REVIEW ARTICLE

Varicose Veins: Recent Updates in Pathophysiology, Diagnosis, and Management

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ABSTRACT

Varicose veins are one of the most prevalent vascular disorders encountered in practice worldwide. It was regarded as a cosmetic concern, but it has been found that varicose veins are a chronic progressive condition associated with significant morbidity and reduced quality of life. This review deals with the current understanding of varicose veins and particular emphasis is placed on emerging non-thermal, non-tumescent ablative techniques and updated classification systems. The endovenous thermal ablation, mechanochemical ablation, and cyanoacrylate-based closure are critically appraised along with conventional surgical and sclerotherapy approaches.

Keywords: Chronic venous disease, Endovenous ablation, Sclerotherapy, Foam sclerotherapy, Cyanoacrylate, Mechanochemical ablation

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INTRODUCTION

Varicose veins are abnormally dilated, tortuous superficial veins of the lower extremities. Usually, they arise as a consequence of sustained venous hypertension and progressive valvular incompetence. They constitute the most visible clinical manifestation of chronic venous disease (CVD), which also includes telangiectasias, reticular veins, oedema, skin changes, and venous ulceration. Despite their high prevalence and well-recognised clinical significance, varicose veins have historically received little attention compared to arterial disorders. The main reason is that varicose veins were considered only of cosmetic importance. But untreated CVD carries a measurable risk of progression, thrombotic complications, and profound impairment of health-related quality of life.[1,2] In Western Europe and North America, varicose veins affect between 25 and 33% of women and approximately 10 to 20% of men over the age of 40 years.[3-5] The condition imposes a substantial economic burden through direct healthcare expenditure [6]

The past decade has witnessed a paradigm shift. The adoption of updated classification systems, advances in high-resolution duplex ultrasonography, and the introduction of minimally invasive endovenous techniques have collectively transformed the standard of care. This review aims to provide a contemporary, evidence-based synthesis of these developments.

Epidemiology and Risk Factors

Prevalence of varicose veins increases substantially with advancing age, rising from approximately 5% in individuals under 30 years to over 50% in those aged 70 years and above.[7] The female predominance, particularly evident in younger age groups, correlates with age, suggesting that hormonal factors related to pregnancy and exogenous oestrogen exposure contribute to early-onset disease in women. The cumulative effect of prolonged standing and gravitational stress becomes the dominant determinant in older cohorts regardless of sex (Fig. 1).[5,6]

Family history constitutes one of the strongest individual risk factors, with first-degree relatives of affected individuals carrying a two- to threefold increased lifetime risk. [7] Occupations requiring prolonged orthostatism, including nursing, teaching, and retail work, are associated with accelerated progression of venous reflux. Obesity, characterised by a body mass index exceeding 30 kg/m^2, exerts compounding effects by increasing intra-abdominal pressure, impairing calf muscle pump function, and promoting a chronic low-grade inflammatory state that weakens venous wall integrity.[8] Multiparity is an independent risk factor, with each successive pregnancy associated with an incremental increase in varicose vein prevalence, attributable to hormonally mediated smooth muscle relaxation within venous walls and the compressive effect of the gravid uterus on the iliac venous system.[7]

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Fig. 1: Varicose veins of Lower limb

Populations consuming low-fibre diets, with associated constipation and elevated intra-abdominal straining, demonstrate higher CVD prevalence in some comparative studies, though confounding by socioeconomic variables limits causal inference. Sedentary lifestyle and reduced ankle joint mobility, a predictor of calf muscle pump dysfunction, have emerged as modifiable risk factors.[9]

PATHOPHYSIOLOGY

Venous Hypertension and Reflux

The fundamental haemodynamic abnormality underlying varicose vein formation is sustained ambulatory venous hypertension. Under physiological conditions, calf muscle contraction during ambulation propels venous blood proximally against a pressure gradient, with competent bicuspid valves preventing reflux during the relaxation phase. When valvular incompetence develops, reflux generates an elevated venous pressure column during standing that is transmitted to the dependent superficial venous system, initiating a cascade of structural and cellular changes that perpetuate disease.[10,11,12] Reflux is defined by the Society for Vascular Surgery as retrograde venous flow lasting more than 500 milliseconds in superficial veins. It is more than 1,000 milliseconds in deep veins following the release of manual compression or a Valsalva manoeuvre during duplex ultrasonographic assessment.[13]

