Chronic Venous Insufficiency

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Contents

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Abstract

The underpinning of chronic venous insufficiency (CVI) is valvular dysfunction, which manifests on a spectrum depending on the severity of insufficiency and duration of the disease. In older adults, special consideration must be taken during the patient encounter to account for age-related factors. In this chapter, we discuss the clinical presentation, diagnosis, and mimics of CVI, with a focus on older adults. The epidemiology, risk factors, disease burden, and grave complications—such as thrombosis and ulceration—are reviewed. The physiological impacts of CVI are described, providing the background for treatment strategies, including noninvasive, medical, and surgical therapies.

Keywords

Venous insufficiency · Venous disease · Venous ulcers · Peripheral edema · Lower extremity edema · Compression treatment · CVI · Valve dysfunction · Diagnosis · Treatment · Compression bandaging

1 Introduction

The intrinsic burden of chronic venous insufficiency (CVI) includes skin changes such as hyperpigmentation, eczema, and lipodermatosclerosis and also lower extremity edema, cramping, pain, and the development of varicose veins [1–4]. Additionally, and importantly, CVI is a very common precursor of venous leg ulcer development [5]. Such ulcers pose a considerable burden including pain [6–8] and difficulties in management [9–12].

A link between CVI and venous ulcers is well demonstrated in a study in which a CVI prevalence of 31.6% was found in patients who had CVI due to combined venous obstruction and superficial venous reflux, and who had either an active or a healed venous ulcer [13]. In that study, the most common independent predictor of the CVI was a history of deep venous thrombosis (DVT).

Conditions that influence the factors that contribute to CVI, such as reflux in superficial and perforating veins, increase with age [14] with the link between CVI and venous ulcers also increasing with advancing age [15]. Further, a 2015 study reported that about a third of patients develops CVI from an initial varicose vein and this rate increased as the patient aged [16]. Evidence also indicates that the prevalence of CVI is more common in Western Europe, the USA, and other industrialized nations than in undeveloped regions, perhaps influenced by lifestyle [17, 18]. In the USA, it is estimated that there are more than 25 million adults with CVI and 6 million individuals with advanced CVI [19].

In this chapter, multiple issues associated with CVI in the geriatric population will be discussed, starting with a description of the overall signs and symptoms of CVI and its clinical manifestations, followed by lower extremity conditions that may mimic various aspects of CVI. Various imaging

methods for diagnosing and evaluating CVI are discussed. The epidemiology and risk factors for CVI, pathophysiology in the geriatric population, and consideration of compression and other treatment modalities for CVI are detailed.

1.1 Epidemiology

The prevalence of CVI is variable depending on the population, geographical location, classification, and methodology used, and it is unclear if males or females are at a higher risk of CVI. Most studies report it to be more prevalent in women [16, 18–21] but at least one proposed that CVI is more prevalent in men [21]. Overall, estimated CVI prevalence varies from <1% to 40% in women and <1% to 17% in men [21]. It has been reported that CVI is more prevalent in women until the age of 45 but more prevalent in men over the age of 55; the prevalence in the 55–65 age group is higher in men (25.3%) than in women (12.3%) [22]. The Edinburgh Vein Study conducted in 2015 reported that approximately one in every three patients develops CVI from an initial varicose vein diagnosis, the rate increasing with age [16]. Further evidence also indicates that the prevalence of CVI varies by region and is more common in Western Europe, the USA, and other industrialized nations than in undeveloped regions, perhaps due to lifestyle [17, 18]. In the USA, it is estimated that there are more than 25 million adults with CVI and 6 million individuals with advanced CVI [19]. International CVI prevalence assessed in 99,359 persons was reported as 29.9%, 26.6%, 24.9%, and 19.8%, respectively, in Eastern Europe, Latin America, Western Europe, and Asia [23].

2 Clinical Presentation

CVI causes persistent venous hypertension in the lower extremities [24]; the signs and symptoms depend on the severity and duration of the pathology contributing to the disease. Chronic venous disease (CVD) represents a spectrum of conditions, ranging from benign spider veins to cutaneous changes and ulceration. The most common manifestations of CVD are telangiectasias (spider veins) and reticular veins, which are the initial presentations of the disease [21]. As vessels are caused to widen due to refluxed blood, they become more tortuous and enlarged, becoming varicose veins [25]. The disease often presents with pitting edema, initially at the malleoli and thereafter ascends the leg [20]. Major clinical features of CVI are dilated veins, edema, pain, or discomfort, along with skin changes. An example of skin changes is shown in Fig. 1 of a patient that has also has a VLU near his medial malleolus as a consequence of his CVI.

