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UNRAVELING CHRONIC VENOUS INSUFFICIENCY: CLINICAL PROFILES, VENOUS PATHOPHYSIOLOGY, AND THE ROLE OF ENDOVENOUS LASER ABLATION

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ABSTRACT

Introduction: Chronic venous insufficiency (CVI) is a progressive manifestation of chronic venous disease driven by sustained venous hypertension, commonly caused by superficial reflux and valvular incompetence. Endovenous laser ablation (EVLA) is a key minimally invasive treatment for symptomatic truncal reflux, particularly great saphenous vein (GSV) incompetence. This review synthesizes current evidence on clinical profile, CEAP classification, reflux anatomy, pathophysiology, and cardiometabolic burden in CVI patients undergoing EVLA.

Methods: This narrative–integrative review included recent evidence from observational studies, systematic reviews, clinical trials, and guidelines on CVI, CEAP classification, duplex ultrasound, GSV reflux, EVLA, and cardiometabolic risk.

Results: EVLA-treated CVI patients are commonly middle-aged or older, frequently female, and often have obesity, hypertension, dyslipidemia, or diabetes mellitus. Most candidates demonstrate primary, reflux-dominant superficial venous disease, especially GSV involvement, supporting EVLA as an anatomically and biologically appropriate intervention.

Discussion: Current evidence links CVI progression to aging, obesity-related venous hypertension, endothelial inflammation, and cardiometabolic dysfunction, while guidelines emphasize duplex mapping, CEAP staging, compression, and risk-factor optimization alongside ablation.[1,2,3,9,11,25]

Conclusion: EVLA-treated CVI represents a reflux-dominant superficial venous phenotype shaped by systemic cardiometabolic risk.

Keywords: Cardiometabolic Risk; CEAP; Chronic Venous Insufficiency; Endovenous Laser Ablation; Great Saphenous Vein

1 INTRODUCTION

Chronic venous insufficiency (CVI) represents the clinically significant spectrum of chronic venous disease, in which sustained venous hypertension produces lower-extremity manifestations including varicose veins, edema, hyperpigmentation, lipodermatosclerosis, and venous ulceration [1,11-14,30]. The pathophysiological basis of CVI is impaired venous return caused by venous valve incompetence, obstruction, calf-muscle pump dysfunction, or combined abnormalities [1,11-14,30]. Because the clinical spectrum ranges from mild venous manifestations to active ulceration, standardized reporting is required to support diagnosis, treatment selection, and comparison across studies [2-4,25].

The CEAP classification is the most widely applied framework for categorizing chronic venous disorders according to clinical class, etiology, anatomy, and pathophysiology [2-4,25]. The 2020 CEAP update refined documentation of recurrent varicosities, corona phlebectatica, and anatomic-pathophysiological descriptors, making it particularly relevant for studies of EVLA-treated cohorts in which reflux anatomy is central to patient selection [2-4,25]. In clinically oriented reviews, CEAP should be interpreted not merely as a descriptive table, but as a structured language that links presentation, reflux distribution, disease mechanism, and therapeutic rationale [2-4,25].

CVI is common in adult populations and carries a substantial clinical burden. Recent systematic and population-based evidence demonstrates wide global variation in chronic venous disease prevalence according to diagnostic definition, geographic setting, and method of ascertainment, while consistently identifying age, obesity, pregnancy-related factors, and prolonged postural exposure as relevant determinants of disease burden [6-10,32]. Importantly, recent Asian community-based evidence also confirms that chronic venous disorders represent a relevant regional health problem rather than an exclusively Western condition [32]. Thus, CVI should be interpreted as a prevalent and progressive vascular disorder with functional and quality-of-life consequences, not merely a cosmetic concern [1,6-9,32].

EVLA has become one of the preferred minimally invasive interventions for symptomatic superficial truncal reflux, particularly great saphenous vein (GSV) reflux, because it enables ultrasound-guided closure of an incompetent vein with limited tissue trauma [3,19,23,25,31]. Contemporary systematic reviews, long-term randomized follow-up, and practice guidelines support endovenous ablation as an effective treatment strategy that improves disease-specific outcomes in appropriately selected patients, although recurrence and post-procedural outcomes remain influenced by anatomy, technique, patient risk profile, and duration of follow-up [3-4,19-25,31]. EVLA is most appropriate when superficial reflux is the dominant hemodynamic lesion, whereas longer-term outcomes remain influenced by systemic cardiometabolic risk and adjunctive care [3-4,10,15,19-25,31,33].

