Chronic Venous Insufficiency

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11.1 Introduction and Epidemiology

The prevalence of leg edema (Fig. 11.1) as a manifestation of chronic venous insufficiency has been reported between 7.4 and 17.1% in men and 4.9–20.3% in women. Furthermore, the prevalence of CVI has been estimated from between <1 and 17% in men and between <1 and 40% in women [1]. Some of the variance in these values is due to differing criteria for what constitutes chronic venous insufficiency and sampling bias in the epidemiological studies. However, it is also known that different populations with exposure to different genetic and environmental factors show significant differences in the incidence of chronic venous insufficiency [2]. Regardless of the exact percentages, venous insufficiency is a common cause of both unilateral and bilateral leg edema. Its peak incidence is in the 5th decade of life, and it is approximately twice as common in females compared to males.

11.2 Anatomy and Physiology

Approximately 90% of blood from the lower limb is drained directly by deep veins, which follow the arteries supplying the lower limb. 10% is drained by superficial veins, which run above the deep fascia for most of their course [3] The physiological ejection fraction of the calf muscle pump is 65% and 15% for the thigh muscle pump [3–5]. When calf muscle dysfunction occurs, it impacts on the valve function too. One must keep in mind that treating valve function without addressing calf pump may lead to treatment failure.

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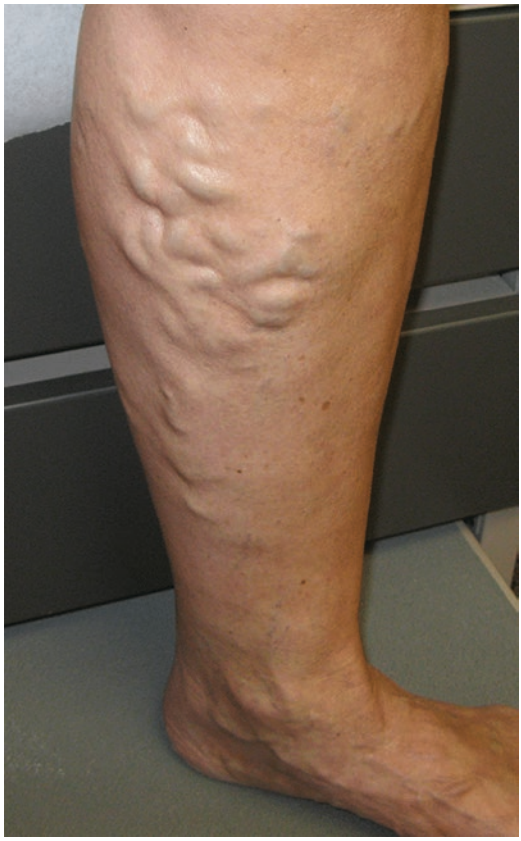

Fig. 11.1 CEAP C3 varicose veins with edema

The superficial venous system is responsible for temperature regulation, as a reservoir of blood and to deliver blood to the deep venous system. Medially, the great saphenous vein (GSV) originates from the dorsal venous arch at the ankle and runs to the pelvis, emptying into the femoral vein at the sapheno-femoral junction (SFJ). Its notable tributaries include the anterior accessory saphenous vein and the lateral accessory saphenous vein [3, 6]. The small saphenous vein (SSV) arises just underneath the lateral malleolus and travels up the leg posteriorly to join the popliteal vein at the sapheno-popliteal junction (SPJ). There are a multitude of perforators through which blood flows from superficial to deep vessels in physiological conditions [3] (Fig. 11.2a and b).

The common pathway of drainage of all the blood of the lower limb is through the femoral vein, up the external followed by the common iliac, into the inferior vena cava. In the case of IVC obstruction blood can drain through the ascending lumbar veins to the azygous and hemiazygos veins, as well as up the epigastric veins to the superior vena cava [7, 8].

Obstruction of the main pathways of venous return and the use of these small diameter collaterals results in a higher pressure distal to the pathology. More blood

is pushed into the superficial venous system, which is more capable of distention as it is not constricted within muscle and dense fascia. However, in a system that only handles 10% of the lower limb blood flow, the introduction of more blood volume can lead to the maladaptive changes characteristic of chronic venous insufficiency.

11.3 Etiopathophysiology of Lower Leg Edema in CVI

The mechanism behind chronic venous insufficiency (CVI) is fundamentally an inflammatory state induced by the stress that venous hypertension places on cells of the vessel wall. As with injury in regions elsewhere in the body, vasoactive substances are released from the endothelium, adhesion molecules, chemokines, matrix metalloproteinases, and inflammatory mediators are expressed to create a local inflammatory response. Adhesion factors such as ICAM-1 have been associated