Venous Wall Remodelling

Primary varicose veins are not simply the passive result of elevated intraluminal pressure but reflect active structural remodelling of the venous wall driven by a complex interplay of mechanical, inflammatory, and molecular mechanisms. Histological analysis of varicose vein specimens shows focal disruption of the smooth muscle cell architecture, fragmentation of elastin fibres, and irregular collagen deposition. There is increased activity of matrix metalloproteinases (MMPs), particularly MMP-1, MMP-2, and MMP-9.[6,7] These proteolytic enzymes degrade structural extracellular matrix components, resulting in progressive weakening and dilatation of the vessel wall. [14-17]

Venous wall hypoxia, generated by turbulent flow and stagnation in varicose segments, stimulates the expression of hypoxia-inducible factors and inflammatory cytokines, including transforming growth factor-beta, interleukin-1 beta, and tumour necrosis factor-alpha. These mediators recruit leucocytes, particularly monocytes and mast cells, into the venous wall, establishing a chronic inflammatory microenvironment that sustains MMP activation and perpetuates wall degradation.[8] Endothelial dysfunction, manifesting as impaired nitric oxide bioavailability and upregulation of adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), further amplifies leucocyte transmigration and oxidative stress within the vessel wall. [9,18,20]

In diseased veins, smooth muscle cells transition from a contractile to a synthetic phenotype, increasing production of extracellular matrix proteins while losing their capacity for coordinated vasoconstriction. This phenotypic shift may explain the altered haemodynamic responsiveness of varicose veins and their tendency toward progressive dilatation despite pharmacological intervention.[8]

Valvular Incompetence: Primary versus Secondary

A critical distinction in the pathophysiological framework is whether valvular incompetence is primary or secondary in origin. Primary incompetence, accounting for the majority of varicose vein presentations, involves intrinsic structural weakness of the valve leaflets and supporting vein wall, arising in the absence of prior thrombotic disease. Secondary incompetence follows deep vein thrombosis, with recanalisation producing fibrotic, scarred valve leaflets incapable of competent closure. The management implications differ substantially, as secondary venous insufficiency may require treatment directed at deep venous outflow obstruction in addition to superficial ablation.[10]

CLASSIFICATION SYSTEMS

The CEAP Classification

Standardised clinical communication and research in CVD have been facilitated by the Clinical-Aetiological-Anatomical-Pathophysiological (CEAP) classification, originally developed in 1994 and subsequently revised in 2004 and most recently updated in 2020.[14,15] The clinical component stratifies disease severity from C0 (no visible venous disease) through C1 (telangiectasias or reticular veins), C2 (varicose veins), C3 (oedema), C4 (skin changes including pigmentation, eczema, lipodermatosclerosis, and atrophie blanche and ankle flare), C5 (healed ulceration), to C6 (active venous ulceration). A subscript of 'A' denotes asymptomatic disease and 'S' denotes symptomatic disease at each clinical class.[21,22]

The 2020 revision introduced several refinements of practical importance, including the addition of C2r and C6r to designate recurrent varicose veins following prior intervention, recognising the clinical significance and management challenges posed by recurrent disease. The ankle flare which was initially thought to be C1 diseases has been shifted to C4c. The aetiological component distinguishes congenital (Ec), primary (Ep), and secondary (Es) causes, with the addition of En for cases where no venous aetiology is identified. These

updates have enhanced the precision of clinical documentation and facilitated more granular comparative research across international cohorts.[11] (Fig. 2 and 3)

Venous Clinical Severity Score

The venous clinical severity score (VCSS) provides a quantitative, examiner-administered instrument for measuring disease burden and tracking treatment response over time. The VCSS evaluates ten clinical attributes, including pain, varicose veins, oedema, skin pigmentation, inflammation, induration, ulceration, and compression use, each scored on a four-point scale from absent to severe. Validated sensitivity to change makes the VCSS particularly valuable in clinical trials assessing the efficacy of therapeutic interventions.[10] (Fig. 4)

