Cardiovasc Intervent Radiol (2014) 37:889–897
DOI 10.1007/s00270-014-0875-4

CIRSE Standards of Practice Guidelines on Iliocaval Stenting

Andreas H. Mahnken · Ken Thomson · Michiel de Haan · Gerard J. O'Sullivan

Received: 5 September 2013 / Accepted: 2 November 2013 / Published online: 15 March 2014

Abstract Chronic venous insufficiency (CVI) as an advanced stage of chronic venous disease is a common problem that occurs in approximately 1–5 % of the adult population. CVI has either a nonthrombotic (primary) or postthrombotic (secondary) cause involving reflux, obstruction, or a combination of both. The role of venous obstruction is increasingly recognized as a major cause of CVI, with obstructive lesions in the iliocaval segment being markedly more relevant than lesions at the levels of the crural and femoral veins. Approximately 70–80 % of iliac veins develop a variable degree of obstruction following an episode of acute deep venous thrombosis. Nonthrombotic iliac vein obstruction also known as May-Thurner or Cockett's syndrome is the most common cause of nonthrombotic iliac vein occlusion. While compression therapy is the basis of therapy in CVI, in many cases, venous recanalization or correction of obstructive iliac vein lesions may result in resolution of symptoms. This document reviews the current evidence on iliocaval vein recanalization and provides standards of practice for iliocaval stenting in primary and secondary causes of chronic venous disease.

Keywords Venous stent · Deep vein thrombosis · Postthrombotic syndrome · Chronic venous disease · Chronic venous insufficiency

A. H. Mahnken (✉)
Department of Diagnostic and Interventional Radiology, University Hospital, Philipps-Marburg, Baldingerstraße, D-35043 Marburg, Germany
e-mail: mahnken@med.uni-marburg.de

K. Thomson
Department of Radiology, Alfred Hospital, University of Melbourne, 55 Commercial Road, Melbourne, VIC 3004, Australia
e-mail: k.thomson@alfred.org.au

M. de Haan
Department of Radiology, Maastricht University Medical Center, P. Debyelaan 25, 6229 HX Maastricht, The Netherlands
e-mail: dehaan-romijn@planet.nl

G. J. O'Sullivan
Section of Interventional Radiology, Department of Radiology, University College Hospital Galway, Newcastle Road, Galway, Ireland
e-mail: gerard.osullivan2@hse.ie

Introduction

Rationale of Iliocaval Stenting

Chronic venous disease (CVD) covers a wide range of symptoms from cosmetic problems to more severe symptoms, such as ulceration. Chronic venous insufficiency (CVI), defined as advanced CVD is a common problem which occurs in approximately 1–5 % of the adult population with healed or active ulcers being reported in approximately 1 % of the adult population [1–3]. The incidence of CVI increases with age [1]. CVD has either a nonthrombotic (primary or idiopathic) or postthrombotic (secondary) cause. Either type can involve reflux, obstruction, or a combination of both [4, 5]. The role of venous obstruction is increasingly recognized as a major cause of CVD, with obstructive lesions in the iliocaval segment being markedly more relevant than lesions at the levels of the crural and femoral veins [4–8]. Iliac vein obstruction is most commonly due to insufficient recanalization following an episode of acute deep venous thrombosis (DVT), with approximately 70–80 % of veins

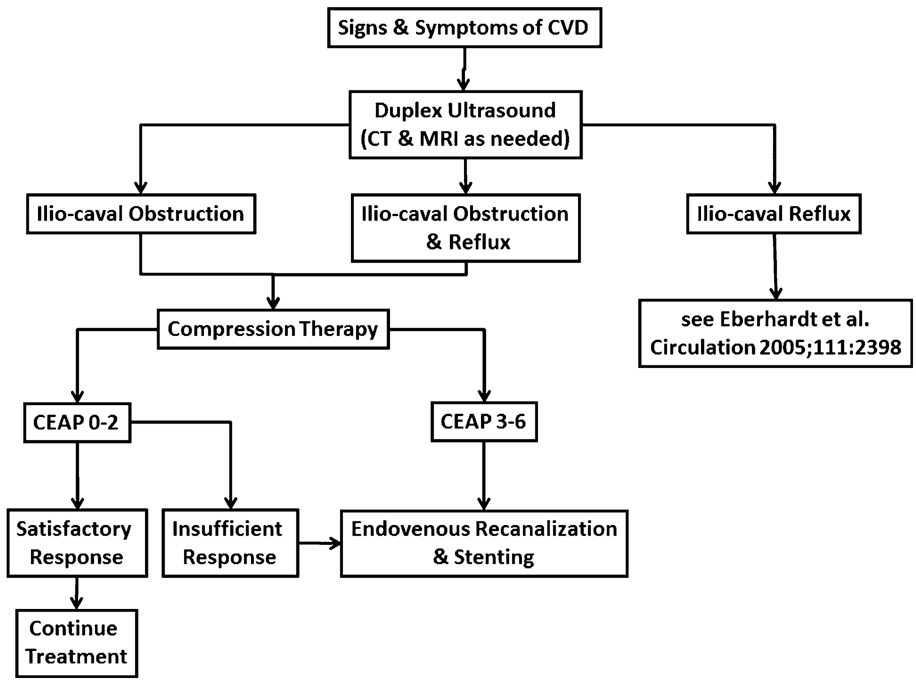

Fig. 1 Suggestion for a simplified treatment algorithm for the diagnosis and treatment of chronic venous obstruction. Different underlying pathophysiological mechanisms may necessitate a combination of therapies

