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Case Report

Aortoiliac occlusion mimicking cauda equina syndrome, a diagnostic dilemma: A case report and review of the literature

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ABSTRACT

Background: Cauda equina syndrome (CES) is a consequence of a variety of etiologies. CES is most commonly due to compression of the thecal sac and nerve roots by a massive disc herniation. However, it rarely presents secondary to aortic occlusion. Aortoiliac occlusive disorder is usually associated with chronic claudication, erectile dysfunction, and diminished lower limb pulses. Acute aortic occlusion, however, is associated with serious complications such as spinal cord infarction and ischemia. It is also associated with a high risk of morbidity and mortality. Moreover, it poses a diagnostic challenge and may be overlooked. This report emphasizes the importance of considering vascular etiology as a differential diagnosis for CES.

Case Description: This case report describes a unique case of aortic occlusion mimicking CES in a 56-year-old female patient.

Conclusion: For patients presenting with cauda equina symptomatology, it is critical to consider vascular etiology, especially for those with cardiovascular risk factors. Spine surgeons and emergency physicians should maintain a high index of suspicion for vascular etiologies and consider appropriate imaging studies to promote early diagnosis and intervention to prevent subsequent neurological and life-threatening consequences.

Keywords: Abdominal aorta, Acute aortoiliac occlusion, Cauda equina syndrome, Emergency physician, Spine surgeon

INTRODUCTION

Cauda equina syndrome (CES) is an uncommon condition that affects <1 in 100,000 patients annually. This syndrome is a consequence of a variety of etiologies, most commonly compression of the thecal sac and nerve roots by massive disc herniation.[13] CES is relatively rare and challenging to diagnose, with no agreed-upon clinical diagnostic criteria.[4,9] Common signs and symptoms of CES may include pain in the lower back, sphincter (bowel and urinary) dysfunction, saddle anesthesia, and lower limb weakness and radicular pain.[5] CES is a spinal surgical emergency that, if not recognized and emergently treated, may progress to paraplegia and/or permanent sphincter dysfunction.[1] The clinical presentation may mimic other differential diagnoses, including acute aortoiliac occlusions.

This report emphasizes the importance of considering vascular etiology as a differential diagnosis for CES. Here, we present a challenging clinical scenario of a unique case of aortic occlusion mimicking CES.

CASE PRESENTATION

A 56-year-old female patient with hypertension, hyperlipidemia, and ischemic heart disease underwent coronary artery bypass grafting in 2016. She had a recent history of non-ST-segment elevation myocardial infarction and dual antiplatelet therapy. She was brought to the emergency department with the chief complaint of acute onset of severe lower back pain, bilateral lower limb weakness, non-dermatomal bilateral lower limb pain, and urinary incontinence. Upon examination, the patient experienced distress and pain despite receiving appropriate analgesic medications, including narcotics. The patient was conscious, febrile, and hypertensive (220/104). The power in the left lower limb was 0/5, and 3/5 in the right lower limb in all muscle groups. She had absent deep tendon reflexes and diminished sensation in both lower limbs. The anal tone was weak, but no saddle anesthesia was present. The lower limbs were not cold, and pedal pulses were palpable but diminished. An emergent magnetic resonance imaging (MRI) of the lumbar–sacral spine was performed and showed an L4–L5 diffuse disk bulge indenting the ventral thecal sac and causing bilateral mild-to-moderate neural foraminal stenosis, mild spinal canal stenosis, and bilateral facet arthrosis [Figure 1]. Cervical and thoracic MRIs were also included, given that she presented with a fever and had a recent central catheter insertion to rule out a spinal abscess. However, the results were unremarkable. MRI of the whole spine at this point was not suggestive of any spinal-related etiology. A repeated physical examination a few hours after presentation revealed 0/5 power in both lower limbs, complete loss of sensation, cold extremities, and absent pulses in both lower limbs. Bedside lower limb Doppler ultrasound showed no pulses. Repeated lactic acid increased from 1.3 mmol/L to 6.8 mmol/L. Computed tomography (CT) abdomen with contrast was conducted, and it showed extensive diffuse atherosclerotic changes affecting the abdominal aorta with branches extending to the lower limbs arterial system, which were more severe on the left. Nonopacification of the aorta from the level below the renal arteries extended to the common iliac arteries, and the contrast continued in the external iliac arteries (likely through collaterals) opacifying the right lower limb arteries with the presence of segments of severe atherosclerotic contrast attenuation and faintly opacifying the left lower limb arteries. Hypodensity in the spleen likely represented partial splenic infarction [Figure 2].

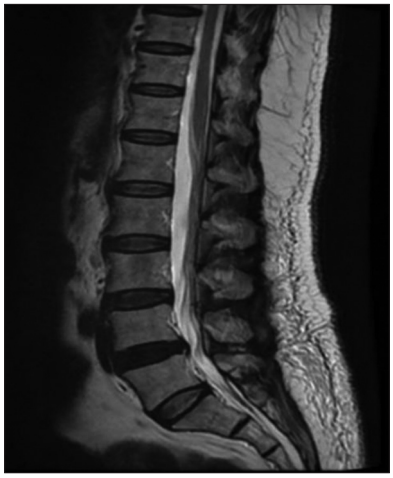

Figure 1: Magnetic resonance imaging of the lumbar–sacral spine without contrast shows multilevel lower lumbar spine degenerative disc disease that is more pronounced at L4–5.