The most common symptom of CVI reported was leg discomfort, including a feeling of leg heaviness in 15–70% of patients and aching or cramping pain in 50% of patients [26–28]. This discomfort is aggravated with prolonged standing and alleviated with leg elevation due to the increased intra-compartmental pressure and subcutaneous volume in the leg. The areas along the varicose veins may become tender due to venous distention or may develop superficial thrombophlebitis—with painful, indurated, and inflamed areas along varicose veins. Further, painful venous claudication may occur with ambulation due to obstruction of the deep venous system [24]. Other symptoms include pruritus or paresthesia, in about 20% of patients [26].

As the edema leads to venous stasis, hemosiderin is deposited in the skin leading to skin changes: hyperpigmentation and eczematous dermatitis [25]. At more advanced stages, CVI may exhibit lipodermatosclerosis due to inflammation

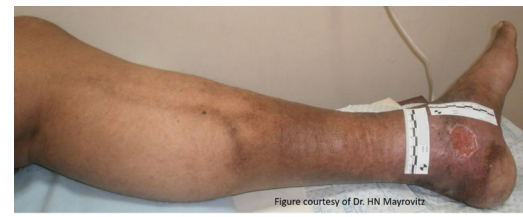

Fig. 1 Skin changes associated with chronic venous insufficiency (CVI). Skin hyperpigmentation in the area of the involved veins is shown together with a venous ulcer that developed as a consequence of the patient's CVI. (Figure courtesy of Dr. HN Mayrovitz)

of subcutaneous fat [29]; lipodermatosclerosis is a disorder where the skin, subcutaneous tissue, and deep fascia may become indurated and adherent [30]. Acutely, lipodermatosclerosis may present with a tender, erythematous indurated plaque, while subacute and chronic forms are non-tender and hyperpigmented [29, 30]. Systemic symptoms such as fever are usually absent [30]. The lower extremity may have an "inverted wine bottle" appearance due to a firm cuff of tissue surrounding the lower calves. Diffusion of fibrinogen into the dermis results in a woody texture [29]. At this stage, the appearance of previously healed ulcers may show stellate or porcelain-white scars surrounded by punctate telangiectasias, known as "atrophie blanche" (white atrophy) [30, 31]. Advanced CVI may contribute to developing lymphedema, and there is an increased risk of cellulitis, ulcer development, and poor wound healing [24]. A venous ulcer due to CVI is shown in Fig. 2 as it looked initially and then after healing from 12 weeks of treatment that included compression bandaging.

3 Diagnosis/Imaging

3.1 Clinical, Etiologic, Anatomic, and Pathophysiologic (CEAP) Classification

CVI is diagnosed based on history, clinical presentation, and diagnostic tests. The gold standard for classifying CVD (and CVI) is the CEAP (clinical, etiologic, anatomic, and pathophysiologic) classification [32]. A summary of the CEAP classification **is shown in** Table 1. CVI diagnosis requires a C3 or above presentation in the "C" of CEAP [20]. Noninvasive testing can categorize the etiology, anatomy, and pathophysiology ("EAP"), including reflux or obstruction [32, 33]. Duplex ultrasound is currently the gold standard, but other diagnostic tests may be helpful in particular circumstances [34]. Besides the clinical presentation ("C"), CEAP does not assess the severity of the disease but can be used to estimate the venous severity scores, which can be used to monitor changes in the patient's condition [35].

