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Case Report

Acute aortic occlusion leading to spinal cord ischemia in a 73-year-old: A case report

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ABSTRACT

Background: Cauda equina syndrome (CES) is typically caused by a compressive etiology from a herniated disk, tumor, or fracture of the spine compressing the thecal sac. Here, we report a CES mimic – acute aortic occlusion (AAO), a rare disease that is associated with high morbidity and mortality. AAO can compromise spinal cord blood supply and leads to spinal cord ischemia.

Case Description: Our patient presented with an acute onset of bilateral lower extremity pain and weakness with bowel/bladder incontinence, a constellation of symptoms concerning for CES. However, on initial imaging, there was no compression of his thecal sac to explain his symptomology. Further, investigation revealed an AAO. The patient underwent an emergent aortic thrombectomy with resolution of symptoms.

Conclusion: AAO can mimic CES and should be considered in one's differential diagnosis when imaging is negative for any spinal compressive etiologies.

Keywords: Acute aortic occlusion, Cauda equina syndrome, Spinal cord ischemia

INTRODUCTION

Cauda equina syndrome (CES) is caused by compression of the thecal sac and nerve roots due to a herniated disk at L4–L5 or L5–S1.[15] Other etiologies of CES include inflammatory conditions, traumatic injuries, tumors, and degenerative changes.[14,16] CES commonly presents with bowel and bladder dysfunction, saddle anesthesia, and sexual dysfunction due to compression of sacral nerve roots; however, the presentation is highly variable with many patients reporting severe back pain and radiculopathy.[14,15] CES can present both acutely and with chronic symptoms, with the timing of symptom onset and progression being important prognostic factors. The management of CES requires urgent neurosurgical intervention. Studies indicate that decompressive surgery should be performed within 48 h of patient presentation to prevent long-term complications.[14,16] If a patient presents with symptoms mimicking CES with no compressive etiologies on magnetic resonance imaging (MRI) images to explain these symptoms, one should be aware of vascular etiologies that can mimic CES.

Spinal cord infarction is a serious complication that can develop following an acute aortic occlusion (AAO) and comprises 1–2% of all strokes.[24] Spinal cord infarction presents with acute onset of a constellation of symptoms, including upper and/or lower extremity paralysis, bowel/bladder dysfunction, pale skin, and/or absent pain and temperature perception with preserved proprioception and vibratory sensation.[17,24] AAO is an uncommon condition that carries high morbidity and mortality. A large study conducted by Grip *et al*. revealed the incidence of AAO to be 3.8/1 million person-years with a mean age of 69.7 years.[11] Other studies have reported the incidence of the primary AAO to be 1–4%. According to earlier studies, the 30-day mortality rate in patients with AAO ranges between 19.9% and 52.5%. Complications that result as a consequence of spinal cord ischemia include lower extremity ischemia and paralysis, bowel ischemia, and bladder/bowel incontinence.[9,10,11,22,25,26] Risk factors that are commonly associated with AAO include cardiac disease, hypertension, diabetes mellitus, tobacco use, and female gender.[10,21]

We present a case of a 73-year-old male with acute onset of bilateral lower extremity pain and weakness as well as urinary incontinence, concerning for CES, who was subsequently found to have an AAO caused by an aortic thrombus, resulting in a spinal cord ischemia.

CASE REPORT

A 73-year-old male with a history of atrial fibrillation on warfarin (INR 1.9) presented with an acute onset bilateral lower extremity weakness, pain, and sensory changes, with a loss of bowel and bladder function and saddle anesthesia. The symptoms began spontaneously while resting in a seated position. The patient's physical examination was remarkable for significant weakness in the right distal lower extremity, and left proximal lower extremity, with bilateral lower extremity loss of fine touch and pinprick sensation from the knee down. The patient was incontinent with poor perineal sensation and absence of any rectal tone. Emergent MRI of his neuroaxis was performed given the concern for CES or spinal cord injury. MRI was unremarkable for any acute neurologic process, cord edema, or disk herniation. Simultaneously, the patient was treated with intravenous dexamethasone and titration of his mean arterial pressure above 90 mmHg. His examination improved significantly in a matter of hours with some residual pain, weakness, and sensory changes in his lower extremities. Interestingly, the patient also tested positive for COVID-19 on arrival.

