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Acute Aortic Occlusion Presenting as Incomplete Spinal Cord Syndrome: A Case Report

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Case Report

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Abstract

Background:

Acute aortic occlusion (AAO) is a rare vascular emergency that usually presents with lower limb ischemia. Spinal cord ischemia from AAO is uncommon and can mimic primary spinal cord or cauda equina syndromes particularly in the presence of preserved pulses, creating a significant diagnostic challenge.

Case Presentation:

A 75-year-old male with coronary artery disease presented with sudden bilateral lower extremity weakness, paresthesia, and urinary retention while exercising. Examination revealed complete lower limb paralysis, patchy sensory loss, absent Achilles reflexes, and decreased perineal sensation, consistent with an incomplete spinal cord syndrome. Notably, bilateral femoral pulses were normal, thereby delaying early consideration of vascular etiology. CT angiography showed acute abdominal aortic occlusion. The patient underwent emergent surgical revascularization, resulting in significant improvement of motor function.

Conclusion:

AAO can present primarily with neurologic deficits in the absence of classic signs of limb ischemia. Early recognition and urgent revascularization are critical to prevent permanent spinal cord injury and enable neurologic recovery.

Background

Acute aortic occlusion (AAO) is a rare but devastating vascular emergency defined by the sudden interruption of blood flow within the abdominal aorta. The condition is most commonly due to either embolic occlusion at the aortic bifurcation or acute thrombosis superimposed on chronic aortoiliac occlusive disease (Hines & Liu, 2021). Studies estimate an incidence of approximately 3 to 4 cases per million person-years, with a mean age of 69.7 years and a high prevalence of underlying cardiovascular disease (Grip et al., 2019).

Despite advances in vascular surgery and perioperative care, AAO continues to carry substantial morbidity and mortality. Reported 30-day mortality rates range from 20% to over 50%, depending on patient comorbidities, level of occlusion, and time to revascularization (Grip et al., 2019; Hines & Liu, 2021). Patients might experience severe complications, including limb loss, renal failure, bowel ischemia, and neurologic deficits.

This case is unique because acute aortic occlusion presented primarily as incomplete spinal cord syndrome, with sudden bilateral lower extremity paralysis, sensory deficits, and urinary retention, despite preserved femoral and distal pulses and no classic signs of acute limb ischemia. This atypical presentation closely mimicked cauda equina syndrome or spinal cord compression, creating a significant diagnostic challenge. Early recognition of a possible vascular cause, prompt CT angiography, and urgent revascularization allowed substantial neurologic and functional recovery.

Case presentation

This is the case of a 75-year-old male with a history of coronary artery disease status post six coronary stents, maintained on aspirin and clopidogrel, and benign prostatic hyperplasia, who presented with acute lower extremity paralysis.

The patient, an active runner, was performing his usual running routine when he experienced sudden-onset bilateral lower extremity paresthesia and cramping around an hour prior to presentation. These symptoms rapidly progressed to complete paralysis. He was transported to the emergency department by emergency medical services. Further history revealed urinary urgency with inability to void.

His vital signs upon arrival were as such: Temperature 37°C, Blood Pressure 197/62 mmHg, Heart Rate 70 beats/min, Respiratory Rate 16 breaths/min, Oxygen saturation 100%.

On physical examination, the patient was awake, alert, and able to follow commands. Cranial nerve examination was grossly intact. Motor strength was 5/5 in the bilateral upper extremities and 0/5 in the bilateral lower extremities. Pinprick sensation was decreased in the right lower extremity up to the mid-thigh and in the left lower extremity up to the mid-shin. Sensory examination also demonstrated patchy loss of sensation over the lower abdomen. Vibration sensation was decreased up to the ankles bilaterally, with absent proprioception in both toes. Bilateral Achilles reflexes were absent. There was decreased perineal sensation. Both resting anal sphincter tone and voluntary anal contraction were absent.

Vascular examination revealed palpable symmetric femoral pulses. Distal pedal pulses were present bilaterally and were detectable by Doppler examination, with no significant asymmetry. The lower limbs were warm, with preserved capillary refill and no clinical signs of acute limb ischemia. There was no skin mottling or discoloration at the time of initial assessment. The patient reported no lower extremity pain, and there was no muscular tenderness on palpation of the lower limbs. Overall, there were no clinical signs suggestive of acute limb ischemia at the time of initial assessment.