developing a variable degree of obstruction [4, 9]. Non-thrombotic iliac vein obstruction occurs where it is crossed by the iliac or hypogastric artery a condition known as May-Thurner or Cockett's syndrome [5]. It typically involves the left common iliac vein, where it is crossed and compressed by the right iliac artery. Although these lesions often are occult, they often are the basis of symptoms in patients with postthrombotic disease. Such lesions are present in approximately 60 % of the asymptomatic general population [10, 11] but are found in more than 90 % of symptomatic patients [5]. While compression therapy is the basis of therapy in CVD, in many cases, venous recanalization or correction of obstructive iliac vein lesions may result in resolution of symptoms [6].

Benign iliofemoral venous occlusion may be difficult to treat. There are several surgical options, depending on the level and extent of disease. The femoro-femoral venous bypass, also known as Palma procedure is used for venous reconstruction in patients with unilateral iliac vein obstruction. Whenever possible autologous vein should be used, alternatively an 8- or 10-mm, externally supported expanded polytetrafluoroethylene (ePTFE) graft is considered an alternative. A temporary arteriovenous fistula is often placed to improve flow and to aid patency. While clinical improvement is often achieved, long-term patency is quite poor with a secondary patency rate of 83 % at 48 months for autologous vein and 0 % in PTFE conduits [12]. Orthotopic iliac or iliocaval reconstruction can be performed for either unilateral venous occlusion when autologous conduit is not available or bilateral iliocaval, or inferior vena cava occlusion (IVC). Experience with femoro- and iliocaval bypass grafts is limited, but 2-year primary and secondary patency rates of 37 and 54 % have been reported [13]. Considering these poor results, endovascular reconstruction has emerged as an alternative in treating chronic venous occlusions.

History of Iliocaval Stenting

While leg ulcers due to CVI are known from the time of Aesculapius himself [14], it took until the 19th century to make the connection between skin changes and a history of deep vein thrombosis. In 1866, John Gay pointed out the connection between skin changes with or without leg ulcers and postthrombotic disease [15]. For centuries, therapy has focused on compression, which still is the baseline therapy. Surgical techniques for bypassing venous obstructions were developed in the mid-20th century, with femorofemoral venous bypass as described by Palma and Esperon in 1960 being the longest used technique [16]. Considering the disappointing results of venous surgery other techniques were used and in 1985, the first use of a self-expanding stent was described in the venous system of a dog [17]. Following this early animal experience, initial studies in humans reported encouraging results [18, 19]. Subsequent studies indicated the value of endovascular therapy in treating CVI. A large study in 447 limbs treated by stenting between 1996 and 2002 showed the importance of venous obstruction for CVI [6]. The combination of reflux and obstruction was shown to be particularly problematic. Based on these results endovenous therapy became a mainstay in the treatment of CVI and PTS.

Table 1 CEAP classification

Classification	Description/definition
C, clinical (subdivided into A for asymptomatic, S for symptomatic)	
0	No venous disease
1	Telangiectases
2	Varicose veins
3	Edema
4	Lipodermatosclerosis or hyperpigmentation
5	Healed ulcer
6	Active ulcer
E, etiologic	
Congenital	Present since birth
Primary	Undetermined etiology
Secondary	Associated with postthrombotic, traumatic
A, anatomic distribution (alone or in combination)	
Superficial	Great and short saphenous veins
Deep	Cava, iliac, gonadal, femoral, profunda, popliteal, tibial, and muscular veins
Perforator	Thigh and leg perforating veins
P, pathophysiological	
Reflux	Axial and perforating veins
Obstruction	Acute and chronic
Combination of both	Valvular dysfunction and thrombus

Definitions

Assisted Primary Patency expresses cases in which a revision of the revascularization method is applied before vessel occlusion occurs. It refers to prophylactic interventions [20].

Complications (minor/major). Treatment-related adverse event requiring nominal therapy or no treatment with or without overnight hospitalization for observation = minor complication. Treatment-related adverse event requiring further therapy with increase in the level of care or prolonged hospitalization = major complication [21].

CEAP (Classification). Clinical classification for CVI to provide a basis for uniform reporting. It comprises four different classes (Clinic, Etiology, Anatomy, Pathophysiology). The clinical classification has seven categories (0 to 6) and is further categorized by the presence symptoms (Table 1) [22].

CVI (chronic venous insufficiency) describes advanced CVD, with functional abnormalities of the venous system resulting in edema, skin changes, and/or venous ulcers [23]. It covers different etiologies, including venous reflux and venous obstruction.

PTS (postthrombotic syndrome) Chronic venous signs and symptoms secondary to deep vein thrombosis. The combination of pain, edema, skin changes, and ulceration secondary to an episode of deep vein thrombosis is commonly referred to as PTS.