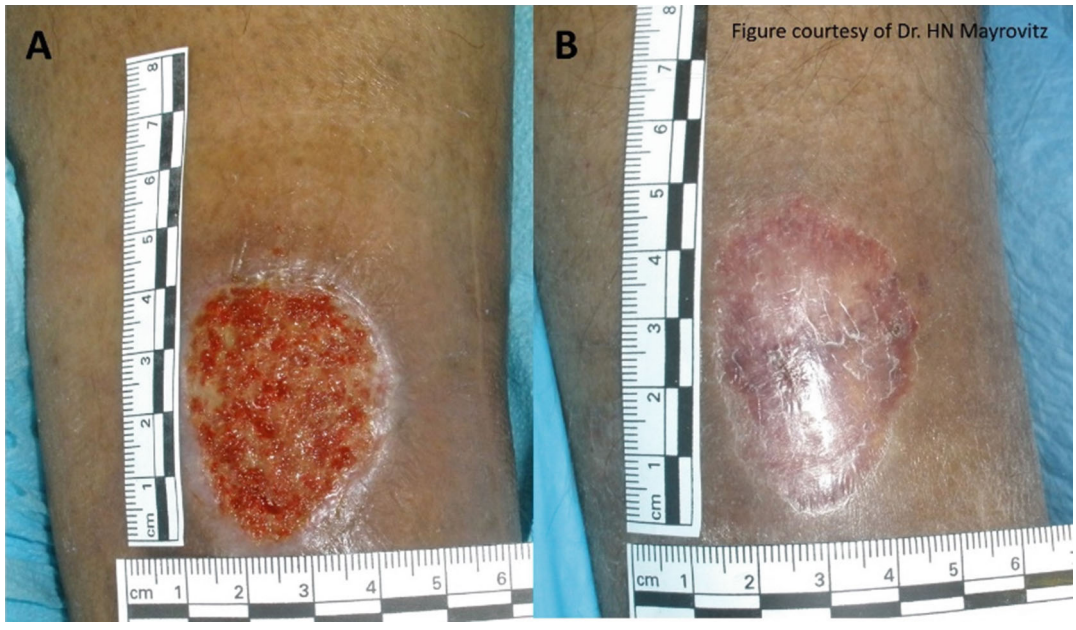

Fig. 2 Venous ulcer attributable to chronic venous insufficiency. (**a**) Initial ulcer after debridement showing granulation tissue. (**b**) After 12 weeks of treatment including compression bandaging. (The figure is courtesy of Dr. HN Mayrovitz)

Table 1 CEAP classification of chronic venous disease (CVD)

C		E		A		P	
Clinical[a]		*Etiology*		*Anatomy*		*Pathology*	
$\mathbf{C_0}$	No visible signs	$\mathbf{E_c}$	Congenital	$\mathbf{A_s}$	Superficial	$\mathbf{P_r}$	Reflux
$\mathbf{C_1}$	Spider veins, telangiectasias, or reticular veins[b]	$\mathbf{E_p}$	Primary	$\mathbf{A_d}$	Deep	$\mathbf{P_o}$	Obstruction and thrombosis
$\mathbf{C_2}$	Varicose veins without clinical signs of CVD[b]	$\mathbf{E_s}$	Secondary	$\mathbf{A_p}$	Perforator	$\mathbf{P_{r,o}}$	Reflux and obstruction
$\mathbf{C_3}$	Varicose veins with edema[c]	$\mathbf{E_n}$	Not identified	$\mathbf{A_n}$	Not identified	$\mathbf{P_n}$	Not identified
$\mathbf{C_{4a}}$	Varicose veins with trophic skin lesions[c]						
$\mathbf{C_{4b}}$	Pigmentation, purpura, and eczema[c]						
$\mathbf{C_5}$	Lipodermatosclerosis and atrophie blanche[c]						
$\mathbf{C_6}$	Healed venous ulcer[c]						

[a]The descriptor A (asymptomatic) or S (symptomatic is placed after the clinical class)
[b]Telangiectasias considered <3 mm diameter; varicose veins considered >3 mm diameter
cClinical class C_3 and above represents chronic venous insufficiency

3.2 Doppler Ultrasound

As previously stated, duplex ultrasound is the gold standard, noninvasive modality for assessing the structure and function of the venous system concerning CVI. Duplex ultrasound combines B-mode imaging of the deep and superficial veins with pulsed Doppler assessment of flow direction with provocative maneuvers to detect, localize, and evaluate valvular incompetence and chronic venous obstruction. The B-mode image allows veins to be visualized directly. At the same time, the Doppler signal is used to survey blood flow, including the presence and direction, by color flow imaging [33]. The absence or disturbance of flow due to chronic deep vein thrombosis or venous stenosis may be directly observed in the venous lumen or inferred from alteration in spontaneous flow characteristics [24].

The posterior tibial, popliteal, superficial femoral, common femoral, and greater saphenous vein should all be assessed via duplex ultrasound for patency and valvular competence [36]. Valvular competence and reflux can be evaluated in the reverse Trendelenburg position during a Valsalva [24]. But a rapid cuff inflation-deflation technique from the standing position is preferred as it more closely approximates physiological conditions [37, 38]. Reflux can be identified by inversion of the blood flow color pattern—blue flow toward the heart or red toward the periphery. Any significant flow toward the feet suggests reflux [24, 33]. The incompetency of a vein is determined via reflux time, with a duration of reflux greater than 0.5 s and 1.0 s for superficial and deep veins, respectively. A longer reflux time indicates a more severe disease, but this does not necessarily determine worse clinical manifestations [39, 40]. The diagnostic limitation of duplex ultrasound is that it cannot assess the pelvic veins and, depending on the patient's anatomy, may be unable to perceive the deep veins of the lower extremity [20].

3.3 CT or MRI

Computed tomography (CT) and magnetic resonance imaging (MRI) are well-standardized, reproducible, and noninvasive methods but may be less accessible in routine clinical practice [41]. However, they may provide valuable information for assessing the etiology and grading of CVI: They are superior in determining the proximal veins, such as the pelvic veins, for intrinsic obstruction or extrinsic compression and can detect changes in the skin, subcutaneous fat, muscles, tendons, periosteum, and bones [20, 41, 42].