Given the patient's clinical picture and the lack of MRI findings of cord compression, the differential diagnosis widened to include a diagnosis of a potential spinal cord stroke. Arterial duplex examination of both LEs demonstrated minimal flow bilaterally. Diminished pulses were noted in the common femoral, profunda, and popliteal arteries bilaterally. The blunted bilateral signal indicated more proximal obstruction. On second review, his lumbar spine MRI depicted signal change in the aorta concerning for a thrombus [Figure 1]. The vascular surgery team was immediately consulted, and computed tomography (CT) angiography scan of the chest and abdomen was ordered. CT angiography findings showed an occlusion of the infrarenal abdominal aorta and bilateral common iliac arteries [Figure 2]. The patient was taken for emergent aortoiliac thrombectomy by vascular surgery. They also performed emergent bilateral 4-compartment fasciotomies at the bedside. After revascularization, his strength improved, and his distal pulses were palpable. The patient was started on a heparin drip and was transferred to the Intensive Care Unit for close monitoring. Due to the improvement in his neurological examination and need for anticoagulation, a lumbar drain was not placed.

One week following presentation, angiography was performed and demonstrated resolution of the aortic thrombus, as shown in Figure 2b. At his 2-month follow-up, he was ambulatory with a walker and only complained of mild dysesthesias in the left foot.

DISCUSSION

AAO is a rare vascular condition that is associated with a mortality rate as high as 75%.[16,21,24] This vascular emergency may result from a variety of etiologies, such as a saddle embolism at the aortoiliac bifurcation, acute *in situ* thrombosis on a background of severe occlusion, aortic dissection, acute thrombus development following aortic dissection or aneurysm,[28] or aortic trauma. Atypical causes include hypercoagulability, vasculitis, and fungal infections.[2,5,13,19,29] In a recent retrospective series of 29 cases of AAO in a single institution, thrombosis was found as the cause in 76% of cases, and more than 40% of patients had a hypercoagulable state due to either antiphospholipid antibody syndrome (17%) or cancer (24%).[9] Although some studies have found, the primary factor influencing mortality is the amount of time to revascularization,[2] other studies have found that the neurovascular examination of the extremities at presentation is more strongly associated with mortality.[10]

Spinal cord ischemia from AAO can present as acute onset paralysis, bowel and bladder dysfunction, pain, and absent temperature sensation, similar to what our patient presented with. The anterior two-thirds of the spinal cord, including the anterior horns, anterior commissure, anterior funiculi, and, to a lesser extent, the lateral funiculi, are primarily supplied by the anterior spinal artery.[20] The anterior spinal artery receives blood flow from thoracic intercostal branches and lumbar radicular arteries. A large segmental posterior radicular artery, the artery of Adamkiewicz, usually arises

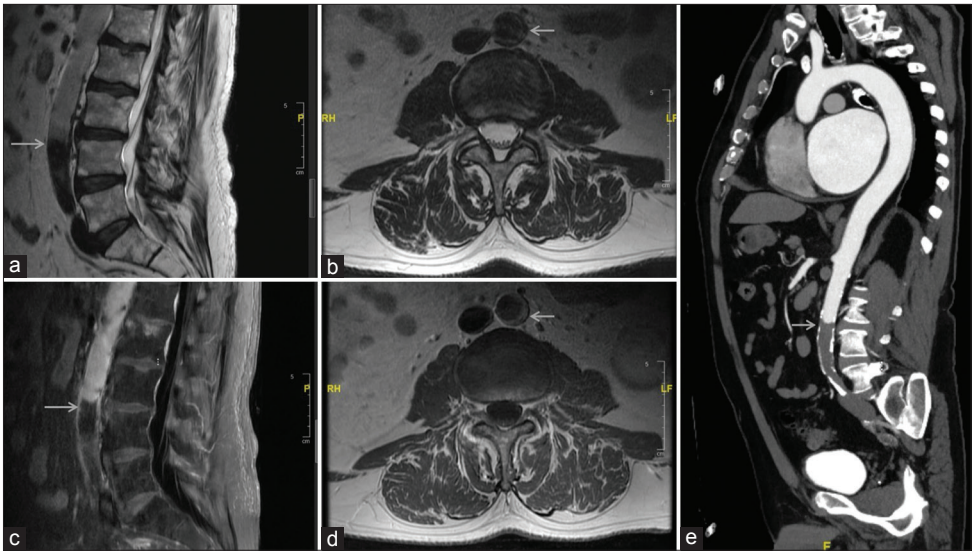

Figure 1: Magnetic resonance imaging of the lumbar spine. (a) T2-weighted image, sagittal. (b) T2-weighted image, axial. (c) T1 postcontrast, sagittal. (d) T1 postcontrast, axial. (e) computed tomography angiography of the chest, sagittal. The arrows depict the abrupt lack of contrast in the aorta, confirming acute aortic occlusion.

