flux. The presence of NIVCLs is an important reason underlying symptom recurrence after EVLA for superficial reflux in the left lower extremity.

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INVITED COMMENTARY

Long-Term Outcomes of Stent Placement for Symptomatic Nonthrombotic Iliac Vein Compression Lesions in Chronic Venous Disease

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ABBREVIATION

NIVL = nonthrombotic iliac vein lesion

Ye et al (1) report the long-term outcomes of iliac vein stent placement in a large series of patients with primary disease. Their experience broadly parallels results from other centers and typifies some of the surprising features of stent application in this disease. Of particular note is the near absence of stent thrombosis, as reported in several other series, even those that used only aspirin for long-term stent maintenance. This is remarkable, considering the natural potential for thrombosis that exists in the venous system. Also noteworthy is the excellent clinical outcome, particularly the healing of venous ulcers. The authors do not reveal the incidence of deep reflux in their study group; only the number with superficial reflux is

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S.R. has stock and ownership in Veniti (St. Louis, Missouri), a medical device company, and a royalty agreement on the sale of venous stents, which are not yet approved for sale.

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provided. In the author's experience, approximately 50% of patients have deep reflux alone or in combination with superficial reflux. The belief that venous ulcers are caused by reflux is widely held. Healing of venous ulcers with stent treatment therefore brings into question conventional concepts. More surprising, such ulcers seem to heal with stent placement alone, even in the presence of severe reflux that is left uncorrected (2).

Nonthrombotic iliac vein lesions (NIVLs) have many odd features, and their origin remains an enigma. A postthrombotic etiology is definitely ruled out based on histologic features. There is evidence that some of these lesions are ontogenic, as they occur at embryologic fusion planes, and lesions have been found in fetuses and infants (3). However, the majority are probably traumatic from repeated pulsations of the intimately associated artery. Many have intraluminal spurs, bands, webs, and membranes. Some may not have a single lumen at the lesion site but multiple ones separated by bands. As the lesion is a combination of external compression, mural fibrosis, and intraluminal obstructions, the term NIVL (4) is preferred over "nonthrombotic iliac vein compression lesions" as used by the authors (1), which misleadingly implies that the lesion is only compressive. Lesions occur on both sides, though favoring the left side at a ratio of approximately 3:1. Lesions also occur at locations distal to the "classic site," particularly near the hypogastric orifice and further distally behind the inguinal ligament.

As the authors point out (1), the diagnostic sensitivity of venography is poor. This may be improved somewhat by including oblique views, but significant mural and intraluminal features may be missed, which is also true for other external imaging modalities. Collateral vessels are present in only approximately one third of cases and are a poor guide to selection of cases. Intravascular ultrasound (US) has a diagnostic sensitivity approaching 90%, as all the components of the pathologic process as described can be discerned. Associated distal lesions are better appreciated with intravascular US than with venography. Some membranous lesions near the hypogastric orifice can be missed by intravascular US and become detectable by routine "balloon-sizing" maneuvers. The authors' exclusive reliance on external imaging modalities may explain the relatively low incidence of primary disease in their chronic venous disease case mix. Missing associated lesions during stent correction of NIVL is a significant factor in causing residual or recurrent symptoms (particularly swelling) that require repeat interventions (5). All major and minor lesions are therefore treated at the first instance, extending the stent below the inguinal ligament if necessary. The latter practice has met with no adverse sequelae, unlike in the arterial system (6).

The concept of "significant" or "critical" stenosis is derived from the arterial side and is based on the degree of stenosis required to restrict peripheral (tissue) perfusion. Tissue perfusion is not an issue in central venous stenoses, but peripheral venous hypertension is, which is the basis of symptoms. An increase of only a few mm Hg of blood pressure at the venular end of the capillary can result in edema and other symptoms of chronic venous disease. There is no published study to define a stenotic threshold for a single focal lesion or for a combination of lesions in series necessary to increase peripheral venous pressure. Nevertheless, stent correction of the lesion should restore the vein to normal anatomic caliber to ensure adequate peripheral venous decompression. The common practice of incomplete correction of arterial stenoses ("perfect is the enemy of good") serves poorly in the venous system. The authors correctly employed large-caliber stents appropriate for the location in this series (1). The use of smaller-caliber stents may result in residual symptoms. Rupture and bleeding from aggressive dilation of NIVLs seldom occurs.

Patients' self-assessment of leg swelling as employed in this study (1) can differ substantially from objective assessment because of the influence of associated pain. Subjective perception of swelling is exaggerated when associated with pain and understated when the associated pain is relieved by the stent placement procedure. A placebo effect is always a factor to be considered when using subjective measures of outcome.

The study of Ye et al (1) adds to the growing literature supporting stent correction as an attractive choice in patients with chronic venous disease caused by NIVLs.

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