Primary Patency is defined as uninterrupted vessel patency with no procedure performed on the treated segment [12].

Secondary Patency is defined whenever maintenance of patency required a secondary intervention at the target lesion [12].

Technical success is defined as patency of the treated vessel segment with <20 % residual stenosis and without dissection or extravasation.

Venous clinical severity score (VCSS) is a complement to the CEAP classification providing a more detailed categorization of the clinical symptoms (Table 2) [24].

Pretreatment Imaging

Venous duplex imaging is the first line imaging method in the diagnostic workup of CVD. It provides information about the type (reflux with or without obstruction) and anatomic extent of disease. However, its use for assessing the iliac veins is limited and additional pelvic imaging studies, such as computed tomography (CT) or magnetic resonance (MR) imaging, should be obtained not only to assess the extent of disease in the iliocaval segment, but also to exclude extravascular disease causing obstruction, such as neoplasms or retroperitoneal fibrosis. The initial ultrasound workup also should include measurement of the ankle-brachial-index (ABI) to exclude the presence of obstructive arterial disease.

Currently ambulatory venous pressure (AVP) measurement is considered the hemodynamic standard of reference for assessing CVI. It involves insertion of a needle into a pedal or hand vein and connection to a pressure transducer. The pressure is determined at rest and after exercise such as toe raises. AVP measurements are often normal in patients with chronic venous obstruction, although a resting hand-foot pressure differential of ≥5 mm Hg is considered indicative of a significant obstruction [25]. An invasively obtained pressure gradient over a venous stenosis or pressure increase below an obstruction on exercise may indicate hemodynamic relevance of an obstruction. Unfortunately, these pressure differences often are as low as 2–3 mm Hg, which may be difficult to measure accurately [26]. Thus, a positive hemodynamic test may indicate hemodynamic relevance of a stenosis, whereas a normal test does not exclude it.

Considering these diagnostic limitations, the diagnosis of outflow obstruction largely depends on anatomic rather than hemodynamic criteria. In such a setting, ascending phlebography is still considered the reference method for assessing chronic venous obstruction before therapy

Table 2 Venous clinical severity score: calculated by adding the individual component scores

Variable	Score			
	0	1 (mild)	2 (moderate)	3 (severe)
Pain	None	Occasional; no use of analgesics	Daily; occasional use of nonnarcotic analgesics	Constant use of narcotic analgesics
Varicose Veins	None	Few, scattered	Multiple	Extensive
Edema	None	Limited	Afternoon, above ankle	Morning above ankle
Hyperpigmentation	None	Mild	Diffuse over lower third of leg	Wide distribution
Inflammation and cellulitis	None	Focal	Moderate	Severe
Induration	None		Less than lower third of leg	Entire lower third of leg or more
Active ulcers (no)	0	1	2	>2
Duration of active ulceration (mo)	None	<3	3–12	Not healed at >12
Diameter of active ulcer (cm)	None	<2	2–6	>6
Use of stockings	None	Occasional	Most days	Constant

although otherwise it has been mostly replaced by noninvasive imaging methods. Ascending phlebography involves the injection of contrast media distal to the suspected pathology (usually in the dorsum of the foot) with tourniquets to limit superficial vein filling and subsequent imaging of the contrast propagation through the deep venous system. For assessing chronic venous obstruction ascending phlebography should include oblique projections to properly visualize eccentric stenoses. The presence of collaterals is indicative of a relevant stenosis.

Iliocaval obstruction and associated abnormalities can also be detected by magnetic resonance (MR) venography and computed tomography (CT) venography, which may replace invasive studies in the future [27, 28]. Both, MR imaging and CT are suited, although not perfect, for diagnosing May-Thurner syndrome, with the left common iliac vein being compressed by the overlying right common iliac artery [29, 30]. Among all imaging tests intravascular ultrasound (IVUS) appears to be most accurate in estimating the morphological degree and extent of iliac vein stenosis and visualizing details of intraluminal lesions, such as intraluminal trabeculation in postthrombotic vessels [31].

Pretreatment imaging is also needed for treatment planning. For this purpose, a color Doppler ultrasound (CDUS) looking specifically at the right internal jugular vein, both popliteal and both common femoral veins is prudent in terms of planning the most efficient site to safely access the critical lesion. If the vein does not look completely normal, then it must be assumed to be scarred from prior DVT, and this access site should be avoided unless others are not available.

Indications for Treatment and Contraindications

Indications

Patients with CEAP clinical class 3–6 and chronic venous outflow obstructions should be considered for interventional therapy. In patients with CEAP clinical class 3 revascularization should be performed if compression therapy has failed. Endovascular revascularization therapy also may be considered in cases of successful compression therapy, as it treats the underlying cause of disease (Fig. 1). While previous treatment algorithms limited indication for revascularization to advanced clinical stages (CEAP clinical class 4-6) or failure of compression therapy [32], this is an extension of generally accepted treatment algorithms that appears to be justified by the low complication and good clinical success rates of endovascular therapy (see 9.1).

