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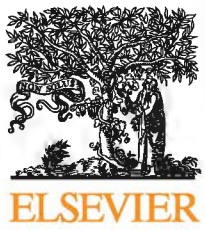

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Treatment of iliac-caval outflow obstruction

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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

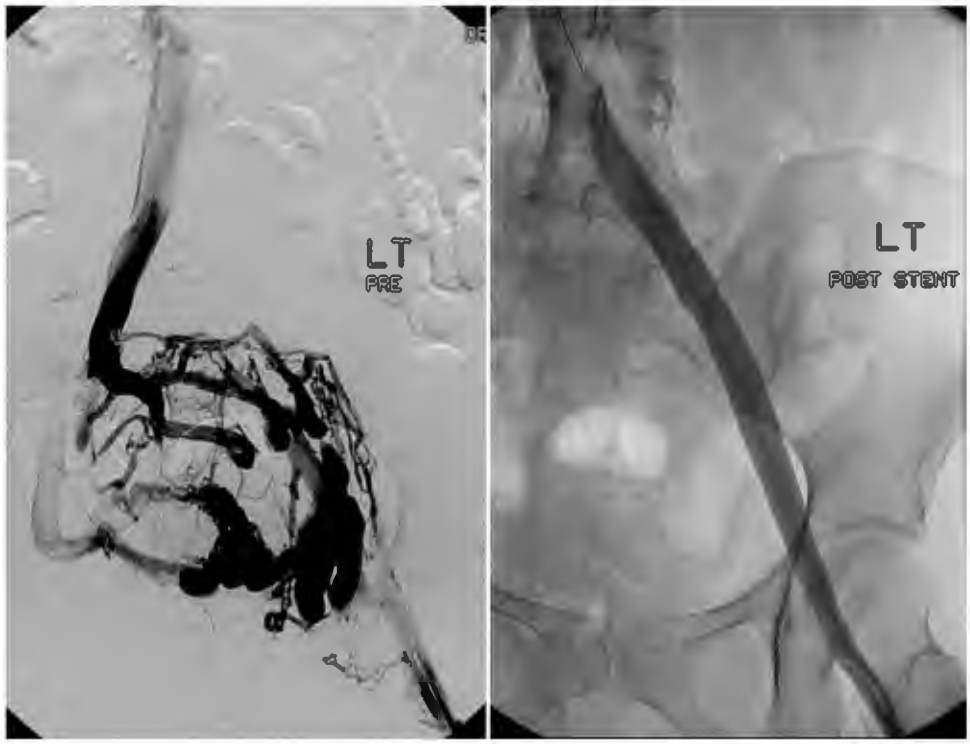

Fig. 1 – Venogram images of a left iliac vein occlusion before (left) and after stent angioplasty (right). Note the absence of collateral veins after stenting. LT, left.

the geriatric population. Patients often harbor excessive fear of blood clots and "circulation problems." The relatively benign nature of the disease in general, and that the purpose of the intervention is to provide symptom relief to improve quality of life and is not essential for preservation of limb or life, must be clearly explained to the patient to obtain informed consent.

