

## Different Methods of Treatment of Varicose Veins: Review Article

Walid Mohammed Gamal<sup>1,\*</sup>, Ayman Elsaed Hassaballah<sup>2</sup>, Abdelrahman Gamal Monofy<sup>1</sup>,  
Abdelraheem Fathy Mohammed<sup>1</sup>

<sup>1</sup> Department of Vascular surgery, Faculty of medicine, South valley University, Egypt.

<sup>2</sup> Department of Vascular surgery, Faculty of medicine, Assuit University, Egypt.

### Abstract

Venous diseases exhibit a diverse range of symptoms, contrasting with arterial occlusive illness. Diagnosis of venous hemodynamic dysfunction lacks a widely accepted noninvasive method like the ankle-brachial index. Understanding lower extremity venous anatomy is crucial for chronic venous disease diagnosis and treatment. Venous walls, with thinner layers than arteries, consist of intimal, medial, and adventitial layers. Venous valves and their role in superficial, deep, and perforating veins are essential for proper venous function. The superficial venous system includes reticular veins, large and small saphenous veins, and their tributaries. Varicose veins affect a substantial portion of the population, with risk factors such as hormonal, lifestyle, acquired, and hereditary elements. Varicose veins can lead to chronic venous insufficiency and venous ulceration, causing a significant strain on healthcare resources. Foam sclerotherapy, in particular, has gained popularity as a cost-effective and highly beneficial treatment for various forms of varicose veins. It induces fibrosis, accelerates ulcer healing, and provides lasting results. Catheter-directed foam sclerotherapy (CDFS) has emerged as a potential therapy with higher complete ablation rates compared to ultrasound-guided foam sclerotherapy (UGFS). Despite challenges in data interpretation and standardization, CDFS offers benefits in controlled foam distribution and reduced complications. Overall, understanding the anatomy, pathophysiology, and treatment options is crucial for effectively managing venous diseases and improving patient outcomes.

**Keywords:** Catheter Foam Sclerotherapy, Lower Limbs, Primary, Varicose Vein

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### 1. Introduction

Compared to arterial occlusive illness, venous disease has a wider range of symptoms, from asymptomatic telangiectasias to edema, skin abnormalities, and ulceration [1]. There is no commonly acknowledged noninvasive diagnostic for venous hemodynamic dysfunction like the ankle-brachial index. Like the vascular system, proper flow requires a working pump and conduits [2]. Venous illness may result from conduit occlusion and directional incompetence, unlike arterial disease [3]. Lower extremity venous architecture is more varied and complicated than arterial anatomy. Chronic venous illness diagnosis, therapy, and pathogenesis need a thorough grasp of this architecture [4].

### 2. Lower Extremity Venous Anatomy

#### 2.1 General Anatomy

Venous walls are thinner than arteries yet have intimal, medial, and adventitial layers. The basement membrane-based intimal monolayer generates antithrombogenic chemicals. Endothelial disturbance may expose neutrophil receptor ligands by promoting

procoagulant activity and suppressing anticoagulant [5]. Three smooth muscle layers containing collagen and elastin make up the adrenergically innervated medial layer [6]. Veins are less elastic and muscular than arteries. The thickest layer, the adventitia, has more collagen, making veins stiffer than arteries. The elliptical cross section of lower leg veins increases venous system capacitance, which is needed for calf "muscle pump" function [7]. Endothelial folds generate bicuspid valves in superficial, deep, and perforating veins. Most valves in the distal leg split the hydrostatic blood column and assure flow from superficial to deep and caudal to cephalad [8]. Reversing the resting antegrade transvalvular pressure gradient closes valves. Normal reflux lasts < 0.5 seconds, but retrograde flow > 0.5 seconds is deemed abnormal in upright posture. Lower extremity veins meet the muscle fascia in the superficial or deep compartment [9]. Deep veins under the muscular fascia drain lower extremity muscles, superficial veins above it drain cutaneous microcirculation, and perforating veins link them. Nomenclature changes have simplified definitions and deleted eponyms for communicating veins [10].

