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Acute Limb Ischemia After Spinal Decompression Unmasking Occult Aortoiliac Occlusive Disease: A Case Report

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Case Report

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Acute Limb Ischemia After Spinal Decompression Unmasking Occult Aortoiliac Occlusive Disease: A Case Report

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Abstract

Background: Distinguishing neurogenic claudication caused by lumbar spinal stenosis from vascular claudication caused by aortoiliac occlusive disease remains difficult, as both conditions produce similar exertional lower-limb symptoms in older patients with degenerative spinal disease and atherosclerotic risk factors. This report differs from prior cases by identifying two specific, actionable gaps: a screening threshold that was not applied before surgery, and a plausible perioperative trigger for decompensation that has not been previously proposed in this context. This case is notable for two under-reported findings: mildly abnormal ankle-brachial index values were present but not acted upon before elective spinal surgery, and acute limb ischemia developed within days of decompression, raising the possibility that perioperative factors contributed to thrombotic progression of previously compensated arterial disease.

Case presentation: A 70-year-old man with hypertension, type 2 diabetes, and a 40-pack-year smoking history presented with a six-month history of progressive bilateral lower-limb claudication. Ankle-brachial indices were mildly reduced (0.85 right, 0.88 left), and lumbar magnetic resonance imaging showed severe L4–L5 canal stenosis with complete cerebrospinal fluid block. Because the spinal findings were considered sufficient to explain the presentation, the patient underwent decompressive laminectomy without preoperative vascular imaging. Within 72 hours postoperatively, he developed acute right lower-limb ischemia, with ankle-brachial index falling to 0.35. Doppler ultrasound identified occlusion at the right common femoral artery, and emergency embolectomy was performed. Histopathology showed organized thrombus overlying atherosclerotic plaque. Distal perfusion recovered fully after revascularization, and three-month

follow-up confirmed sustained patency and improved function. Notably, the ischemic presentation was strictly unilateral, and no imaging of the aortoiliac segment was obtained, leaving the diagnosis of true bilateral aortoiliac disease unconfirmed and isolated common femoral artery thrombosis a plausible alternative explanation.

Conclusions: This case highlights two clinical implications beyond the well-known overlap between neurogenic and vascular claudication. First, mildly reduced ankle-brachial index values in patients with multiple atherosclerotic risk factors should prompt vascular imaging before elective spinal decompression rather than being dismissed as incidental. Second, the close temporal relationship between surgery and acute ischemia suggests that perioperative hemodynamic and positional factors may accelerate decompensation of previously compensated arterial disease, warranting closer perioperative vascular monitoring in at-risk patients. A standardized preoperative vascular screening threshold may help prevent both ineffective spinal surgery and postoperative limb-threatening complications.

Keywords: Aortoiliac occlusive disease; Common femoral artery thrombosis; Neurogenic claudication; Lumbar spinal stenosis; Ankle-brachial index; Perioperative ischemia; Case report

Introduction

Aortoiliac occlusive disease (AIOD), commonly termed Leriche syndrome, is a chronic atherosclerotic disorder producing partial or complete obstruction of the distal aorta and/or iliac arteries, most often at the aortic bifurcation (1). Its classic triad, including lower-extremity claudication, diminished femoral pulses, and erectile dysfunction, is frequently incomplete or masked by extensive collateral circulation, which delays recognition until advanced ischemic complications occur (1, 2). Prevalence rises with age and cardiovascular risk burden, including smoking, diabetes, hypertension, and hyperlipidemia (2, 3). Chronic narrowing reduces pelvic and