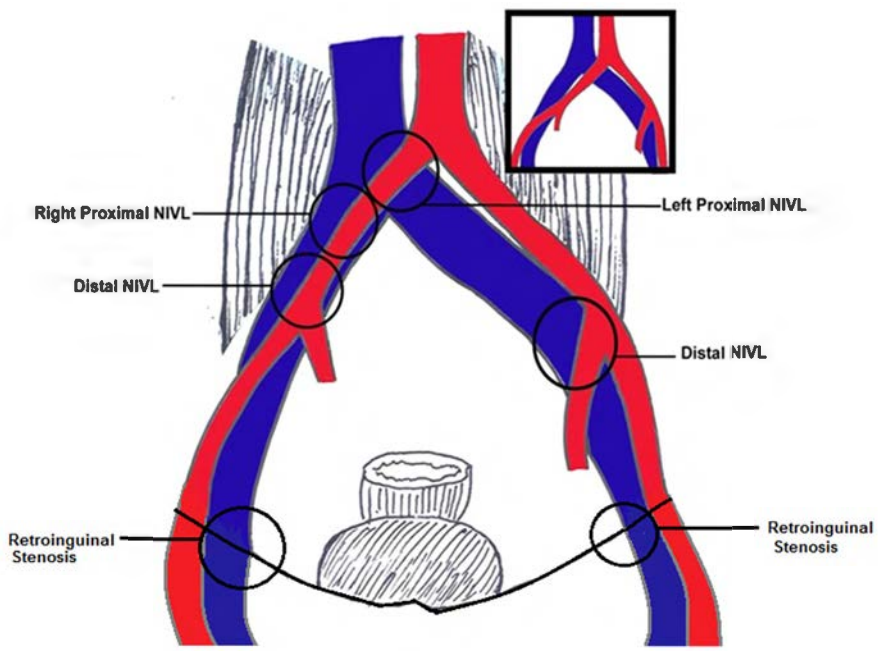

Fig. 2 – Schematic of iliocaval venous segment showing sites of venous obstructive conditions in patients with May-Thurner syndrome. Obstructive lesions (circles) are found using intravascular ultrasound behind arterial and ligamentous crossover points. The lesions occur in both sexes, both sides and in all age groups. NIVL, non-thrombotic iliac vein lesions.

4. Diagnosis

Traditional venography even via the transfemoral route has only about 50% sensitivity because diffuse lesions of the Rokitanski type are easily missed (Fig. 3) and focal nonthrombotic lesions frequently occur only in the anteroposterior plane and are obscured in frontal projections [7,12,13]. The sensitivity of transfemoral venography can be increased by biplane imaging and by measurement of femoral venous pressure measurements during venography [13,14]. In my experience, imaging techniques (eg, duplex, magnetic resonance venography, computed tomographic venography) have proven to be similarly insensitive. Recent work (unpublished) indicates that the sensitivity of imaging techniques can be considerably increased if diameter measurements of the iliac vein segments are included for comparison with anatomic norms for assessment.

5. Technique

Principles of venous stenting are different and, in many ways, are opposite of arterial stenting basics [15]. Access can be obtained in deep locations, as the low venous pressure results in low incidence of access complications, particularly if sealing devices are used routinely. Our favorite approach is the femoral vein at mid-thigh level, which allows room for infrainguinal extension of the stent. This is a direct short antegrade route to the obstruction, which enhances ease of instrumental manipulation. The supine position is an advantage in an increasingly supine population, as respiratory compromise that can occur in the prone position for the popliteal approach is avoided. The popliteal and the jugular can be used as alternative approaches in special circumstances. General anesthesia provides for superior cardiopulmonary control, particularly in the geriatric population with comorbidities. Balloon dilation is often exceedingly painful under local anesthesia, even with intravenous supplementation. Recoil of lesions after balloon dilation of iliac vein stenosis is nearly universal and stenting is required [16]. Unlike in the arterial system, hyperdilation and use of large balloons and stents are safe. Rupture and hemorrhage are extremely rare, even in recanalization of occluded iliac vein cords, because of restrictive perivenous fibrosis and anatomic coverings, and low venous pressure. The purpose of venous stenting is to reduce peripheral venous pressure, which is the basis of symptoms, therefore, large-bore stents approximating normal anatomy should be used for effective peripheral venous decompression. The following diameters are anatomic minima in normal adults: IVC, 20 mm; common Iliac vein, 16 mm; external iliac vein, 14 mm; and common femoral vein, 12 mm. It is common arterial practice to size the stent according to the normal adjacent segment. This will result in serious understenting in iliac veins because adjacent segments are often diffusely stenotic, as described earlier. My colleagues and I routinely size stents by 2 mm over anatomic minimums to allow for later hyperdilation during reinterventions, if required. In contrast, even slight undersizing, for example, use of a 14-mm stent in the iliac vein (16 mm) will result in an iatrogenic stenosis of about 25%, even greater if in-stent restenosis of 20% to 25%, which is frequently seen, develops over time [17]. Use of an 8-mm or 10-mm stent in the iliac vein based on arterial practice (perfect is the enemy of good) is a nearly irretrievable iatrogenic stenosis that is not easily corrected. All lesions >20% (area) should be stented without skip areas to avoid residual symptoms and interstent stenosis. This will result in stenting from IVC to the common femoral vein below the inguinal ligament in most cases, whether thrombotic or nonthrombotic. We seldom see stent fracture or erosions in braided Wallstents from crossing the groin crease or increased stent thrombosis [18]. Extensive metal load in the iliac-caval-femoral system appears to be well tolerated (Fig. 4). Ignoring lesions to minimize stent length often results in stent thrombosis or residual symptoms.

Unlike in the arterial CTO lesions, subintimal dissection is not feasible or practiced in recanalizing iliac vein CTO lesions.

