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SPECIAL REPORT

Consensus Statement on the Management of Nonthrombotic Iliac Vein Lesions From the VIVA Foundation, the American Venous Forum, and the American Vein and Lymphatic Society

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ABSTRACT: A nonthrombotic iliac vein lesion is defined as the extrinsic compression of the iliac vein. Symptoms of lower extremity chronic venous insufficiency or pelvic venous disease can develop secondary to nonthrombotic iliac vein lesion. Anatomic compression has been observed in both symptomatic and asymptomatic patients. Causative factors that lead to symptomatic manifestations remain unclear. To provide guidance for providers treating patients with nonthrombotic iliac vein lesion, the VIVA Foundation convened a multidisciplinary group of leaders in venous disease management with representatives from the American Venous Forum and the American Vein and Lymphatic Society. Consensus statements regarding nonthrombotic iliac vein lesions were drafted by the participants to address patient selection, imaging for diagnosis, technical considerations for stent placement, postprocedure management, and future research/educational needs.

Key Words: disease management ■ iliac vein ■ lower extremity ■ stents ■ venous insufficiency

A nonthrombotic iliac vein lesion (NIVL) is defined by extrinsic compression of the iliac vein, most typically occurring between arterial structures and the vertebral body of the spine. This compression results in intrinsic venous luminal stenosis (Figure 1A), characterized by vessel wall fibrosis and intraluminal webs or spurs.[1,2] Although comprehensive population-based prevalence studies are lacking, smaller computed tomography (CT)-based investigations have reported anatomic compression in up to 70% of the asymptomatic population.[3,4] Symptoms of lower extremity chronic venous insufficiency or pelvic venous disease can develop secondary to NIVL. Factors that determine symptomatic manifestations of anatomic compression remain unclear. Symptoms may present along a spectrum, including asymmetrical edema, pain (manifested when walking or standing for extended periods of time), secondary varicose veins, and venous ulcerations.[5] Prevalence estimates from single-center studies suggest that NIVL occurs in 53% to 87% of patients with Clinical-Etiology-Anatomy-Pathophysiology class 4 to 6 venous disease.[6,7] Thus, patient selection based on symptoms is a key factor, given that anatomic compression has been observed in both symptomatic and asymptomatic patients.

Venous duplex ultrasound, insufficiency (reflux) examinations, and axial imaging are most commonly used to assess for the presence of a NIVL. Venography and intravascular ultrasound (IVUS) are the mainstays for endovascular assessment of NIVL and planning before stent placement.[8] IVUS has become the primary modality by which NIVLs are evaluated and an important tool for the evaluation of lesion severity, as well as an adjunct

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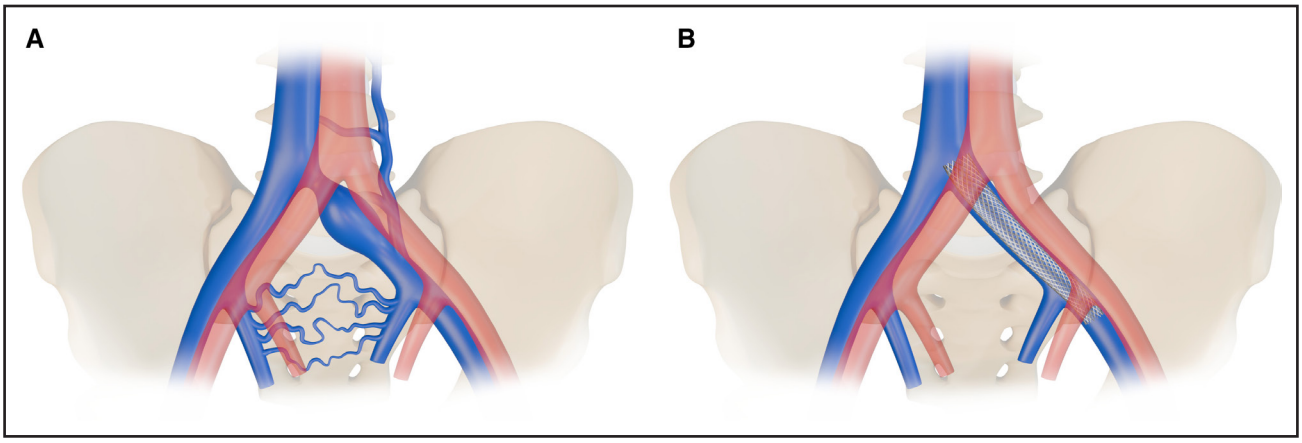