DIAGNOSTIC EVALUATION

Clinical Assessment

The diagnostic evaluation of varicose veins begins with a thorough clinical history incorporating symptom characterisation, assessment of functional impairment, identification of risk factors, and documentation of prior venous interventions. Symptoms attributable to chronic venous hypertension include heaviness, aching, throbbing, pruritus, and cramps in the lower limbs, typically worsening with prolonged dependency and improving with limb elevation or compression hosiery. Physical examination should include

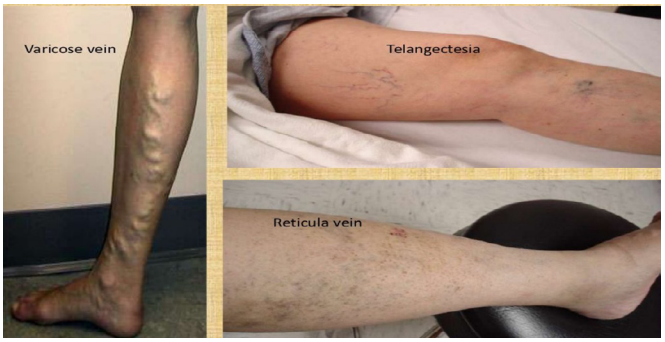

Fig. 2: Varicose vein, Tejengiectasia, Reticular veins

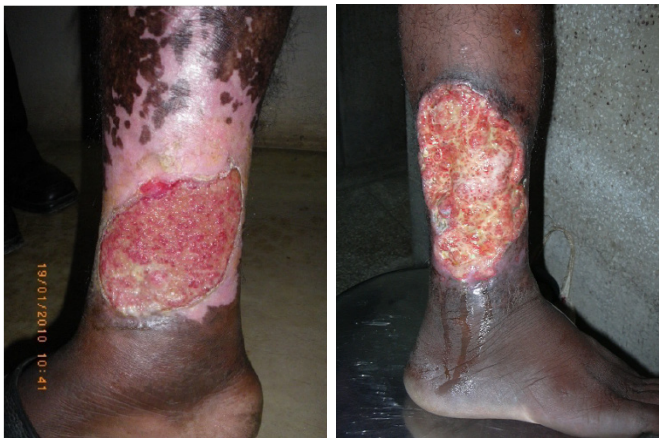

Fig. 3: Venous Ulcer

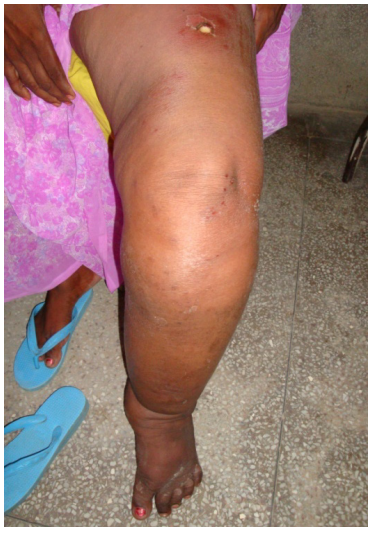

Fig. 4: DVT with secondary varicosity

inspection and palpation of the entire lower extremity venous system in both standing and supine positions, with documentation of the distribution and characteristics of visible varices, skin changes, and oedema.[23,24]

The Trendelenburg test, historically used to localise sites of incompetence, has been largely superseded by duplex ultrasonography as the standard diagnostic modality. However, clinical assessment retains value in identifying obvious sapheno-femoral or sapheno-popliteal junctional incompetence and in planning the sequence of ultrasonographic interrogation. [25]

Duplex Ultrasonography(Fig. 5)

Duplex ultrasonography combining B-mode anatomical imaging with colour flow and spectral Doppler interrogation has become the cornerstone of varicose vein assessment,