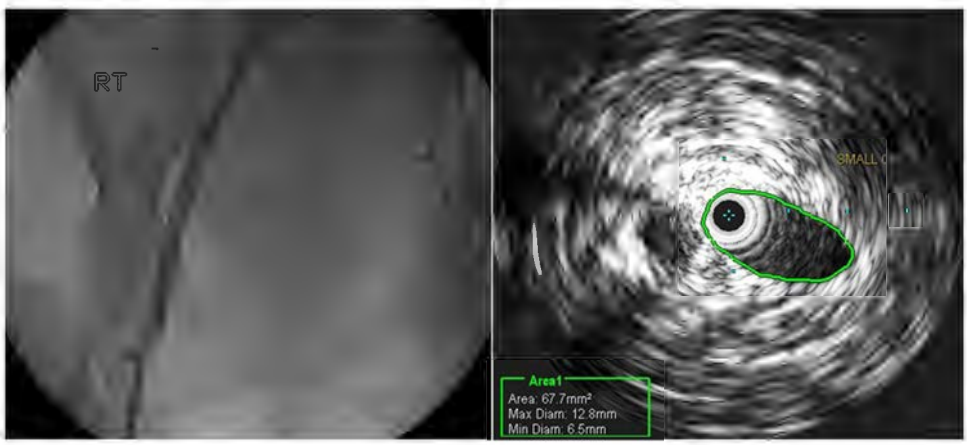

Fig. 3 – Venogram of a long diffuse (Rokitanski) stenosis (left). Note the absence of collaterals. Diameter reduction is not readily apparent by venography, but cross-sectional lumen reduction by intravascular ultrasound shows 70% area stenosis (right). Focal lesions often occur in association with Rokitanski stenosis, and can be underestimated if percent of stenosis is calculated based on the adjacent segment measurements. The degree of stenosis is best calculated based on anatomic minima for the location (see text). CIV, common iliac vein.

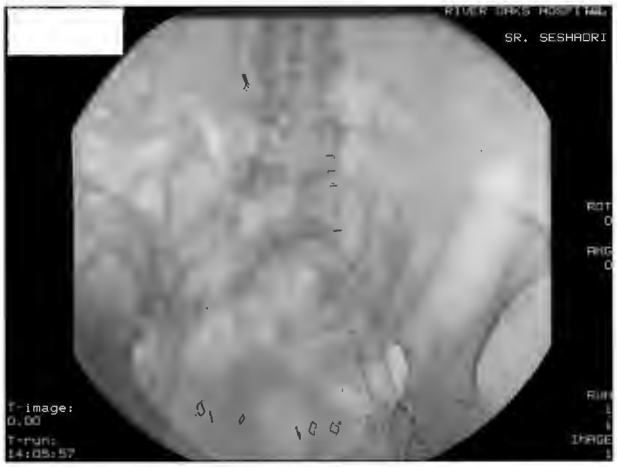

Fig. 4 – Radiography image after recanalization of occluded inferior vena cava secondary to chronic filter thrombosis. The filter was compacted by balloon dilation and then stented across. The stent stack extends into the common femoral vein bilaterally crossing the groin crease. Extensive metal load is well tolerated but missed lesions often lead to stent thrombosis due to poor inflow/outflow. Stenting (braided) across the groin crease has not been associated with stent fracture or erosion.

The post-thrombotic vein wall is a thick fibrous tissue without discernable layers; headway is made by threading a hydrophilic guide wire through the maze of trabaculae, sometimes dense and often not. Because contrast does not flow easily through such trabeculae, seemingly daunting long lesions on venography can often be recanalized with surprising ease. Even extensive CTO lesions involving the entire IVC have been recanalized safely with little morbidity (no renal or hepatic compromise) [19,20]. Guide-wire perforations are rare during recanalization if sharp or rigid ends are not used and the hydrophilic guide wire is advanced along the projected normal course of the vein. When the guide wire veers off course, it can be withdrawn and readvanced along the proper course without aborting the procedure for fear of hemorrhage. The low venous pressure and perivenous covers limit bleeding.

It is nearly always necessary to cross the iliac-caval junction, which is a common choke point. If the stent is not extended for 2 to 3 cm cephalad, squeezing of the stent distally with recurrence of stenosis is to be expected. It is nearly impossible to place the stent precisely at the iliac vein ostium beyond the stenosis with venographic control alone. The lesion at this location is often spiral, without clear end points; the level of the confluence is variable; and identifying clues, such as the contralateral iliac ostium, are often not opacified on venography. Intravascular ultrasound remains the mainstay of iliac vein stenting [21]. The ability to use intravascular ultrasound planimetry to identify and gauge stenotic lesions; absence of radiation hazard, which allows repeated use of stenting to ensure defect-free stent placement; and the ability to identify iliac vein confluence with confidence are major advantages over venography.