## 2.2 Superficial Veins

The superficial venous system includes reticular veins, large and small saphenous veins, and their tributaries. Lower extremity skin and subcutaneous tissue are drained by parallel reticular veins between the saphenous fascia and dermis. Direct connection with deep veins occurs in 60% of individuals with severe thigh telangiectasias via perforators [11]. The dorsal pedal venous arch gives rise to the great saphenous vein, which ascends anterior to the medial malleolus, passes posteromedial to the knee, and joins the common femoral vein in the thigh. Ultrasound shows its 'Egyptian eye' path in the saphenous compartment in the thigh [12]. [13] found true duplication of the great saphenous vein in 8-10% of the thigh and 25% of the calf, with probable varicose veins from fascia support loss. The dorsal pedal arch's tiny saphenous vein rises posterolaterally beyond the lateral malleolus to the popliteal vein. It connects to the great saphenous vein or other veins with 7-10 valves. Chronic venous disease can result from various superficial-to-deep drainage systems, including lateral superficial veins in Klippel-Trenaunay syndrome, sciatic drainage, lateral subdermic system, obturator veins, and round ligament routes [14, 15]. Most people have valves at the saphenofemoral junction and external iliac–common femoral segment, while the great saphenous vein has at least six [16].

## 2.3 The Deep Veins

The primary deep veins of the lower leg follow arterial routes and vary greatly, with classic morphology in just 16% of limbs. Tibial, peroneal, soleal, and gastrocnemial calf deep veins create a plexiform configuration surrounding arteries [18]. Calf muscle pumps depend on 1 to 18 soleal sinuses and a gastrocnemial network emptying into the popliteal vein. The deep femoral vein links directly to the popliteal vein in 38% of limbs and via a tributary in 48% [19]. The femoral vein substitutes "superficial femoral vein," and up to 27% of extremities may have duplicated internal iliac veins [20]. Internal iliac veins drain several tributaries and may be important in ilioacaval occlusion [21]. Varicosities and pelvic congestion syndrome are caused by pelvic venous insufficiency and inadequate internal iliac vein tributaries [22]. In 50% of venograms, the left common iliac vein is compressed between the sacroiliac spine and right common iliac artery to generate the inferior vena cava [23]. The inferior vena cava develops from fetal posterior cardinal, subcardinal, and supracardinal veins, with 1% of patients having disorders that cause interruptions or duplications. Valve distribution varies between the iliac and deep venous systems, however iliac veins seldom have valves [24]. Deep venous valves grow from cranial to caudal, and comprehensive dissections demonstrate different valves in common, external, and internal iliac veins [25]. Popliteal valve competence is important because muscle sinusoids, valveless but typically emptying into valved veins, help pump calf muscle [26].

## 2.4 The Perforating Veins

Cadaveric investigations show 64 direct or indirect perforating veins between the ankle and groin, always with arteries and in intramuscular septa [27]. The many and varied perforating veins of clinical relevance may be divided into foot, medial and lateral calf, and thigh groups, each directing flow to superficial or deep veins [28]. Clinically important *Gamal et al., 2023*

medial calf perforators are paratibial and posterior tibial. Avoid Sherman, Boyd, and Cockett eponyms for perforators. Approximately 14 veins per leg align in a 3-cm-wide track ascending the medial calf, with 52% being direct perforators [29]. The upper perforators are paratibial, whereas the lower ones are posterior tibial. Direct perforators are clustered into five groups around the medial malleolus, whereas indirect perforators are randomly dispersed [30]. Paratibial perforators, especially, may necessitate subfascial endoscopic closure with fascia incision between the superficial and deep posterior compartments. Four or five knee-to-ankle paraperoneal perforators are common in the lateral calf. Hunterian femoral canal perforators link the great saphenous vein proximal to the knee to the distal superficial femoral or proximal popliteal vein, which might cause medial thigh varicosities when competent [31].