lower-limb perfusion, while acute thrombotic occlusion can precipitate critical limb ischemia requiring emergent intervention (3). Delayed diagnosis carries substantial morbidity, including irreversible ischemic injury and major vascular reconstruction (4-6). A central diagnostic challenge is the clinical overlap between vascular claudication and neurogenic claudication caused by lumbar spinal stenosis, both of which produce exertional lower-limb pain, weakness, and paresthesia in older patients with degenerative spinal disease (4). Several studies have shown that symptom-based differentiation alone is unreliable: vascular claudication can present with low-back pain, buttock discomfort, and gait limitation resembling spinal pathology (7), and classic clinical discriminators such as posture dependency, relief with sitting, and symptom distribution have been shown to have poor diagnostic accuracy when used in isolation (8). As a result, radiological confirmation of degenerative spinal disease, even when genuinely severe, does not exclude a coexisting or causative vascular pathology, and reliance on imaging concordance alone can lead to unnecessary spinal surgery and delayed vascular management (9, 10). What remains comparatively under-addressed in the existing literature is when, specifically, a mildly abnormal ankle-brachial index (ABI) in an at-risk patient should trigger vascular imaging before elective spinal decompression, and whether the perioperative period itself, through hemodynamic changes, positioning, and reduced mobility, can accelerate decompensation of previously compensated arterial disease (3, 11). Prior reports of Leriche syndrome unmasked by spinal surgery have generally framed the vascular event as an incidental, temporally coincidental finding rather than examined this potential causal relationship in detail (3). Clarifying both the screening threshold and the plausibility of a perioperative trigger has direct relevance for neurologists and neurosurgeons, who are typically the first clinicians to evaluate claudication in older patients and who must decide whether additional vascular workup is warranted before attributing symptoms to

spinal pathology alone. This report makes two specific contributions to that gap: first, a concrete ABI-based threshold for triggering preoperative vascular imaging; second, evidence supporting the plausibility that the perioperative period itself, not only the pre-existing vascular disease, can precipitate acute decompensation. Because the initial clinical decision in such presentations, whether spinal imaging findings sufficiently explain lower-limb symptoms, or whether concurrent vascular workup is warranted, is typically made by neurologists and neurosurgeons rather than by vascular specialists, clarifying a practical screening threshold has direct and immediate relevance to neurological and neurosurgical decision-making, independent of the ultimate vascular diagnosis. We report a case in which a 70-year-old man with multiple atherosclerotic risk factors and mildly reduced ABI values underwent spinal decompression for severe, symptom-concordant imaging findings, and developed acute limb-threatening ischemia within 72 hours of surgery. We use this case to propose a concrete ABI-based screening threshold for preoperative vascular imaging and to discuss the plausibility of a perioperative contribution to thrombotic progression, rather than to restate the known overlap between neurogenic and vascular claudication simply. This case report was prepared in accordance with the CARE guidelines (12).

Case presentation

A 70-year-old man presented with a six-month history of progressive bilateral lower-extremity pain, heaviness, and intermittent numbness, exacerbated by walking short distances and relieved by rest, with marked worsening over the preceding month that severely limited his mobility. He reported occasional cold sensation in his feet but denied rest pain or erectile dysfunction at presentation. The timeline of clinical events, from initial presentation through three-month follow-up, is summarized in Table 1. His medical history included long-standing hypertension, type 2 diabetes mellitus, and a 40-pack-year smoking history, without prior cardiovascular or

thromboembolic disease. There was no relevant family history of vascular, spinal, or thromboembolic disease, and no psychosocial factors (e.g., occupational exposure, limited healthcare access) were identified as contributory to his presentation. He was hemodynamically stable on examination, with preserved motor strength in both lower limbs. Sensory impairment to light touch and pinprick was present bilaterally below the knees. Peripheral pulses (femoral, popliteal, dorsalis pedis, and posterior tibial) were palpable but slightly diminished bilaterally. Ankle-brachial indices were 0.85 on the right and 0.88 on the left, consistent with mild peripheral arterial insufficiency. Lumbar magnetic resonance imaging demonstrated severe degenerative canal stenosis at L4–L5 with complete cerebrospinal fluid block (Figure 1). The differential diagnosis at presentation included neurogenic claudication secondary to lumbar spinal stenosis, peripheral arterial disease, and diabetic peripheral neuropathy; the severity and symptom-concordance of the MRI findings were considered most consistent with a primary neurogenic etiology. Given the severity of these findings, the multidisciplinary team proceeded to L4–L5 decompressive laminectomy and medial facetectomy; preoperative vascular imaging (CT or MR angiography) was not obtained, as the mildly reduced ABI values were, at that time, not considered to independently warrant further vascular workup relative to the severity of the spinal pathology. Surgery was uncomplicated, with preserved hemodynamics throughout. Postoperatively, the patient experienced only minimal transient symptom relief, with persistent claudication and sensory complaints. Over the following 72 hours, he developed progressive right lower-limb ischemic symptoms, including increasing rest pain, pallor, delayed capillary refill, and eventual cyanosis of the right foot. Repeat pulse examination showed loss of both dorsalis pedis and posterior tibial pulses on the right, while left-sided pulses remained palpable. The right-sided ABI declined to 0.35, indicating acute critical limb ischemia. Urgent vascular surgery consultation was