This review synthesizes evidence on epidemiology, CEAP staging, duplex ultrasonography, GSV reflux, endothelial inflammation, obesity, cardiometabolic comorbidities, and EVLA outcomes. It examines EVLA-treated CVI as a clinical phenotype shaped by reflux-dominant superficial disease and systemic risk factors rather than solely as a procedural population [1-25].

2 MATERIAL AND METHODS

2.1 Review Design

This narrative-integrative literature review was developed to provide a clinically oriented synthesis of CVI in patients considered for or treated with EVLA. This design was selected because the clinical relevance of EVLA cannot be interpreted through procedural outcomes alone; it requires integration of epidemiological patterns, CEAP-based

disease characterization, venous hemodynamics, duplex ultrasonographic findings, pathobiological mechanisms, cardiometabolic comorbidity burden, and therapeutic implications.

The methodological development of the review was informed by the Scale for the Assessment of Narrative Review Articles (SANRA). SANRA is a six-domain appraisal instrument that emphasizes the importance of the review question, explicit aims, transparent literature identification, appropriate referencing, scientific reasoning, and relevant presentation of data and clinical endpoints [5]. These principles were used to maintain a focused objective, prioritize clinically applicable evidence, and keep interpretation proportionate to the available literature.

2.2 Literature Identification

Relevant evidence was identified from PubMed-indexed literature and major evidence-based clinical guidance. Priority was given to original observational studies, randomized controlled trials, systematic reviews and meta-analyses, international clinical practice guidelines, consensus documents, and high-quality narrative reviews. Search concepts included chronic venous insufficiency, chronic venous disease, CEAP classification, superficial venous reflux, great saphenous vein, endovenous laser ablation, duplex ultrasonography, venous hypertension, endothelial dysfunction, obesity, hypertension, diabetes mellitus, dyslipidemia, compression therapy, and Venous Clinical Severity Score (VCSS). To emphasize contemporary evidence, references published from 2016 through 2026 were retained for the manuscript.

2.3 Evidence Selection and Synthesis

Evidence was considered relevant when it contributed to interpretation of CVI among patients potentially suitable for, or treated with, EVLA, particularly publications describing superficial truncal reflux, GSV incompetence, CEAP clinical staging, duplex-based assessment, associated cardiovascular or metabolic conditions, EVLA indications and outcomes, or adjunctive management. Publications focused exclusively on cosmetic management without meaningful clinical, hemodynamic, diagnostic, or therapeutic relevance were not prioritized. Because this was not designed as a formal systematic review or meta-analysis, no quantitative pooling was undertaken.

The evidence was synthesized thematically according to the clinical pathway of patients with CVI undergoing EVLA. Principal themes comprised demographic and cardiometabolic profile, CEAP-based clinical severity, anatomical and pathophysiological distribution of venous disease, the diagnostic role of duplex ultrasonography, mechanisms linking venous hypertension with inflammation and endothelial dysfunction, the procedural rationale for EVLA in superficial reflux-dominant disease, adjunctive management strategies, and remaining evidence gaps.

3 RESULTS AND DISCUSSION

3.1 Epidemiology and Risk Profile of CVI

CVI increases with age because venous valve degeneration, venous wall remodeling, and cumulative orthostatic exposure develop progressively over time [1,6-9,14,32]. Recent epidemiological syntheses and community-based studies consistently show that chronic venous disease is common in adult populations, although prevalence differs according to whether disease is defined by symptoms, visible varicosities, clinical examination, or duplex-confirmed reflux [6-9,32]. An EVLA cohort from a tertiary hospital therefore represents a selected symptomatic subset of a substantially larger community burden [6-9,32].

Sex differences in chronic venous disease are complex. Female predominance is frequently reported in clinical and international survey-based cohorts and may reflect pregnancy-related venous pressure, hormonal influences, symptom perception, and healthcare-seeking patterns, whereas variation across population studies indicates that sex alone cannot fully explain disease severity [6-9,32]. Female predominance in an EVLA-treated cohort should therefore be interpreted as a combination of biological susceptibility and referral or treatment-selection factors rather than as an isolated causal observation [6-9,32].