## 2.5 Varicose Veins

About 23% of US people have varicose veins, including spider telangiectasias, reticular veins, and genuine varicosities. With spider telangiectasias and reticular veins, 80% of males and 85% of women have them. Varicose veins afflict 22 million women and 11 million men aged 40–80 [32]. Chronic venous insufficiency will cause venous ulceration in 2 million of these people. High incidence of varicose veins and high expenses of treating consequences, especially chronic venous ulcers, strain healthcare resources [33]. Varicose veins cause chronic venous ulcerations, which cost the US \$3 billion and rob 2 million workdays. Even varicose veins without chronic venous insufficiency lower quality of life [34].

## 3. Risk Factors, Anatomy, and Pathophysiology

Risk factors for varicose veins include hormonal, lifestyle, acquired, and hereditary variables. Estrogen, especially in women, may exacerbate varicose veins. Smoking is a modifiable risk factor for varicose veins and chronic venous illness, including ulceration. Post-thrombotic syndrome after deep vein thrombosis (DVT) may cause varicose veins without underlying venous illness [36]. Small perforator veins link superficial veins to deep veins for lower extremity venous drainage. Disease in any of these venous systems may cause varicose veins, with symptoms and severity increasing with further system involvement. Various pathophysiological causes cause vein wall weakness and varicosity. Varicosities may form in branch vessels as well as larger and smaller saphenous veins. Large varicose veins may develop from iliac vein or inferior vena cava obstruction [38]. Venous hypertension, valvular incompetence, vein wall structural changes, inflammation, and shear stress are the main causes of varicose veins. Venous hypertension is caused by reflux from valve incompetence, venous outflow blockage, or calf-muscle pump failure. Valve leaflet deformation, ripping, thinning, and adhesion may cause incompetence. Structural alterations weaken and dilate vein walls. The histology of varicose veins shows collagen type I overproduction, collagen type III synthesis reduction, and smooth muscle cell and elastin fiber disruption. Varicose vein specimens may have higher tissue inhibitors of matrix metalloproteinases, which may enhance extracellular matrix deposition. In animal models, high-pressure venous valves have more inflammatory cells and matrix metalloproteinases, causing poor remodeling. Inflammatory and prothrombotic

alterations from turbulent flow, flow reversal, and lower shear stress compromise the vein wall and valve leaflets' structural and functional integrity [39-41].

#### 4. Clinical Presentation

CEAP classifies venous varicosities by class (C1–6), etiology (E), anatomy (A), and pathophysiology (P). Spider telangiectasias and reticular veins (C1) are identified as dilated intradermal venules (<1 mm) and subdermal veins (1–3 mm). True varicose veins (C2) are rope-like, palpable subcutaneous veins (>3 mm). All varicose vein classes may be cosmetically problematic [42]. Localized pain, burning, pruritus, and dry skin might progress to leg heaviness, weariness, cramping, hyperpigmentation, edema, lipodermatosclerosis, and ulceration in higher CEAP classes [43]. Varicose vein analysis comprises a physical exam and CEAP score. Presenting, architecture, etiology, and pathophysiology classify clinical manifestations from cosmetic to severe [44].

Uncomplicated instances need cosmetic treatments. Fatigue, heaviness, bleeding, local discomfort, pores, edema, and skin ulceration characterize complicated varicose veins. Venous ulcerations may take over 9 months to heal and 66% linger over 5 years, affecting everyday life. A thorough categorization system helps clinicians diagnose and compare symptoms to determine the lower limb varicose vein problem stage using CEAP clinical grades [45, 46]. Diagnostic testing includes patient history, physical examination, Doppler studies, duplex scanning, venous pressure checks, plethysmography, varicography, ascending/descending phlebography, and subjective questionnaires to assess venous disease's impact on social, physical, psychological, and pain quality of life (QOL).