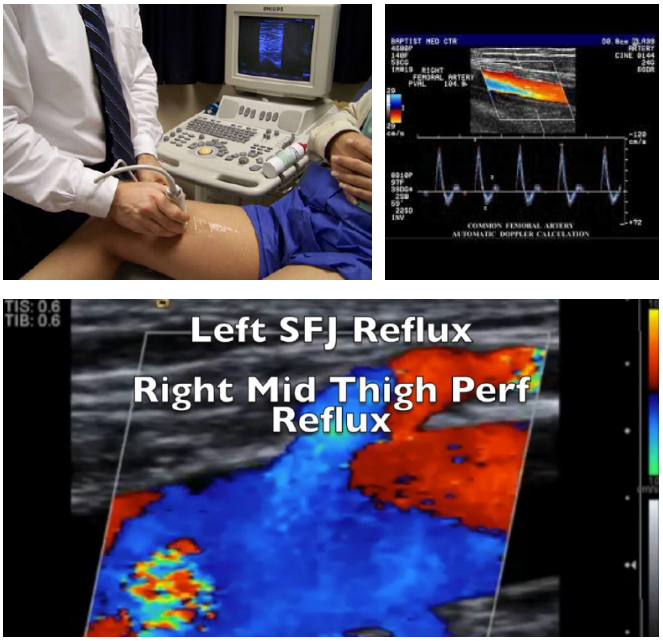

Fig. 5: Colour Dopler/Duplex

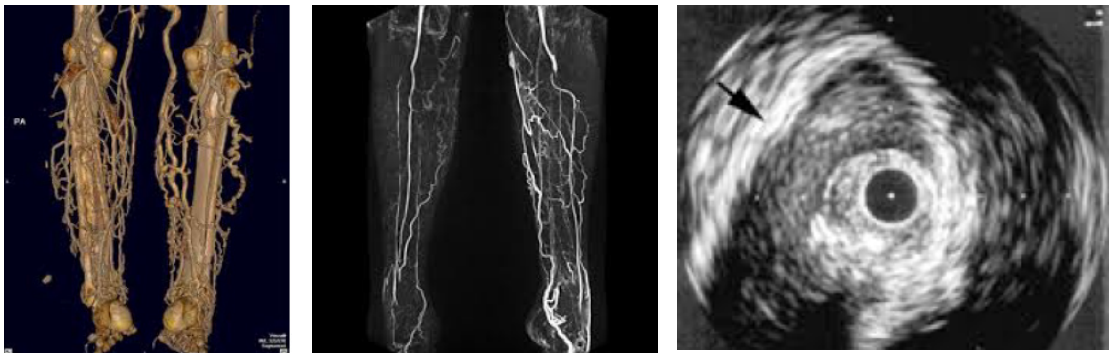

Fig. 6: CT Venograpghy, MR Venography, IVUS

essential for accurate anatomical mapping, identification of reflux sites, and pre-procedural planning. The examination should systematically evaluate the great saphenous vein (GSV) from its junction with the common femoral vein at the saphenofemoral junction (SFJ) to the ankle, the small saphenous vein (SSV) and its termination at the saphenopopliteal junction (SPJ), the deep venous system for evidence of prior thrombosis or ongoing obstruction, and any accessory or perforating veins contributing to the superficial disease burden.[26]

Reflux is provoked by standardised augmentation manoeuvres, typically distal compression and release or a Valsalva manoeuvre at the SFJ. The diameter of the GSV at specific anatomical landmarks, including 3 cm below the SFJ in the upright position, provides additional prognostic information, as larger diameters are associated with higher rates of reflux and greater symptom burden. Detailed mapping of the GSV course is a prerequisite for thermal and non-thermal endovenous ablative procedures, as accurate knowledge of the vein's diameter, depth, and proximity to adjacent structures determines the choice of energy settings, technique, and tumescent anaesthetic volume required [25,26]. Sometimes CT and MR venography may be needed, especially in deep venous insufficiency and recurrent Veins. Intravascular ultrasound is helpful in deep venous problems. (Fig. 6)

MANAGEMENT OF VARICOSE VEINS

Conservative Measures

Conservative management constitutes an appropriate first-line approach for patients with mild to moderate symptoms, those with contraindications to invasive intervention, and those who decline procedural treatment. Graduated elastic compression hosiery, prescribing external support typically in the range of 20 to 30 or 30 to 40 mmHg at the ankle, reduces ambulatory venous pressure, attenuates oedema, and may relieve symptomatic heaviness and aching, particularly when worn consistently throughout the day.[27,28] Evidence supporting a disease-modifying effect of compression therapy on structural venous pathology is limited, and compression should be regarded as symptom management rather than curative treatment.