Stenting of the iliac veins also should be considered in the presence of nonthrombotic obstructive venous lesions in the iliocaval segment with a degree of stenosis of more than 30 % and the presence of venous collaterals. This type of lesions has long been considered as a common but clinically irrelevant finding. More recent data, however, indicated this morphologically low degree of stenosis to be an important factor in CVD. Accordingly, stenting in CVD provided good clinical results despite the presence of untreated reflux [5] and nonthrombotic iliac vein lesions may now be considered a contributing factor in CVD.

Iliocaval stenting should always be considered as an adjunct to interventional or surgical management of iliocaval thrombosis. With approximately 80 % of patients with iliofemoral thrombosis showing extrinsic iliac compression as seen on CT venography [27], a high rate of stenting after interventional therapy of iliocaval thrombosis is to be expected.

Contraindications

There are only few absolute contraindications for iliocaval stenting including uncorrectable coagulopathy and local or systemic infection.

Patient Preparation

A thorough clinical assessment is mandatory. Obtaining an in depth patient history looking for any hints of previous thrombosis is of particular importance. Clinical assessment includes CEAP and CVS scores. Inflammation at any potential puncture sites as well as systemic infection should be excluded. Preinterventional blood testing includes platelets, coagulation panels, and renal and thyroid function. The more common thrombophilic disorders (e.g., factor V Leiden) should be ruled out.

In cases of impaired renal function, patients should be treated according to the current European Society of Urogenital Radiology (ESUR) guidelines [33]. Patients with known allergic reactions to contrast material should be prepared according to international guidelines [34]. The platelet count should be >50.000/cm^3 and INR <1.5. However, patients with known venous obstructions are commonly anticoagulated with INR in the range of 2.5–3.5. While in cases of reduced platelet count, appropriate measures to correct the coagulation state should be undertaken, one may perform endovenous therapy despite an elevated INR. Because many of these patients are on warfarin, they should be switched to heparin for the procedure. Peripheral intravenous access must be obtained and the urinary bladder should be emptied before the procedure. If patients are unable to stay flat and immobile, general anaesthesia should be considered.

Equipment Specifications

In order to perform venous revascularisation procedures, a dedicated state-of-the-art digital subtraction angiography (DSA) C-arm unit with a large detector providing high-quality imaging and sufficient magnification is indispensable. A colour-Doppler ultrasound with different linear and/or curvilinear ultrasound probes (3–9 MHz) should be on hand for ultrasound-guided puncture of the access vessels.

Standard materials include:

1) 4–5 Fr catheters, typically with Multipurpose, Cobra or Sidewinder configuration.
2) 0.035″ standard guidewires. For treating chronic venous occlusions, an additional variety of hydrophilic and Teflon coated 0.035″ guidewires with differently shaped tips and varying degrees of stiffness need to be on hand.
3) 5- to 12-Fr sheaths with lengths ranging from 10 to 45 cm.
4) Standard over-the-wire balloon catheters with diameters ranging from 6–24 mm.
5) High-pressure balloon catheters with diameters of 12–18 mm.
6) Snares with loop diameters ranging from 10 to 25 cm.
7) A variety of large diameter self-expanding stents (12–28 mm) with a very high radial force. Dedicated venous stents up to 18 mm diameter are available for the iliofemoral segment. In case of caval obstructions larger stents are needed.

In selected cases additional materials may be required:

1) In case of caval reconstruction with stent-grafts up to 18-Fr sheaths may be needed.
2) Availability of either a reentry device or a Roesch-Uchida needle may be needed in chronic occlusions.
3) Stent grafts with diameters from 14 to 28 mm (usually aortic stent-grafts of iliac extensions) may be used.
4) Large balloon-expandable stents should be on hand to provide an extra radial power if needed (e.g., in restenosis due to external compression).

Procedural Features and Variations of the Technique

– Preparation of the patient according to the “CIRSE IR Patient Safety Checklist” [35].
– Sterile skin preparation of both groins is recommended. The site and direction of the venous access (ipsilateral retrograde femoral or popliteal or contralateral retrograde femoral) depends on the preference of the interventionalist. The ipsilateral femoral approach should be preferred for starting the procedure as it is easy to access and provides a good pushability of catheters, sheaths, and stents; however, if there has been prior DVT, this vein may be scarred and consideration should be given to popliteal venous access. One should be ready to include an additional right jugular venous access if needed.
– The use of ultrasound guidance for venous puncture is recommended and has largely eliminated access site complications.

