of the artery of Adamkiewicz resulting in spinal cord ischemia and infarction. The intraluminal clot noted in the aorta may have obstructed the ostia of the artery of Adamkiewicz or propagated into this artery. It is also possible that diminished flow in the artery of Adamkiewicz caused stasis and primary intraluminal thrombus formation in this artery. The exact answer will not be known since the specimen was not saved for pathological evaluation after the abdominal aortic aneurysm was repaired.

CONCLUSION

An abdominal aortic aneurysm should be included in the differential diagnosis of any patient presenting with acute, nontraumatic, spinal cord infarction. Rapid identification of this condition followed by immediate surgical repair may prevent permanent paraplegia.

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None

CONFLICT OF INTEREST

The author declares that he has no conflicts of interest.

STATEMENT OF HUMAN & ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by the author. This report was found by the institutional review board to be exempt (#69-03)



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