

Lumbar Spinal Stenosis Presenting as Refractory Bilateral Lower Extremity Edema: Rapid Resolution After Unilateral Biportal Endoscopic Surgery—A Case Report and Literature Review

Ping Xu

55741831@qq.com

Wuhan Hospital of Traditional Chinese Medicine

Haixiang Xi

Wuhan Hospital of Traditional Chinese Medicine

Yuhui Zhang

Wuhan Hospital of Traditional Chinese Medicine

Xinyu Mai

Wuhan Hospital of Traditional Chinese Medicine

Case Report

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Abstract

Purpose

Bilateral lower extremity edema is a common clinical symptom, often caused by cardiac, hepatic, renal, or venous disorders. However, the etiology of some refractory cases remains occult, and in such instances, lumbar spinal stenosis—a spinal etiology—is easily overlooked. Prolonged lower extremity edema secondary to lumbar spinal stenosis has rarely been reported in the literature, and the underlying pathophysiological mechanisms remain elusive. This case presents a unique instance where severe L4/5 lumbar spinal stenosis manifested solely as a five-year history of refractory bilateral calf edema, occurring in the complete absence of low back pain, sciatica, or neurogenic claudication. Notably, the edema resolved rapidly and completely following unilateral biportal endoscopic (UBE) decompression, providing direct in vivo evidence that stenosis-related edema is reversible after neural decompression. By documenting this rare presentation and its dramatic treatment response, this report aims to (1) raise awareness of a treatable spinal etiology in cryptogenic bilateral edema, (2) advocate for lumbar MRI as a screening modality, even in patients lacking overt spinal symptoms, and (3) provide mechanistic insights regarding epidural venous plexus compression and neurogenic inflammation, thereby expanding the etiological spectrum of lower extremity edema.

Case Presentation

A 76-year-old female presented with a five-year history of persistent symmetrical pitting edema of both calves, in the absence of low back pain, radicular pain, or motor deficits. Routine medical workup was unremarkable, with cardiac, hepatic, renal, and vascular etiologies excluded. Multiple therapeutic trials involving volume depletion strategies and diuretics proved ineffective. Subsequently, lumbar MRI and CT demonstrated severe lumbar spinal stenosis at the L4/L5 level.

Results

Following L4/5 unilateral biportal endoscopic (UBE) decompressive surgery (partial laminectomy with ligamentum flavum resection and central/lateral recess decompression under general anesthesia), bilateral calf edema markedly subsided on the first postoperative day and completely resolved by postoperative day 9. A repeat lower extremity ultrasound showed resolution of subcutaneous soft tissue edema in both calves. At the 4-week follow-up, no recurrence of lower extremity edema was observed.

Conclusion

This case illustrates that lumbar spinal stenosis represents a rare but reversible cause of refractory lower extremity edema. Proposed mechanisms may involve impaired venous return due to compression of the epidural venous plexus and potentially neurogenic inflammation. Lumbar MRI should be considered a screening tool for unexplained, refractory lower extremity edema. The UBE technique, as a minimally invasive and precise decompression approach, provides an effective treatment option for such patients and broadens the clinical understanding of the etiological spectrum of lower extremity edema.

Introduction

Bilateral lower extremity edema is a common clinical symptom, and its differential diagnosis typically centers on cardiac, hepatic, renal, and vascular etiologies. When routine examinations fail to identify a definitive cause, establishing a diagnosis of cryptogenic edema often proves challenging. Notably, the concept of lower extremity edema of spinal origin remains underrecognized among clinicians. In particular, long-standing, refractory edema caused by lumbar spinal stenosis (LSS) has only been reported in exceedingly rare instances in the literature.

The typical clinical manifestations of LSS include neurogenic claudication and/or lumbosacral radiculopathy, a condition that commonly occurs in older adults [1, 2].

However, the possibility of lumbar spinal stenosis as a rare cause of lower extremity edema is often overlooked in clinical practice. The primary pathophysiology of this condition involves compression of nerve roots and blood vessels resulting from spinal canal narrowing [3]. Animal models have confirmed that such compression can lead to decreased nerve root blood flow and local ischemia [4]; furthermore, vasoactive agents have been employed to ameliorate related symptoms [5]. Evidence suggests that lower extremity swelling in LSS patients is an independent risk factor for inadequate intraoperative somatosensory evoked potential baselines, indicating that neurogenic edema may be correlated with neurological functional status [6]. These lines of evidence suggest that compression of spinal origin affecting the nerve roots or the cauda equina may induce or exacerbate lower extremity edema by impacting local microcirculation, venous return, or axonal transport. Nevertheless, the specific mechanisms remain to be elucidated.