#### 5. Diagnosis

Varicose veins are examined standing up for erythema, discomfort, or induration, suggesting superficial vein thrombosis. Identify severe chronic venous disease symptoms include edema, hyperpigmentation, lipodermatosclerosis, and ulceration [48]. A thorough pulse examination includes the Brodie-Trendelenberg test to detect superficial and profound venous insufficiency. The Perthes test detects deep venous insufficiency or blockage [49]. If the reason is unknown or intervention is contemplated, venous ultrasonography is advised. It checks for superficial and deep venous reflux, thrombosis, and iliac or inferior vena cava problems. CT, MRI, or venography may be needed [50]. Continuous-wave, pulsed-wave, color, power, and spectral Doppler ultrasound methods assess blood flow in different ways. Standard color duplex ultrasound scans allow noninvasive hemodynamic and morphological evaluation of treated veins and early detection of recurring varicose veins. It uses color pictures to detect irregularities and quantify blood flow. Duplex ultrasonography can monitor clinical progression following CVD therapy by revealing recurrent occurrences [51].

#### 6. Prognosis

Varicose veins may worsen without therapy and correction of underlying causes. Advanced chronic venous insufficiency, such as lower extremities edema and venous ulcers, may worsen quality of life and function. When big,

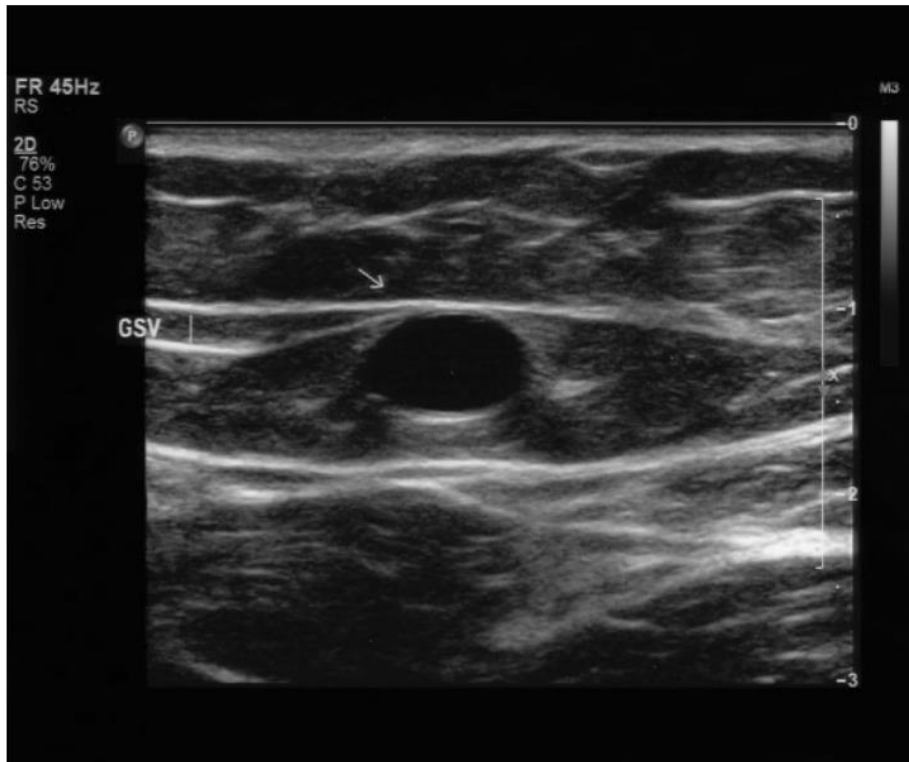
damaged, or over bony prominences, varicose veins may thrombose or burst and bleed. In a large observational cohort study, varicose veins elevated DVT risk 7-fold. [52].