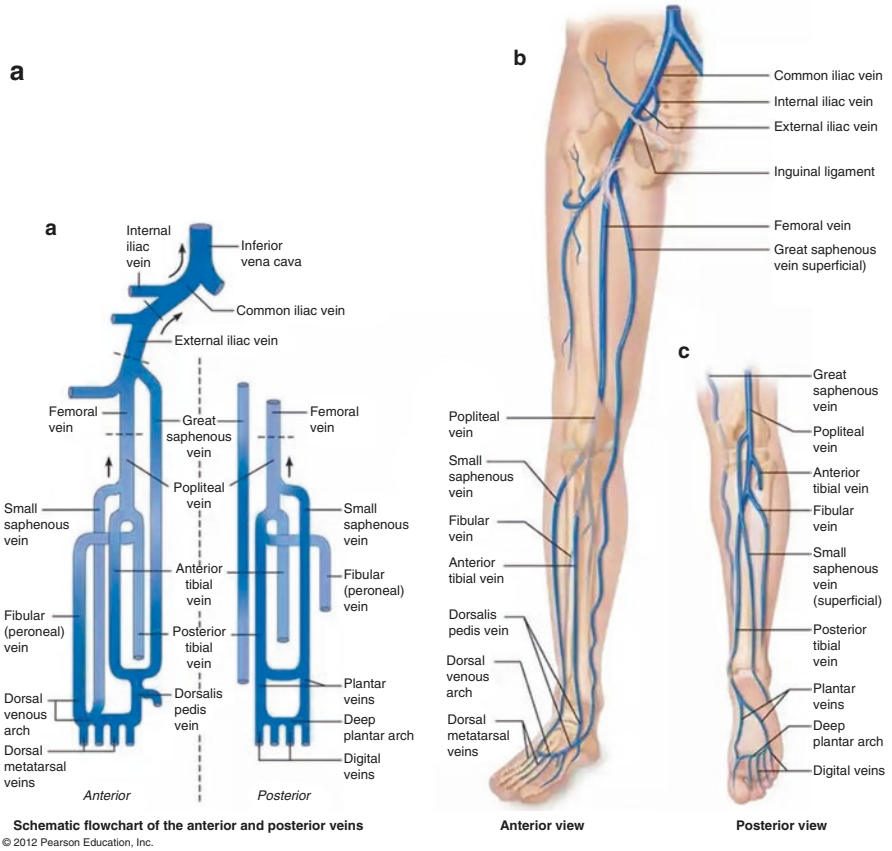

Fig. 11.2 (**a**) Anatomy of venous system of lower limbs; (**b**) Varicose veins and vein valve function

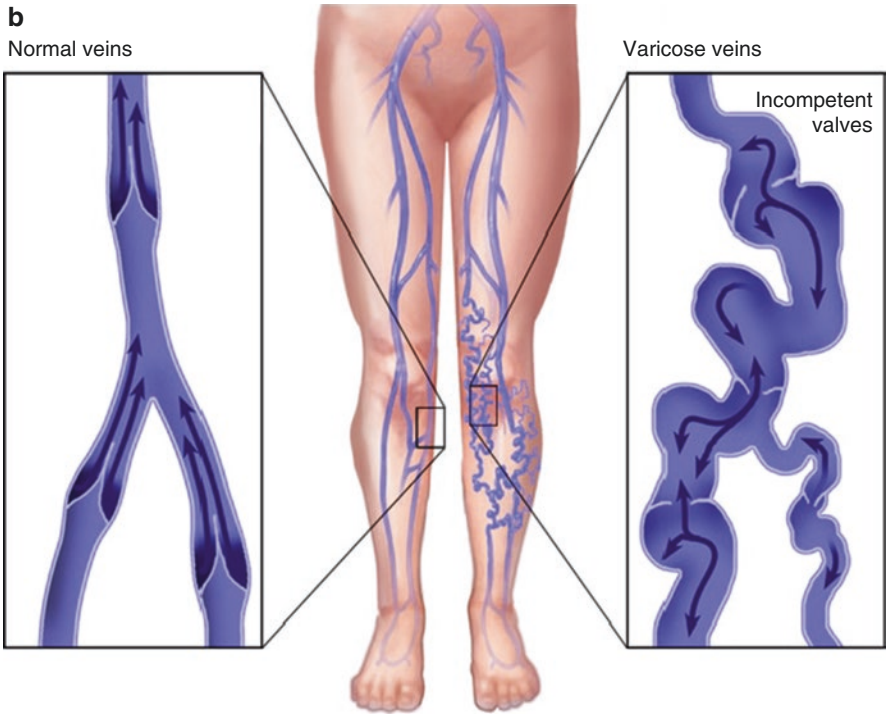

Fig. 11.2 (continued)

with the invasion of venous vales and vessel walls by monocytes and macrophages [9]. The overall result is disruption and disorganization of vessel walls with fibrosis, all of which leads to decreased vein compliance and further damage [5, 9]. Furthermore, stretching of the vessel wall may lead to a vicious cycle of further valvular incompetence causing more hypertension, dilation, and fluid leakage setting up a negative cycle in which the condition chronically progresses (Fig. 11.3).

Following from the pathophysiology, anything that increases lower limb venous pressure may contribute to the development of chronic venous insufficiency. These may include prolonged standing, musculo-venous pump failure, thoraco-abdominal pump failure, heart failure, decreased sympathetic tone, and obesity (increased intra-abdominal pressure) [2, 10].

An important cause of increased capillary hydrostatic pressure is estrogen. Estrogen inhibits vascular smooth muscle tone and proliferation, resulting in a greater diameter of vessels. The result is more fluid and hence more capillary hydrostatic pressure. This is why chronic venous insufficiency has a much greater prevalence in the female population [11]. During pregnancy, levels of estrogen are further elevated, and the developing fetus obstructs the inferior vena cava resulting in an increased risk of developing symptoms of chronic venous insufficiency.