- The use of arterial angiographic techniques, particularly the use of DSA and power injection of contrast material is useful to improve the image quality of diagnostic phlebography. It is mandatory to identify venous anomalies such as inferior vena cava discontinuity [36], because these variations may not allow for interventional therapy or require additional therapeutic measures to avoid early rethrombosis, such as stenting of collateral vessels or additional shunt surgery.
- For crossing a chronic occlusion, initially standard hydrophilic wires and catheters are used, if necessary followed by dedicated chronic total occlusion (CTO) wires; 0.018″ diameter in a variety of tip weights (4–30 g). Finally, the use of sharp recanalization techniques may be required. These advanced techniques may include the off-label use of medical devices and should be limited to experienced centers.
- When crossing long lesions, repeated injection of contrast material and image acquisition in different oblique projections is recommended to make sure that intraluminal recanalization is performed. In case of partially extraanatomic recanalization, the use of stent-grafts should be considered.
- When a catheter will not follow a hydrophilic guidewire, snaring and tightening the wire from the opposite direction (e.g., via jugular or contralateral approach) usually helps to provide sufficient support to allow replacement of the hydrophilic guidewire by an extra-stiff wire for stenting.
- Predilatation is essential in chronic occlusions, particularly with modern purpose designed laser-cut self-expanding stents. Some interventionalists perform predilatation up to the nominal diameter of the subsequently used stent [37, 38]. Predilatation may be painful; this is normal and does not indicate impending rupture as it does in arterial interventions.
- It is important that the proximal and distal end of the stents lie in a normal/healthy venous segment. As the true lesion often exceeds the extent as seen on phlebography, the stent should cover an area longer than the lesion as seen on phlebography. The use of IVUS is helpful to visualize the true extent of a venous lesion. If IVUS is not available the stent should exceed the lesion as seen on phlebography by at least 5 mm in both directions. When multiple stents are needed, they should be implanted in an overlapping manner with a minimum overlap of 1.5 cm. Short skip areas in between two stents must be avoided.
- Stenting of a lesion adjacent to the confluence of the common iliac veins, such as in May-Thurner syndrome, requires the stent to be placed well, but not too far (generally $\sim$1 cm) into the IVC to avoid early restenosis. This technique does not usually limit the venous flow from the contralateral limb.
- In bilateral iliac or iliocaval obstruction with bilateral stenting, either the kissing stent (or double barrel) technique or the inverted Y-technique, may be used. Available data comparing the kissing stent and inverted Y-stenting suggest that the kissing stent technique the better alternative in terms in primary and secondary patency [39].
- Stenting across the inguinal ligament should be avoided. Although there is no data on venous stent fractures in this vessel segment, stenting across the inguinal ligament is a risk factor for early in-stent restenosis [40]. However, stenting down to a normal flow segment is more important than avoiding crossing the inguinal ligament. If stenting of this vessel segment cannot be avoided, the use of dedicated segmented venous stents is suggested.
- In general, large self-expanding stents should be used (iliac vein: 14–16 mm; IVC: 18–24 mm) and overdilatation of the vein seems to be no problem. Even in chronic occlusions the veins accept large stents without rupture. Dilatation of the stent almost up to the nominal diameter is recommended to achieve a good lumen. An optimal wall apposition is thought to be beneficial as it permits quick endothelialisation of the metal struts.
- If the venous inflow is poor, particularly if the deep femoral vein is affected by the venous obstruction, an additional arteriovenous fistula below the stent, typically between the common femoral artery and vein and/or endophlebectomy is recommended to improve flow and to avoid early rethrombosis due to insufficient flow.

Medication and Periprocedural Care

During the procedure, routine monitoring of vital function is mandatory. ECG-monitoring, pulse oximetry, and repeated blood pressure measurements are strongly recommended.

Although not mandatory, some premedication is recommended to enhance tolerance to treatment. A low-dose benzodiazepine (e.g., 1–3 mg of midazolam) may be applied as an anxiolytic agent directly before the treatment. Dilatation of chronic venous obstructions often is painful, and slow infusion of potent i.v. analgesia, such as pethidine or piritramide, is recommended. While these substances are generally well tolerated, one has to be aware of its typical adverse reactions, which typically are those of opioids, including nausea, vomiting, sedation, dizziness, and diaphoresis. Overdosage can cause muscle flaccidity, respiratory depression, cold and clammy skin, hypotension, and coma. Antiemetic drugs, such as dimenhydrinate, should be generously used to manage the typical side effects of opioids. General anesthesia should be considered if multiple or repeated dilatations are needed.

Postprocedural Follow-up Care

A thorough posttreatment assessment is mandatory including assessment of the CEAP classification, the CVSS, and Doppler ultrasound. Careful imaging and clinical surveillance is mandatory at regular intervals to identify recurrent stenosis and to guide the need for reintervention [41]. Surveillance intervals may include regular visits at 1, 3, 6, and 12 months and every 12 months thereafter.

The use of compression stockings should be continued, with the degree of compression depending on the CEAP score [32]. Continuous anticoagulation with warfarin aiming at a target International Normalized Ratio (INR) range of 2.5–3 is strongly recommended, although there is no evidence from controlled studies on this issue. Platelet aggregation is known to be important in high-flow, high-shear environment, such as in the coronary arteries, whereas coagulation may be more important in the fibrin-rich thrombi characteristic of the low-flow, low-shear venous circulation [42]. The relative importance of antiplatelet agents versus anticoagulants has never been evaluated in clinical trials and is largely based on extrapolation from the arterial system and an understanding of the venous system. Based on clinical data on stenting of chronic iliocaval occlusions, long-term warfarin is recommended in patients with long occlusions, underlying thrombophilia, suprarenal occlusions, and previous long-term anticoagulation and poor inflow on completion angiogram [43, 44]. With postthrombotic lesions being more prone to restenosis, the use of anticoagulants appears to be useful in this subgroup. Thus, although the use of antiplatelet agents and anticoagulants has not been studied systematically, there seems to be a role for these drugs.