Lifestyle modification counselling should encompass weight reduction in obese patients, avoidance of prolonged orthostatism, regular physical activity emphasising calf muscle strengthening, and limb elevation during periods of rest. Pharmacological agents, including micronised purified flavonoid fraction (MPFF) and horse chestnut seed extract (HCSE), have demonstrated modest efficacy in randomised controlled trials for reduction of CVD-related symptoms and oedema, though their effect on reflux or structural venous disease is not established.[26]

Conventional Surgical Treatment

High ligation and stripping of the GSV was the standard surgical procedure for varicose veins for much of the twentieth century and remains a valid treatment option in appropriate candidates. The operation involves disconnection of the GSV at the SFJ through a groin incision, followed by removal of the GSV trunk using a vein stripper, and avulsion of visible tributary varices through multiple small punctures. Randomised controlled trials conducted in the 1990s and early 2000s established that surgical stripping was superior to high ligation alone in reducing recurrence rates, attributable to the removal of the GSV reflux source.[27,28]

Surgical treatment carries a recognised risk profile including wound complications, saphenous nerve injury (occurring in up to 40% of cases following below-knee stripping due to the intimate anatomical relationship of the nerve and vein), haematoma formation, and the morbidity associated with general or regional anaesthesia. Systematic reviews comparing surgery with endovenous ablation techniques have consistently demonstrated equivalent or inferior efficacy of surgery for anatomical success and symptom improvement at medium-term follow-up, alongside higher complication rates and longer recovery periods.(17, 27.28,)

Endovenous Thermal Ablation

Endovenous thermal ablation (EVTA), encompassing radiofrequency ablation (RFA) and endovenous laser ablation (EVLA), has emerged as the dominant treatment modality for GSV and SSV incompetence in contemporary phlebological practice, endorsed by national and international guidelines as the preferred interventional approach in eligible patients.[19,26]

Both techniques deliver controlled thermal energy to the

Fig. 7: RFA, Laser, Moca, Glue

venous endothelium and wall through a catheter or optical fibre advanced to the target segment under ultrasound guidance, inducing irreversible fibrosis and occlusion of the treated vein. Tumescent local anaesthesia, instilled along the vein tract through a series of perivenous infiltrations, serves the dual purposes of compressing the vein around the catheter to optimise energy transfer and providing a protective thermal barrier shielding adjacent nerves, skin, and fascia from heat injury.

Radiofrequency ablation, commercialised predominantly through the ClosureFAST system, delivers segmental bipolar radiofrequency energy at 120 degrees Celsius via 7-centimetre treatment cycles along the vein. In the landmark CLASS trial, RFA demonstrated non-inferiority to GSV surgery at five years with superior early recovery, reduced post-procedural pain, and lower rates of haematoma and nerve injury.[18] Endovenous laser ablation delivers laser energy at wavelengths typically ranging from 810 to 1470 nm, with higher wavelengths targeting water-containing haemoglobin and venous wall tissue with greater selectivity and a more favourable perioperative pain and ecchymosis profile than earlier shorter-wavelength devices.[19,20,29]

Meta-analyses encompassing multiple randomised controlled trials have demonstrated anatomical occlusion rates exceeding 90% at one to five years for both RFA and EVLA, broadly equivalent to or superior to those achieved with GSV surgery. With the added advantages of outpatient delivery under local anaesthesia, same-day mobilisation, and rapid return to normal activity.[25] Clinical effectiveness in terms of symptom relief and quality of life improvement has been similarly demonstrated across multiple validated outcome instruments including the Aberdeen Varicose Vein Questionnaire and EuroQol EQ-5D. (Fig. 7)

Foam Sclerotherapy

Ultrasound-guided foam sclerotherapy (UGFS) represents an established minimally invasive technique in which a sclerosant agent, most commonly sodium tetradecyl sulphate or polidocanol, is prepared as a microfoam by agitation with gas and injected into the target varicose vein under real-time ultrasound guidance. The foam formulation displaces intraluminal blood, maximising sclerosant contact time with the venous endothelium and enhancing the inflammatory response that produces the desired fibrous occlusion.[30]

UGFS offers particular versatility for the treatment of large, tortuous tributary varices, recurrent varicose veins following prior intervention, and perforating veins that are challenging to approach surgically. It can be delivered in an outpatient or office setting without anaesthesia and is associated with low upfront procedural costs. However, UGFS demonstrates lower anatomical occlusion rates for GSV truncal disease compared with EVTA at medium-term follow-up, with several randomised trials reporting superior duplex-confirmed occlusion rates for thermal techniques at one to three years.[31,32]