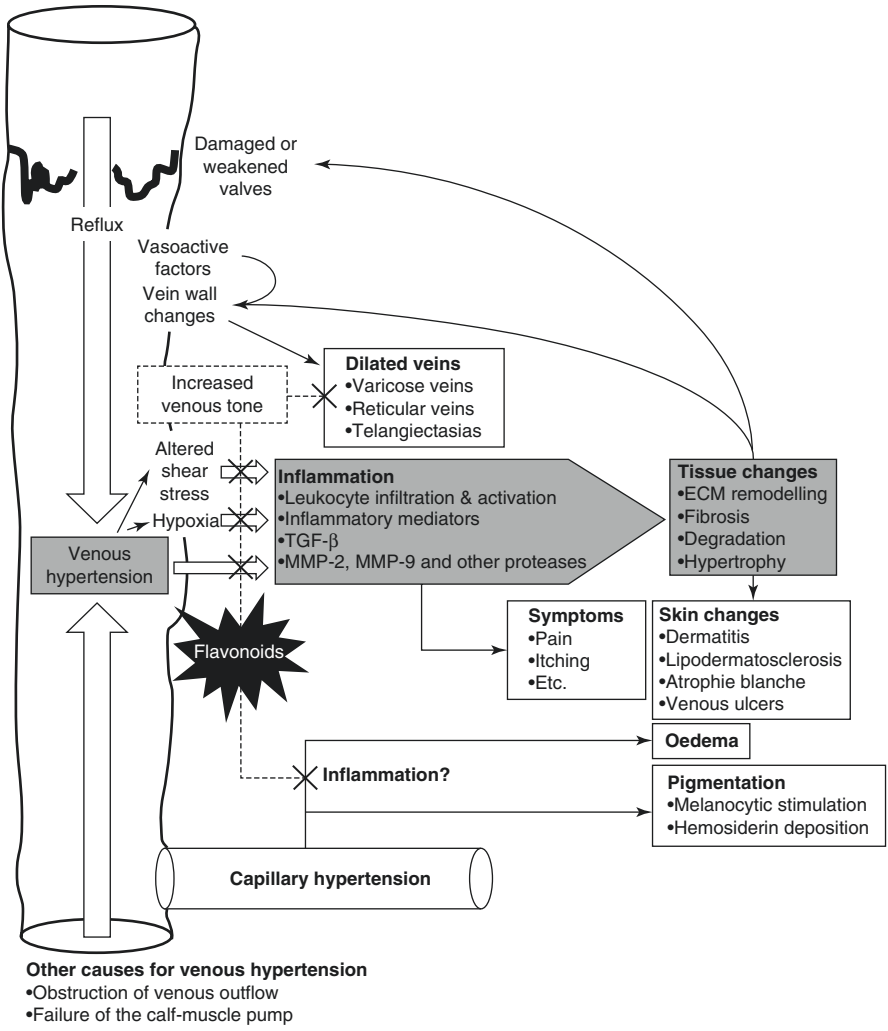

Fig. 11.3 Pathophysiological mechanisms in venous hypertension

While these underlying conditions may certainly contribute to chronic venous insufficiency, the most common cause of venous hypertension is deep venous obstruction (DVO). The etiology of DVO is broadly classified into deep vein thrombosis or scarring after DVT, or non-thrombotic iliac vein lesions (NIVL) [2].

A common cause of NIVL is May-Thurner syndrome. Also known as Cockett syndrome or Iliac vein compression syndrome, it occurs when the right common iliac artery compresses the left common iliac vein against the sacral promontory. This can lead to endothelial irritation and the formation of intraluminal "spurs" or

"bands" which further affect blood flow [12]. It has been implicated in the etiology of leg edema and refractory leg ulcers and has been associated with varicose veins of the pelvic organs [12, 13]. It is present in 22–32% of the general asymptomatic population and 18–49% of patients with left lower limb DVT [14].

Other risk factors that make veins more vulnerable or weak to changes in venous pressure can also contribute to chronic venous insufficiency: advancing age, smoking, previous lower extremity trauma, and genetic disorders resulting in abnormal vein or connective tissue characteristics like Ehlers-Danlos type IV syndrome, Klippel-Trenaunay syndrome, hyperhomocysteinemia, and FOXC2 mutations (venous valve failure ➔ varicose veins) [15]. Additionally, a number of multifactorial genetic or epigenetic changes may predispose one to CVI through deformed, shrunken, or generally abnormal valves in the venous system of their lower limb. Such a family history of chronic venous insufficiency is a risk factor for the development of CVI in an individual. Marfan's syndrome only affects the arterial system and as such is not implicated in the development of chronic venous insufficiency.

Due to this, the approach to chronic venous insufficiency is largely symptom based, most commonly classified using CEAP scoring, which qualitatively grades the level of venous disease based on external symptoms, etiology, anatomic distribution, and pathophysiology.

Patients presenting with edema are by definition C3 patients.

11.4 Diagnosis of CVI in Patients Presenting with Lower Leg Edema

The general symptoms of chronic venous insufficiency include leg pain or cramps, fatigue, pruritis, and heaviness. When present, edema has a tendency to occur in the evening and decrease with walking or elevation above the level of the patient's heart (e.g., when supine and resting their leg on a pillow). Its likelihood may be further assessed with questioning directed at the risk factors related to the pathophysiology of CVI discussed previously. Risk factors for the development of venous insufficiency include pregnancy, smoking, obesity, trauma, DVT, superficial thrombophlebitis, and inactivity. Finally, chronic venous insufficiency is positively correlated with age, as are a multitude of other medical conditions that may present with lower limb edema [16]. Therefore, it is important to consider the individual holistically and thoroughly assess for comorbid conditions that may contribute to lower limb edema.

The signs of chronic venous insufficiency in the lower limbs manifest as a spectrum, from telangiectasias and spider veins, to edema and varicose veins, lipodermatosclerosis, and finally venous ulcers. Edema is a constant presentation of C3-6 venous insufficiency.