obtained. Arterial Doppler ultrasound revealed acute occlusion at the level of the right common femoral artery with absent distal flow. The patient underwent emergent surgical embolectomy using a Fogarty catheter; a large thromboembolic cast was extracted and sent for pathological assessment. Histopathology confirmed organized thrombotic material overlying atherosclerotic plaque, favoring in-situ arterial thrombosis over a cardioembolic source (Figure 2). Transthoracic echocardiography subsequently identified no embolic source, supporting a local atherosclerotic-thrombotic mechanism. Following revascularization, distal perfusion improved markedly, with restoration of limb warmth and strong palpable distal pulses. The patient was started on dual antiplatelet therapy (aspirin 81 mg and clopidogrel 75 mg daily) and a high-intensity statin, per vascular surgery recommendations. The patient tolerated dual antiplatelet therapy and high-intensity statin treatment without adverse effects, and adherence was confirmed at the three-month follow-up visit. No imaging of the aortoiliac segment (CT or MR angiography) was performed at any point during admission; the presumption of underlying chronic aortoiliac disease was therefore based on the patient's atherosclerotic risk profile, clinical course, and femoral-level histopathology rather than confirmed by direct imaging of the aortic bifurcation or iliac vessels, a gap addressed further in the Discussion and Limitations. Aortoiliac imaging was not pursued after revascularization because the patient's presenting symptoms and distal pulses had normalized and he had no indication for further intervention at that time; in retrospect, however, confirmatory imaging would have clarified the diagnosis and is now recommended as standard practice in our institution for similar cases. The remainder of the postoperative course was uneventful. At three-month follow-up, the patient reported markedly improved walking capacity and a return to daily activities he had abandoned over the preceding year, with no recurrent ischemic symptoms. Duplex ultrasonography confirmed maintained distal arterial patency.

Discussion

This case highlights two related but separable problems: the well-recognized difficulty of distinguishing vascular from neurogenic claudication, and a less-examined question of whether spinal surgery itself can precipitate thrombotic decompensation of pre-existing, compensated arterial disease. Diagnostic overlap. Lumbar spinal stenosis produces posture-dependent neurogenic claudication typically eased by flexion or sitting (9), whereas Leriche syndrome, the classic, typically bilateral and symmetric form of aortoiliac occlusive disease, reflects chronic aortoiliac obstruction with reduced lower-limb perfusion (1, 10). Table 2 summarizes the key clinical discriminators between the two entities, although these discriminators individually have limited diagnostic accuracy (8). In practice, this distinction is unreliable: vascular claudication can present with low-back pain, buttock discomfort, and gait limitation resembling spinal pathology (7), nd Nadeau et al. demonstrated that classic clinical discriminators including posture dependency, relief with sitting, and symptom distribution have poor diagnostic accuracy when used in isolation (8). In our patient, severe imaging-confirmed canal stenosis was sufficient on its own to explain the presenting symptoms, and this imaging concordance likely reduced the perceived need for further vascular workup despite mildly abnormal ABI values. Our patient's unilateral presentation makes the diagnosis of Leriche syndrome presumptive rather than confirmed. Why was preoperative vascular imaging not obtained, and was this justified? This is the central methodological question raised by this case. Bilaterally reduced ABI values of 0.85 and 0.88 meet formal criteria for mild-to-moderate peripheral artery disease under current guideline-based thresholds (ABI 0.41–0.90) (13), yet such values are frequently interpreted in clinical practice as an incidental, age-related finding rather than a marker of clinically significant disease. However, ABI can substantially underestimate the severity of proximal aortoiliac disease when