Advancements in spinal minimally invasive techniques, particularly the widespread adoption of unilateral biportal endoscopic (UBE) surgery, have established an ideal clinical model for studying the resolution of remote symptoms following neural decompression. The UBE technique facilitates efficient and precise spinal canal decompression through two independent working channels, and its clinical efficacy has been extensively validated. The present case clearly demonstrates that, in the absence of other identifiable causes, long-standing and severe lower extremity edema can be solely attributed to LSS. Successful spinal canal decompression resulted in the resolution of this edema, supporting the classification of edema of spinal origin as a distinct clinical entity. This finding not only expands the clinical understanding of the etiological spectrum of bilateral lower extremity edema but also provides a new diagnostic and therapeutic perspective for patients with chronic unexplained edema.

Case Description

A 76-year-old female was admitted with a chief complaint of persistent bilateral calf edema for five years. The presenting symptom was symmetrical edema of both calves, with a five-year duration and recent exacerbation. She had no history of common internal medical conditions such as hypertension, diabetes mellitus, cardiac disease, liver disease, or renal disease, nor did she have a history of varicose veins or deep vein thrombosis. Physical examination revealed symmetrical pitting edema of both calves, with normal skin temperature, no erythema, and no limitation in ankle joint range of motion. The right dorsalis pedis artery pulse was diminished, while the left was normal. Neurological examination showed no typical radicular pain or motor deficits.

Prior Diagnostic and Therapeutic Course: Over the past five years, the patient had sought medical attention multiple times due to recurrent bilateral lower extremity edema. Comprehensive examinations including cardiac, hepatic, renal, and thyroid function tests, autoimmune markers, coagulation studies, and lower extremity venous ultrasound had been performed. The results only revealed stenosis of the right anterior tibial artery, and common systemic diseases as well as vascular edema due to impaired venous return were largely ruled out. Regarding treatment, the patient had attempted diuretics, leg elevation, compression stockings, local massage, and traditional Chinese medicine therapies, all of which failed to provide improvement. This refractory edema, unresponsive to conventional treatment, strongly suggests that the etiology is not attributable to common systemic diseases or local venous/lymphatic drainage impairment (Fig. 1).

Imaging Findings and Diagnostic Process: Lumbar CT revealed disc herniation at the L4/L5 level, hypertrophy of the ligamentum flavum, osteophyte formation at the posterior vertebral margins, hypertrophy and convergence of the facet joints, and secondary spinal stenosis. The axial T2-weighted MRI images clearly demonstrated compression of the dural sac at the L4–L5 level with a "cloverleaf" deformity, hypertrophy of the ligamentum flavum, and narrowing of the bilateral lateral recesses and nerve root canals. As the patient lacked typical radicular symptoms (such as radiating leg pain or intermittent claudication), a spinal origin was not initially considered the primary diagnosis. After systematic exclusion of common systemic causes such as cardiac, hepatic, renal, and nutritional disorders, a review of the literature suggested that LSS may induce edema by affecting venous and lymphatic return from the lower extremities. Furthermore, the edema was unresponsive to diuretic therapy but rapidly resolved following surgical decompression, suggesting that the underlying mechanism may involve mechanical venous outflow obstruction or neurogenic drainage impairment (Fig. 2).

Surgical Intervention and Clinical Outcome: The patient underwent unilateral biportal endoscopic (UBE) surgery under general anesthesia. At the L4/L5 level, partial laminectomy, resection of the ligamentum flavum, and resection of hypertrophic bone were performed to achieve decompression of the central canal and lateral recesses. Under endoscopic visualization, the dural sac and L5 nerve root were observed to be significantly compressed; after decompression, the dural sac demonstrated restored

pulsation and the nerve root regained mobility. On postoperative day 1, the patient reported significant symptom relief in both calves, and physical examination revealed substantial resolution of the pre-existing bilateral lower extremity pitting edema, with prominent skin wrinkling over the calves and dorsum of the feet. By postoperative day 9, the edema had completely resolved, skin elasticity had returned, and repeat bilateral lower extremity color Doppler ultrasound confirmed the complete absence of edema. At the 4-week follow-up, bilateral lower extremity edema had not recurred, and the skin appeared normal (Fig. 3,4).