At this stage, the differential diagnosis included cauda equina syndrome, acute spinal cord infarction, acute transverse myelitis, compressive spinal cord pathology (e.g., epidural abscess or hematoma), and acute aortic occlusion.

Initial laboratory investigations demonstrated hemoglobin 14.8 g/dL, white blood cell count 7,900/mm³, and platelet count 217,000/mm³. Coagulation studies were within normal limits (INR 1.1, PTT 26.1 s). Serum creatinine was 1.0 mg/dL with an estimated GFR of 78 mL/min/1.73 m². Electrolytes showed potassium 3.5 mmol/L. Lactate was 2.2 mmol/L. Arterial blood gas revealed a pH of 7.36. Creatine phosphokinase (CPK) was markedly elevated at 28,537 IU/L.

Given the predominant neurological presentation and the presence of palpable, symmetric femoral pulses, MRI of the lumbosacral spine was initially considered to evaluate the working diagnosis of compressive pathology such as cauda equina syndrome. However, recognizing the significant clinical overlap between compressive spinal lesions and vascular causes of acute spinal cord ischemia—and the potential for catastrophic consequences if vascular occlusion were missed—a multidisciplinary discussion with the neurology team was undertaken. It was agreed to proceed first with CT angiography to evaluate the aorta and major arterial branches. CTA was also considered capable of rapidly identifying compressive etiologies, including epidural hematoma or abscess, thereby allowing simultaneous assessment of both vascular and structural causes. Subsequent evaluation with MRI was planned for further spinal cord characterization, but CTA was prioritized given the acuity and potential severity of the presentation.

CTA chest, abdomen and pelvis showed:

A 3-cm hypodense thrombus in the infrarenal abdominal aorta, beginning approximately 5 cm below the origin of the left renal artery. The thrombus progressed to complete occlusion over a short 2.5-cm segment and involved the iliac bifurcation. Extension of intramural thrombus into the origin of the left common iliac artery was noted over a length of approximately 1.4 cm, resulting in greater than 50% luminal narrowing. Mild narrowing of the right common iliac artery was also observed.

CT angiography of the chest, abdomen, and pelvis (coronal reconstruction) demonstrating complete occlusion of a 2.5-cm segment of the infrarenal abdominal aorta extending to the aortic bifurcation.

Following an urgent multidisciplinary discussion involving neurology, neurosurgery, and vascular surgery, the patient was immediately initiated on therapeutic heparin and underwent emergent limb-saving thromboendarterectomy.

Overall, the clinical picture was suggestive of incomplete cord syndrome due to acute aortic occlusion.

The patient underwent urgent open revascularization via a transabdominal approach. Following systemic anticoagulation with intravenous heparin, the infrarenal abdominal aorta was exposed and clamped distal to the inferior mesenteric artery and proximal to the bifurcation. Extensive thrombus and atherosclerotic plaque were evacuated from the distal abdominal aorta, followed by reconstruction using an ePTFE tube graft with restoration of brisk bilateral femoral pulses. Total lower limb ischemia time from presentation to revascularization was approximately 1 hour 47 minutes. Prophylactic fasciotomies were not required, as intraoperative assessment demonstrated viable musculature and satisfactory

distal perfusion. Postoperatively, the patient was monitored in the intensive care unit for reperfusion-related complications.

Perioperative laboratory evaluation showed a transient lactic acidosis. Initial lactate was 2.2 mmol/L with a pH of 7.36. During the intraoperative course, lactate increased to 6.1 mmol/L with a pH of 7.30. By postoperative day 1, the acidosis had resolved, with lactate decreasing to 2.1 mmol/L and pH normalizing to 7.45.

No perioperative complications such as acute kidney injury or reperfusion syndrome occurred. Therapeutic anticoagulation was resumed postoperatively.