Figure 1. Schematic rendering of nonthrombotic iliac vein lesion causes.
A, Compression of the left common iliac vein by the left common iliac artery with collateral formation; and **B**, poststent placement.

to endovascular intervention.[9] In appropriately selected patients with moderate or severe symptoms, stent placement (Figure 1B) can result in improved pain, swelling, quality of life (QOL), and, when present, the healing of venous stasis ulcers.[5] Stent patency is well preserved in the majority of cases, with a low incidence of clinically driven need for reintervention.[10] However, inappropriate stent placement or inappropriate stent sizing can result in undesired outcomes, such as a lack of symptom improvement or stent migration.

To provide guidance for providers treating patients with NIVL, the VIVA Foundation convened a multidisciplinary group of leaders in venous disease management with representatives from the American Venous Forum and the American Vein and Lymphatic Society. The consensus statements published here were drafted by the participants and reflect the agreement of at least 80% of participants regarding patient selection for treatment, imaging considerations for diagnosis, technical considerations for stent placement, optimal postprocedure medical therapy and surveillance, and future directions in research and education.

PATIENT SELECTION FOR NIVL STENT PLACEMENT

Consensus recommendations:

1. Stent placement for NIVL may be appropriate in the presence of asymmetrical edema significantly affecting QOL, after excluding other systemic causes of edema and primary lymphedema.
2. Stent placement for NIVL may be appropriate in the presence of progressive Clinical-Etiology-Anatomy-Pathophysiology class 4 to 6 venous disease or venous claudication with minimal superficial venous disease or following previous treatment of underlying superficial venous reflux.
3. Stent placement for NIVL is inappropriate in patients with minimal to no symptoms.
4. Prophylactic stent placement for NIVL is inappropriate in asymptomatic patients to prevent possible future venous thromboembolism events.
5. Stent placement for NIVL may have a role in some cases with QOL-impacting chronic pelvic pain (CPP) of venous origin in the presence of dilated parauterine veins with or without pelvic venous reflux.

NIVL typically leads to asymmetrical swelling and seldom presents with symmetrical bilateral edema. Rarely, asymptomatic compression may be present bilaterally and at the iliac confluence.[5] Bilateral edema, when encountered, is generally attributable to factors such as medications (ie, calcium channel blockers), lymphedema, bilateral superficial venous reflux, or other systemic causes (Table).[11–13] Before intervening on a NIVL, it is critical to evaluate and exclude other potential causes of bilateral edema. Significant edema extending to the thigh that affects QOL may warrant intervention, whereas limited ankle edema may not warrant intervention, and other potential etiologies should be investigated.

The treatment of NIVL has primarily relied on data derived from single-center cohort studies and investigational device exemption studies. Studies evaluating iliac vein stent placement in investigational device exemption trials have demonstrated sustained improvements in outcomes, including Venous Clinical Severity Score and QOL, for the NIVL population that are comparable to those observed in the postthrombotic syndrome population.[14] However, there is significant heterogeneity among studies, including inclusion/exclusion criteria and outcomes reporting/assessment. These inherent limitations serve as a cautionary reminder against relying exclusively on these trials for guiding patient selection.

