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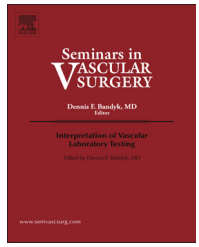

Treatment of iliac-caval outflow obstruction

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ABSTRACT

The importance of the obstructive component in chronic venous disease (CVD) with ulceration has been emphasized recently for a venous condition that has primarily focused on the reflux component. Modern imaging techniques, particularly intravascular ultrasound, have shown the frequency of the obstructive element in both post-thrombotic and nonthrombotic disease. The emergence of iliac vein stent angioplasty and its good results in the treatment of large vein and other diverse CVD subsets has strengthened the role of obstruction. Lower-limb symptom diminution after iliac vein stenting in patients with concomitant reflux has been surprising, and has prompted a better understanding of CVD pathology. The technique of venous stenting differs from arterial in both technique and purpose. Mere restoration of forward flow is not sufficient; adequate decompression of the peripheral veins with reduction in ambulatory venous hypertension must be achieved. This requires implantation of large-diameter stents approximating normal anatomy. Stent recanalization of chronic total occlusions of the iliac-caval segments—even long occlusions involving the entire inferior vena cava (IVC)—can be successfully carried out, supplanting prior difficult open techniques, and this approach is applicable to patients with thrombosed IVC filters. Iliocaval stent angioplasty is safe, with low mortality and morbidity (<1%), and a cumulative patency ranging from 90% to 100% and 74% to 89% for nonthrombotic and post-thrombotic disease, respectively, at 3 to 5 years. Clinical relief of pain ranged from 86% to 94% and relief for swelling ranged from 66% to 89%; and 58% to 89% of venous ulcers healed. Procedural success in recanalization of chronic total occlusion lesions ranged from 83% to 95%, but long-term patency of stents in recanalized chronic total occlusion lesions is 10% to 20% lower than for stenotic lesions. Initial stent treatment does not preclude later open correction of obstruction or reflux in case of stent failure. These features, combined with the minimally invasive nature of the stent technique, have opened this avenue of treatment to a larger portion of the symptomatic CVD population.

1. Introduction

The central theme in chronic venous disease (CVD) in the past half century has been reflux in pathophysiology, diagnosis, and treatment. There was recognition that obstructive pathology occurred in post-thrombotic disease and less often in primary disease (eg, May-Thurner syndrome); but the affected subsets were believed to be relatively small. This underestimation was a result of the use of diagnostic techniques that were insufficiently sensitive to obstructive lesions. The recent advent of intravascular ultrasound and high-resolution imaging techniques has led to the realization that obstruction of the iliac-caval segments occurs ubiquitously in both post-thrombotic and nonthrombotic subsets [1,2].

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Development of percutaneous stent technology has allowed clinical validation of correcting central venous obstruction in CVD patients [3]. Being minimally invasive with a high safety profile, the stent technique is more readily applied to a larger pool of patients than was possible with open surgical techniques to correct deep venous pathology. An unexpected observation was that patients who also had reflux—even those with axial or multisegmental reflux—were relieved of their symptoms after stent correction of obstruction, despite the presence of uncorrected reflux [4]. This observation suggested that stent treatment should be offered to most patients with CEAP (clinical, etiology, anatomical, pathophysiology) clinical class 3 and higher before correction of deep reflux is considered.

2. Pathophysiology

Strandness and colleagues showed in a landmark prospective study that combined obstruction/reflux was the eventual outcome in the majority (≈90%) after deep venous thrombosis (DVT) [5]. Factors that predispose patients to developing post-thrombotic syndrome include DVT involvement of the of the iliac vein, its poor resolution, and recurrent DVT. The lesions can vary from obstructive stenosis to chronic total occlusion (CTO). A special form of diffuse stenosis was described by Rokitanski (Rokitanski stenosis) in which a restrictive post-thrombotic perivenous envelope develops to retard collaterals and narrow the lumen. This form is often found in combination with focal stenosis [6].