compensated by collateral circulation, a well-recognized limitation of ABI in slowly progressive AIOD (2, 11). In retrospect, the combination of advanced age, diabetes, hypertension, and a 40-pack-year smoking history constituted sufficient pretest probability to justify at least a screening CT or MR angiogram before proceeding to elective spinal surgery, independent of the ABI value in isolation. We propose, as a practical threshold, that any ABI below 0.9 in a patient with two or more atherosclerotic risk factors should prompt vascular imaging before elective spinal decompression, rather than being treated as an incidental finding. Was surgery itself a contributing trigger? The 72-hour interval between decompression and acute limb ischemia raises a mechanistic question that is easy to overlook if the case is framed only as "unmasking" of pre-existing disease. Perioperative hypotension, prolonged prone positioning, and reduced postoperative mobility can all transiently compromise collateral flow in a limb already dependent on collateral circulation for perfusion, potentially converting subclinical, compensated stenosis into an acute thrombotic event (3, 14). Prone positioning has been independently associated with a significantly higher risk of severe intraoperative hypotension in large retrospective spine-surgery cohorts (14), and position-related occlusion of blood flow is a recognized contributor to perioperative thrombotic risk more broadly (15). The histopathological finding of organized thrombus overlying atherosclerotic plaque supports a two-stage process: a chronic, pre-existing thrombotic component at the site of plaque, upon which acute occlusion was superimposed in the immediate postoperative period. We cannot establish causality from a single case, but this temporal relationship is clinically important: it suggests that patients with borderline ABI values may warrant closer perioperative vascular monitoring, not only preoperative screening—when undergoing spinal surgery under general anesthesia. Does this case actually confirm Leriche syndrome? This limitation deserves explicit acknowledgment. Leriche syndrome, by definition, reflects aortoiliac occlusive disease typically

bilateral and symmetric. In this patient, ischemia was strictly unilateral, the left limb remained clinically unaffected throughout, and no imaging of the aortic bifurcation or iliac vessels was obtained at any stage to confirm proximal aortoiliac involvement. The diagnosis of underlying AIOD was inferred from the patient's atherosclerotic risk profile and femoral-level histopathology rather than demonstrated directly. An equally plausible alternative interpretation is isolated right common femoral artery thrombosis on a background of diffuse but non-critical atherosclerosis, without true aortoiliac bifurcation disease. We present this case as illustrative of the diagnostic overlap between vascular and neurogenic claudication while explicitly flagging that the aortoiliac diagnosis itself remains unconfirmed by direct imaging. It is the distinction future similar reports should resolve with routine aortoiliac imaging.

Clinical implications: Regardless of the precise anatomical diagnosis, this case supports incorporating a standardized vascular screening step including pulse examination, bilateral ABI, and a defined imaging threshold into the preoperative evaluation of claudication in neurosurgical practice, together with multidisciplinary input from vascular surgery when risk factors or borderline ABI values are present (7, 11). Previous reports have similarly emphasized that timely revascularization is the cornerstone of favorable outcomes in Leriche syndrome (16); our case extends this by suggesting that the preoperative and perioperative periods, not only the post-diagnostic period, are critical windows for risk mitigation.

Strengths and limitations: The principal strength of this report lies in the detailed temporal and histopathological documentation linking a specific perioperative window to a confirmed thrombotic event, which allowed us to move beyond a purely descriptive account and propose a testable screening threshold. Nonetheless, several limitations warrant consideration. As a single-patient report, our findings cannot establish generalizable diagnostic or management protocols,

and the proposed ABI threshold requires validation in larger cohorts before adoption into practice. We explicitly acknowledge that proceeding to elective spinal decompression without baseline vascular imaging, in a patient with a reduced ABI and multiple atherosclerotic risk factors, represents a deviation from an ideal perioperative workup rather than a defensible standard of care; we present this case specifically so that the threshold we propose can help other clinicians avoid a similar gap prospectively rather than retrospectively. Larger prospective studies are needed to better characterize clinical red flags that reliably differentiate neurogenic from vascular claudication and to formally evaluate the safety and yield of routine preoperative vascular screening in candidates for elective spinal decompression.

Conclusions

Vascular claudication can closely mimic neurogenic claudication and, as this case illustrates, may remain under-investigated even when mildly abnormal ABI values are present alongside severe, symptom-concordant spinal imaging findings. We propose a concrete threshold (ABI below 0.9 in the presence of two or more atherosclerotic risk factors) as an indication for vascular imaging before elective spinal decompression. This case additionally raises the possibility that perioperative hemodynamic and positional factors may accelerate thrombotic decompensation of compensated arterial disease, an association meriting further prospective study. Because unilateral ischemia and the absence of aortoiliac imaging leave the underlying diagnosis in this case only presumptively consistent with Leriche syndrome, we recommend that future similar reports confirm aortoiliac involvement directly rather than inferring it from risk profile and distal histopathology alone. Clinicians evaluating claudication in the setting of degenerative spinal disease should maintain a high index of suspicion for coexisting arterial disease and apply a defined vascular screening threshold rather than relying on symptom-imaging concordance alone.