Indirect evidence suggests the potential benefits of stent placement for NIVL in patients with venous ulcers. A 2020 meta-analysis, encompassing both retrospective and prospective studies, compared standard medical therapy (a variable combination of compression therapy

Table. Other Causes of Lower Extremity Edema[11]

Primary cause	Mechanism of action	Unilateral	Bilateral
Cardiac: right heart failure	Increased central venous hypertension leading to increased capillary permeability and an increase in plasma volume		X
Biventricular failure			X
Heart failure with preserved ejection fraction			X
Hepatic	Decreased protein synthesis and decreased plasma oncotic pressure leading to increased systemic venous hypertension and capillary permeability		X
Renal	Increased protein loss leading to decreased plasma oncotic pressure and increased plasma volume through sodium/water retention		X
Thyroid and adrenal disorders	Abnormal water excretion and hyponatremia		X
Obstructive sleep apnea	Increase in pulmonary vascular resistance, pulmonary hypertension, and resultant capillary hydrostatic pressure		X
Allergic cause: angioedema and urticaria	Increased capillary permeability		X
Malabsorption and malnutrition	Decreased protein synthesis and decreased plasma oncotic pressure		X
Pregnancy related	Increased plasma volume		X
Premenstrual edema	Increased plasma volume		X
Idiopathic	Unknown		X
Drugs: calcium channel blockers, vasodilators, NSAIDS, antiepileptics, antidepressants, antipsychotics, hormone therapy, corticosteroids, alpha adrenergic blockers, chemotherapy, thiazolidinediones	Various mechanisms including increased capillary permeability from vasodilation, increased plasma volume by sodium/water retention, and increased capillary permeability		X
Lipedema	Adipose tissue accumulation		X
Lymphedema*	Excessive accumulation of lymphatic fluid. This chronic and advancing buildup of protein-rich fluid in the interstitial and fibro-adipose tissues surpasses the lymphatic system's ability to effectively transport this fluid	X	X
Chronic venous insufficiency*	Increased venous hypertension and capillary permeability	X	X
IVC or iliac vein obstruction/deep vein thrombosis/superficial vein thrombosis*	Increased venous hypertension and capillary permeability	X	X
Cellulitis*	Increase capillary permeability	X	X
Complex regional syndrome*	Increased capillary permeability is mediated by neurogenic/proinflammatory cytokines	X	X
Tumor/mass/radiation therapy*	Increase local venous hypertension	X	X
Veno-venous or lympho-venous malformations	Increased venous hypertension and capillary permeability	X	
Compartment syndrome	Local venous hypertension resulting in increased capillary permeability	X	
Ruptured baker's cyst	Extravascular fluid accumulation and increased capillary permeability	X	
Ruptured calf muscle/intramuscular hematoma	Extravasation of blood and inflammation-related increased capillary permeability	X	

IVC indicates inferior vena cava; and NSAIDs, nonsteroidal anti-inflammatory drugs.
*These conditions can present as unilateral or bilateral edema based on the underlying pathology.

and anticoagulation) to endovascular revascularization with stent placement for iliac vein obstruction.[15] This analysis revealed a 62% ulcer healing rate (mean healing time: 3 months) and a 10% recurrence rate for standard medical therapy. In contrast, stent placement exhibited a higher healing rate of 76% (mean healing time: 2.2 months) and a lower medical therapy recurrence rate of 2%. It should be noted, however, that this cohort included both NIVL and thrombotic etiologies. Stent placement for NIVL may be of value in patients presenting with C4-6 disease, specifically in patients experiencing lifestyle-limiting venous stasis symptoms where there is minimal superficial venous reflux or persistent symptoms despite prior treatment for superficial venous reflux.

NIVL has been associated with symptoms beyond lower extremity venous stasis. A single retrospective study demonstrated the presence of NIVL in a significant number of patients who had cryptogenic strokes due to a patent foramen ovale.[16] However, the optimal management of this association is uncertain. More commonly, CPP has been associated with NIVL. CPP

impacts up to 26% of women worldwide at some point during their lives. The cause of CPP may be a nongynecologic pathogenesis in up to 80% of patients,[17] and venous causes (formerly termed pelvic congestion syndrome) may account for nearly a third of cases.[18] CPP from pelvic venous disease can be caused by reflux (gonadal or internal iliac vein), compression (left renal vein or left common iliac vein), or a combination of reflux and obstruction.[19,20] The specific role of NIVL, when present, is of growing clinical interest, where it is postulated that it may result in increased pressure in the pelvic reservoir as a primary or secondary cause of CPP.[21,22] A single-center retrospective review of 271 women presenting with CPP and pelvic venous disease found that patients with a combination of gonadal vein reflux and NIVL experienced improved symptom relief with either simultaneous or staged iliac vein stent placement and ovarian vein embolization relative to ovarian vein embolization alone.[23] Smaller single-center series have likewise shown improvement in CPP with NIVL treatment.[21,24] However, the indeterminate causative role of reflux and obstruction, when both are present, has not been fully characterized and requires further study to determine optimal treatment strategies.