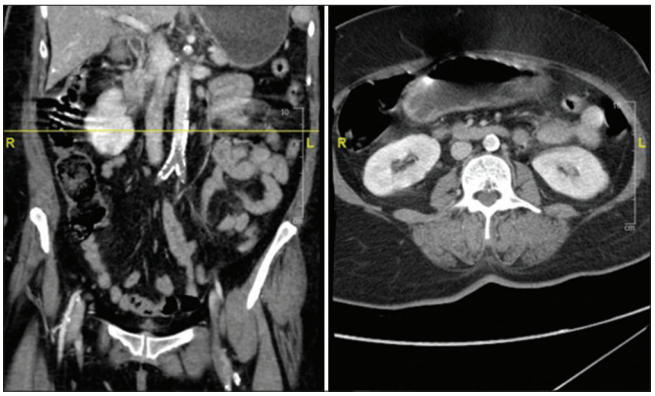

Figure 2: Computed tomography angiography abdomen aortogram multiple cuts showing a filling defect in the abdominal aorta starting below the level of the renal arteries extending the bilateral proximal part of the common iliac arteries. (The hallmark - Filling defect).

The patient then underwent emergent bilateral aortoiliac embolectomy and bilateral four-compartment lower limb fasciotomy. Postoperatively, there was no improvement in neurological function. She was started on heparin infusion, and dual antiplatelet therapy was initiated. The patient's condition continued to deteriorate a few days later, and she died due to a cardiac event.

DISCUSSION

Acute aortic occlusion (AAO) is an uncommon vascular disorder and emergency that is associated with a mortality rate ranging from 20% to 75%.[10,14,16,18] AAO etiologies include embolization into the aortoiliac bifurcation, acute aortoiliac thrombosis, aortoiliac dissection or aneurysm-

related acute thrombus formation, and aortoiliac trauma.[19] Atypical causes include hypercoagulable state, vasculitis, and fungal infections.[6,12,15,20] Cardiovascular disorders are the most commonly associated comorbidities with AAO.[10]

Chronic aortoiliac occlusive disorder is likely to manifest as chronic erectile disorder, claudication, diminished lower limb pulses, cool extremities, cyanotic extremities, compartment syndrome, and myonecrosis.[3] AAO, however, is associated with serious complications ranging from ischemic neuropathy and spinal cord dysfunction to permanent spinal cord infarction. In addition, it imitates CES, poses a diagnostic challenge, and may be overlooked.[17] AAO may present with profound systemic response, including tachycardia and diaphoresis. Furthermore, it may present with cold extremities, ischemic cutaneous manifestations, and faint or pulselessness in addition to out-of-proportion abdominal pain and oliguria/anuria if there is concomitant renal or visceral involvement.[2,8] CT is the modality of choice due to its wide availability and it is quick to perform. It shows the extent and mechanism of the disease, vessel patency, and signs of poor perfusion.[11]

Certain aspects of management, including distinguishing between embolism and thrombosis etiologies before surgery, the use of aortography, the best initial treatment modality, the preferred course of management, and the role of long-term or permanent anticoagulation, are subjects of debate.[2,8] Optimizing cardiac function, administration of anticoagulants, and optimization of hydration status is recommended.[3] AAO is a surgical condition that requires an emergent revascularization/reperfusion procedure along with anticoagulation unless contraindicated.[19] Nonoperative management has a mortality rate of up to 75%, whereas emergent operative management has significantly lower rates.[3,19]

Revascularization/reperfusion interventions may include thrombectomy, embolectomy, thrombolysis, extra-anatomic bypass, and direct repair.[3,19] The choice between endovascular and open vascular approaches is largely dependent on the surgeon's expertise, disease extension, and patient comorbidities.[7] The open surgical approach is the gold standard for AAO management. It has a higher primary patency rate and a relatively higher perioperative mortality rate ranging from 0% to 7%.[7] However, the endovascular approach has an increasing role in the management of AAO, with a similar secondary patency rate, higher costs, lower hospitalization stay period, and lower perioperative morbidity and mortality risks.[7] Thrombolysis is usually an adjunct to other treatment modalities.[7] The neurovascular status upon presentation (severity of the ischemia) and the duration of ischemia, along with early detection and expeditious intervention, are all factors associated with a lower risk of morbidity, mortality, and improved recovery.[2,3,8,10,19]

Patients with AAO may present with clinical pictures mimicking CES, which may lead to a diagnostic puzzle. To preclude misdiagnosis, emergency medicine physicians and spine surgeons must have a high index of suspicion regarding vascular etiology. Along with a detailed neurological examination, peripheral vascular examination should not be overlooked. The consideration of vascular etiologies should be even higher, especially if a spinal MRI does not suggest a compressive neurological etiology and/or a spinal cord lesion.

CONCLUSION

For patients presenting with cauda equina symptomatology, it is critical to consider vascular etiology, especially for those with cardiovascular risk factors. Spine surgeons and emergency physicians should maintain a high index of suspicion for vascular etiologies and consider appropriate imaging studies to promote early diagnosis and intervention to prevent subsequent neurological and life-threatening consequences.

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Ethical approval

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Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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