Discussion

The greatest diagnostic challenge in this case was that bilateral lower extremity edema was the sole clinical manifestation, with no typical spinal symptoms such as low back pain or sciatica. This isolated presentation may predispose clinicians to focus exclusively on cardiac, renal, or vascular etiologies, potentially leading to the oversight of a spinal cause[6]. Currently, no specific test can directly establish a causal relationship between spinal stenosis and lower extremity edema; diagnosis often depends on excluding other common causes and is retrospectively confirmed by the rapid resolution of edema following surgical intervention. This "therapeutic diagnosis," while effective, introduces diagnostic uncertainty. While the mechanisms underlying LSS-induced lower extremity edema likely involve venous outflow obstruction and neurogenic edema, this pathophysiological association remains frequently underappreciated in clinical practice. Diagnostic confirmation typically necessitates excluding other common causes and observing the rapid resolution of edema post-surgical decompression, which serves to retrospectively validate the pathophysiologic connection[7]. This concept of "therapeutic diagnosis," though effective, also increases uncertainty and risk during the diagnostic process. This case clearly demonstrates that long-standing, severe edema can be caused solely by LSS in the absence of other identifiable triggers, and that successful neural decompression leads to marked resolution—thereby establishing the possibility of "spinal edema" as a distinct clinical entity.

The core mechanism by which lumbar spinal stenosis leads to lower extremity edema involves impaired venous return within the spinal canal. When the lumbar spinal canal, lateral recesses, or intervertebral foramina undergo narrowing due to degenerative changes or osteophyte formation, the epidural venous plexus is directly compressed, resulting in pathologically elevated venous pressure within the spinal canal. This high-pressure state is retrograde-transmitted to the deep venous system of the lower extremities, obstructing normal venous return from the legs to the iliac veins and vena cava. Chronic venous stasis substantially elevates capillary hydrostatic pressure in the lower limbs, resulting in tissue fluid filtration exceeding the compensatory capacity of lymphatic drainage, ultimately culminating in persistent, refractory lower extremity edema [6]. Moreover, the occurrence of new-onset postoperative lower extremity pain following lumbar spinal stenosis surgery is frequently attributed to lower extremity venous thrombosis, which underscores the critical role of venous outflow obstruction in the pathophysiology of this condition [8]. Therefore, relieving venous compression within the spinal canal is paramount for alleviating such edema.

Beyond direct mechanical compression, neurogenic inflammation constitutes a pivotal mechanism in lower extremity edema associated with lumbar spinal stenosis. Stenosis-induced nerve root compression stimulates the release of proinflammatory mediators, such as substance P and calcitonin gene-related peptide (CGRP), from nerve endings [9]. Through an axon reflex mechanism, these substances markedly enhance the permeability of local blood vessels—particularly postcapillary venules—allowing plasma proteins and other macromolecules to leak into the interstitial space. This establishes a steep osmotic gradient that drives fluid extravasation, culminating in edema formation. Concurrently, dysfunction of compressed sympathetic nerve fibers may lead to dysregulated constriction of precapillary sphincters and impaired contraction of lymphatic smooth muscle in the lower extremities, hindering lymphatic drainage and further aggravating tissue edema [10]. Thus, neurogenic inflammation and lymphatic outflow obstruction jointly contribute to the complex pathophysiology of neurogenic edema.

Given the atypical presentation of this case, lumbar spine MRI is recommended in the diagnostic workup for patients presenting with unexplained, refractory, symmetric bilateral lower extremity edema, even in the complete absence of low back pain or sciatica. MRI is the gold standard for evaluating spinal stenosis [11], which enables accurate assessment of osseous canal narrowing and nerve root compression [12]. Particular attention should be paid to the L4/L5 and L5/S1 levels, as these segments contain large nerve roots that are vulnerable to compression and are also closely associated with the pelvic venous network via extensive collateral channels. Severe stenosis at these levels may also result in cauda equina ischemia, presenting as neurogenic claudication [13].

Conclusion

This case report describes an exceptionally rare clinical phenomenon: severe lumbar spinal stenosis may present as the exclusive etiology of refractory, symmetric bilateral lower extremity edema. Following the exclusion of common medical and vascular etiologies, lumbar spine MRI emerged as the pivotal diagnostic modality. Severe central canal and lateral recess stenosis at the L4/L5 level was confirmed; subsequent decompression via unilateral biportal endoscopy (UBE) not only substantiated the hypothesis that mechanical compression was the primary driver but also demonstrated the efficacy of minimally invasive surgery. This case underscores the importance of including lumbar spinal stenosis in the differential diagnosis for patients presenting with unexplained, symmetric lower extremity edema refractory to conventional treatment, warranting prompt lumbar spine imaging.

Declarations

Ethics approval and consent to participate

The procedures followed in this case report were in accordance with the Declaration of Helsinki. This case report was approved by the Ethics Committee of Wuhan Hospital of Traditional Chinese Medicine

(Approval No. WHTCM-LW2026-002). Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Availability of data and materials

The data and materials presented in this case report were obtained from the clinical records of the patient who underwent surgical treatment at Wuhan Hospital of Traditional Chinese Medicine.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

X.P. and X.H.X. performed the procedures. Z.Y.H. and M.X.Y. supervised the patient treatment. X.P. drafted the manuscript. X.H.X., Z.Y.H., and M.X.Y., helped draft the manuscript and critically revised it. The authors have read and approved the final manuscript.