Managing the iliac vein confluence during iliac vein stenting is currently unsatisfactory due to the absence of suitable stent designs [22]. We have routinely extended braided stents for 3 to 5 cm beyond the confluence to avoid the problems mentioned. Acute jailing of the opposite iliac seldom occurs with contralateral flow maintained through and around the stent, but chronic subclinical jailing remains a concern. The vena-caval extension does complicate subsequent contralateral stenting, which is required in roughly 20% of patients, as the disease is often bilateral. These concerns can be ameliorated by deploying a Z-stent with widely spaced struts across the iliac-caval junction at the upper end of the Wallstent stack [23]. This technical modification makes sequential or simultaneous bilateral stenting much easier than other techniques (Fig. 5). The “double-barrel” technique is not suitable for delayed sequential stenting as the newly placed limb is prone to be compressed by the older stent, which has become stiffer with tissue encrustment. Late compression of one of the limbs by its companion in the double-barrel technique also occurs with time (unpublished data).

Many patients with CVD have combined superficial and deep reflux. The superficial system is generally addressed first. In some instances, a combined ablation and stenting procedure makes sense and is easily carried out [24]. For instance, saphenous vein is likely to be only a minor contributor to pathology, if it is of small caliber (<5 mm) or the clinical presentation (eg, massive swelling, advanced lipodermatoscleorosis, severe diffuse orthostatic pain) is obviously centered on deep venous pathology.

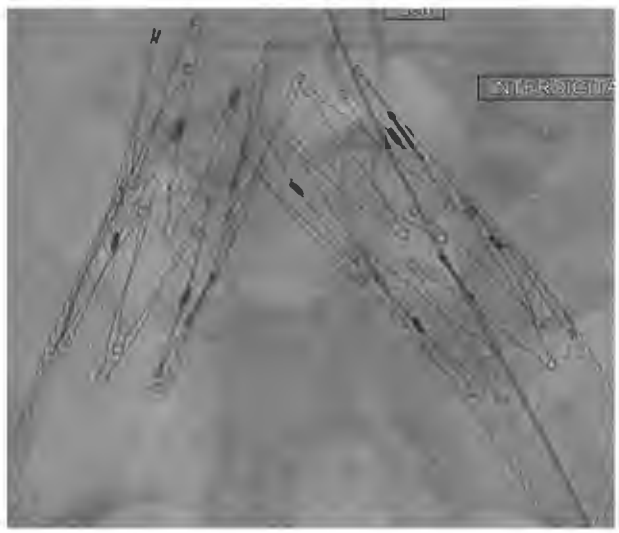

Fig. 5 – Radiography image showing Z-stent modification to restore normal patency to the common iliac vein bifurcation. A Z-stent is deployed inside of the Wallstent stack at the upper end. The upper half of the Wallstent projects into the inferior vena cava, which simplifies bilateral stenting. Interdigitation of the bilateral Z-stent struts keeps the overall diameter within bounds within IVC. The lower half of the Z-stent is restrained by the Wallstent, preventing erosion while adding to the radial strength at the iliac-caval junction, which is a critical choke point. The Z-stent should be oversized relative to the Wallstent to prevent embolization.

6. Postoperative anticoagulation and stent surveillance

Heparin or Bivalrudin is used intraoperatively and low-molecular-weight heparin for short duration postoperatively for standard prophylaxis. The stenting procedure per se does not require chronic anticoagulation (ie, warfarin or newer generation). Acetylsalicylic acid has been adequate for stent maintenance, particularly in nonthrombotic cases. Chronic anticoagulation is used for standard indications (eg, thrombophilia, recurrent thrombosis, and unprovoked thrombosis) in post-thrombotic cases. We have used chronic anticoagulation in extensive IVC stenting as a precautionary measure.

Duplex surveillance of the stent has proven to be reliable both for detecting stent thrombosis and development of in-stent restenosis. Stent comrpression from outside elements with or without in-stent restenosis is unique to iliac vein stenting [16]. Stent compression is easily missed if stent diameter measurements are not a routine part of the examination. Duplex surveillance is carried out the day after the procedure, and 4 weeks, 3 months, and yearly thereafter. Recurrent or residual symptoms require interval examination. Most thrombosed stents can be reopened if detected within 4 weeks of onset and occasionally even beyond this window (a few cases even a year later). A trial at reopening the stent by wire passage and balloon dilation would be worthwhile, even late after stent thrombosis.

