

Chronic Venous Ulcer Treated With Iliac Vein Stenting for Proximal Deep Vein Compression

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A 79-year-old man with a history of type 2 diabetes mellitus and hypertension presented with a 5-month history of poorly healing venous ulcers on the anterior and medial aspect of the left calf, which had been recurring over 14 years.

History. The ulcers were limited to the patient's left lower extremity and did not involve the contralateral limb, despite his having advanced venous stasis skin changes and swelling on both calves. He reported having leg heaviness and constant pain that is alleviated by elevation of his lower extremities. Additionally, he reported having debilitating venous claudication symptoms, which he described as left calf and thigh pain that radiates up to the flank and buttock upon walking 2 blocks.

He had previously been treated with compression stockings and Unna boots at a local wound-care center with minimal success; the ulcers had recurred, along with functionally debilitating symptoms consistent with venous hypertensive disease. He had had several consultations and vascular Doppler studies at various vein centers; however, he had not undergone intravascular ultrasonography (IVUS) guided endovascular therapy for possible proximal deep vein compression.

Physical examination. Physical examination revealed visually remarkable calf-size asymmetry, extensive hyperpigmentation, stasis dermatitis, indurated skin, and active open and healed ulcers on the left leg (**Figure 1**). The left lower extremity demonstrated +3 (of 4) pretibial and ankle pitting edema extending to the mid-thigh, compared with +2 pitting edema limited to below the knee in the right lower extremity. There were palpable anterior tibial (AT), posterior tibial (PT), and dorsalis pedis (DP) pulses bilaterally.



Figure 1. Pathologic asymmetry in calf size associated with extensive venous stasis hyperpigmentation, stasis dermatitis, indurated skin (consistent with lipodermatosclerosis), and active open and healed ulcers on the left leg.

Diagnostic tests. Lower-extremity arterial Doppler ultrasonography ruled out the presence of peripheral arterial disease. Venous Doppler ultrasonography findings showed no reflux in the

superficial and deep vein systems. There was neither acute nor chronic deep vein thrombosis. Given the patient's cardiovascular risk factors and bilateral leg swelling, transthoracic echocardiography was performed to rule out a cardiac etiology by confirming normal biventricular function.

The decision was made to proceed with ilio caval venography and subsequent examination using IVUS. Under US guidance, the left great saphenous vein (GSV) was accessed using a micropuncture needle and a 4F sheath and subsequent upgrade to an 11F sheath using a Bentson guidewire. The wire was then advanced proximally into the left common iliac vein (CIV) up to the inferior vena cava (IVC); injection of contrast dye showed no apparent venous stenosis (**Figure 2**).



Figure 2. *The left GSV was accessed using a micropuncture needle and a 4F sheath and subsequent upgrade to an 11F sheath using a Bentson guidewire. The wire was then advanced proximally into the left CIV up the IVC; injection of contrast dye showed no apparent venous stenosis.*

However, on evaluation with IVUS, extrinsic compression of the CIV and external iliac vein (EIV), with approximately 70% reduction in diameter compared with the adjacent normal reference segment, was identified (**Figures 3 and 4**).

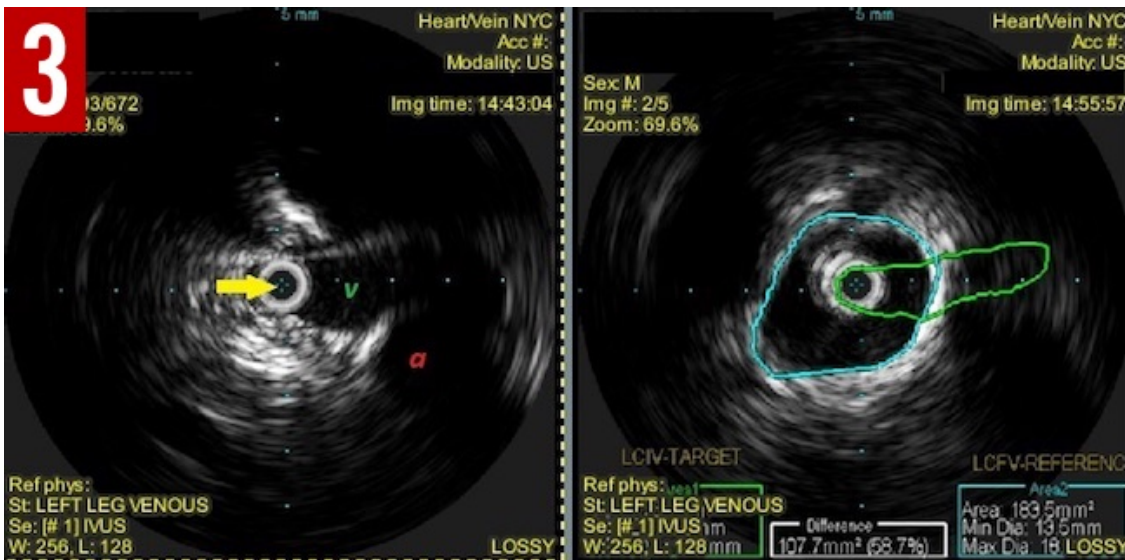


Figure 3. IVUS demonstrating nonocclusive external compression of left CIV (green v) at the level of crossing of the right CIA (red a), described as iliac vein compression. More than 58% luminal narrowing was measured compared with the reference vessel size of the left common femoral vein. Note the IVUS catheter in the CIV (yellow arrow).