Etiologic evaluation favored in situ aortic thrombosis rather than embolic disease, supported by intraoperative findings; transthoracic echocardiography showed no intracardiac thrombus, and cardiac rhythm monitoring revealed no atrial fibrillation. Further hypercoagulability testing was deferred given the acute presentation and underlying atherosclerotic burden.

Clinical Timeline

Time From ED Arrival	Event
T0 (Arrival)	Patient presented with sudden bilateral lower extremity paralysis and urinary retention. Vital signs obtained.
T + 5 min	Physical and neurologic exam: complete bilateral lower extremity paralysis, sensory deficits, absent anal sphincter tone. Distal pulses palpable and symmetric.
T + 10 min	Laboratory investigations ordered (CBC, coagulation profile, metabolic panel, lactate, CPK, arterial blood gas).
T + 20 min	Multidisciplinary discussion regarding imaging strategy — MRI for compressive pathology vs CTA to exclude vascular occlusion.
T + 30 min	CT angiography of chest, abdomen, and pelvis performed.
T + 45 min	CTA demonstrated infrarenal abdominal aortic occlusion at the aortic bifurcation.
T + 60 min	Urgent multidisciplinary meeting (neurology, neurosurgery, vascular surgery) and therapeutic heparin infusion initiated.
T + 107 min	Patient transferred emergently to the operating room for limb-salvage thromboendarterectomy.

Outcome and Follow-up

On postoperative day (POD) 1, the patient was extubated. He reported improvement in bilateral lower extremity weakness; however, movement remained limited due to pain. Physical examination

demonstrated improved motor strength in both lower extremities, with motor power improving to at least 3/5 bilaterally compared with 0/5 on presentation.

On POD 4, a trial of void following Foley catheter removal was unsuccessful, and the patient was discharged with an indwelling urinary catheter. He was discharged on aspirin and clopidogrel.

On POD 8, the Foley catheter was removed, and the patient was able to void spontaneously despite a weak urinary stream.

At discharge, the patient required assistance with transfers, ambulation, and activities of daily living.

At 5-month follow-up, the patient demonstrated marked functional recovery. He was able to ambulate independently without assistance, void normally, and reported complete resolution of lower extremity numbness.

Discussion

Classically, acute aortic occlusion (AAO) presents with bilateral lower extremity ischemia characterized by the “six Ps”: pain, pallor, pulselessness, paresthesia, paralysis, and poikilothermia. Absent or markedly diminished femoral pulses are traditionally regarded as a key diagnostic feature, often prompting immediate vascular imaging and surgical consultation. However, this classic presentation is not universal. In rare cases, collateral circulation or partial distal vessel patency may preserve femoral pulses, thereby obscuring early clinical recognition of AAO (Surowiec et al., 1998; Grip et al., 2019).

This case illustrates an atypical and diagnostically challenging presentation of AAO, in which the patient presented predominantly with acute neurological deficits mimicking incomplete spinal cord or cauda equina syndrome. Unlike the classic presentation with overt limb ischemia and absent pulses, femoral pulses were preserved, significantly delaying consideration of a vascular etiology.

Neurologic manifestations of AAO are uncommon but particularly deceptive. Spinal cord ischemia may occur when blood flow through the radiculomedullary arteries—most notably the artery of Adamkiewicz—is compromised. The spinal cord is especially vulnerable to hypoperfusion because of its limited collateral supply, particularly in the thoracolumbar region (Novy et al., 2006). Consequently, AAO may present with acute paraplegia, sensory disturbances, and bowel or bladder dysfunction, closely resembling acute myelopathy or cauda equina syndrome.

Spinal cord infarction is rare, accounting for approximately 1–2% of all ischemic strokes (Novy et al., 2006). Recognized vascular causes include aortic pathology, severe systemic hypotension, embolic events, and complications of aortic surgery. Clinically, spinal cord ischemia typically presents abruptly with motor weakness—complete or incomplete—variable sensory loss, and prominent autonomic dysfunction. Although classic descriptions emphasize dissociated sensory loss with preservation of vibration and proprioception, real-world presentations are frequently incomplete and heterogeneous (Robertson et al., 2012).