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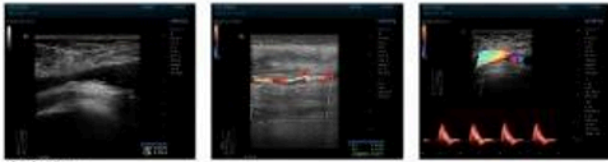
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Figures

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姓名: 潘** 性别: 女 年龄: 76岁 住院号: 病区: 检查部位: 双下肢四肢血管彩色多普勒超声 床号: 科室: 超声科



A

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姓名: 性别: 女 年龄: 76岁 住院号: 病区:

检查部位: 双下肢四肢血管彩色多普勒超声 床号: 科室:



B

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Figure 2

Preoperative T2-weighted sagittal and axial MR images showing spinal stenosis at the L4/L5 level.

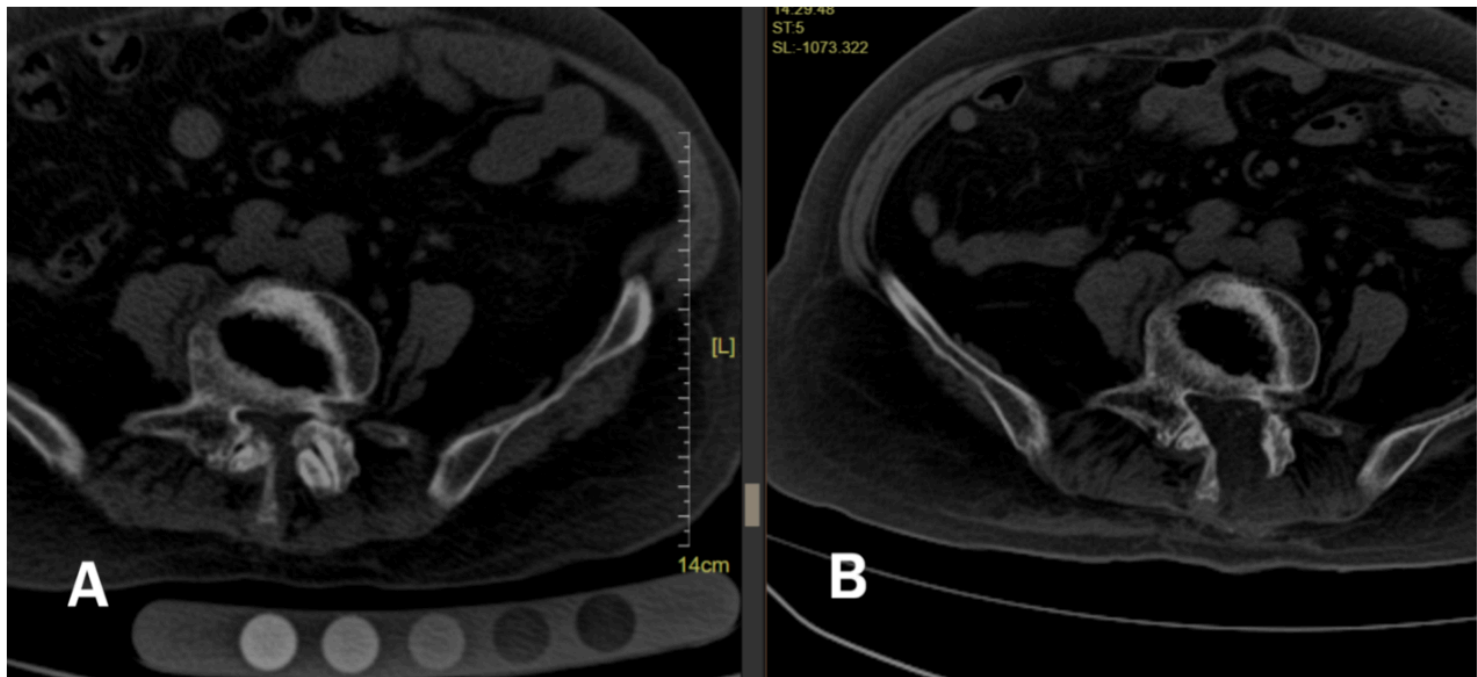


Figure 3

Comparative pre-operative (A) and post-operative (B) CT images at the L4/L5 level after UBE surgery



Figure 4

Resolution of bilateral calf swelling postoperatively on day 1