Edema due to chronic venous insufficiency presents as a pitting edema. If the disease has been present for a long time however, the edema may be non-pitting or brawny as lymphatic obstructive elements get involved with subcutaneous

inflammatory changes. It can be elicited by palpating the skin over distal shaft of the tibia and over medial malleolus of the tibia and compressing the area for 15 s with the thumb. Tenderness may be present, so it is important to take patient comfort into consideration. Edema due to hypoalbuminemia, which also presents with pitting edema, generally refills more quickly than edema due to CVI. It is important to note the presence and characteristics of any ulcers, varicosities, or skin changes present, as these give important indicators of the severity and possible etiology of the disease. If chronic venous insufficiency is the cause of lower limb edema, it is almost certain that varicosities (a less severe manifestation of the disease) will be present.

Clinically, there are three special tests to assess the competence of a patient's lower venous system: the sapheno-femoral and sapheno-popliteal junctional cough impulse test, the Trendelenburg (or Brodie-Trendelenburg) test, and Perthes' test. The cough impulse test can uncover an incompetent sapheno-femoral junction if a fluid thrill is felt in the proximal great saphenous vein (medial to the femoral vein) after a patient is prompted to cough. The Trendelenburg test may be useful in indicating the locations of superficial venous reflux points that result in varicose veins. The Trendelenburg test involves elevating a patient's affected leg while they are lying supine and applying pressure to the great saphenous vein just distal to the SFJ. The patient is then asked to stand up with the practitioner continuing to apply constant pressure to the area. If the sapheno-femoral junction is incompetent but the rest of the distal deep venous system is fine, the varicose veins would not re-engorge with blood as the practitioner will have occluded the area of reflux. However, if the varicose veins fill up with blood while the proximal great saphenous is occluded, it indicates that there is reflux in the more distal regions of the superficial venous system.

Perthes' test is an extension of the Trendelenburg test which tests the function of the musculo-venous pump in the legs. While standing, some pressure is released from the GSV allowing for more blood flow in the limb. The patient is then asked to perform several leg raises. If the perforating veins of the leg have competent valves, the pump will function effectively and superficial veins of the leg will appear less rigid compared to baseline [17].

Note that these tests may also be referred to as "tourniquet tests" as tourniquets can be used to occlude patient's superficial veins instead of manual occlusion. In practice however, these tests have largely been supplanted by venous duplex scans that provide more objective information.

11.4.1 Deep Vein Thrombosis in the Context of CVI and Leg Edema

Thrombotic etiologies of CVI result in symptoms ranging from mild edema to the classical red hot and swollen cellulitic limb with or without leg tenderness on examination. Lack of signs and symptoms of a current or past DVT should not preclude a thorough history for symptoms or risk factors of DVT. Previous DVT causing scarring and valvular dysfunction is a common cause of CVI known as

post-thrombotic syndrome. Secondly, patients with past DVT are at increased risk of future DVT due to altered blood flow and endothelial injury in the recanalized segment of the vein [18]. Hence, there must always be a high index of suspicion for DVT.

11.4.2 Investigations

The preferred method for diagnosis of CVI is color flow venous duplex ultrasound. It is effective in evaluating venous reflux times and has a sensitivity and specificity of 91 and 99%, respectively, for diagnosing proximal DVTs using venous compression criterion. A reflux time of greater than 1 s in deep veins or greater than 0.5 s in superficial veins is diagnostic of reflux. Individual efficacy, however, is operator dependent, and a number of caveats must be taken into consideration. The patient must stand upright during ultrasound in order to elicit maximal reflux using gravity. The angle of insolation must be continuously adjusted in correspondence with the tortuous course of veins and kept under 60 degrees to ensure the accurate detection of doppler shift. Continuous wave doppler can be used to screen and mark varicose veins, however. If a definitive etiology is doubtful on duplex ultrasound, further investigations may be employed.

Air plethysmography testing is similar to Trendelenburg and Perthes' tests mentioned previously but now with a quantitative way of measuring venous volume. Specifically, an inflated cuff around the patient's calf can detect changes in pressure exerted by veins on the cuff and uses this information to calculate changes in volume. The rate of vein refill is known as the venous filing index. If, when a limb is moved from an elevated to a dependent position, the venous filling index is greater than >4 mL/s per second, the vein is incompetent and refilling too fast. A healthy vein has a maximum venous filling index of 2 mLs per second. Occlusion of superficial veins at different levels of the leg can indicate where the reflux is occurring. Similar to Perthes' test, changes in lower limb venous volume after activation of the musculo-venous pump of the calf can also be quantified using air plethysmography.

While largely given up except for research purposes, ambulatory venous pressure (AVP) can be measured by inserting a needle connected to a pressure transducer into the foot. As with previous tests and investigations, the use of gravity, cuffs or occlusion, and exercise or activation of the musculo-venous pump are used in conjunction with the investigation to ascertain the nature of the underling CVI [4, 9].

Air plethysmography is also mainly used as a research tool and for evaluating outcomes of treatments for venous reflux and obstruction.

Computed tomography (CT) and magnetic resonance (MR) contrast venography can be utilized in patients with suspected iliac vein and inferior vena caval pathology. They are ideal to screen for extrinsic etiologies of deep venous obstruction including May-Thurner syndrome, tumors, and cysts. Furthermore, they are useful in visualizing the venous system in patients with complex anatomical variants.

Invasive testing can be used to further visualize an individual's venous system and inform management. Digital subtraction ascending venography visualizes the flow of contrast and provides anatomical clarity and may help distinguish primary and secondary venous disease. Descending venography again can be used to define the anatomic extent of the reflux (Kistner classification) and can be used as a quantitative version of the "cough impulse test" in which the patient performs a valsalva maneuver to identify reflux in the deep venous system.

Intravascular ultrasound (IVUS) has a superior functionality to ascending and descending venograms, allowing 2- and 3-dimensional accurate measurement of deep vein venous stenosis and intraluminal anatomy and pathology.