Outcome

Effectiveness

Technical success rates of venous stenting are very high with approximately 84–93 % in chronic postthrombotic iliac vein obstruction [29, 45]. Technical success rates decrease to 66 % in postthrombotic lesions with total occlusion of the IVC [46]. In case of acute thrombosis with or without external compression, either due to May–Thurner syndrome or malignancy, technical success rates are usually up to 100 % even when the IVC is involved [46–48].

Long-term results are very encouraging. Two major series from different groups on stenting in postthrombotic iliac vein obstruction report primary, assisted primary and secondary patency rates of 57, 80, and 86 % at 6 years [49] and 83, 89, and 93 % at 10 years, respectively [50]. A detailed analysis of the outcome of stenting in postthrombotic iliocaval obstruction can be found in a recently published review [51]. In patients with primary iliac vein obstruction, long-term results are even better with primary, assisted primary, and secondary patency rates of 79, 100, and 100 % at 6 years [49]. Titus et al. also stress the significance of underlying disease for long-term results, as they report a significantly better 2-year outcome in patients with May-Thurner syndrome when compared with external compression or thrombophilia as underlying cause of disease [52].

Technical success is mirrored by clinical improvement with marked improvement in VCSS and ulcer healing. In a series by Hartung et al., all but 2 of 47 (96 %) ulcers healed after venous stenting [53]. Neglén et al. reported a long-term ulcer healing rate of 55 % [49]. These clinical results were proven by several recent studies, which reported significant decrease in the VCSS after stenting of iliocaval obstructions [54] or reduced swelling and pain levels after endovenous treatment [47, 55]. Clinical improvement appears to be long lasting, although long-term results after treating acute DVT seem to be better than stenting in chronic venous obstruction [56].

Complications and Their Management

The mortality rate after endovenous revascularization is low with mortality rates ranging from 0–1 % [49, 57]. The most common complications are early (<30 days) and late rethrombosis with 1.5–3 % for early and roughly 5 % for late events [49, 58]. Extension of the stent into the IVC is associated with contralateral iliac vein occlusion in approximately 1 % of patients [49]. However, failure to extend the stent in the IVC and thereby completely covering the underlying lesion in May-Thurner syndrome results in an ipsilateral restenosis or occlusion rate of 36 % [59].

Rare complications include venous tears, injury of an adjacent artery, stent fracture, and stent dislocation either due to undersizing or due to the guidewire being caught in the stent. All of these complications were reported to only occur sporadically. However, from personal experience stent fractures may be underreported in the literature. In case of a nonultrasound guided puncture technique access site complications, such as hematoma, arteriovenous fistula, or arterial injury, were reported to occur in up to 7 % of patients [49], whereas access site complications are almost zero with ultrasound-guided puncture.

Conclusions

Stenting in chronic iliocaval obstruction is safe and effective. It provides excellent long-term results with respect to target vessel revascularization as well as symptom relief, therefore improving the quality of life. In selected patients, it appears to even reverse established PTS. During the past decade, venous outflow obstruction has become recognized as more relevant in CVD than previously anticipated and endovascular correction of outflow obstructions should be liberally indicated.

Conflict of interest Dr. O'Sullivan received consultant fees from Cook Medical, Covidien, and Bayer Medrad. He is a speaker for a variety of companies involved in the venous area, including Cook Medical, Covidien, and Bayer Medrad and Optimed. None of the other authors have a conflict of interest or a financial disclosure to declare.