IMAGING CONSIDERATIONS FOR NIVL DIAGNOSIS

Consensus recommendations:

1. In a patient considered for NIVL treatment, an invasive diagnosis with the complementary use of venography and IVUS is recommended.
2. Dynamic IVUS evaluation of NIVL is recommended; this includes breath hold and maneuvers that increase intra-abdominal pressure. Fixed lesions are more likely to be pathological, whereas dynamic compressions vary with such maneuvers and are less likely to be pathological.
3. Using thresholds of >50% area reduction or >61% diameter stenosis on IVUS at the NIVL is correlated with symptom improvement following venous stent placement. Intervention below the stated thresholds is not recommended.
4. The use of venography thresholds alone for the diagnosis and treatment of NIVL is less well established.
5. Axial imaging with CT or magnetic resonance imaging can help confirm the presence of anatomy that may be associated with a clinically significant NIVL. The final diagnosis and intention to treat, however, are based on clinical evaluation and venography/IVUS.

The use of IVUS for NIVL intervention has become commonplace, primarily for its greater sensitivity for venous pathology, particularly compression, over 2-dimensional/multiplanar venography.[5] The limitations of venography are most notable in the anterior-posterior projection, where detection of lesions is limited on single-plane venography.[25] A study of 345 consecutive limbs with suspected NIVL demonstrated that venography underestimated the median degree of stenosis by 30% in comparison with IVUS. In this study, the sensitivity of venography in comparison with IVUS for detection of a stenosis >70% was only 45%.[26] Similarly, in the prospective VIDIO study (Venogram Versus IVUS for Diagnosing Iliac Vein Obstruction), IVUS identified 30% more stenotic lesions of 50% severity or greater compared with venography alone.[27] IVUS is also sensitive for detecting vessel wall features, including mural thickening, residual thrombus, synechia, trabeculation, and nonfunctional valves. Dynamic lesions, where the severity of stenosis may vary with factors that include hydration, respiratory phasicity, and variation in intra-abdominal pressure, may not warrant routine treatment, and caution should be applied before stent placement.[28] Further study is needed to determine the impact of identifying these features on intraprocedural decision-making.[28]

The definitive threshold for treatment to improve symptoms among patients with NIVL remains an area of ongoing debate and investigation. Historically, an area reduction of >50% at a NIVL has been applied as a metric for patient selection.[5] The VIDIO study demonstrated that among 48 patients with NIVL, an IVUS diameter reduction of >61% at the lesion was significantly predictive of clinical success; area as a metric was not found to be predictive (Figure 2).[29] An isolated measurement alone is often not sufficient to predict clinical improvement. In this cohort, among 68 patients undergoing venous intervention for advanced chronic venous disease, including both NIVL and postthrombotic etiologies, a preintervention cross-sectional area reduction of >54% by IVUS best predicted clinical improvement. Thus, further investigation is needed to determine optimal measurement methods and treatment thresholds. Furthermore, the effects of hydration and patient positioning need to be further studied. Fixed lesions are not affected by patients' breathing or position; however, we caution against establishing a diagnosis of NIVL in dynamic lesions.