11.5 Classification of CVI in the Context of Lower Limb Edema

The CEAP classification was developed by the American Venous Forum and is the most commonly used classification system for chronic venous disorders. The name is an acronym for "clinical manifestations," "etiology," "anatomy," and "pathophysiology." Spider veins have already been discussed as C1. C2 denotes the presence of varicose veins—>4 mm diameter palpable tortuous dilations of the superficial veins of the lower limbs in which retrograde flow and blood stasis may occur. Patients presenting with edema are classified C3 (Fig. 11.1). If patients have pigmentation or eczema in their lower limbs they are classified as C4a. C4b is for lipodermatosclerosis, the chronic inflammation and fibrosis of skin and subcutaneous tissues of the lower leg, or atrophie blanche, which present as white coin-sized to palm-sized atrophic plaques. C4c is corona phlebectatica; abnormally visible cutaneous blood vessels at the ankle with nous cups, blue and red telangiectasias, and capillary "stasis spots." Patients with healed venous ulcers are C5, C6 represents an active venous ulcer, and C6r denotes a recurrent active venous ulcer. Observe how CEAP classification system directly corresponds to the pathophysiologic "march" of chronic venous insufficiency. Varicose veins lead to edema, and edema contributes to pathologic skin changes.

In the etiological diagnosis, E_p denotes a primary etiology, where a degenerative process of the venous valves or wall leading to weakness and dilatations results in pathologic reflux. This does not involve scarring or post-thrombotic syndrome, which is a secondary intravenous cause. E_{si} refers to any intravenous secondary cause of venous disease. These include DVT, arteriovenous fistulas, and primary intravenous sarcomas. E_{se} on the other hand refers to extravenous secondary causes of CVI, such as central venous hypertension from obesity, congestive heart failure nutcracker syndrome, or extrinsic compression such as from tumors, fibrosis, May-Thurner syndrome, or poor musculo-venous pump function. Congenital conditions like Klippel-Trenaunay syndrome or arteriovenous malformations are classified as E_c, and finally, E_n is used for apparently idiopathic chronic venous insufficiency.

Anatomical classification is $A_{s,}$ A_D, or A_p for disease in superficial, deep, or perforator veins, respectively. Disease may occur in novel combinations of the

superficial, perforating, and deep venous systems in which case their corresponding subscripts can be combined.

Note that the limb affected should also be recognized with [L] or [R] [4, 9, 19].

Further detailed classification can be made using abbreviations for the specific veins in which there is pathology. While too numerous to all be listed here, 3 important ones that inform appropriate use criteria for management are whether the pathology is in the small saphenous vein (SSV), great saphenous vein above the knee (GSVa), or great saphenous vein below the knee (GSVb) [5, 20].

Finally, the pathophysiologic classification is separated into P_r, P_O, $P_{R,O}$, and P_n for "reflux," "obstruction," "reflux and obstruction," and "no pathophysiology identified," respectively [4, 9, 19].

While CEAP is best used to describe the severity of chronic venous insufficiency, the revised venous clinical severity score (rVCSS) is a more sensitive measure of the change in severity of CVI over time. It is calculated using parameters, viz. pain, varicose veins, venous edema, pigmentation, inflammation, induration, number of active ulcers, duration of active ulceration, active ulcer size, and use of compressive therapy. Calculators of this score are available for free from various sources on the Internet.

11.6 Conservative Management

Initial treatment is conservative and symptomatic. Elevation of the legs above the level of the heart for 30 min 3-4 times per day can reduce edema caused by CVI. The reduction in pressure in superficial tissues increases perfusion and hence also promotes the healing of venous leg ulcers if present.

Exercise in the form of daily walking and plantar flexion exercises to strengthen calf musculo-venous pump have been shown to improve hemodynamic parameters in patients and improve edema and ulcer healing rates.

There is mixed evidence for using compression therapy in patients with symptomatic varicose veins; however, in patients with more severe CVI such as edema or ulcers, long-term compression therapy has been shown to be beneficial. Elastic compression therapy results in faster ulcer healing compared with inelastic compression therapy. High compression is more effective than low compression, and multilayer bandages are more effective at providing desired compression pressures. An external pressure of 35–40 mmHg at the ankle is necessary to prevent capillary exudation in legs affected by venous disease. Compression stockings used for treating CVI need to exert a minimum of 20–30 mmHg (class II compression garments) at the ankle to be effective, with higher grades of compression stockings used for more severe venous disease. Knee-high stockings are sufficient for most patients. They should not, however, be pulled up into the popliteal fossa where they can cause skin irritation, strictures, and discomfort. Compression therapy should not be used in the presence of active infection or cellulitis. Furthermore, an ankle-brachial index ≤ 0.5 is an absolute contraindication to compression therapy. Compression therapy may also put extra stress on the heart in patients with chronic heart failure [21–23].

Pharmacological therapy can be used as an adjunct to therapy or in those with contraindications to or unaccepting of compression therapy. Venoactive agents which increase venous tone such as rutin and rutosides have been used, as well as medications such as stanozolol and prostacyclin analogues which affect blood flow properties. Hydroxyethylrutoside, a mixture of semisynthetic flavonoids, is effective in reducing leg volume and edema through reducing permeability of the microvascular endothelium. However, its effects have not been assessed in the past 6 months after follow-up. Escin, which is "horse chestnut seed extract," induces vasoconstriction through the release of prostaglandins and reduces leg volume and edema in CVI patients. Micronized purified flavonoid fraction (MPFF) is another flavonoid-based medication which has been shown to reduce ankle circumference, erythema, skin changes, ulcer healing, and overall quality of life. A meta-analysis showed that MPFF reduced lower leg edema more than hydroxyethylrutoside.