7. Reinterventions

Approximately 20% of stented limbs have required reinterventional correction over time for stenting [25]. Up to 25% of lumen compromise by in-stent restenosis (ISR) is common and is without symptoms if large-bore stents had been implanted. The incidence of severe ISR (>50%) is relatively rare and occurs in about 5% of stented limbs, mostly in the post-thrombotic subset [17]. Residual or recurrent symptoms are the indications for reinterventional correction, not the detection of ISR by itself. The pathology of ISR in iliac veins probably differs from that seen in arteries. Venous ISR appears to be largely composed of thrombus layering from sluggish flow.

ISR can usually be cleared by high-pressure balloons (16 atm). It is important to search for inflow or outflow problems that might have promoted development of ISR and correct them. Stent compression is more difficult to correct. In approximately 2% of all stented limbs, the problem becomes recalcitrant to balloon dilation.

8. Results

Iliac vein stenting is safe and effective, with negligible mortality [3]. In my experience, morbidity after iliac vein stenting is mostly minor; about 25% of patients have back pain that is controlled easily with analgesics. The incidence of early (30 days) and late DVT is approximately 3% each, which is not different from the incidence in native disease.

A review of worldwide iliac and IVC stent series found cumulative patency ranged from 90% to 100% and 74% to 89% for nonthrombotic and post-thrombotic disease, respectively, at 3 to 5 years [26]. Clinical relief of pain ranged from 86% to 94% and for swelling ranged from 66% to 89%; and 58% to 89% of venous ulcers healed. Procedural success in recanalization of CTO lesions ranged from 83% to 95%. Long-term patency of stents in recanalized CTO lesions tended to be 10% to 20% lower than in stenotic lesions. Thrombophilia has not been associated with increased stent thrombosis in several series.

A major surprise in our series was complete long-term healing of stasis ulcers in 65% to 80% of limbs, despite the presence of severe reflux (Fig. 6) [27]. Nonhealing occurs largely in a post-thrombotic subset with multisegment (>3 segments) reflux. About half of the patients using stockings are able to discard them after the stenting procedure.

9. Summary

The pathology and clinical orientation in symptomatic CVD has been focused on the reflux element. With newer diagnostic techniques and the emergence of venous stenting, the focus has turned to obstruction, which is now recognized as a common and important element. This is true for both thrombotic and nonthrombotic disease. Iliac-caval stenting has proven to be both safe and very effective, with high stent patency rates. Because the procedure is minimally invasive, it is well tolerated by most, extending treatment to patient subsets that would be normally excluded from more complex open procedure alternatives. A surprising clinical observation is that many stented patients seem to get symptom relief, tolerating untreated residual reflux, even in cases of axial or multisegment reflux. This is an indication that traditional

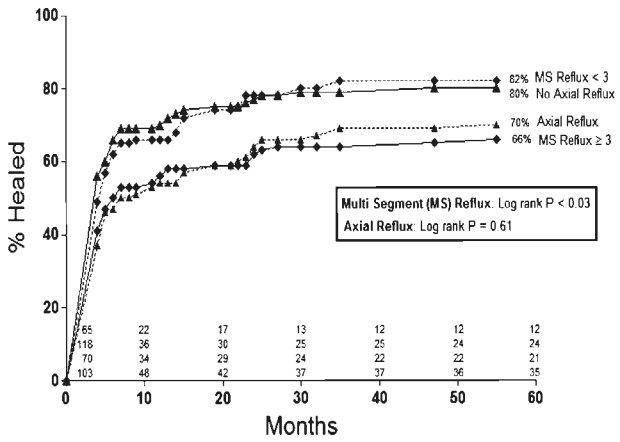

Fig. 6 – Graph showing the percent of leg ulcer healing with time after iliocaval venous stenting for combined obstructive reflux, even if the reflux component is severe. There is no significant difference in cumulative ulcer healing between limbs with and without axial reflux after iliac vein stenting. Ulcer healing rate is significantly lower in limbs with deep reflux in ≥3 segments compared with limbs with reflux in fewer segments. Even so, complete and durable ulcer healing occurs in about two-thirds of the limbs with residual uncorrected reflux in ≥3 deep vein segments.

views of CVD pathophysiology are incomplete and further research is needed.

A major deficiency in current venous stent technology is the absence of a dedicated venous stent optimized for the iliac-caval anatomy and the unique pathology, which is so different from arterial stenotic lesions.

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