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Abstract

The most common cause of chronic venous disease (CVD) is reflux. Reflux is abnormal blood flow direction resulting from venous valve dysfunction. In the lower extremity thigh and calf, abnormal flow is from proximal to distal in deep or superficial veins, or deep to superficial in perforator veins. Reflux is most often primary (unknown etiology and not present at birth), is less often secondary (known cause like thrombosis or trauma), and is rarely congenital. This chapter will discuss how to manage symptomatic venous reflux disease.

4.1 Introduction

The most common cause of chronic venous disease (CVD) is reflux. Reflux is abnormal blood flow direction resulting from venous valve dysfunction. In the lower extremity thigh and calf, abnormal flow is from proximal to distal in deep or superficial veins, or deep to superficial in perforator veins. To account for a normal valve closure time, reflux is defined by consensus opinion to be 0.5 s for veins generally, with the exception of the femoropopliteal deep system, where the value is 1.0 s [1]. Reflux is most often primary (unknown etiology and not present at birth), is less often secondary (known cause like thrombosis or trauma), and is rarely congenital.

Obstruction, typically from thrombosis, is also an important cause of CVD. Anatomic

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Table 4.1 Common venous discomfort symptoms

Tingling
Aching
Burning
Muscle cramps
Swelling
Throbbing
Heaviness
Itching
Restless legs
Tiredness
Fatigue

obstructions, such as vein compression from an artery, as in May-Thurner syndrome, or even a tumor, can also cause CVD. Some unfortunate patients may have combined reflux and obstruction, which produces worse symptoms than either condition alone. Both reflux and obstruction in the infrainguinal lower extremity are accurately diagnosed by duplex ultrasound, and this test is essential to management of potential CVD patients.

This chapter will discuss how to manage symptomatic venous reflux disease. Not all reflux causes symptoms, and not all reflux should be treated. The differential diagnosis of potential venous symptoms will be discussed. Then the principles of conservative and procedural management will be outlined.

4.2 Differential Diagnosis

CVD can manifest itself in a variety of ways. The hallmark symptoms of CVD are pain, discomfort, spider veins, reticular veins, varicose veins, swelling, skin changes, and leg ulcers. Table 4.1 outlines common venous discomfort complaints. Pain, discomfort, and swelling symptoms typically worsen with extremity dependence (i.e., standing) as the day progresses, since reflux is activated by gravity. The symptoms are also worse when the weather is warm, as a result of venous dilatation. Symptoms usually improve with extremity elevation or compression. Swelling, skin changes, and ulcers typically start at the ankle

Table 4.2 Differential diagnosis of lower extremity pain and discomfort

Deep or superficial venous thrombosis
Peripheral arterial disease
Iliocaval obstruction
Pelvic congestion syndrome
Proximal venous reflux (i.e., branches of the internal iliac vein)
Vascular malformation
Nutcracker syndrome
Chronic compartment syndrome
Neuralgia (i.e., sciatica)
Complex regional pain syndrome
Restless legs syndrome
Musculoskeletal (i.e., muscle/tendon/ligament sprain, muscle pain, osteoarthritis, rheumatoid arthritis)
Cellulitis

Table 4.3 Differential diagnosis of unilateral leg swelling

Chronic venous insufficiency
Deep venous thrombosis
Iliocaval obstruction
Lymphedema
Lipedema
Baker's cyst
Cellulitis
Orthopedic trauma

area, where ambulatory venous pressure is highest, but may progress proximally up the calf and thigh.

Table 4.2 outlines differential diagnostic considerations for lower extremity pain and discomfort. Tables 4.3 and 4.4 outline the differential diagnoses of unilateral and bilateral leg swelling, respectively. Table 4.5 outlines the differential diagnosis of leg ulcers. Skin changes in the ankle area are often due to chronic venous insufficiency, but each skin sign has a differential diagnosis of its own, and dermatology consultation should be considered if chronic venous insufficiency cannot be ruled in. Leg ulcers can also be caused by skin cancer, or venous ulcers can become malignant [2]. It is not clear from the literature when to biopsy, but skin biopsy should be considered for leg ulcers which do not heal despite appropriate management and

Table 4.4 Differential diagnosis of bilateral leg swelling

Bilateral chronic venous insufficiency
Congestive heart failure
Pulmonary hypertension
Protein-losing nephropathy
Liver cirrhosis
Obesity

Table 4.5 Differential diagnosis of leg ulcer

Venous ulcer
Peripheral arterial disease
Neuropathic ulcer
Pressure ulcer
Skin cancer

patient compliance and for nonhealing leg ulcers in unusual locations.