Stanozolol is an oral anabolic steroid which has shown efficacy in the treatment of lipodermatosclerosis and venous ulcers. Defibrotide is a DNA derivative with profibrinolytic and antithrombotic properties that was found to significantly reduce ankle circumference over one year of use.

Diuretics have no role in the treatment of lower leg edema but may be used to treat comorbid conditions which may be worsening the edema. Antibiotics are only indicated in the presence of infection.

Basic management of other symptoms of chronic venous insufficiency should also be understood in order to optimize patient care and minimize complications. Stasis dermatitis which has symptoms of pruritis, pigmentation, erythema, and scaling can often be present in patients with advanced venous disease. Patient education of proper skin care techniques including skin cleaning, the use of emollients, and the avoidance of itching or scratching is very important. Moderate use of topical corticosteroids may be considered in patients with difficult to manage symptoms. Allergic contact dermatitis and irritant contact dermatitis often develop in patients with CVI and present as a failure of skin symptom improvement with standard treatment. Diagnosis and avoidance of the offending substances is the main preventive measure.

Any venous ulcers present should be debrided. Surgical, enzymatic, or biological methods may be used. Autolytic agents such as hydrogel, EUSOL, and bio cellulose have been shown to increase wound debridement rates. The presence of bacteria is common, and unless signs of inflammation or lymphangitis are present systemic antibiotics and wound swabbing should be avoided. The use of antibiotics in patients with asymptomatic leg ulcers is associated with the development of resistant strains in individuals. Ulcer dressings come in various forms, and a systematic review found no dressing to be more efficacious than any other. As such decisions regarding their use may be centered on individual patient preferences and managing individual symptoms such as exudate, odor, comorbid skin conditions, and pain. There is insufficient evidence for the use of hyperbaric oxygen, electromagnetic therapy, and therapeutic ultrasound in ulcer care [21, 22].

11.7 Operative Management

In the context of edema, the rationale behind the ablation of superficial veins is that removing or collapsing the superficial venous reflux system, which is responsible for the pathological fluid extravasation, will lead to a reduction in oedema. Furthermore, by reducing venous volume in the limb, the effects of venous hypertension in the superficial tissues may be alleviated. However, if there is an underlying pathology in the deep venous system leading to the cause of varicose veins and edema, then edema and varicose veins may recur over time following surgery.

In general, candidates for venous ablation must be symptomatic and show >0.5 s retrograde flow in superficial axial veins. Axial reflux is defined as uninterrupted reflux from the groin to the calf, whereas segmental reflux is localized retrograde flow anywhere in the venous system of the lower limbs. In mild disease (C2–C3), saphenous reflux is treated with reflux in perforators often resolving naturally as a consequence. However, if persistent and large diameter >3 mm, perforator vessels may need to be ablated as well.

The precise indications for surgical correction in patients with edema in CVI are the most uncertain. In 2020, the American Venous Forum, together with the Society for Vascular Surgery, the American Vein and Lymphatic Society, and the Society of Interventional Radiology, developed appropriate use criteria for surgery of lower extremity venous disease in response to reports of inappropriate venous procedures. 119 scenarios of CVI were rated for appropriateness by an expert panel and the widest distribution of scores was observed for the indication of edema, especially edema in the context of segmental saphenous reflux or when considering proximal deep venous stenting for the relief of edema. This is because of the wide and often multifactorial cause of edema in the lower limbs. Hence when there is only mild evidence of venous pathology in the context of edema, clinical judgment based on unique features of the case must be taken into account in order to discern the best method of management. Ablation of below knee GSV, segmental GSV without SFJ reflux, or AAGSV (anterior accessory great saphenous vein) in patients with edema "may be appropriate" in that treatment may be acceptable or reasonable, but more research or patient information is necessary to classify the appropriateness of the intervention. Ablation of an SSV with evidence of reflux is always appropriate in the context of edema.

Note that this "gray zone" of management only exists at C3. If a patient has edema with any features of C4a-C6 disease, ablation of the GSV is almost always appropriate and is likely to lead to improved outcomes for the patient [20].

Superficial veins are known to display anatomical variance among individuals. These can present challenges in the management of chronic venous insufficiency. In the thigh, the GSV can have a large subcutaneous tributary running in the superficial fascia or have a twin lying in the same plane. These duplications can have a common junction with the femoral vein, join separately into the femoral vein, or be "insula" and merge proximally and distally to form a single GSV [24]. Orsini et al. found collaterals which flow underneath the sapheno-femoral junction which can be difficult to identify and cause sure relapse if ignored during surgery [25]. At the

level of the knee, the long saphenous may once again be seen alone or alongside a large superficial tributary. In 29% of the population, it is absent at the level of the knee. This occurs when the GSV pierces the superficial fascia in the distal thigh. The now subcutaneous vessel now runs down past the knee and only reenters the "saphenous compartment" between the superficial and deep fascia distally in the leg. This variation can also be seen with a superficial branch that continues to run down the leg in superficial fascia near the distal GSV. There are also tributaries of the GSV in the leg that are large enough to look like duplications of the GSV [24]. There is also significant variability in deep veins, where classical anatomy may be present in as few as 16% of limbs [5].

There are a multitude of techniques available for vein ablation, with the field constantly moving toward therapies that are minimally invasive and maximally efficacious. Endovenous ablation is the most common format used, with multiple subcategories existing.