Several prior case reports have described AAO presenting with spinal cord ischemia; however, most highlight diminished or absent distal pulses as a critical diagnostic clue (Taghlabi et al., 2022). In contrast, preserved femoral pulses in this case underscore that a normal peripheral pulse examination does not exclude proximal aortic occlusion. Additional reports describe AAO masquerading as cauda equina syndrome or transverse myelitis, often resulting in delayed diagnosis and poorer outcomes (Soga et al., 2013; Taghlabi et al., 2022).

Given the potentially catastrophic consequences of delayed diagnosis, clinicians evaluating patients with sudden bilateral lower extremity weakness, sensory deficits, and bladder dysfunction—particularly when spinal imaging does not reveal a compressive lesion—should maintain a high index of suspicion for vascular etiologies. In such scenarios, early computed tomography angiography of the aorta is essential for accurate diagnosis and timely intervention.

Conclusion

Acute aortic occlusion can present atypically with predominant neurologic symptoms and preserved femoral pulses, closely mimicking incomplete spinal cord or cauda equina syndromes. This case emphasizes that normal peripheral pulses do not rule out AAO and that early vascular imaging should be considered in any patient with sudden bilateral lower extremity weakness and autonomic dysfunction. Prompt recognition and urgent revascularization are critical to improving neurologic outcomes and preventing permanent disability.

Learning Points

- **Acute aortic occlusion may present as sudden atraumatic paraplegia with preserved distal pulses**, closely mimicking cauda equina syndrome or spinal cord compression.
- **Early CT angiography should be considered in hyperacute bilateral lower extremity weakness** to avoid delays associated with pursuing MRI first.
- **Rapid recognition and urgent revascularization can result in substantial neurologic recovery and functional independence.**

Strengths and Limitations

Strengths

This case report provides a clear example of an uncommon presentation of acute aortic occlusion, in which neurologic symptoms predominated despite preserved peripheral pulses. The report is strengthened by a detailed description of the initial neurologic findings, vascular examination, laboratory abnormalities, CT angiography results, operative management, and postoperative recovery. The inclusion of a clinical timeline also helps demonstrate how early multidisciplinary assessment and rapid vascular imaging contributed to timely diagnosis and treatment.

Limitations

This report describes a single patient, so it cannot establish the frequency of this presentation or define a standard diagnostic approach. Because urgent revascularization was prioritized, MRI was not performed before surgery, limiting direct imaging confirmation of spinal cord ischemia. In addition, some aspects of neurologic recovery were assessed clinically rather than with standardized functional or neurologic scoring systems, and follow-up was limited to 5 months.

Patient Perspective

I found the sudden onset of symptoms and the need for emergency surgery distressing, but I undestood that urgent treatment was necessary to restore blood flow and reduce the risk of permanent neurologic injury. The medical team explained the seriousness of my condition and the reason for proceeding with immediate surgery, which helped reassure me during a frightening situation. The early recovery period was difficult because of pain, weakness and needing assistance with mibilitu and daily activities; however, I gradually noticed improvement in my leg strength and sensation. Over time, I was able to walk independetly again and regain normal urinary function, and I am grateful for the prompt diagnosis, surgical treatment and follow up care that contributed to my recovery.

Declarations

Ethics and Consent to Participate

Ethics approval was not required for this case report in accordance with institutional policies, as it describes the clinical course of a single patient and does not involve experimental intervention or identifiable patient information. The patient provided informed consent for the publication of this case report and accompanying clinical information.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Data availability

All data generated or analyzed during this study are included in this published article. Additional details are available from the corresponding author on reasonable request, while maintaining patient confidentiality.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Haya Farhat and Joseph Assaf contributed to data collection, literature review and manuscript drafting.

Ralph Bou Chebl contributed to and manuscript revision.

Gilbert Abou Dagher supervised the project and critically revised the manuscript.

All authors read and approved the final manuscript.

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Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the author(s) used **ChatGPT (OpenAI)** in order to **assist with language editing, improving clarity, and refining the wording of the manuscript**. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

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Figures



Figure 1

CT angiography of the chest, abdomen, and pelvis (coronal reconstruction) demonstrating complete occlusion of a 2.5-cm segment of the infrarenal abdominal aorta extending to the aortic bifurcation.

Supplementary Files

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