More advanced CVD, measured as increased CEAP class, is associated with more areas of reflux. Saphenous reflux is common in all CEAP classes. The prevalence of perforator and deep venous reflux increases with increasing CEAP class [3]. The most common pattern of saphenous reflux involves the great saphenous vein (GSV) (Fig. 4.1). Reflux in the small saphenous vein (Fig. 4.2) or anterior accessory GSV (Fig. 4.3) is also common. However, cross-over involvement occurs, and each patient with suspected CVD merits a duplex ultrasound to determine if they meet the typical pattern [4]. Non-saphenous reflux (Fig. 4.4) occurs in around 10 % of patients [5].

Venous symptoms can also be caused by venous sources other than lower extremity reflux or obstruction. Iliocaval obstruction should be considered in patients with venous symptoms with minimal or no reflux on infrainguinal ultrasound. Pelvic congestion syndrome can present with pelvic pain or varicosities or lower extremity varicosities which can be followed with ultrasound above the inguinal ligament. A vascular malformation usually presents at birth or puberty (due to hormonal changes) and can also be suggested by unusual anatomy seen on ultrasound. Reflux of tributaries of the internal iliac vein can cause varicosities on the buttocks or pelvic areas.

**Fig. 4.1** The classic great saphenous vein (*asterisk*) pattern

4.3 Medical Management

Multiple conservative measures have been recommended for patients with CVD, including compression, leg elevation, exercise, diet and weight loss, and analgesics. Compression options include elastic compression stockings, inelastic bandaging, and pneumatic compression. Prescription strength compression stockings start at 20–30 mmHg (Class 1) and are followed by 30–40 mmHg (Class 2), 40–50 mmHg (Class 3), and 50+ mmHg (Class 4). In general, compression at 20–30 mmHg seems effective for symptomatic varicosities, while 30–40 mmHg is preferred if tolerated for those with venous ulcers



Fig. 4.2 Classic small saphenous vein reflux pattern

or leg swelling [1]. Knee-high length is often used due to greater ease in getting the stocking on, but thigh and pantyhose styles are also available. Compression therapy improves symptoms and quality of life in patients with simple symptomatic varicosities, but it does not reverse disease [6]. In patients with venous ulcers, compression accelerates healing and reduces ulcer recurrence risk [7]. Compression has not been shown to reduce varicosity recurrence rates or slow disease progression [6].

Compression therapy is contraindicated in patients with significant peripheral arterial disease, congestive heart failure, or active infection at the site. Patient compliance and difficulty getting the stocking on can be a major problem, so providers should carefully explain the benefits to



Fig. 4.3 The anterior accessory great saphenous vein (*asterisk*), when present, runs superficial to the femoral vessels

patients. Interventional ablation of symptomatic reflux is more effective in improving quality of life than compression and lifestyle modification [8]. Despite the data, third-party payers often require a “trial” of conservative measures, such as compression, before ablation can be performed.

Exercise has been advocated under the hypothesis that making the calf muscle stronger, even in the presence of malfunctioning venous valves from reflux, may improve overall calf muscle pump function. In patients with venous ulcers, improving ankle range of motion and muscle strength improves venous hemodynamic parameters but has not yet been clearly shown to affect clinical outcomes [9].