Adverse effects of foam sclerotherapy include superficial thrombophlebitis in treated segments, skin staining at injection sites, and, rarely, neurological symptoms including visual disturbances and migraine-like episodes, postulated to arise from paradoxical embolisation of sclerosant foam through a patent foramen ovale. Serious complications, including stroke and deep vein thrombosis, have been reported but are uncommon at recommended foam volumes and sclerosant concentrations.[33,34]

Non-Thermal Non-Tumescent Techniques

Recognition of the limitations of thermal ablation, principally the requirement for tumescent anaesthesia and the attendant

procedural complexity and patient discomfort, has stimulated the development and clinical adoption of non-thermal, non-tumescent (NTNT) techniques that offer effective venous occlusion without thermal energy delivery. These include mechanochemical endovenous ablation (MOCA) and cyanoacrylate adhesive closure, both of which have achieved regulatory approval and accumulated a growing evidence base over the past decade.[35]

Mechanochemical endovenous ablation, commercialised as the ClariVein system, combines mechanical endothelial disruption via a rotating wire at the catheter tip with simultaneous infusion of a liquid sclerosant, typically polidocanol. The mechanical component induces vasospasm and endothelial damage that potentiates the sclerosant effect, enabling effective occlusion without thermal injury and without the need for tumescent anaesthesia.[36,37] Published series and registry data report one-year occlusion rates of 80 to 90% for GSV treatment with MOCA, somewhat lower than those achieved with optimised EVTA but acceptable within the context of a less invasive procedure that patients tolerate with minimal discomfort.[38]

Cyanoacrylate adhesive closure, delivered through the VenaSeal system, represents the most recent NTNT innovation. The technique involves catheter-based injection of n-butyl cyanoacrylate adhesive at regular intervals along the incompetent vein under ultrasound guidance. It induces immediate mechanical closure through polymerisation of the adhesive upon contact with blood. It is followed by a fibrotic foreign body reaction that maintains long-term occlusion.[38] Critically, the absence of tumescent anaesthesia eliminates the need for multiple perivenous injections, reducing procedural discomfort and duration. The VeClose randomised controlled trial demonstrated non-inferiority of cyanoacrylate closure to RFA for GSV occlusion at 36 months, with comparable symptom improvement outcomes.[39,40]

Both NTNT techniques avoid the theoretical risks of thermal injury to adjacent nerves and skin, making them particularly attractive for the treatment of veins running in close proximity to the saphenous nerve or overlying thin, atrophic skin. The absence of compression therapy requirements post-procedure, particularly for cyanoacrylate closure, represents an additional patient convenience advantage, though the implications for long-term outcomes require continued follow-up from ongoing registry studies.[41,42]

MANAGEMENT IN SPECIAL POPULATIONS

Recurrent Varicose Veins

Recurrent varicose veins following prior intervention represent a clinically challenging and increasingly prevalent problem as the treated population ages and initial interventions accumulate. Recurrence can be attributable to inadequate primary treatment with residual incompetent saphenous segments, neovascularisation at the groin following high ligation creating new channels of reflux, or progression of venous disease in previously uninvolved segments. The 2020 CEAP update, by introducing the C2r designation, has facilitated more precise documentation of this entity and enabled more granular analysis in outcome studies.[18]

UGFS is frequently the preferred technique for recurrent varicose veins given the distorted anatomy at the saphenofemoral junction following prior surgery, which increases the technical complexity of repeat endovenous procedures. However, EVLA and RFA remain feasible in experienced hands. [43]

Varicose Veins in Pregnancy

Varicose veins appearing during pregnancy typically reflect a combination of hormonally mediated venous smooth muscle relaxation and progressive compression of the iliac veins by the enlarging uterus. In the majority of women, varicose veins that develop during pregnancy substantially regress within six to twelve months postpartum. Invasive intervention is generally deferred until after delivery and the post-lactational period. Conservative management with graduated compression hosiery is the mainstay of care throughout pregnancy, reducing symptom burden and the risk of thrombotic complications.[44]

Women in whom significant varicose veins persist beyond twelve months postpartum should be reassessed with repeat duplex ultrasonography and considered for definitive intervention, taking into account planned future pregnancies, as additional gestational events may precipitate recurrence even after successful treatment.