CT venography has previously shown value in the detection of thrombus within abdominal/pelvic venous structures.[30] However, there are no large studies that specifically correlate anatomic features/metrics of NIVL on CT or magnetic resonance imaging with the presence of venous stasis symptoms. A retrospective series of 50 asymptomatic patients found that 24% had >50% diameter compression of the left iliac vein on CT.[3] MR venography demonstrated 90% sensitivity in a retrospective series of 28 patients with NIVL; however, its use is limited by its availability, patient tolerance, and potential for artifacts.[31] Venous duplex ultrasonography

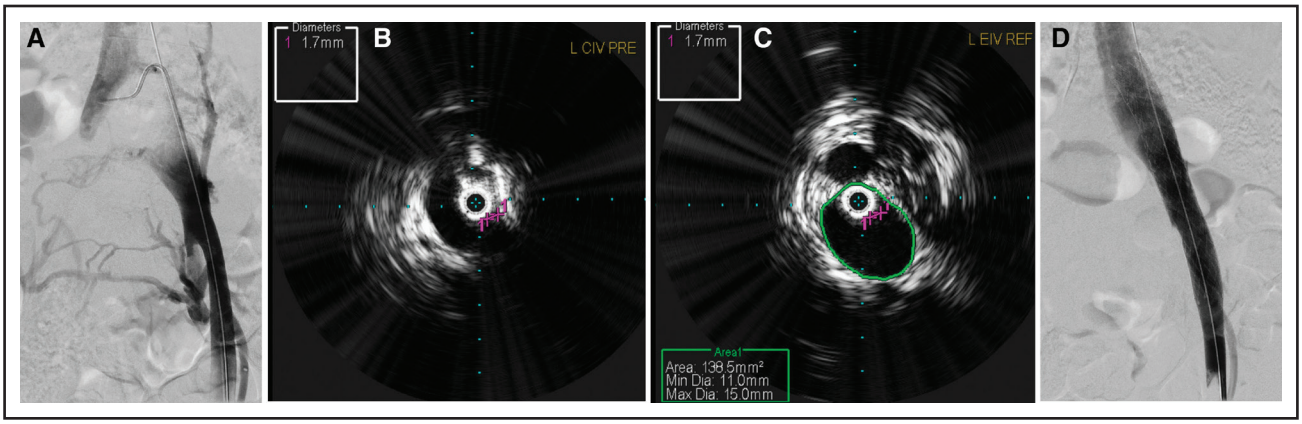

Figure 2. Venographic and intravascular ultrasound (IVUS) images from a nonthrombotic iliac vein lesion (NIVL) treatment procedure.

A, Digital subtraction left external iliac venography demonstrating left common iliac vein lesion with ascending lumbar and cross-pelvic collateral drainage; this patient has no prior history of deep vein thrombosis. **B**, IVUS image demonstrating a left common iliac vein compression lesion caused by the right common iliac artery and underlying vertebral body; purple cursors on the image reflect the minimum diameter at the lesion. **C**, IVUS image demonstrating lumen measurement at the selected reference vessel (left external iliac vein). This demonstrates a >61% diameter stenosis (average of the reference relative to the minimum diameter at the compression lesion relative to the minimum diameter measured in part **B**). Given the average diameter of 13 mm at the reference vessel, a 14 mm diameter stent size was selected. **D**, Digital subtraction left external iliac venography following placement of a 14×120 mm self-expanding venous stent.

may be an alternative method to diagnose NIVL noninvasively in centers with local expertise in this modality. While not predictive of symptoms, these data suggest that anatomic features of NIVL can be identified by axial imaging and may be of value in assessing for NIVL in the presence of symptoms.

TECHNICAL CONSIDERATIONS FOR NIVL STENT PLACEMENT

Consensus recommendations:

1. The choice of stent size and length in NIVL should depend on IVUS for diameter/length measurements with complementary fluoroscopy for length measurements.
2. Stent migration in NIVL can have devastating consequences. Measures to mitigate the possibility of stent migration and complications, including appropriate device diameter and length, are mandatory.
3. Although the approach to selecting stent diameter in NIVL is variable, sizing based on the normal reference vessel (eg, the external iliac vein) is generally recommended. In the presence of a significant compression, prestenotic dilation may be present and should not be used for sizing.
4. Stents for NIVL should be extended into the straight portion of the external iliac vein to limit stent migration and other complications.