#### 7. Therapies for Varicose Veins

Stabbing or microincision phlebectomy, which uses local tumescent anesthesia and leaves little scarring, may cure branch varicosities from main superficial veins [53]. For larger and smaller saphenous vein varicosities, endovenous methods have replaced surgical stripping and vein ligation, which have a higher recurrence rate. Stripping may cause ecchymosis, scarring, hematoma, infection, nerve damage, and DVT. Due to the danger of venous ulcers, surgical stripping and endovenous ablation should be avoided in deep venous patients [54, 55]. Endovenous radiofrequency or laser ablation treatments are effective and lasting alternatives to surgical stripping with better results and fewer problems. Endovenous varicose vein treatments are used more due to patient choice for less invasive procedures, clinician accessibility, and favorable reimbursement [56, 57]. Endovenous ablation is cheaper than surgical stripping. However, recurrence may need microincision phlebectomy. Endovenous treatment failure or anatomical issues may need surgical stripping [58]. Ecchymosis, hematoma, skin burns, DVT, and nerve damage are endovenous ablation complications. Minimizing saphenous venous reflux may resolve or improve additional superficial varicosities, minimizing the requirement for local treatments [59]. Foam sclerotherapy may cure spider telangiectasias and saphenous varicosities. Mixing air with traditional sclerosants increases venous wall contact, even in bigger varicose veins. Previous reports of foam sclerosant embolization following saphenous vein ablation have tempered excitement. Z.-X [60].

#### 8. Foam Sclerotherapy

Lower extremity primary varicose veins are often associated with the big and small saphenous vein basins, and various treatments are employed for radical varicophlebectomy, including crosssectomy and stripping, radiofrequency, laser ablation, or intraluminal sclerosing agents [61]. Since the 2006 European Consensus, the use of liquid or highly viscous foam sclerotherapy, employing sodium tetradecyl sulfate and polidocanol in the EU, has been permitted, with recommended alternatives being sterile air from pre-made setups [54, 62, 63]. Patients prefer foam preparation with biological gases like carbon dioxide and/or oxygen, although this requires specialized medical practice equipment [64]. When introduced into varicose veins, the very viscous foam efficiently desquamates the endothelium with 20–30 micron bubble sizes, followed by limb compression to collapse the treated venous artery into connective tissue [59]. Beneficial outcomes of foam sclerotherapy have been observed since 2012, with success rates exceeding 90% for reticular and reticular vein treatments. The effectiveness depends on factors such as the type of sclerosing agent, foam creation process, viscosity, severity of venous pathology, and proper healing procedures [65].



**Fig. (1).** Transverse ultrasound image of the great saphenous vein. The great saphenous vein lies in a subcompartment bordered superficially by the saphenous fascia and deeply by the muscular fascia. [17]