EMERGING DEVELOPMENTS AND FUTURE DIRECTIONS

Pharmacological Targets

Advances in the molecular pathophysiology of venous wall remodelling have identified several potential pharmacological targets for disease modification in CVD. Inhibitors of specific MMP subtypes, compounds targeting the Notch signalling pathway implicated in venous smooth muscle cell phenotypic switching, and agents modulating the transforming growth factor-beta cascade are under preclinical and early clinical investigation.[8,9] Should effective pharmacological disease modification prove achievable, it could complement procedural treatment by addressing the underlying biological predisposition to recurrent venous disease.

Artificial Intelligence in Vascular Ultrasound

Machine learning algorithms applied to duplex ultrasonographic datasets hold promise for automated reflux detection, venous diameter measurement, and predictive modelling of treatment outcomes based on pre-procedural morphological characteristics. Early validation studies have demonstrated that convolutional neural networks can achieve comparable accuracy to experienced vascular technologists for GSV segmentation and reflux classification, raising the prospect of AI-assisted diagnostic workflows that could improve consistency and efficiency in high-volume phlebology practices.[45]

Patient-Reported Outcome Measures

The field of phlebology has embraced the shift toward patient-centred outcome assessment, with validated patient-reported

outcome measures (PROMs) including the Aberdeen Varicose Vein Questionnaire, VEINES-QOL/Sym, and the chronic venous insufficiency quality of life Questionnaire (CIVIQ) increasingly mandated in clinical trial protocols and national audit programmes. The integration of PROMs with objective haemodynamic and anatomical endpoints is enabling a more complete understanding of the relationship between venous reflux correction and patient well-being, and is highlighting that anatomical occlusion does not invariably translate into proportionate symptom relief.[46]

High-Intensity Focused Ultrasound (HIFU) (Fig. 8)

High-intensity focused ultrasound (HIFU) represents one of the most innovative and genuinely non-invasive modalities. Unlike endovenous thermal ablation, foam sclerotherapy, and even the newer non-thermal, non-tumescent catheter-based techniques, HIFU achieves venous wall destruction without any percutaneous access to the circulation. Focused ultrasound beams are directed transcutaneously from an extracorporeal transducer, converging at a precise focal point within the target vein to generate localised temperatures exceeding 70 degrees Celsius through acoustic absorption. This induces coagulative necrosis of the venous endothelium and smooth muscle layer, resulting in fibrous occlusion of the treated segment. The overlying skin, subcutaneous fat, and adjacent neurovascular structures are largely unaffected by the rapidly dissipating thermal gradient outside the focal zone.[47,48]

The theoretical appeal of a truly non-invasive approach is substantial. The complete elimination of skin puncture, catheter insertion, tumescent anaesthesia, and intravascular instrumentation removes the procedural risks. The chance of haematoma, thrombophlebitis, needle-site infection, nerve injury from perivenous anaesthetic infiltration, and deep vein thrombosis from catheter manipulation remains minimal. Patients with needle phobia, coagulation disorders precluding vascular access, or significant anxiety regarding invasive procedures may represent an ideal population for HIFU. The procedure requires no compression therapy in the immediate post-treatment period and imposes no restrictions on ambulation or daily activity. (49,50,)

Fig. 8: HIFU

The principal HIFU device developed for superficial venous applications is the SonoVein system (Theraclion, France), which combines a therapeutic focused ultrasound transducer with an integrated real-time B-mode ultrasound imaging module, enabling simultaneous visualisation of the target vein and confirmation of focal energy deposition during treatment. The device generates multiple focused energy pulses along the treated venous segment, with the operator advancing the focal zone in stepwise increments under continuous sonographic guidance. Early clinical feasibility studies demonstrated histological evidence of targeted endothelial and smooth muscle cell destruction at the focal zone with preservation of perivascular structures in ex-vivo vein specimens and in initial in-human pilot investigations.[51]

