

Iliocaval Stenting for Advanced Chronic Venous Disease

Stenting ilio caval venous lesions on the basis of clinical suspicion and IVUS.

BY JOSE I. ALMEIDA, MD, FACS, RPVI, RVT, AND CRISTAL BOATRIGHT, MMS, PA-C

The etiology of venous obstruction can be primary (nonthrombotic) or secondary (postthrombotic), with roughly equal prevalence estimates in patients with chronic venous disease (CVD). Signs and symptoms of chronic venous obstruction and reflux overlap with some differences. Varicose veins, edema, lipodermatosclerosis, and ulceration can occur with either pathology. Limb swelling beyond ankle edema is rare with pure superficial reflux alone. Combined reflux/obstruction is commonly present in either etiology;^{1,2} however, obstruction alone without reflux occurs in approximately one-third of primary etiology cases.³

Primary obstruction, often referred to as *nonthrombotic iliac vein lesions* (NIVLs), usually arises from compression of the left common iliac vein by crossing of the overlying right common iliac artery. Other compression sites commonly occur at proximal or distal locations such as the hypogastric artery bifurcations. Webs and membranes

resulting from traumatic injury by pulsations of the intimately related artery are frequently present as well. Such lesions are present in 30% to 50% of the general population, but symptom expression occurs in 3% to 5% of cases when additional insult, such as trauma, infection, or reflux, is added.^{4,5}

Percutaneous endovenous stenting has emerged during the last decade as the method of choice to treat femoroiliocaval venous outflow obstruction due to CVD. Accurate hemodynamic tests are unavailable, thus diagnosis and treatment must be based on clinical suspicion and morphologic ultrasound findings.

METHODS

Forty-two limbs with signs and symptoms of advanced CVD were evaluated via intravascular ultrasound (IVUS) with an intent-to-treat protocol and were followed prospectively. In the operating room, the

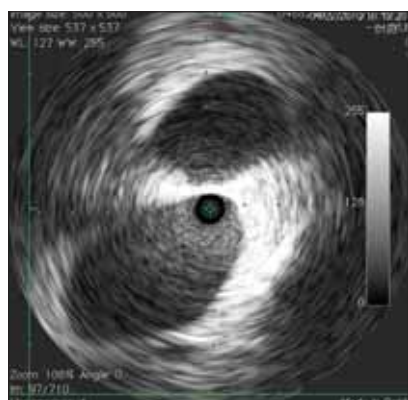


Figure 1. The aorta (above) and the inferior vena cava (below).

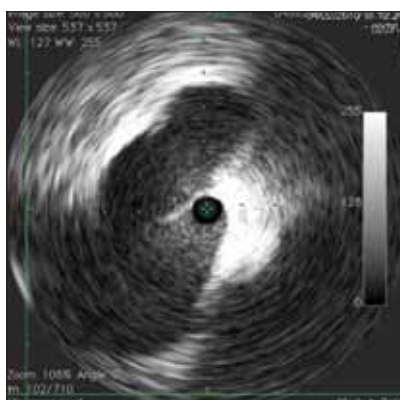


Figure 2. The aortic bifurcation (above) and common iliac vein confluence (below). Notice the right common iliac artery crossing over the left common iliac vein.

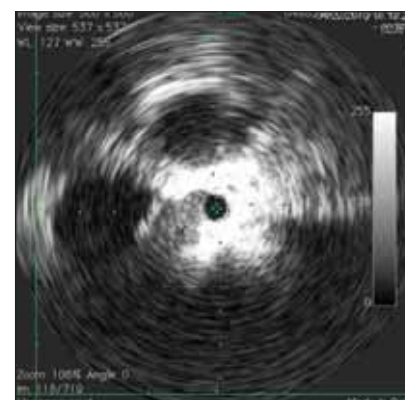


Figure 3. The right common iliac artery (above) compressing the left common iliac vein (below).

femoral vein was accessed percutaneously, and an 11-F sheath was placed. Digital subtraction venography was performed followed by IVUS examination (Figures 1 through 3).

Balloon angioplasty and subsequent stent placement was performed in symptomatic patients who had lesions with > 50% area reduction along the length of the femoroiliacaval outflow tract (Figures 4 through 6). The CEAP (clinical, etiologic, anatomic, pathophysiologic) class was C3 in 18 limbs, C4 in nine limbs, C5 in eight limbs, and C6 in seven limbs (Figure 7); the median age was 57 years (range, 32–74 y); the left-right limb ratio was 1.3:1; the female-male ratio was 1.7:1; and the primary-secondary etiology ratio was 28:14.

RESULTS

Stents were placed in 33 of 42 limbs (79%). Twenty-two of 28 patients (79%) with primary disease were stented. Eleven of 14 patients with secondary disease had stents placed; three patients had chronic total occlusions (CTOs) that could not be crossed. Stenting was performed with no mortality (< 30 d) and low morbidity. Two thrombotic events, both in patients with secondary disease, occurred during the postoperative period (< 30 d). Patency was restored in both cases using thrombolytic therapy. Primary patency was 94%, and secondary patency was 100% in this early series.