Obesity is among the most clinically relevant modifiable factors in CVI and EVLA care. Increased body mass index can increase intra-abdominal and lower-extremity venous pressure, impair venous return, worsen ambulatory venous hypertension, and promote inflammation that may contribute to edema and progressive disease severity [1,9-13,33]. Clinical evidence also indicates that obesity is associated with worse treatment outcomes in patients undergoing management for CVI, supporting weight optimization as part of peri-procedural and longitudinal care [10,33]. Obesity should therefore be considered a mechanistically and prognostically relevant modifier of disease burden and clinical outcome [1,9-13,33].

Hypertension, dyslipidemia, and diabetes mellitus are not primary causes of venous reflux in the same manner as valvular incompetence, but they may amplify vascular dysfunction through endothelial injury, inflammation, oxidative

stress, and impaired microcirculatory repair [11-13,15,33]. This shared vascular biology supports interpretation of CVI within a broader cardiometabolic-risk framework, particularly in cardiovascular and vascular medicine populations [11-13,15,33].

3.2 CEAP Classification As A Clinical and Research Framework

The CEAP system is essential for CVI-EVLA studies because it links visible clinical severity with disease origin, anatomical location, and hemodynamic mechanism [2-4,25]. In EVLA-treated cohorts, clinical classes C2 to C4 are commonly relevant because patients typically seek intervention when varicose veins, edema, discomfort, or skin changes become clinically meaningful [2-4,25]. C5 to C6 disease remains highly important because venous ulceration reflects advanced microcirculatory damage and is associated with increased treatment complexity and follow-up needs [1-2,4,25,30-31].

When C3 disease predominates in an EVLA cohort, the distribution is clinically meaningful rather than merely descriptive. Edema reflects sustained venous hypertension, fluid extravasation, and microcirculatory dysfunction, often indicating that patients seek treatment after disease has progressed beyond uncomplicated visible varicosities [1,11-14,30]. Interpreting CEAP distribution in this way links clinical class to symptom burden, delayed presentation, functional limitation, and possible differences in healthcare access or patient perception [2,6-9,32].

Etiologically, primary venous disease (Ep) commonly dominates EVLA populations because degenerative valvular incompetence within the superficial venous system is a principal indication for endovenous thermal ablation [3-4,19,23,25,31]. Secondary etiologies such as post-thrombotic obstruction remain clinically important but often require different diagnostic and therapeutic strategies, especially when deep venous disease contributes substantially to venous hypertension [3-4,25,31].

Pathophysiologically, reflux (Pr) is the central target of EVLA. Reflux represents retrograde venous flow due to valve incompetence and is evaluated through standardized duplex ultrasound assessment of the superficial and deep venous systems [3,16-18,25,31]. When reflux is the dominant mechanism and the anatomy is superficial truncal disease, EVLA addresses the primary hemodynamic abnormality by closing the incompetent vein [3,19,23-25,31].

3.3 Duplex Ultrasound and Anatomical Mapping Before EVLA

Duplex ultrasound is the primary diagnostic modality for evaluating lower-extremity venous reflux because it combines anatomical visualization with functional flow assessment [3,16-18,25,31]. Contemporary guidelines emphasize duplex scanning before intervention because treatment planning requires identification of the reflux source, junctional involvement, tributary or perforator disease, vein characteristics, and deep venous patency [3,16-18,25,31].

The GSV is the most frequently treated superficial truncal vein in EVLA practice. Contemporary diagnostic and interventional literature identified GSV incompetence as a major anatomical substrate of symptomatic superficial reflux and as a principal indication for catheter-based thermal ablation [3,16-19,23,25,31]. Predominance of GSV involvement in an EVLA cohort is clinically coherent because the anatomy is accessible, functionally relevant, and well suited to targeted reflux elimination [3,19,23-25,31].

Small saphenous vein, perforator, and deep venous abnormalities remain clinically relevant because they may indicate more complex reflux patterns, recurrence risk, or advanced disease [3,16-18,25,31]. Lower rates of deep venous or obstructive pathology in elective EVLA cohorts reflect treatment selection for patients whose dominant abnormality is amenable to superficial endovenous intervention rather than absence of clinical importance [3-4,25,31].

3.4 Pathophysiological Relevance: From Reflux to Inflammation

The central hemodynamic event in CVI is venous hypertension. Sustained venous pressure contributes to venular dilation, disturbed shear stress, capillary leakage, edema, and progressive microcirculatory dysfunction [1,11-14,30,33]. These hemodynamic alterations help explain progression from visible varicosities toward inflammatory skin changes and venous ulceration [1,11-14,30,33].