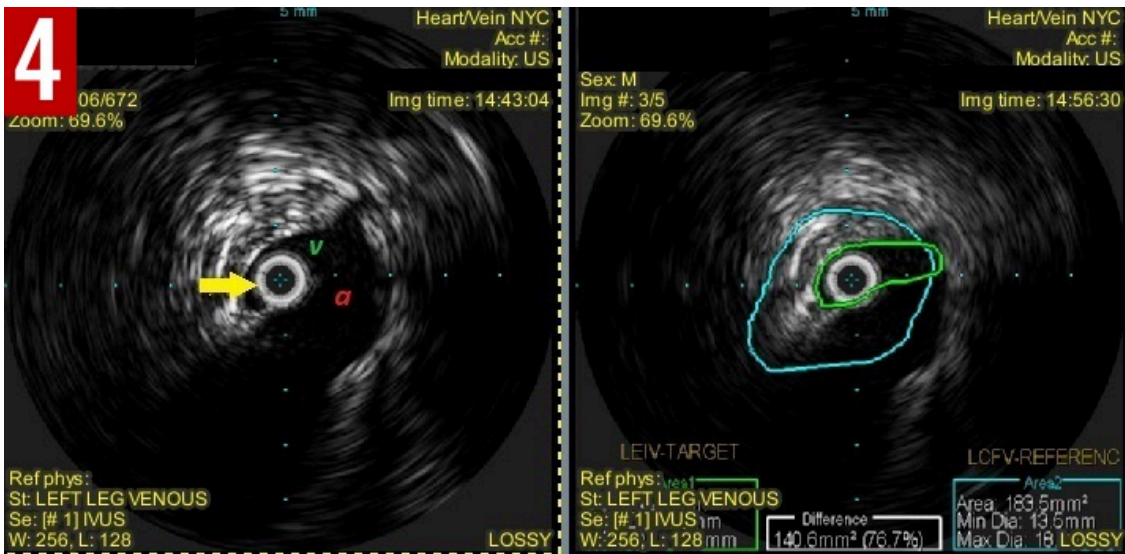


Figure 4. IVUS showing remarkable stenosis of the left EIV (green v) at the arterial-venous crossings (red a). Approximately 76.7% luminal reduction was measured compared with the reference vessel size of left common femoral vein. Note the IVUS catheter in the CIV (yellow arrow).

Balloon dilation with stent deployment was performed successfully. Subsequent IVUS showed a complete resolution of diffuse iliac vein compression by right common iliac artery (CIA) after iliac vein stent implantation (**Figures 5 and 6**). In addition, clear post-procedural improvement to 0% luminal narrowing and wall apposition were demonstrated. Despite this remarkable improvement in luminal patency recorded by IVUS, the post-stent venogram showed no significant difference from the one prior to intervention, demonstrating the important role of IVUS (**Figure 7**) despite no identifiable change in luminogram by contrast fluoroscopy.



Figure 5. IVUS demonstrating the decompressed left CIV after stenting.



Figure 6. Complete resolution of left EIV compression by right CIA after iliac vein stent implantation. IVUS showed clear postprocedural improvement to 0% luminal narrowing and wall apposition.



Figure 7. *The post-stent venogram showed no significant difference from the one prior to intervention, demonstrating the important role of IVUS despite no identifiable change in luminogram by contrast fluoroscopy.*

Discussion. Chronic venous ulcers are a debilitating condition that often substantially impacts the quality of life of those affected. Due to its recalcitrant nature and its tendency to recur, conventional treatments often fail to heal or prevent ulcers. Venous ulcers and associated venous symptoms often restrict mobility. As a result, work productivity and even activities of daily living can be substantially impaired, as in this patient's case.

Nonhealing ulcers resulting from venous disease have many well-described etiologies, including chronic venous insufficiency (CVI) due to incompetent valves, postthrombotic syndrome, ilio caval obstruction, or a combination of these conditions. Genetics and lifestyle also have an important role in developing and remitting venous ulcers.

This patient's ulcers most likely resulted from obstruction due to iliac vein compression, also known as May-Thurner syndrome, which is caused by extrinsic compression at the arterial-venous crossing.¹ Iliac vein compression can cause a full range of CVI classifications, including spider veins, varicose veins, and swelling, hyperpigmentation, and lipodermatosclerosis.²

Delayed investigation of the underlying cause of this patient's venous ulcer resulted in diminished quality of life due to chronic nonhealing ulcers. Iliac vein obstruction has been underappreciated as a cause of venous hypertension and associated venous stasis ulcers due to the lack of noninvasive diagnostic studies that reliably detect obstruction.^{2,3} In one study, 37% of patients with healed or active venous ulcers had evidence of ilio caval venous obstruction.⁴

While conservative management with compression and wound therapy has been known to heal ulcers appropriately, it requires a patient's full adherence, is relatively costly, and can result in unsatisfactory long-term clinical outcomes. Stenting of a compressed iliac vein often results in rapid healing of venous ulcers, as in our patient's case. In a retrospective study, stent

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placement with or without correction of associated reflux resulted in complete venous ulcer healing in up to 76% of cases.²

Furthermore, endovascular evaluation for compression of deep veins is crucial for accurate assessment of luminal patency. As demonstrated in this patient's case, its utility extends beyond effective interventional therapy for nonhealing ulcers, also serving as a valuable diagnostic tool.

Outcome of the case. Iliac vein stenting resulted in rapid healing of this patient's ulcer as well as in significant improvement in limb pain and swelling; those outcomes have continued without further reports of recurrent open ulcers. With relief of orthostatic leg pain and achiness, the patient returned to baseline normal daily activities. His venous stasis hyperpigmentation improved, and his leg swelling was significantly reduced. Stent patency was confirmed by duplex ultrasonography at 1, 3, and 6 months and yearly thereafter.

References:

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