Published clinical data on HIFU for varicose veins, while still limited relative to the extensive evidence base supporting thermal ablation and sclerotherapy, have demonstrated encouraging early results. A prospective multicentre study evaluating HIFU treatment of great saphenous vein incompetence reported complete occlusion in approximately 80% of treated segments at three months, with maintained occlusion in around 70% of limbs at one year of follow-up assessed by duplex ultrasonography.[46,47] Symptom improvement, as measured by validated disease-specific quality of life instruments, was statistically significant compared with baseline assessments, and the procedure was well tolerated with a low rate of significant adverse events. Skin bruising and mild transient discomfort at the treatment site were the most frequently reported side effects, reflecting the extracorporeal energy delivery mechanism. No cases of deep vein thrombosis, nerve injury, or skin burns were observed in the published series, consistent with the predicted favourable safety profile of the focused transcutaneous approach.[48]

However, several important limitations and outstanding questions constrain the current clinical positioning of HIFU. Occlusion rates in published studies, while promising, remain modestly inferior to those reported for optimised radiofrequency ablation and endovenous laser ablation, which consistently achieve greater than 90% anatomical success at one to three years in randomised controlled trials.[19,20] It takes around 30 to 60 minutes per limb, depending on vein length and calibre, and compares unfavourably with the procedural efficiency of endovenous treatment. The technique is additionally limited to veins within an accessible focal depth, generally up to 15-20 mm below the skin surface, so not good in obese patients or deeply situated GSV[49,50]

Ongoing and planned multicentre randomised trials comparing HIFU with RFA and EVLA will be essential to

establish whether the non-invasive procedural advantage translates into clinically meaningful benefits in patient-reported outcomes, recurrence rates, and health economic terms that justify its broader adoption. Reimbursement coverage for HIFU in varicose vein treatment remains limited across most healthcare systems pending the generation of this higher-level comparative evidence, and at present the technique should be regarded as an emerging investigational modality with genuine theoretical and preliminary clinical promise rather than a validated alternative to established first-line interventions.[51,52]

DISCUSSION

The management of varicose veins has undergone a profound transformation over the past two decades, characterised by the progressive displacement of conventional surgery by endovenous techniques that deliver equivalent or superior outcomes with reduced procedural morbidity and faster patient recovery. The cumulative evidence base from randomised controlled trials and systematic reviews now robustly supports endovenous thermal ablation as the first-line interventional treatment for truncal GSV and SSV incompetence, endorsed by professional society guidelines across vascular surgery, interventional radiology, and phlebology.[13,16,31]

The emergence of non-thermal non-tumescent techniques, particularly cyanoacrylate closure and mechanochemical ablation and HIFU, has extended the procedural toolkit available to clinicians and introduced new options for patients in whom tumescent anaesthesia is burdensome or for anatomical settings where the risks of thermal injury are heightened. While these techniques have not yet demonstrated superior long-term efficacy over established thermal methods, their favourable tolerability profile and expanding outcome data from prospective registries and randomised trials position them as legitimate alternatives that will likely capture an increasing share of the varicose vein treatment market as clinician experience accumulates and device refinement continues.[24-28]

Foam sclerotherapy retains a valuable and complementary role, particularly for tributary varices, small-calibre disease, and recurrent presentations, where its flexibility, low cost, and non-thermal mechanism offer advantages that procedural thermal techniques cannot readily replicate. The optimal sequencing and combination of therapeutic modalities in complex or recurrent presentations remains an active area of clinical investigation, and head-to-head comparative data from well-designed multicentre trials with long-term follow-up are needed to guide individualised treatment planning.[50]

CONCLUSION

Varicose veins represent a common, clinically significant, and progressive manifestation of chronic venous disease with a complex multifactorial pathophysiology. It is because of venous hypertension, valvular incompetence, and active venous wall remodelling. Contemporary management is guided by updated classification systems that facilitate precise clinical communication and longitudinal disease tracking. Duplex ultrasonography remains the diagnostic cornerstone, providing the anatomical and haemodynamic data necessary for individualised treatment planning. The therapeutic landscape has evolved substantially, with endovenous thermal ablation now established as the standard of care for truncal incompetence. Foam sclerotherapy is a complementary and expanding array of non-thermal, non-tumescent techniques that offer effective and patient-friendly alternatives. Future advances in pharmacological disease modification, artificial intelligence-assisted diagnosis, and personalised outcome-driven management strategies hold the promise of further improving outcomes.

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