**Table (2).** Risk Factors for Varicose Veins. [37]

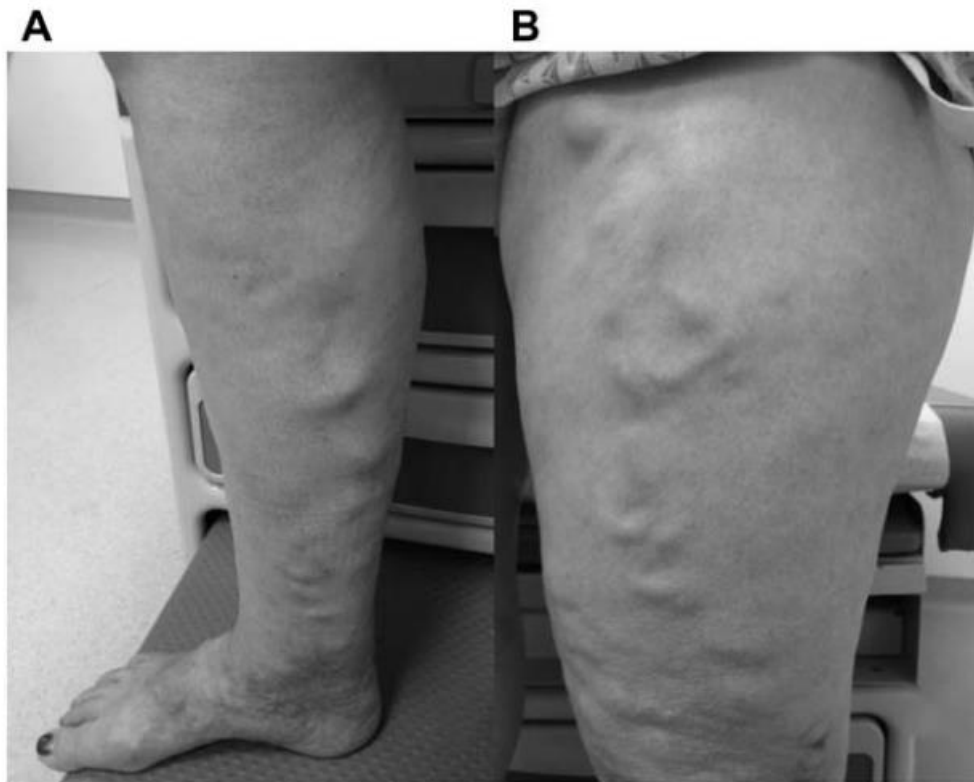
Category	Risk Factor	Proposed Mechanism	
Hormonal	Female gender	High estrogen state Venous hypertension	
Lifestyle	Prolonged standing and/or sitting	Venous hypertension	
Acquired	Smoking	Venous endothelial injury	
	Obesity	Venous hypertension	
	Pregnancy		High estrogen state
			Venous hypertension
	Deep vein thrombosis		Deep venous obstruction
			Venous valvular incompetence
Age		Venous valvular incompetence	
Inherited	Family history	Venous valvular incompetence	
	Tall height	Venous hypertension	
	Congenital syndromes		Venous valvular incompetence
			Venous hypertension
			Deep venous obstruction



**Fig. (2).** Prominent reticular veins in the left popliteal fossa. [35]

**Table (3).** CEAP clinical classification of chronic venous disease. [47]

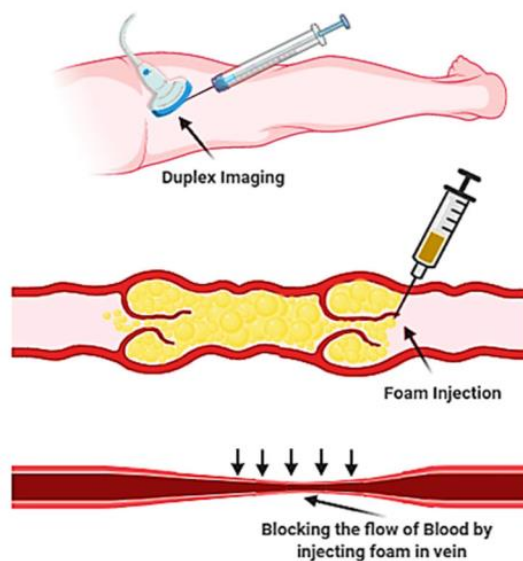
<b>Clinical classification</b>	<b>Description</b>
C0	No visible signs of venous illness
C1	Reticular veins
C2	Varicose veins
C3	Edema
C4a	Changes in skin and Pigmentation or eczema.
C4b	Changes in skin and subcutaneous tissue atrophies blanche
C5	Healed venous ulcer
C6	Active venous ulcer



**Fig. (3).** Large rope-like varicose veins along the right lower leg and left thigh. [9]

**Table (4).** Etiologic, anatomic, and pathophysiologic classification of chronic venous disease. [6]

Etiological classification description		Anatomical classification description		Pathophysiology description	
Ec	Congenital	As	Superficial veins	Pr	Reflux
Ep	Primary	Ap	Perforator veins	Po	Obstruction
Es	Secondary (post-thrombotic)	Ad	Deep veins	Pr,o	Reflux and obstruct ion
En	No venous cause identified	An	No venous location identified	Pn	No venous pathophysiology identifiable