Patient Perspective

The patient reported that his quality of life had been severely limited in the months before surgery, to the point that he had abandoned several routine daily activities because of walking difficulty. He described only brief, partial relief after the spinal decompression, followed shortly afterward by an unexpected and frightening deterioration in his right leg. He noted that he had not been informed, before surgery, that a vascular complication of this nature was possible. Following the emergency vascular procedure, he reported a marked and sustained improvement in walking capacity, and at three-month follow-up stated that he had resumed most of his previous daily activities without recurrent leg pain or coldness.

List of abbreviations

ABI: Ankle-brachial index

MRI: Magnetic resonance imaging

AIOD: Aortoiliac occlusive disease

CTA: Computed tomography angiography

MRA: Magnetic resonance angiography

Declarations

Ethics approval and consent to participate

Formal ethics approval was not required for this single case report, as it involved standard clinical and diagnostic care and did not alter the patient's management pathway. This was confirmed by the Ethics Committee of Isfahan University of Medical Sciences, in accordance with institutional policy for case reports not requiring full ethical review. All procedures performed were in

accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent to participate was obtained from the patient.

Consent for publication

Written informed consent for publication of this case report, including all accompanying clinical images, was obtained from the patient. A copy of the consent form is available for review by the Editor-in-Chief upon request.

Availability of data and materials

All relevant clinical data supporting the findings of this case report are presented within the manuscript. De-identified supplementary materials, including radiological images, are available from the corresponding author upon reasonable request, subject to patient confidentiality.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

MM and MHB conceived and designed the case report. MM was the lead clinician responsible for the patient's neurosurgical management and drafted the initial version of the manuscript. NA contributed to the patient's clinical management, acquisition of clinical data, and coordination of follow-up. AR and RA were involved in the patient's perioperative and vascular surgical

management and contributed to data acquisition. NM performed the histopathological examination of the excised thromboembolic material and provided the pathological interpretation reported in the manuscript. SKS conducted the literature review and contributed to drafting the manuscript. MHB interpreted the clinical and diagnostic findings in the context of the existing literature, substantively revised the manuscript for important intellectual content, and is the corresponding author. All authors read and approved the final manuscript, and each author agrees to be personally accountable for their own contributions and for ensuring that questions related to the accuracy or integrity of the work are appropriately investigated and resolved.

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Figure 1. MR myelography demonstrating severe lumbar canal stenosis at the L4–L5 level with complete cerebrospinal fluid block.