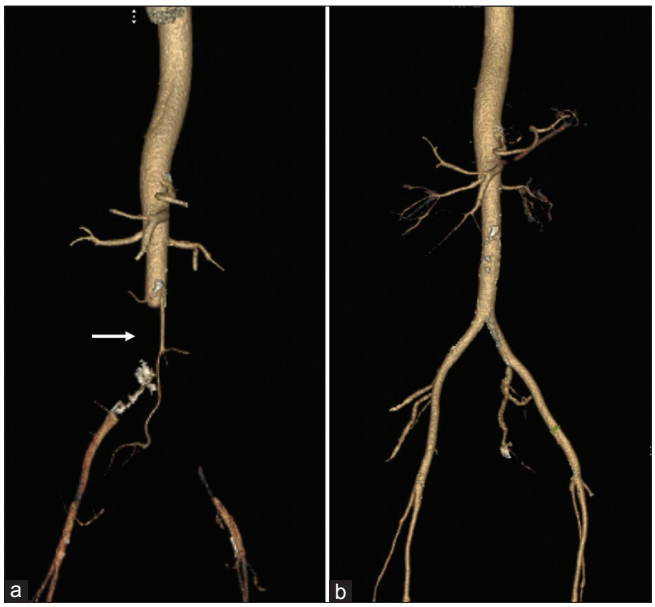

Figure 2: Three-dimensional aortic reconstructions. (a) Preoperative reconstruction demonstrating acute aortic occlusion. (b) Postoperative reconstruction after aortoiliac thrombectomy confirming aortic patency without residual thrombus. The arrow depicts an abrupt filling defect in the aorta confirming acute occlusion.

from the intercostal vessels between T9 and T12 in roughly 75% of patients[8] and supplies perfusion to the lumbar spinal cord and the conus medullaris. Blockage of blood supply in this region can lead to anterior cord syndrome, manifesting with a sudden onset of radicular pain, flaccid paralysis, urinary and fecal incontinence, and diminished pain and temperature sensation with spared proprioception and vibratory sense.[7,23,24,26] This symptomology greatly overlaps with what is seen in CES, which is why CES was initially suspected in our patient.

Imaging plays a key role in the diagnosis of AAO and is valuable for assessment and management.[4,6,18] The gold standard technique used for diagnosing AAO is aortography. Although it is time-consuming, this procedure carries the highest yield as it can simultaneously be utilized for preoperative assessment. Another useful imaging technique used in the prompt diagnosis of AAO is contrast-enhanced CT, as was used in our patient. This study is more readily available in general hospitals than aortography. An added advantage of contrast-enhanced CT is the ability to detect aortic dissections or aneurysms as the cause of occlusion. Deep duplex ultrasonography has also been useful in the evaluation of AAO as it is quick, accessible, and noninvasive and performed rapidly.[3]

The primary objective in the management of AAO is prompt restoration of blood flow. Once a diagnosis of AAO is made, administration of IV hydration and heparin while maximizing cardiac function is crucial steps in the treatment. Surgical intervention is recommended in patients with AAO unless the ischemia is deemed irreversible or the patient is too unstable for surgery. Conservative management with anticoagulation alone is unfavorable due to the high mortality associated with this practice.[2,30] Several factors should be considered before surgical intervention, such as the specific etiology of AAO, anatomic variations, and general surgical risk. Surgical interventions include urgent revascularization

with thrombectomy, direct aortic reconstruction, or anatomic bypass procedures.[22] Patients with infrarenal aortic occlusion may benefit from aortic reconstruction due to the risk of embolus from the distal aorta to the renal and mesenteric arteries proximally.[2]

In our study, the patient had several risk factors that were associated with the development of AAO and subsequent spinal cord ischemia. He had atrial fibrillation being treated with warfarin. Echocardiography performed on admission ruled out embolism of a mural thrombus. The patient also had dyslipidemia, hypertension, a history of prostate cancer, and a positive COVID test that placed him at a higher risk for thrombosis.[27]

Following diagnosis, the patient underwent bilateral aortoiliac thrombectomy. The paralysis in his bilateral Les improved after revascularization. At a follow-up visit after 2 months, he endorsed significant improvement in his strength. He was however still experiencing burning dysesthesias in his left lower extremity that was treated with neuropathic pain medications.

A patient with AAO can present with a constellation of clinical features mimicking CES. To avoid a delay in diagnosis, physicians must consider vascular etiologies and routinely perform neurovascular examinations when imaging for spinal cord or thecal sac compressive etiologies are negative.[2,3,9,12] After prompt revascularization, most cases of AAO with an evolving neurologic deficit achieve partial or total recovery.[1,2,28]

CONCLUSION

AAO is a rare vascular emergency that can mimic CES and should be considered in the differential diagnosis when lumbar imaging is negative for any compressive pathologies of the thecal sac. A thorough neurovascular examination of the lower limbs should be performed to assess distal pulses and ultimately avoid a delayed diagnosis, especially in elderly population. Prompt diagnosis of AAO prevents critical delays in revascularization and can potentially mitigate complications and improve patient outcomes.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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