**Fig. (4).** Treatment of varicose veins using foam sclerotherapy. [84]

### 8.1 General Rules for Foam Sclerotherapy:

Foam sclerotherapy, recognized as an effective intervention for varicose veins, involves a systematic approach. The procedure begins with a comprehensive ultrasound examination to evaluate the venous system in both standing and supine positions [66]. Patients are educated about the process and receive clear post-procedure instructions [67]. The method includes skin disinfection and the application of Esmarch on specific joints [68, 69]. Subsequent steps encompass re-examination using ultrasound and the Veinlite system, facilitating veinipuncture with the guidance of Veinlite or ultrasound [70]. Foam creation is executed with limbs straightened at a 45-degree angle on adapted couches [71, 72]. The introduction of foam occurs within a 60-second timeframe, accompanied by vital sign monitoring and ultrasound assessment [73]. Cannulas are sequentially removed, puncture sites covered with sterile patches, and eccentric compression applied [74, 75]. Measurements for a medical elastic sock are taken while the patient is supine, and the compression sock is subsequently applied [76, 77]. Patients are instructed to engage in a 30-minute walk, and comprehensive follow-up details are provided [78]. Numerous clinical studies since 2012 emphasize the procedure's effectiveness, highlighting the importance of the sclerosing agent, foam characteristics, and proper execution. Foam sclerotherapy achieves over 90% success in reticular vein treatment, surpassing liquid sclerosant outcomes. Although recurrence rates may be slightly higher, long-term studies showcase improved patient quality of life and successful recurrence elimination with repeated foam sclerotherapy. Combining external elastocompression, eccentric compression, and using perforated catheters enhances efficacy, making foam sclerotherapy a cost-effective and highly beneficial treatment for various varicose vein forms [65, 79-82].

Moreover, foam sclerotherapy accelerates ulcer healing, surpasses liquid sclerosant in venous malformation treatment, and proves to be a cost-effective, standalone treatment or a complementary approach after other interventions like laser or radiofrequency ablation [60, 83, 84].

### 8.2 Role of Catheter-directed foam sclerotherapy in treatment of varicose vein

Varicose veins, which affect 40% of people, raise healthcare expenses [62]. Nonsurgical varicose vein treatments including radiofrequency ablation (RFA) and endovenous laser ablation have replaced open surgery [85, 86]. Ultrasound-guided foam sclerotherapy (UGFS) induces fibrosis using foamed sclerosant and is safe and cost-effective [87]. Complete truncal ablation may need numerous UGFS treatments [82]. Catheter-directed foam sclerotherapy (CDFFS) uses duplex ultrasonography to administer sclerosant via an intravenous catheter, reducing hazards and improving effectiveness [65]. With its catheter introduction, CDFFS offers even sclerosant distribution, making it a cost-effective, safe, and similar ablation method [88, 89]. Foam sclerotherapy, which injects sclerosant foam, treats varicose veins well [68, 90]. Sodium tetradecyl sulfate, polidocanol, hypertonic saline, and N-butyl cyanoacrylate are important sclerosing agents that cause damage via several pathways [47, 91, 92].

A meta-analysis shows CDFFS has significantly higher complete ablation rates than UGFS at different intervals [93]. CDFFS is a potential therapy because to enhanced surface contact and reduced venous filling [94, 95]. Variations in procedural approaches and lack of standardization are problems [79, 96]. Data interpretation of full ablation rates is difficult due to incomplete reporting and various confounders [34, 97]. CDFFS has less complications due to controlled foam distribution, smaller doses, and injection pressures [98]. Variations in tributary treatment, procedure, and chemical usage affect findings [61, 96]. Results interpretation is further complicated by chronic venous illness severity distribution and the lack of direct comparisons between UGFS and CDFFS [79]. Despite these problems, CDFFS is a potential varicose vein therapy that has benefits over UGFS.

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