Endothelial dysfunction provides a mechanistic bridge between local reflux and progressive tissue injury. Current reviews and contemporary clinical research describe how venous hypertension is associated with endothelial activation, inflammatory mediator expression, extracellular matrix remodeling, and progressive valvular dysfunction [11-14,33]. These processes can establish a self-perpetuating cycle in which venous hypertension promotes inflammation and structural deterioration, which in turn aggravate reflux and clinical progression [11-14,33].

Cardiovascular-oriented reviews place venous disease within a broader systemic context. CVI and cardiovascular comorbidities may coexist through overlapping mechanisms involving congestion, endothelial dysfunction,

inflammatory activation, and hemodynamic vulnerability, particularly in patients with heart failure or substantial cardiometabolic burden [15]. This framework is clinically useful when interpreting CVI cohorts with frequent hypertension, diabetes mellitus, dyslipidemia, obesity, or cardiac disease [10-15,33].

This pathophysiological cascade helps clarify why EVLA is rational in superficial reflux-dominant disease. By eliminating axial superficial reflux, EVLA targets a major hemodynamic driver of ambulatory venous hypertension and can contribute to improvement in symptoms and disease-specific outcomes [3-4,19-25,31]. Nevertheless, because systemic inflammatory and cardiometabolic risk may persist after vein closure, EVLA should be integrated with compression, physical activity, weight management, and vascular-risk optimization [4,25,28-31,33].

3.5 EVLA: Mechanism, Evidence, and Fit with the CVI Phenotype

EVLA is an ultrasound-guided endovenous thermal ablation technique in which laser energy induces heat-mediated endothelial injury, vein-wall contraction, and subsequent fibrotic occlusion of an incompetent vein [19]. Its procedural rationale is strongest when the reflux source is a superficial truncal vein, especially the GSV, because the diseased axial segment can be treated under imaging guidance with limited surgical trauma [3,19,23-25,31].

Contemporary evidence syntheses and long-term randomized follow-up support EVLA as an effective treatment option for GSV incompetence and symptomatic superficial truncal reflux. Systematic reviews of randomized trials and long-term comparative evidence indicate clinically meaningful anatomical and quality-of-life outcomes after endovenous management, while recent guideline documents position ablation as a central intervention for appropriate superficial reflux disease [19-25,31]. Differences in recurrence, pain, complications, and anatomical closure remain influenced by comparator treatment, technique, device generation, and follow-up duration [19-24].

Current international guidelines support endovenous ablation as a major treatment option for symptomatic superficial truncal reflux [3-4,25,31]. This evidence supports the clinical coherence of EVLA cohorts dominated by primary reflux and GSV involvement because that phenotype closely corresponds to the population for whom EVLA is anatomically and therapeutically appropriate [3-4,19,23,25,31].

EVLA is effective and minimally invasive, but outcomes depend on venous anatomy, procedural technique, adjunct treatment of tributaries, compression adherence, and follow-up duration [19-25,31]. Recent comparative evidence indicates broadly similar efficacy between endovenous thermal modalities for truncal reflux, although specific complication and recurrence profiles may differ across techniques and study settings [24].

3.6 Cardiovascular and Metabolic Comorbidities in EVLA-treated CVI

A high prevalence of hypertension in a CVI-EVLA cohort should be considered within a shared vascular-dysfunction framework rather than treated as a coincidental observation. Hypertension may coexist with endothelial dysfunction, vascular stiffness, and inflammatory activation, which overlap with mechanisms implicated in chronic venous disease [11-13,15,33]. Although arterial hypertension is distinct from venous hypertension, coexistence of arterial and venous vascular disease may identify patients with broader circulatory vulnerability [11-13,15,33].

Dyslipidemia and diabetes mellitus may further impair endothelial biology and microvascular repair. Diabetes mellitus is associated with oxidative stress, impaired tissue repair, and microvascular dysfunction, which are clinically relevant in advanced CEAP classes and ulcer risk [11-13,30,33]. Dyslipidemia may contribute to systemic inflammatory and endothelial abnormalities, supporting cardiometabolic assessment in patients with CVI undergoing EVLA [11-13,33].

Obesity warrants specific emphasis because it can increase lower-extremity venous pressure while also contributing to chronic low-grade inflammation [1,9-13,33]. In EVLA-treated populations, obesity may therefore operate as a dual-mechanism amplifier: it can worsen reflux-related hemodynamic burden and may adversely affect treatment outcomes or longer-term symptom improvement [10,33].