Thermal ablation involves denaturing the proteins of the vessel wall, causing collapse. Both laser ablation (Fig. 11.4a) and radiofrequency ablation (Fig. 11.4b) are modes of thermal ablation. Due to the high temperatures used, a large volume of dilute local anesthetic must be administered along the length of the vein being ablated to create a "heat sink." However, this in itself, known as tumescent infiltration, may be uncomfortable for the patient.

Various forms of non-thermal ablation are also in use, with advantages of a decreased likelihood of surrounding structure injury such as nerve injury and a decreased requirement for anesthesia. Mechanical occlusion chemically assisted (MOCA) ablation uses a rotating wire to damage the vein wall from inside with simultaneous application of a liquid sclerosant. In cyanoacrylate embolization (Fig. 11.5a, b), "glue" is introduced to the diseased vein and triggers an innate immune response that results in fibrotic occlusion of the vein.

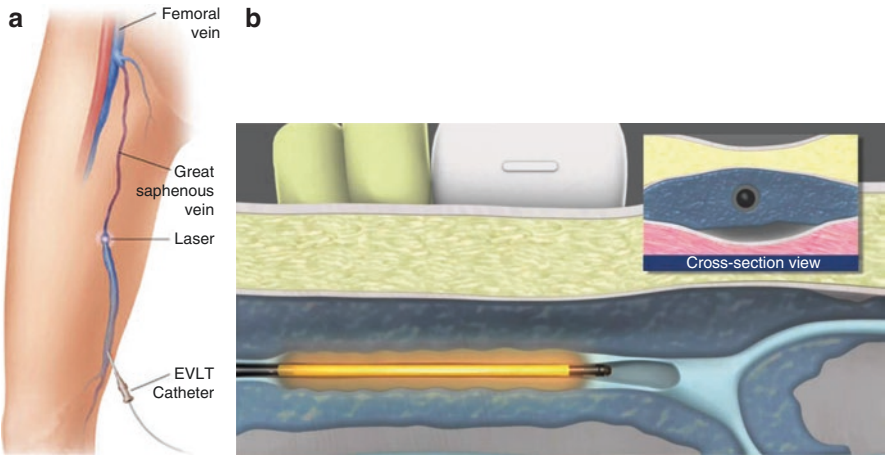

Fig. 11.4 (**a**) Endovenous laser ablation of varicose veins; (**b**) Endovenous radiofrequency ablation of varicose veins

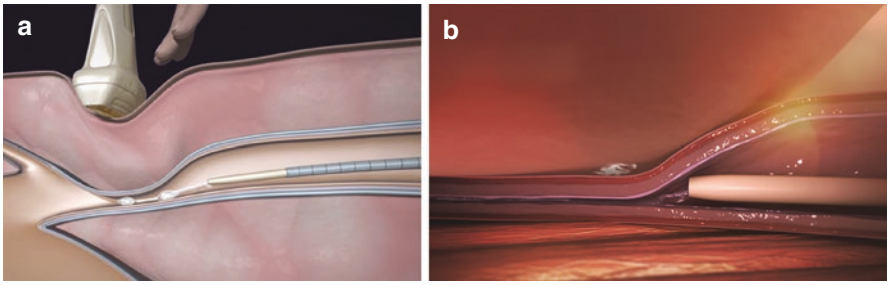

Fig. 11.5 (**a**, **b**) Endovenous cyanoacrylate glue ablation of varicose veins

Another example of endovenous ablation is polidocanol endovenous microfoam, where a mixture of oxygen, carbon dioxide, and 1% polidocanol solution is delivered into the diseased vein causing the formation of microfoam bubbles.

Despite being less invasive and achieving lower rates of recurrence than surgery, endovenous ablation is not always possible. Chronic phlebitis can result in the formation of fibrous adhesions known as synechiae. These can obstruct the passage of a catheter, making endovenous ablation impossible. Similarly, severe tortuosity of a vessel may have an obstructive effect on endovenous surgery. There are also specific contraindications to thermal ablation as the ablation of veins too close to the skin surface may result in burns. As such, target veins that are not at least 1 cm deep to the skin after tumescent anesthesia is administered should not be ablated. Veins over 1cm in diameter have an increased risk of non-closure so more advanced techniques may have to be used in order to ensure adequate closure. Saphenous veins with very large diameters at the sapheno-femoral junction may be at risk for heat-induced thromboembolism. Acute deep vein thrombosis and superficial vein thrombophlebitis are a contraindication to endovenous ablation for similar reasons. Pregnancy also puts patients in a prothrombic state and as such venous interventions should be delayed to at least 6 weeks after delivery.

Saphenous stripping and ligation have been virtually replaced by endovenous ablation techniques.

Great saphenous vein stripping at the level of knee or below it is rarely performed in order to avoid saphenous nerve injury. If peripheral artery disease is present and of a severity that would impede wound healing (ABI<0.5, absolute ankle pressure <60 mHg), it should first be treated before performing any venous intervention or surgery. Patients with genetic or congenital venous abnormalities such as Klippel-Trenaunay syndrome should generally not undergo venous surgery [21, 22].

11.8 Management of Deep Vein Obstruction in the Context of Oedema

In patients with known deep venous insufficiency, the incidence of varicose vein, leg ulceration, and recurrent ulcer recurrence rates are much higher.

Primary investigation of deep venous obstruction utilizing venous duplex ultrasound at 2–3 MHz to evaluate the iliac veins and IVC, CT, and MR venography is useful for estimating the location and severity of obstruction as outlined above. Intravascular ultrasound (IVUS) is the most sensitive diagnostic test and is performed during diagnostic venography or therapeutic intervention to confirm obstruction and select the location of the lesions and size of stents prior to proceeding with angioplasty and stenting (Fig. 11.6a, b).