References

1. Callam MJ (1994) Epidemiology of varicose veins. Br J Surg 81:167–173
2. Rabe E, Pannier-Fischer F, Bromen K, Schuldt K, Stang A, Poncar C, Wittenhorst M, Bock E, Weber S, Jöckel KH (2003) Bonn Vein Study by the German Society of Phlebology: epidemiological study to investigate the prevalence and severity of chronic venous disorders in the urban and rural residential populations [German]. Phlebologie 32:1–14
3. Jawien D, Gorzela T, Ochwat D (2003) Prevalence of chronic venous insufficiency in men and women in Poland: multicenter cross-sectional study in 40,085 patients. Phlebology 18:110–121
4. Johnson BF, Manzo RA, Bergelin RO, Strandness DE Jr (1995) 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 21:307–312
5. Raju S, Neglén P (2006) High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogenicity. J Vasc Surg 44:136–143
6. Neglén P, Thrasher TL, Raju S (2003) Venous outflow obstruction: an underestimated contributor to chronic venous disease. J Vasc Surg 38:879–885
7. Labropoulos N, Volteas N, Leon M, Sowade O, Rulo A, Giannoukas AD, Nicolaides AN (1997) The role of venous outflow obstruction in patients with chronic venous dysfunction. Arch Surg 132:46–51
8. Raju S, Hollis K, Neglén P (2006) Obstructive lesions of the inferior vena cava: clinical features and endovenous treatment. J Vasc Surg 44:820–827
9. Akesson H, Brudin L, Dahlstrom JA, Eklof B, Ohlin P, Plate G (1990) Venous function assessed during a 5-year period after acute iliofemoral venous thrombosis treated with anticoagulation. Eur J Vasc Surg 4:43–48
10. Cockett FB, Thomas ML, Negus D (1967) Iliac vein compression—its relation to iliofemoral thrombosis and the post-thrombotic syndrome. Br Med J 2:14–19
11. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao J, Matsumura J (2004) Iliac vein compression in an asymptomatic patient population. J Vasc Surg 39:937–943
12. Jost CJ, Gloviczki P, Cherry KJ Jr, McKusick MA, Harmsen WS, Jenkins GD, Bower TC (2001) Surgical reconstruction of iliofemoral veins and the inferior vena cava for nonmalignant occlusive disease. J Vasc Surg 33:320–328
13. Gloviczki P, Cho JS (2005) Surgical treatment of chronic occlusions of the iliocaval veins. In: Rutherford RB (ed) Rutherford's vascular surgery, 6th edn. Elsevier, Philadelphia, pp 2303–2320
14. Stanton JA (1999) Aesculapius: a modern tale. JAMA 281: 476–477
15. Bergan JJ (1991) Historical highlights in treating venous insufficiency. In: Bergan JJ, Yao JST (eds) Venous disorders. W.B. Saunders, Philadelphia, pp 3–18
16. Palma EC, Esperon R (1960) Vein transplants and grafts in the surgical treatment of the postphlebitic syndrome. J Cardiovasc Surg 1:94–107
17. Wright KC, Wallace S, Charnsangavej C, Carrasco CH, Gianturco C (1985) Percutaneous endovascular stents: an experimental evaluation. Radiology 156:69–72
18. Zollikofer CL, Largiader I, Bruhlmann WF, Uhlschmid GK, Marty AH (1988) Endovascular stenting of veins and grafts: Preliminary clinical experience. Radiology 167:707–712
19. Antonucci F, Salomonowitz E, Stuckmann G, Stiefel M, Largiade'r J, Zollikofer CL (1992) Placement of venous stents: clinical experience with a self-expanding prosthesis. Radiology 183: 493–497
20. Dormandy JA, Rutherford RB (2000) Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Concensus (TASC). J Vasc Surg 31:S1–S296
21. Sacks D, McClenny TE, Cardella JF, Lewis CA (2003) Society of Interventional Radiology clinical practice guidelines. J Vasc Interv Radiol 14:S199–S202
22. Porter JM, Moneta GL (1995) Reporting standards in venous disease: an update. International Consensus Committee on Chronic Venous Disease. J Vasc Surg 21:635–645
23. Eklof B, Perrin M, Delis KT, Rutherford RB, Gloviczki P (2009) American Venous Forum; European Venous Forum; International Union of Phlebology; American College of Phlebology; International Union of Angiology. Updated terminology of chronic venous disorders: the VEIN-TERM transatlantic interdisciplinary consensus document. J Vasc Surg 49:498–501
24. Rutherford RB, Padberg FT, Comerota AJ, Kistner RL, Meissner MH, Moneta GL (2000) Venous severity scoring: an adjunct to venous outcome assessment. J Vasc Surg 31:1307–1312
25. Neglén P, Raju S (1993) Detection of outflow obstruction in chronic venous insufficiency. J Vasc Surg 17:583–589
26. Albrechtsson U, Einarsson E, Eklöf B (1981) Femoral vein pressure measurements for evaluation of venous function in patients with postthrombotic iliac veins. Cardiovasc Intervent Radiol 4:43–50
27. Chung JW, Yoon CJ, Jung SI, Kim HC, Lee W, Kim YI, Yae HJ, Park JH (2004) Acute iliofemoral deep vein thrombosis: evaluation of underlying anatomic abnormalities by spiral CT venography. J Vasc Interv Radiol 15:249–256
28. Fraser DG, Moody AR, Martel A, Morgan PS (2004) Re-evaluation of iliac compression syndrome using magnetic resonance imaging in patients with acute deep venous thromboses. J Vasc Surg 40:604–611
29. Oguzkurt L, Tercan F, Pourbagher MA, Kizilkilic O, Turkoz R, Boyvat F (2005) Computed tomography findings in 10 cases of iliac vein compression (May-Thurner) syndrome. Eur J Radiol 55:421–425
30. McDermott S, Oliveira G, Ergül E, Brazeau N, Wicky S, Oklu R (2013) May-Thurner syndrome: can it be diagnosed by a single MR venography study? Diagn Interv Radiol 19:44–48