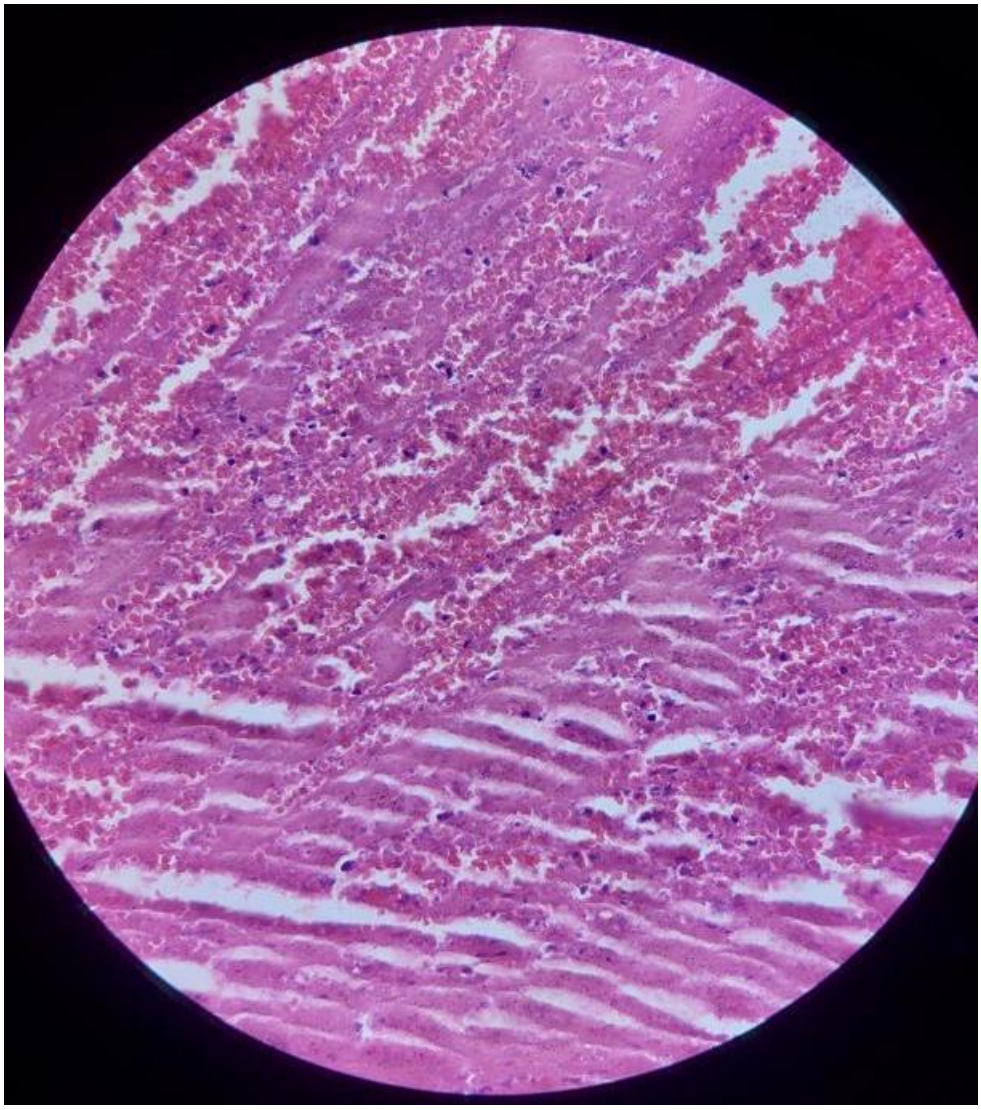


Figure 2. Microscopic pathological view of the thrombus retrieved during lower limb embolectomy (×40), showing diffuse areas of hemorrhage and fibrinoid material.

Table 1. Timeline of clinical presentation, intervention, and outcomes.

Time point	Clinical event
6 months before admission	Onset of progressive bilateral lower-limb claudication
1 month before admission	Marked worsening of symptoms and mobility
Admission	ABI 0.85 (right)/0.88 (left); MRI: severe L4–L5 stenosis with CSF block
Day 0	L4–L5 decompressive laminectomy and medial facetectomy
Day 3 postoperative (72 h)	Progressive right lower-limb ischemia recognized clinically; ABI declined to 0.35; Doppler confirmed right common femoral artery occlusion; emergent Fogarty embolectomy performed
Month 3 postoperative	Improved walking capacity; Duplex ultrasonography confirmed sustained arterial patency

Table 2. Clinical features distinguishing neurogenic claudication from vascular claudication due to aortoiliac occlusive disease (AIOD)

Clinical Feature	Neurogenic Claudication (Lumbar Spinal Stenosis)	Vascular Claudication (Aortoiliac Occlusive Disease, AIOD)
Relationship to posture	Usually improves with lumbar flexion or sitting	Not related to posture
Pain characteristics	Diffuse pain, numbness, heaviness, or paresthesia	Localized cramping muscle pain, typically induced by exertion
Peripheral pulses	Usually normal	Reduced or absent
Ankle–Brachial Index (ABI)	Usually normal	Typically reduced (<0.9)
Lumbar MRI findings	Lumbar canal and/or foraminal stenosis	Lumbar stenosis may coexist (diagnostic overlap is common)
Associated clinical findings	Low back pain, radiculopathy	Cold extremity, erectile dysfunction, skin discoloration, diminished perfusion

Note: These distinguishing features demonstrate substantial clinical overlap and have limited diagnostic accuracy when used individually (8), highlighting the central diagnostic challenge illustrated by this case.