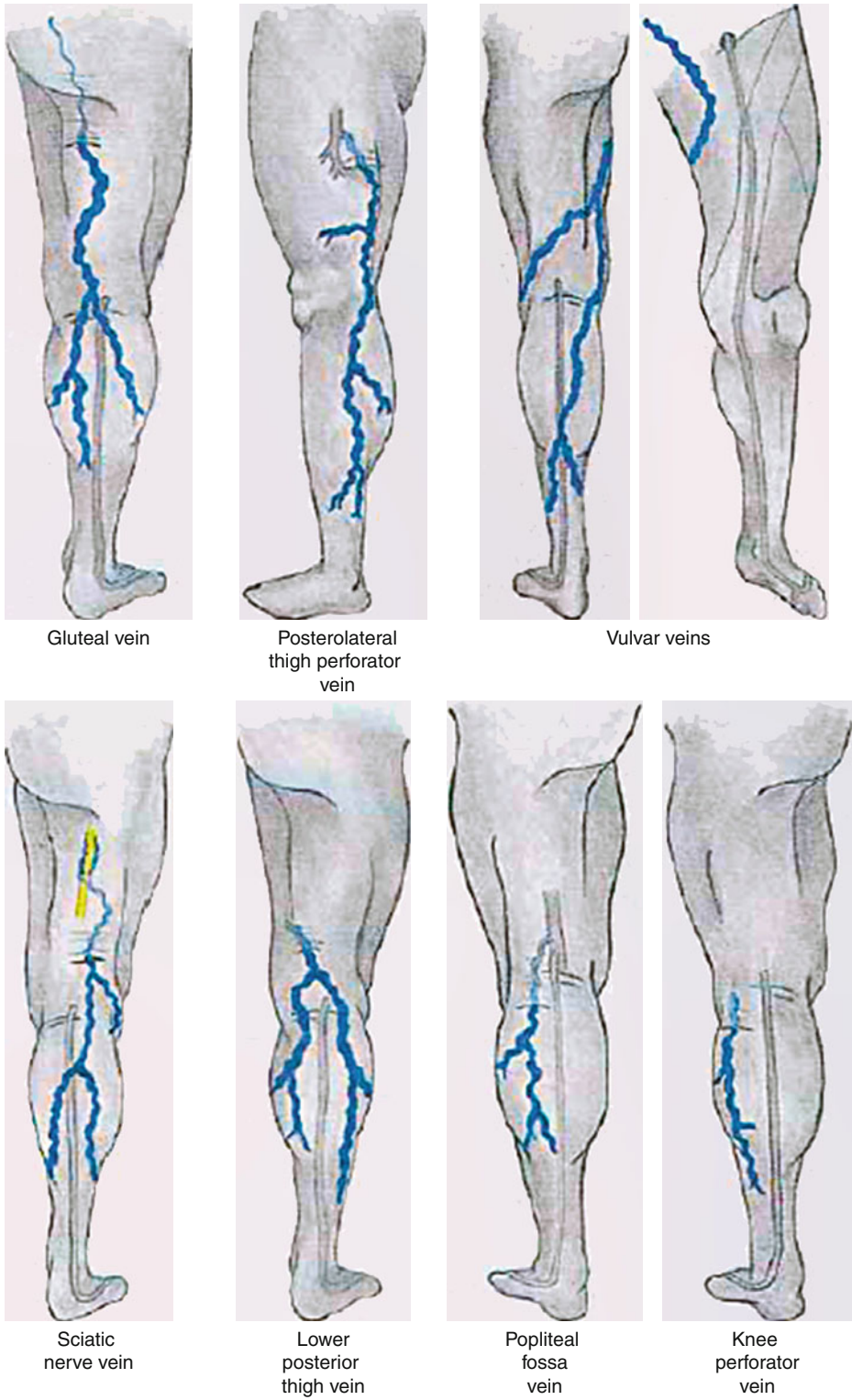


Fig. 4.4 Non-saphenous vein reflux patterns (Used with permission) [5]

Table 4.6 Venoactive medicines

Horse chestnut seed extract (aescin)
Flavonoids – rutosides, diosmin, hesperidin
Micronized purified flavonoid fraction (MPFF)
French maritime pine bark extract
Calcium dobesilate, naftazone
Benzarone

Table 4.6 lists venoactive medicines. Studies on these medicines are limited, none are FDA approved for venous reflux disease, and many are not available in the USA [1].

4.4 Interventional Strategies

In general, symptomatic and refluxing veins are treated in the following order: saphenous, then epifascial (saphenous tributaries and localized varicosities), then perforator veins, and then deep veins. This order is based upon assessment of benefits and risks. Ablation of symptomatic saphenous reflux has been shown to improve quality of life in patients with symptomatic varicosities [10]. In patients with healed or active ulcers, it has been shown to reduce ulcer recurrence by 25 % at 4 years, from 50 to 25 % [11]. It is not clear if saphenous ablation improves ulcer healing rates, with positive and negative results reported [11, 12].

Incompetent perforator vein (IPV) management is controversial. IPVs are associated with worse venous disease, based on CEAP score [3]. The clinical benefit with treatment of IPVs, however, has not been shown independent of saphenous vein treatment [13]. Clinical improvement after IPV treatment may be hard to demonstrate because isolated perforator reflux is rare. The ankle blowout syndrome of a leg ulcer with a nearby incompetent perforator was described in 1953 [14]. Consensus opinion still favors treatment in this setting [1].

Some deep vein treatments, such as ilio caval stenting for obstruction or gonadal vein ablation for pelvic congestion syndrome, carry a high benefit at low risk. Other deep vein disease treatments, such as those for mixed obstruction and reflux, carry significant morbidity and require

specialized skills, allowing performance only at specialized centers.