Stent sizing differs based on design. Nitinol stents are more likely to reach their rated diameter compared with elgiloy stents. The final diameter of elgiloy stents is a function of the deployed length and adequate fixation at the ends of the stent. Length determinations can be aided with the use of marker catheters or markings on IVUS catheters during fluoroscopy. Measurement of vessel diameters is most accurate utilizing IVUS, as previously noted.

A literature review of 31 studies examining 54 instances of venous stent migration demonstrated a significant number of cardiopulmonary stent migrations.[32] Migration of stents to the heart occurred in 56% (n=30), and 24% (n=13) migrated to the pulmonary artery. The overall mortality rate in this cohort was 16.2% (n=6/37 with available mortality data). Notably, among the migrating stents with reported sizing information, 82.6% (n=38/46) were shorter than 60 mm, and none were longer than 100 mm. Furthermore, 44 of 47 migrating stents measured 14 mm or smaller in diameter. In a parallel observation of the Manufacturer and User Facility Device Experience database, the majority of reported migrating venous stents, spanning various manufacturers, were implanted for NIVL.[33]

Guidelines for stent sizing in investigational device exemption trials ranged from 1 to 4 mm oversizing compared with the normal reference vessel segment chosen. For example, in both the ABRE (Medtronic, Minneapolis, MN) and VIRTUS (Boston Scientific, Maple Grove, MN) clinical trials, stent sizing was established based on an operator-defined 2-mm oversizing compared with the normal reference vessel segment chosen.[14,34] The Zilver Vena trial (Cook Medical, Bloomington, IN) recommended oversizing ranging from 1 to 4 mm in comparison with the normal reference vessel.[35] Consistent with other investigational device exemption studies, Venovo's (Becton Dickinson, Tempe, AZ) instructions for use also proposed a 1- to 3-mm oversizing concerning the selected normal reference vessel.[36]

Pre- and poststent placement dilation to match the reference vessel was also recommended in these trials. Given the variability in approaches for sizing, we recommend following the manufacturer's instructions for use.

Evidence from a meta-analysis indicates a higher propensity for stent migration among stents shorter than 60 mm.[32] Although previous studies linked longer stent length with an increased risk of in-stent re-thrombosis and stent occlusion,[37] these findings were likely reflective of postthrombotic disease, and this risk may not apply to NIVL. In a subset analysis of 41 patients with NIVL from a recent investigation, patency was 98% at 6 months, independent of stent length.[38]

OPTIMAL MEDICAL THERAPY AND SURVEILLANCE FOR PATIENTS WITH NIVL

Consensus recommendations:

1. The routine use of anticoagulation or antiplatelet therapy for untreated NIVL is not supported.
2. In treated patients with NIVL with no evidence of previous venous thromboembolism (either by imaging or history), there is no consensus that anticoagulation or antiplatelet therapy is necessary.
3. An assessment of thrombotic risk in patients with NIVL should be made. If anticoagulation or antiplatelet therapy is indicated, the agent, dose, and duration should be tailored accordingly.
4. Routine early and long-term clinical surveillance, including imaging of patients with NIVL following stent placement, should be performed. Imaging to assess the stent is per practitioner preference (eg, ultrasound or axial imaging). Attention should be paid to stent-related adverse events such as migration and stenosis/thrombosis.

The incidence of iliac vein compression in an asymptomatic population has been estimated between 25% and 66%. One series reported that nearly 25% of asymptomatic patients evaluated for abdominal pain in the emergency department had >50% diameter compression and up to 66% had >25% diameter compression (correlating to 50% area stenosis); none had a history of prior deep vein thrombosis (DVT).[3] In 1 retrospective series of patients with acute iliofemoral DVT, 84% had evidence of iliac vein compression.[39] In another study, 65% of patients with a DVT and iliac compression had additional contributing risk factors for DVT development.[4] However, the presence of compression alone as a solitary risk factor has not been described; indeed, most patients with anatomic compression will never have a DVT. Thus, antithrombotic prophylaxis for anatomic compression alone is not currently warranted.