The mean initial Venous Clinical Severity Score (VCSS) was 11.2. VCSS improved to a mean value of 8.2 (range, 1–17) at the 1-month follow-up visit and was 7.5 (range, 2–13) at the 6-month follow-up visit.

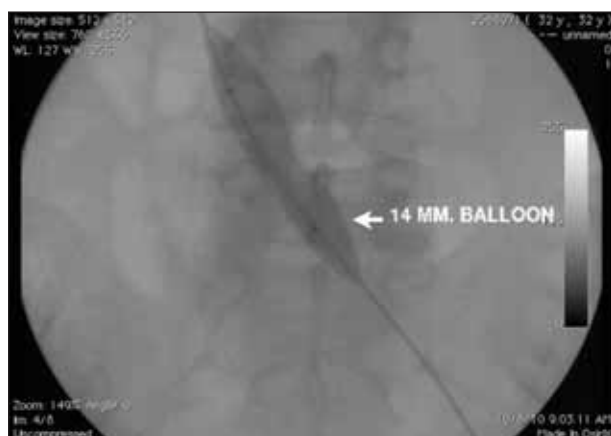


Figure 5. Balloon angioplasty of left common iliac vein. Waste was seen during balloon inflation.

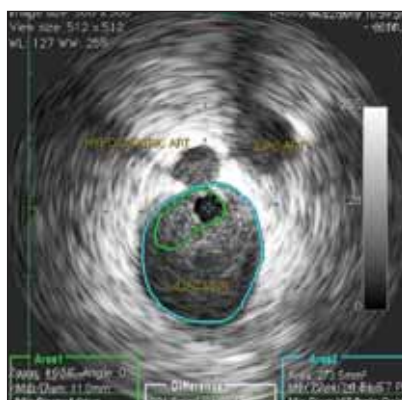


Figure 4. IVUS measurement. The compressed iliac vein (superimposed, green color) area is measured against the normal adjacent external iliac vein segment (blue). There is an 81% area reduction of the left common iliac vein resulting from right common iliac artery compression.

DISCUSSION

In an analysis of 982 iliac vein stent placements from a group in Mississippi,⁶ there was no death, and morbidity was minor. Early (< 30 d) deep vein thrombosis occurred in 1.5% and, later (> 30 d), in 1%. Twenty-three stents (3%) became occluded during the observation period. Stent thrombosis was exclusive to postthrombotic obstruction, and there were no occlusions in limbs stented for NIVLs. Cumulative long-term stent patency was 100% in primary limbs and 86% in postthrombotic limbs at 6 years. Aspirin was used for stent maintenance (the majority of limbs) except in cases of thrombophilia or previously instituted warfarin therapy. The beneficial clinical outcome occurred regardless of

the presence of remaining deep reflux.²

The fundamental problem with these cases lies in the lack of a standard diagnostic modality to identify and quantify the degree of hemodynamically significant venous stenosis. Several studies have shown that IVUS is superior to single-plane venography in detecting the extent and morphologic degree of stenosis.⁷⁻¹⁰ This case series describes the diagnosis and management of iliac vein stenosis in highly selected patients with advanced CVD based on a 50% area reduction on IVUS. The 50% area reduction threshold for treatment was chosen by the Mississippi group 1 decade ago and has been validated through rigorous long-term clinical follow-up.⁸

Transcutaneous duplex ultrasound is unreliable, does not provide accurate measurements, and has a high false-nega-



Figure 6. Stent placement relieving left common iliac vein compression.

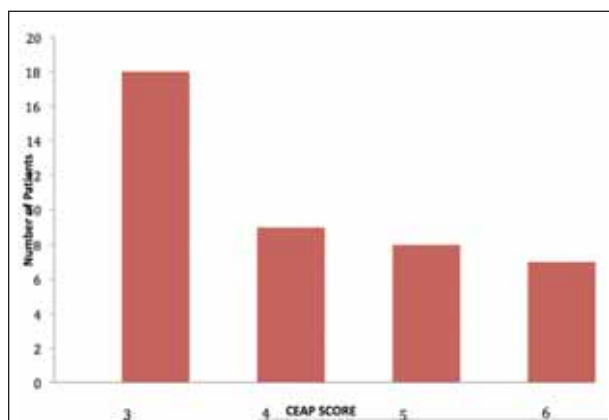


Figure 7. CEAP class distribution.

tive rate.¹¹ High-resolution cross-sectional imaging techniques (magnetic resonance, computed tomography) can detect iliac vein obstructive lesions, but their sensitivity and specificity are currently unknown. Traditional ascending venography via pedal injection has poor diagnostic yield due to the dilution of contrast higher up in the limb. Contrast delineation of the iliac vein segments can be improved with transfemoral injection; nonetheless, diagnostic sensitivity is only approximately 50% because proximal NIVLs are often not circumferential and are obscured in frontal projections.^{3,5}

A number of qualitative (eg, absence of duplex phasicity) and pressure-based techniques (eg, arm/foot pressure, exercise femoral pressure, femoral pressure increment with papaverine femoral artery injection) have been described, but sensitivity of individual tests is only 50% or less.²

The Maimonides group¹² has chosen a 50% diameter reduction by IVUS (75% area reduction) as their trigger point for treatment. In their series, just more than 50% of patients were found to have lesions justifying venous stenting. In contrast, more than 90% of the patients in the Mississippi group's series underwent stent placement. The Maimonides group noted a particular benefit in C6 patients, of which more than 50% managed to maintain healing of their ulcers after iliac vein stenting.