Renal disease, anemia, and heart failure, when present, may complicate interpretation of lower-limb edema. Swelling in patients with CVI may arise from venous reflux, while systemic contributors such as cardiac failure, renal impairment, hypoalbuminemia, or medication effects may coexist [1,15,30]. Duplex-confirmed reflux is therefore important when attributing symptoms to venous disease, while systemic comorbidities may influence treatment planning and follow-up [3,16-18,25,31].

3.7 Clinical Implications for Patient Care and Outcome Assessment

The principal clinical implication is that patients with CVI undergoing EVLA should be assessed both as procedural candidates and as individuals with vascular-risk factors. Before EVLA, clinicians should document CEAP class, duplex reflux mapping, vein anatomy, deep venous patency, symptom burden, BMI, cardiovascular comorbidities, and relevant

treatment context [2-4,16-18,25,31]. This integrated assessment connects descriptive patient characteristics with clinical decision-making rather than treating them as isolated frequencies.

CEAP is primarily a descriptive classification, whereas the VCSS and disease-specific quality-of-life instruments can help quantify clinical burden and changes after treatment [2-4,25-27,31]. Incorporating VCSS, patient-reported symptoms, quality-of-life instruments, ultrasound-confirmed occlusion, recurrence, and complications can substantially increase the scientific value of future CVI-EVLA studies [19-27,31].

Adjunct management remains important after EVLA. Compression therapy, leg elevation, exercise, weight reduction, and management of cardiometabolic risk factors remain relevant because closure of an incompetent vein does not eliminate all contributors to chronic venous disease progression or recurrence [4,25,28-31,33]. Venoactive drugs such as micronized purified flavonoid fraction may improve selected symptoms, edema, and quality of life in chronic venous disease, but should be considered adjuncts rather than substitutes for treatment of clinically significant truncal reflux [28-29].

Taken together, an older age profile, female predominance, obesity, and GSV reflux identify a treatable superficial-reflux phenotype embedded within systemic vascular and metabolic risk rather than a collection of unrelated baseline characteristics [6-10,16-19,23,25,32-33].