Collaterals are seen on venography in only about one-third of patients with iliac vein stenosis; collateral formation is poorly understood. Visualized collaterals are of high resistance, as evidenced by their prompt disappearance when the main venous tract is stented (Fig. 1). This might be because collateralization (eg, transpelvic, ascending lumbar, pudendal) depends on the tributaries in which flow has to be reversed to drain into the opposite iliac vein or vena cava. Even though collateralization might appear profuse on venography, the poor conductance of smaller-caliber veins compared with the normal iliac vein to carry venous outflow is not fully appreciated. Because conductance is related to the fourth power of radius (Poiseulle), a total of 256 collaterals, each one-fourth the size of the normal iliac vein, will be required to equal its flow and normalize distal venous hypertension. The clinical features of venous obstruction are related to peripheral venous hypertension. Collateralization is superior around obstructive lesions of the IVC or the femoral vein. The azygus and profunda femoris veins are embryologic predecessors of adult structures; collateral flow is in the same direction (not against valves, if present). These embryologic analogues develop rapidly to equal the caliber of the occluded vein—the reason why symptoms recede in a matter of months after onset of DVT or even after ligation and excision of the IVC and femoral veins, respectively. Thrombotic involvement of the vena cava or the femoral vein in neonates or during childhood might produce no symptoms at all, only to be discovered as incidental findings during imaging studies later in life.

In contrast, nonthrombotic obstructive lesions in the iliac-caval vein segments are due to fibrotic or membranous stenosis that develop where the vein is crossed by arteries or fibrous ligaments, as shown in Figure 2. The lesions may be ontogenic in some instances, as the location often corresponds to embryologic fusion planes. Most others are thought to be largely a result of traumatic injury from repeated pulsations of the closely related artery. They take time to develop, as they are seen only rarely in the pediatric age group. A thrombotic etiology is ruled out, as the pathognomic vascular invasion is absent. The lesions are typically subsegmental and focal. Although commonly referred to as “iliac vein compression syndrome,” a term popularized by Cockett, it is an unfortunate misnomer because compression is just one component of the lesion [7]. Focal strictures, trabecular strands, and membranes involving mural and luminal elements of the vein at the location are dominant aspects of the pathology. The lesion was described first by McMurrich as early as in 1905 later amplified by Ehrich and Krumbhaar [8,9]. The lesion is commonly referred to as May-Thurner syndrome, after May and Thurner brought it to prominence in the 1950s by detailed autopsy studies [10]. Controversy regarding the pathophysiologic significance has persisted from the start because the descriptive autopsy material came in asymptomatic individuals. In a modern replication of the earlier autopsy studies, Kibbe et al found the lesion in as much as two-thirds of asymptomatic individuals in incidental imaging studies carried out for other reasons [11]. Some texts describe the lesion as a “normal” anatomic variant. Most symptomatic patients with CVD harbor the lesion, however, it is present in approximately 80% of the symptomatic subset [12,13]. Based on modern intravascular ultrasound findings, both sexes, both sides, and all age groups are now known to be affected, not merely the left lower limb of young females as was once thought. There is no question that stent correction of the lesion in these individuals provides symptom remission.

Why does the lesion remain asymptomatic in so many while its correction in symptomatic patients provide symptomatic relief? A plausible explanation is that the lesion is permissive, remaining silent until additional insult, such as trauma, cellulitis, or additional pathology (eg, reflux, DVT, sedentary leg dependency) precipitates symptoms. It is not uncommon to encounter patients who develop leg swelling and pain related to the iliac vein lesion after events such as fracture, knee replacement, or an attack of cellulitis. The iliac vein lesion in these patients has undoubtedly preceded the secondary event. Permissive pathologies are ubiquitous in human disease and many remain silent until onset of secondary injury. One well-known example is ureteric reflux, which can be asymptomatic until onset of infection. Other examples include obesity and diabetes, hypertension and heart failure, carotid stenosis and stroke, and obesity and diabetes. In all such cases, correction of the permissive lesion is the first line of treatment and is often curative.

3. Indications

Most patients with CEAP clinical class 3 and higher who have failed conservative therapy with severe symptoms can be considered. The procedure has proven to be low risk, even in