High patency rates have been achieved using a variety of antithrombotic approaches, including no anticoagulation, no antiplatelet, low-dose oral anticoagulants, and short-duration low molecular weight heparin. A 2018 Delphi consensus statement recommended anticoagulation during the first 6 to 12 months (low molecular weight heparin for the first 2–6 weeks and a direct oral anticoagulant thereafter) as the preferred treatment and concluded that there was no consensus for antiplatelet therapy.[40] More recent studies have concluded that anticoagulation and antiplatelet therapy are not needed in patients with NIVL, with cohort studies and editorials supporting this approach.[41,42] Therefore, it may be reasonable to limit or discontinue antithrombotic therapy following treatment for NIVL.

If stent-bearing patients with NIVL have other factors that increase thrombotic risk, these factors take precedence over the presence of a stent for NIVL.[43] Thrombotic risk factors may include inherited thrombotic disorders, active cancer, and chronic inflammatory conditions.

Appropriate patient selection and proper stent placement technique are integral to preventing potentially life-threatening events, such as stent migration.[32,44] Nonetheless, well-placed stents can have complications and failures in the short or long term. Imaging surveillance assesses these events and should be a part of ongoing clinical follow-up. The long-term patency of stents placed for NIVL ranges from 96% to 99%, as observed in multicenter cohorts.[14,34,45] While some studies suggest that extended long-term surveillance for patients with NIVL may not be necessary,[46] the lack of long-term data on the performance of dedicated venous stents argues for continued clinical and imaging surveillance.

FUTURE DIRECTIONS IN RESEARCH AND EDUCATION

Consensus recommendations:

1. Evidence-based appropriateness of treatment and longitudinal management of patients with NIVL should be supported by long-term prospective trials, to include the following:
   a. Outcomes focusing on patient QOL measures
   b. Appropriateness emphasizes patient selection, intervention technique, and postprocedure optimal medical therapy and surveillance.
2. Future directions in NIVL research include the establishment of consensus guidelines with multisocietal endorsement.
3. Directions in NIVL education include the dissemination of future appropriateness guidelines to providers treating NIVL and to referring practitioners as the standard of care through societal endorsement.
   a. A comprehensive evaluation of patients with NIVL includes expertise in other etiologies possibly contributing to patient symptoms. With limited venous exposure in current training paradigms, additional postgraduate training may be necessary.

b. In addition to societal endorsement, physicians should adhere to the standard of care and appropriate guidelines. Physicians must participate in tracking and reporting their quality outcomes.

The challenge of developing consensus documents for the treatment and management of patients with NIVL stems from the paucity of rigorous data supporting a specific treatment strategy.

Studies need to focus on defining who benefits the most from the treatment of NIVL and defining the determinants of treatment success. Some retrospective data suggest that endovascular therapy of NIVL is associated with benefits. However, other studies suggest that some patients show no improvement or show clinical deterioration following stent placement.[47] There is a dearth of research comparing medical therapy to endovascular intervention.[48–52] Determinants of success should include not only technical outcomes but also QOL measures. The focus on patient-centric outcomes is essential, but no consensus exists on which QOL instrument most accurately reflects the clinical benefit of NIVL treatment.[53] The choice of postintervention anticoagulation may not be associated with rates of restenosis, and the optimal regimen may need to be tailored to the patient.[43,54] Similarly, surveillance protocols need to be defined by exploring thresholds that contribute to restenosis and the need for intervention.

Specific research questions, such as intervention thresholds, optimal postoperative pharmacological strategies, and recommended surveillance intervals, will inform future guidelines documents. Additionally, the impact of NIVL on at-risk populations and underrepresented minority populations needs further study to optimize clinical outcomes. Ultimately, multi-specialty endorsement is essential for not only the dissemination of these recommendations but also the widespread acceptance required to mitigate the inconsistent management of patients with NIVL.

A comprehensive evaluation of patients with NIVL includes expertise not only in venous education but also in other etiologies possibly contributing to patient symptoms. With limited venous exposure in current training paradigms,[55,56] additional education for individuals treating NIVLs may be necessary. Physicians should adhere to evidence-based guidelines to ensure adherence to the standard of care. Furthermore, appropriateness of care requires accountability, starting with physicians tracking their quality outcomes.[57]

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