Similar to operative management of the superficial venous system, the American Venous Forum is cautious with its recommendation for iliac vein or IVC stenting as a first-line therapy for a patient with C3 venous disease. 24% of patients with no history of lower extremity DVT or any other associated symptoms have at least 50% obstruction of the left common iliac vein [26]. Therefore, the presence of significant deep vein obstruction in a patient with lower limb edema does not necessarily imply causation. As such, other causes of edema and the relative impact of deep vein obstruction on the patient's edema must be considered. Treatment may improve the outcome, but the risks of surgery must be weighed against uncertain reward. If edema is present with lipodermatosclerosis, atrophie blanche, or ulceration, stenting is indicated (>C4a-6) for both NIVL and post-thrombotic obstructions of ilio-caval system (Fig. 11.7a, b).

For patients with superficial venous reflux and deep vein reflux, iliac vein obstruction correction is necessary to prevent superficial venous reflux recurrence—which could lead to a recurrent edema. In a 207-patient cohort with deep vein obstruction, patients who only received endovenous laser ablation had a high rate of superficial venous reflux recurrence when compared to patients who received both endovenous laser ablation and iliac stent placement [27].

If endovascular stenting fails, open surgery may be required to relieve symptoms of edema associated with c4a-C6 disease. This may include sapheno-femoral cross-over bypass, cross-pelvic venous bypass, femorofemoral or ilioiliac prosthetic bypass, and femorocaval and aortic elevation. Postoperative management is comprehensive with long-term anticoagulation and compression therapy.

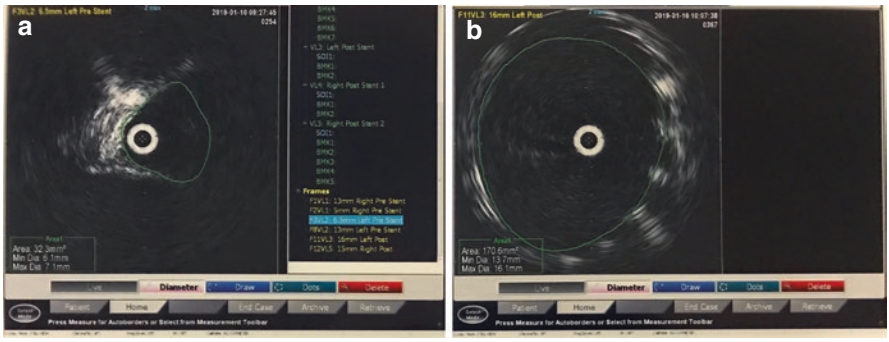

Fig. 11.6 (**a**) Intravascular ultrasound showing narrowed iliac vein; (**b**) Intravascular ultrasound showing widely open stented iliac vein

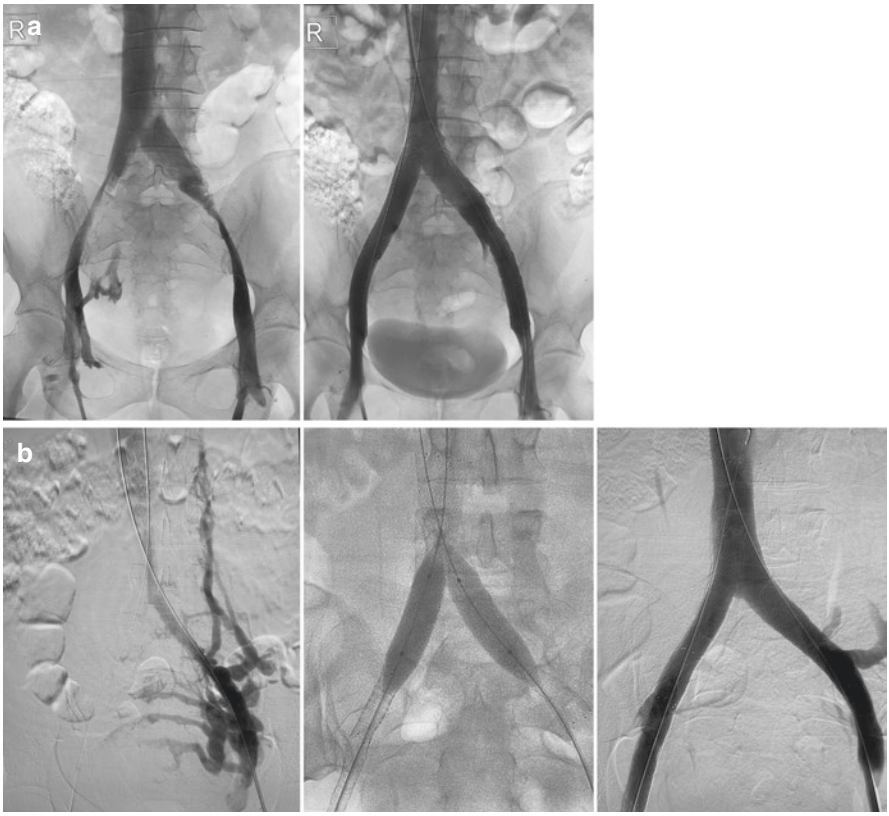

Fig. 11.7 (**a**) NIVL obstruction of bilateral Iliac veins Fig. 11.5b. After Iliac vein stents; (**b**) Post-thrombotic obstruction of left common iliac vein; (**b** and **c**) After Iliac vein balloon angioplasty and stents

Finally, in the case of more novel causes of deep vein obstruction like tumors, cysts, and osteophytes, conservative symptomatic treatment of chronic venous insufficiency should occur in tandem with treatment of the primary etiology.

Conflict of Interest None.

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