3.8 Conceptual Framework

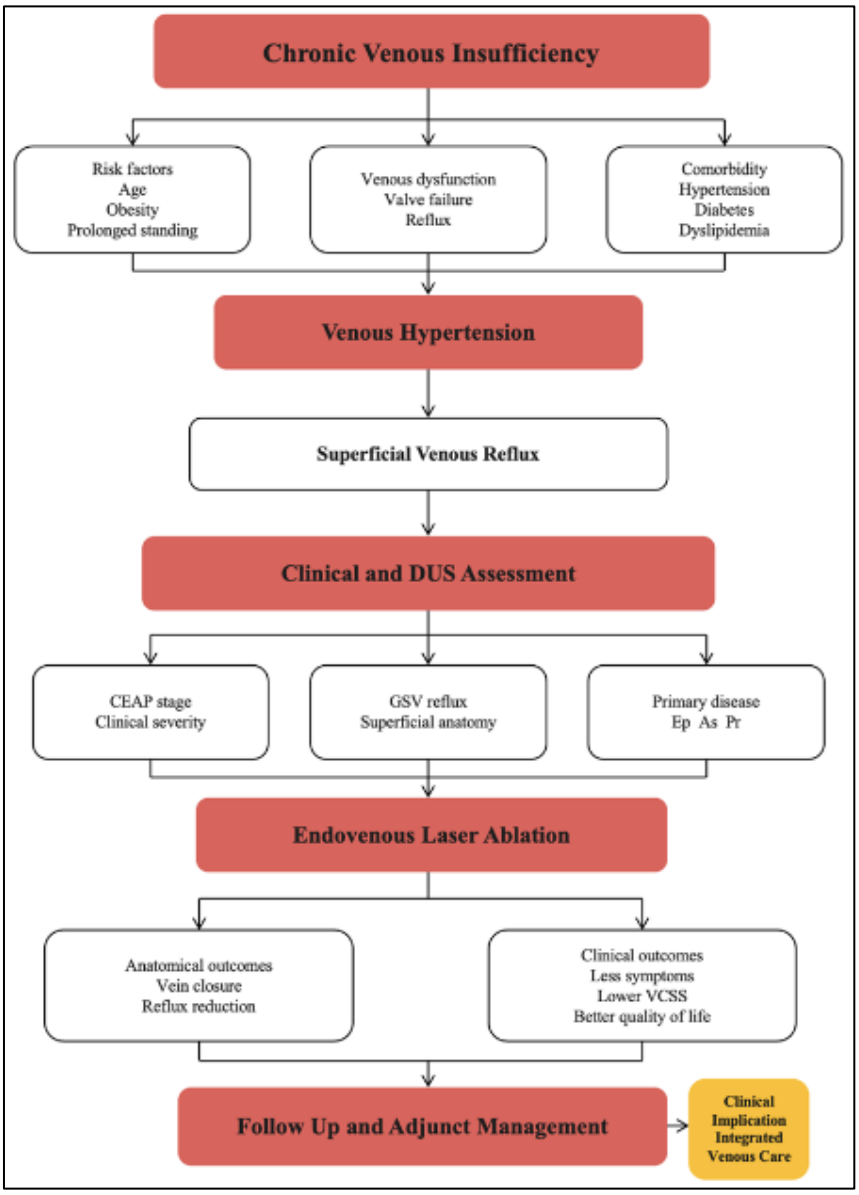

Figure 1: Relationship between chronic venous insufficiency risk profile, reflux-dominant disease trait, pathophysiological progression, and rationale for endovenous laser ablation

Patient-level risk factors and systemic comorbidities interact with venous wall weakness and valvular incompetence to produce chronic venous hypertension and superficial reflux [1,9-15,33]. This hemodynamic state manifests clinically as varicosities, edema, or skin changes and anatomically as superficial venous involvement, most often affecting the GSV [3,16-19,23,25,31]. EVLA fits this phenotype because it targets the refluxing superficial truncal vein, while optimal longer-term outcomes require adjunct management of obesity, compression adherence, physical activity, and cardiometabolic risk [4,25,28-31,33].

Table 1: Evidence Map Supporting the Literature Review

Theme	Main evidence base	How it strengthens the literature review	Key citations
CEAP and classification	CEAP 2020 update; SVS/AVF/AVLS and ESVS guidelines	Frames severity, etiology, anatomy, and reflux mechanism in a standardized language that directly informs treatment rationale.	[2-4,25]
Epidemiology and risk	Global pooled epidemiology; international survey; recent Asian community evidence	Demonstrates that CVD is common and clinically relevant, with regional variation in burden and risk profile.	[6-9,32]
Obesity and severity	Clinical studies of BMI, treatment outcomes, and inflammatory burden	Positions obesity as both a mechanical amplifier of venous hypertension and an inflammatory/outcome-modifying factor.	[9,10,33]
Pathophysiology	Contemporary reviews of venous hypertension, endothelial dysfunction, inflammation, and progression	Links reflux to edema, tissue injury, inflammation, and advanced clinical manifestations.	[1,11-15,30,33]
Diagnostic work-up	Contemporary duplex reviews and international guidelines	Supports duplex ultrasound before EVLA to confirm reflux anatomy, guide intervention, and evaluate deep venous involvement.	[3,16-18,25]
EVLA effectiveness	EVLA review; systematic reviews; long-term randomized follow-up; contemporary guidelines	Supports EVLA as an evidence-based intervention for symptomatic superficial truncal reflux, especially GSV disease.	[3,4,19-25,31]
Outcome measurement	VCSS and quality-of-life studies; guideline recommendations	Strengthens future local studies through standardized assessment of severity, patient-reported burden, and treatment response.	[3,25-27,31]
Adjunct therapy	Guidelines and evidence on compression, risk modification, and venoactive therapy	Emphasizes that procedural closure should be integrated with conservative and cardiometabolic management.	[4,25,28-31,33]

4 CONCLUSION

CVI in patients considered for or treated with EVLA is best understood as a clinically meaningful phenotype characterized by symptomatic superficial reflux, frequent GSV involvement, primary venous disease, and a relevant cardiometabolic burden. CEAP classification provides standardized clinical staging, duplex ultrasonography defines reflux anatomy, and pathophysiology links sustained venous hypertension to inflammation, edema, tissue injury, and disease progression.

EVLA directly addresses the dominant superficial hemodynamic lesion, but durable care extends beyond vein closure. Compression, physical activity, weight management, cardiometabolic risk optimization, and structured follow-up remain important. Future studies should link baseline phenotype to post-EVLA VCSS, quality of life, duplex-confirmed occlusion, recurrence, complications, and longer-term outcomes, particularly in Southeast Asian populations.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of Conflict of Interest

The authors declare that there is no conflict of interest.

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