Relative contraindications to superficial vein treatment include severe medical comorbidities which limit patient quality of life benefits from treatment. Inability to walk reasonably (i.e., at least 5 min/h) may increase clot risk with treatment. Although the opinion has been challenged, treatment of the superficial system in the presence of deep venous obstruction is generally considered contraindicated, since the superficial system could be functioning as collateral circulation [15]. Acute thrombosis is generally a contraindication to superficial treatment, except in cases like saphenofemoral junction ligation of proximal GSV thrombosis in order to reduce embolization risk. Anticoagulation, however, can be considered in this case as well.

Some advocate concomitant instead of staged therapies of saphenous and epifascial systems. The main benefit of the concomitant strategy is that the patient can be treated in one session, resulting in a faster improvement in quality of life, although the improvement is not sustained in the longer term [16]. This strategy may be particularly useful for patients who travel a long distance for their appointments, who lack the time for repeated visits, or who are undergoing ambulatory phlebectomies.

Others advocate staged treatments. After GSV ablation, attached varicosities often become smaller, and some disappear [17]. Even small saphenous vein (SSV) reflux sometimes corrects after GSV ablation [18]. Presumably these improvements are due to reduction of the volume of reflux moving distally into these veins after successful ablations. Remaining varicosities are then easier to treat [19]. Some have even recommended waiting 4 months after saphenous ablation before treating remaining varicosities due to less need for treatment with this waiting period [20].

It is important for patients to understand that any chronic venous disease management strategy does not cure vein disease, but can often make a big difference in clinical endpoints such as quality of life and ulcer recurrence. Still, varicosity or ulcer recurrence remains a risk. The patient

who presents with recurrent chronic venous disease needs a reassessment, including duplex ultrasound, to determine the cause of recurrence before a successful treatment strategy can be implemented.

4.5 Alternative Strategies

Some advocate treatment of the superficial tributaries before the saphenous in many cases. The saphenous-first strategy was based on a pathophysiologic model that reflux begins at saphenous-deep junctions, such as the saphenofemoral junction, and then progresses distally gradually, usually over several years. More recent ultrasound studies challenge that belief [21]. Based on this new information, some advocate treating the refluxing tributaries and localized varicosities first, before the refluxing saphenous vein, in many cases. Retrospective data on this technique, termed ASVAL (ambulatory selective varices ablation under local anesthesia), is intriguing [22].

An additional challenge to the standard saphenous then tributaries model comes from some who advocate disconnecting points where reflux crosses from deep to saphenous (like the saphenofemoral junction) or from saphenous to *epifascial* (like the saphenous-tributary junction) but to otherwise preserve these refluxing veins in order to preserve venous drainage and thus prevent disease recurrence [23]. CHIVA (for the French, “cure conservatrice et hemodynamique de l’insuffisance veineuse en ambulatoire” and in English, “conservative hemodynamic treatment for chronic venous insufficiency”) utilizes surgical ligations for disconnection [24]. CHIVA has been shown in two randomized, controlled trials to reduce recurrence in comparison to surgical high ligation and stripping [25, 26].

4.6 Ablation Techniques

Thermal (endovenous laser or radiofrequency), surgical (high ligation with or without stripping), and chemical (ultrasound-guided foam

sclerotherapy) are all safe and effective techniques to ablate a symptomatic saphenous vein. There are few studies comparing clinical endpoints between these options. The American Venous Forum and Society for Vascular Surgery recommend thermal ablation as first choice in a consensus opinion because it is minimally invasive and has similar or better early-term results, and equivalent midterm results, as surgery [1]. Techniques for ultrasound-guided foam sclerotherapy are rapidly improving, but results are not yet as good as those seen with thermal ablation and surgery [1].

Ablation techniques for epifascial veins include chemical (sclerotherapy with or without ultrasound guidance) and surgical (microphlebectomy or powered phlebectomy) [19]. Chemical ablation is fully reviewed in Chap. 11 and surgical techniques in Chap. 12. Thermal ablation (laser or radiofrequency), reviewed in Chap. 10, can also be used in some cases if the vein is straight and long enough for technical success, but this technique is less commonly used for these veins [19].

Surgical endoscopic perforator surgery (SEPS), thermal ablation, and ultrasound-guided foam sclerotherapy are all technically successful therapies for incompetent perforating veins [1]. Some deep vein problems, such as ilio caval obstruction and pelvic congestion syndrome, are now also amenable to endovascular treatments. Other deep vein diseases require sophisticated techniques like valvuloplasty or even venous bypass, which are performed only at specialized centers.

Conclusions

Venous disease occupies a wide spectrum of severity and possible treatments. The keys to success remain the same: identify the source of the symptoms; treat in order to achieve a durable improvement in quality of life for patients; and minimize venous disease recurrence. The history of phlebology is far from written. New technologies, instrumentation, and knowledge of the subject continue to alter our understanding of the disease.

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