31. Neglen P, Raju S (2002) Intravascular ultrasound scan evaluation of the obstructed vein. J Vasc Surg 35:694–700
32. Eberhardt RT, Raffetto JD (2005) Chronic venous insufficiency. Circulation 111:2398–2409
33. Stacul F, van der Molen AJ, Reimer P, Webb JA, Thomsen HS, Morcos SK, Almén T, Aspelin P, Bellin MF, Clement O, Heinz-Peer G (2011) Contrast Media Safety Committee of European Society of Urogenital Radiology (ESUR). Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. Eur Radiol 21:2527–2541
34. Bush WH Jr (1999) Treatment of acute contrast reactions. In: Bush WH Jr, Krecke KN, King BF Jr, Bettmann MA (eds) Radiology Life Support (RAD-LS). CRC Press, Boca Raton, pp 31–51
35. Lee MJ, Fanelli F, Haage P, Hausegger K, Van Lienden KP (2012) Patient safety in interventional radiology: a CIRSE IR checklist. Cardiovasc Intervent Radiol 35:244–246
36. Bass JE, Redwine MD, Kramer LA, Huynh PT, Harris JH Jr (2000) Spectrum of congenital anomalies of the inferior vena cava: cross-sectional imaging findings. Radiographics 20: 639–652
37. Raju S, McAllister S, Neglén P (2002) Recanalization of totally occluded iliac and adjacent venous segments. J Vasc Surg 36:903–911
38. Raju S, Oglesbee M, Neglén P (2011) Iliac vein stenting in postmenopausal leg swelling. J Vasc Surg 53:123–130
39. Neglén P, Darcey R, Olivier J, Raju S (2010) Bilateral stenting at the iliocaval confluence. J Vasc Surg 51:1457–1466
40. Knipp BS, Ferguson E, Williams DM, Dasika NJ, Cwikiel W, Henke PK, Wakefield TW (2007) Factors associated with outcome after interventional treatment of symptomatic iliac vein compression syndrome. J Vasc Surg 46:743–749
41. Raju S, Tackett P Jr, Neglén P (2009) Reinterventions for nonocclusive iliofemoral venous stent malfunctions. J Vasc Surg 49:511–518
42. Meissner MH (2013) Indications for platelet aggregation inhibitors after venous stents. Phlebology 28(Suppl 1):91–98
43. Kurklinsky AK, Bjarnason H, Friese JL, Wysokinski WE, McBane RD, Misselt A, Moller SM, Gloviczki P (2012) Outcomes of venoplasty with stent placement for chronic thrombosis of the iliac and femoral veins: single-center experience. J Vasc Interv Radiol 23:1009–1015
44. Raju S, Neglén P (2009) Percutaneous recanalization of total occlusions of the iliac vein. J Vasc Surg 50:360–368
45. Wahlgren CM, Wahlberg E, Olofsson P (2010) Endovascular treatment in postthrombotic syndrome. Vasc Endovascular Surg 44:356–360
46. Raju S, Hollis K, Neglén P (2006) Obstructive lesions of the inferior vena cava: clinical features and endovenous treatment. J Vasc Surg 44:820–827
47. Ye K, Lu X, Li W, Huang Y, Huang X, Lu M, Jiang M (2012) Long-term outcomes of stent placement for symptomatic nonthrombotic iliac vein compression lesions in chronic venous disease. J Vasc Interv Radiol 23:497–502
48. te Riele WW, Overtoom TT, van den Berg JC, van de Pavoordt ED, de Vries JP (2006) Endovascular recanalization of chronic long-segment occlusions of the inferior vena cava: midterm results. J Endovasc Ther 13:249–253
49. Neglén P, Hollis KC, Olivier J, Raju S (2007) Stenting of the venous outflow in chronic venous disease: long-term stent related outcome, clinical, and hemodynamic result. J Vasc Surg 46: 979–990
50. Hartung O, Loundou AD, Barthelemy P, Arnoux D, Boufi M, Alimi YS (2009) Endovascular management of chronic disabling ilio-caval obstructive lesions: long-term results. Eur J Vasc Endovasc Surg 38:118–124
51. Hartung O (2011) Results of stenting for postthrombotic venous obstructive lesions. Perspect Vasc Surg Endovasc Ther 23:255–260
52. Titus JM, Moise MA, Bena J, Lyden SP, Clair DG (2011) Iliofemoral stenting for venous occlusive disease. J Vasc Surg 53:706–712
53. Hartung O, Lugli M, Nicolini P, Boufi M, Maleti O, Alimi YS (2010) Stenting for iliac veins post-thrombotic obstructive lesions: results of a multicentric retrospective study. J Vasc Surg 51:790
54. Hölper P, Kotelis D, Attigah N, Hyhlik-Dürr A, Böckler D (2010) Long-term results after surgical thrombectomy and simultaneous stenting for symptomatic iliofemoral venous thrombosis. Eur J Vasc Endovasc Surg 39:349–355
55. Nayak L, Hildebolt CF, Vedantham S (2012) Postthrombotic syndrome: feasibility of a strategy of imaging-guided endovascular intervention. J Vasc Interv Radiol 23:1165–1173
56. Alhadad A, Kölbel T, Herbst A, Holst J, Alhadad H, Gottsäter A (2011) Iliocaval vein stenting: Long term survey of postthrombotic symptoms and working capacity. J Thromb Thrombolysis 31:211–216
57. Broholm R, Panduro Jensen L, Baekgaard N (2010) Catheter-directed thrombolysis in the treatment of iliofemoral venous thrombosis. A review. Int Angiol 29:292–302
58. Hartung O, Otero A, Boufi M, Decaridi G, Barthelemy P, Juhan C, Alimi YS (2005) Mid-term results of endovascular treatment for symptomatic chronic nonmalignant iliocaval venous occlusive disease. J Vasc Surg 42:1138–1144
59. Neglén P, Raju S (2000) Balloon dilation and stenting of chronic iliac vein obstruction: technical aspects and early clinical outcome. J Endovasc Ther 7:79–91