CTOs of the iliac vein have been reported to be percutaneously recanalized in as many as 85% of cases—although only in 50% with concomitant inferior vena cava occlusions.^{13,14} Our experience has only yielded a 40% (two of five cases) successful crossing rate. CTO crossing devices have recently become commercially available and may yield higher wire passage rates in the future.

In this series, VCSS scores improved during the postoperative period. It should be noted that, anecdotally, pain relief often occurred immediately; however, it took some patients as long as 3 to 6 months to observe noticeable improve-

ment in their symptoms, especially for those with edema. In two patients with venous ulceration, the ulcers healed initially postprocedure but quickly reopened and then rehealed and remained healed. This suggests that there is a lag period at the microcirculatory level for venous decompression to occur.

CONCLUSION

Our approach to patients with C3 through C6 disease is to treat their superficial reflux first. In patients with persistent signs and symptoms of CVD who wish to explore the possibility of a deep venous outflow problem, we offer an IVUS examination with an intent-to-treat approach. Because information from cross-sectional imaging studies is expensive and nondefinitive, we omit these studies from our protocol. Venous stenting can be performed with low morbidity and mortality rates under IVUS control and has resulted in major symptom relief in patients with advanced CVD. Validation with objective clinical scores and quality-of-life questionnaires are needed for future studies. Currently, open surgical techniques are used only in cases of stent failure. This treatment strategy should be considered in patients with advanced CVD. ■

Jose I. Almeida, MD, FACS, RPVI, RVT, is Director of the Miami Vein Center and Voluntary Associate Professor of Surgery at the University of Miami-Jackson Memorial Hospital in Miami, Florida. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Almeida may be reached at dralmeida@mac.com.

Cristal Boatright, MMS, PA-C, is with the Miami Vein Center in Miami, Florida. She has disclosed that she holds no financial interest in any product or manufacturer mentioned herein.

1. Johnson BF, Manzo RA, Bergelin RO, Strandness DE Jr. Relationship between changes in the deep venous system and the development of the postthrombotic syndrome after an acute episode of lower limb deep vein thrombosis: a one- to six-year follow-up. *J Vasc Surg.* 1995;21:307-312; discussion 313.
2. Raju S, Darcey R, Neglén P. Unexpected major role for venous stenting in deep reflux disease. *J Vasc Surg.* 2010;51:401-408; discussion 408.
3. Raju S, Neglén P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogenicity. *J Vasc Surg.* 2006;44:136-143; discussion 144.
4. Kibbe MR, Ujiki M, Goodwin AL, et al. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg.* 2004;39:937-943.
5. Negus D, Fletcher EW, Cockett FB, Thomas ML. Compression and band formation at the mouth of the left common iliac vein. *Br J Surg.* 1968;55:369-374.
6. Neglén P, Hollis KC, Olivier J, Raju S. Stenting of the venous outflow in chronic venous disease: long-term stent-related outcome, clinical, and hemodynamic result. *J Vasc Surg.* 2007;46:979-990.
7. Neglén P, Raju S. Balloon dilation and stenting of chronic iliac vein obstruction: technical aspects and early clinical outcome. *J Endovasc Ther.* 2000;7:79-91.
8. Neglén P, Raju S. Intravascular ultrasound scan evaluation of the obstructed vein. *J Vasc Surg.* 2002;35:694-700.
9. Forauer AR, Gemmette JJ, Dasika NL, et al. Intravascular ultrasound in the diagnosis and treatment of iliac vein compression (May-Thurner) syndrome. *J Vasc Interv Radiol.* 2002;13:523-527.
10. Satokawa H, Hoshino S, Iwaya F, et al. Intravascular imaging methods for venous disorders. *Int J Angiol.* 2000;9:117-121.
11. Hurst DR, Forauer AR, Bloom JR, Greenfield LJ, et al. Diagnosis and endovascular treatment of ilioacaval compression syndrome. *J Vasc Surg.* 2001;34:106-113.
12. Hingorani A. Iliac-femoral venous stenting for lower extremity venous stasis symptoms. *J Vasc Surg.* In press.
13. Raju S, Hollis K, Neglén P. Obstructive lesions of the inferior vena cava: clinical features and endovenous treatment. *J Vasc Surg.* 2006;44:820-827.
14. Kolbel T, Lindh M, Akesson M, et al. Chronic iliac vein occlusion: midterm results of endovascular recanalization. *J Endovasc Ther.* 2009;16:483-491.