These modalities may require intravenous (IV) contrast material, with appropriate timing of image acquisition, to obtain a venogram with better visualization to assess deeper venous structures, as well as obstructive disease, varicose veins, perforating veins, and other venous malformations [24]. Contrast may be contraindicated in some older adults with acute or chronic kidney disease. With spiral CT, large zones with multiple scans can be imaged quickly. The IV contrast administration can be timed through multiple scans so that different veins opacify depending on the blood flow and proximity to central veins allowing for sequential views [33]. In advanced CVI, osseous changes and soft tissue calcifications can be better visualized by CT. Also, fibrosis of the dermis in the lower legs can be seen as hyperdense condensation [41].

MRI provides multiplanar imaging and high spatial resolution to visualize soft tissue changes. In CVI, MRI is sensitive in detecting subcutaneous edema, lipodermatosclerosis, fatty muscle transformation, and periosteal ossification. On T1-weighted images, periosteal hyperostosis will be hyperintense, and subcutaneous fat will appear more hypointense due to fibrosis. Metaplastic calcifications in the cutaneous fat, if present, can be visualized. On T2-weighted images, edema appears hyperintense due to water content [41]. Magnetic resonance venography (MRV) can visualize the vascular system without needing nephrotoxic contrast media [43]. Compared to CT or duplex ultrasound, MRV is reported superior in determining overall thrombus burden in smaller branching veins and diagnosing pelvic vein thrombosis [33].

3.4 Dermoscopy

Dermoscopy, also known as epiluminescence, is a powerful diagnostic tool used in dermatological examinations. By magnifying and illuminating the skin, dermoscopy enhances the visualization of subcutaneous structures and reveals important morphological features that may not be visible to the naked eye. Within the vasculature of the dermal papillae and papillary dermis, the most important chromophore is hemoglobin, the pigmented protein of red blood cells [44]. At low magnification (x10), red globules and a scaly surface may be seen in stasis dermatitis [45]. However, at high magnification (x30), glomerular vessels—"large-caliber reddish dots formed by larger-caliber reddish dots formed by tortuous capillaries curled up into a ball or resembling the glomerular apparatus of the kidneys"—can be seen and may often be distributed in clusters or through the lesions [45, 46]. Histologically, this corresponds to a convolution of frequently dilated capillaries in the dermis [45]. Additionally, a rainbow pattern may also be seen. However, this can also be seen in other lesions, such as Kaposi sarcoma, dermatofibroma, pyogenic granuloma, melanoma, and lichen planus [47, 48].

4 Mimics and Masqueraders

Several conditions may mimic aspects of CVI. These include the presence of cellulitis, lymphedema, thyroid dermatopathy, volume overload edema, and lipoedema. A discussion follows.

4.1 Cellulitis

Cellulitis is a bacterial infection of the deep dermis and subcutaneous tissue that disrupts the skin barrier [31, 49]. Patients with cellulitis commonly present with the cardinal signs of inflammation: calor (warmth), rubor (erythema), tumor (edema), and dolor (pain). The inflammation develops over a few days [49–51]. More severe forms of cellulitis may present with blisters, hemorrhagic bullae, or pustules that may ulcerate [52]. Superficial edema may result in peau d'orange, a loss of normal skin wrinkling, giving an orange peel-like appearance [50, 53]. Systemic involvement corresponds to the severity of the infection. Mild signs and symptoms include fever and leukocytosis. While more severe symptoms, including tachycardia, hypotension, confusion, and sepsis, may appear before visible changes in the skin [51, 53]. Painful regional lymphadenopathy may also be present [50]. Laboratory findings that may be seen may include elevated white blood cell

counts (in 34–50% of patients), erythrocyte sedimentation rate (in 59–91%), and C-reactive protein (in 77–97%) [50, 54]. In older populations, pseudocellulitis, a condition mimicking cellulitis or stasis dermatitis, may occur [31, 55].

Risk factors for cellulitis include a history of cellulitis, obesity, diabetes, and prior saphenous venectomy [49, 52, 56]. However, venous insufficiency and lymphedema are two major risk factors for lower-extremity cellulitis. Venous insufficiency nearly doubles the odds of having cellulitis (odds ratio = 2.9 [1.0–8.7]) [57]. In these diseases, venous and lymphatic drainage are obstructed, preventing the clearing of microbes that have breached the skin. Thus, bacteria collect in the skin causing local infection and spread through superficial tissue [58].

Clinical assessment is the gold standard for diagnosing cellulitis [59, 60], a point requiring emphasis. Therefore, clinicians must be cognizant of pathognomonic features suggesting other diseases, essentially the differential diagnosis. Pseudocellulitis, especially in the older population, must be diagnosed promptly due to comorbidities, a higher risk for antibiotic-related adverse events, and multidrug-resistant organisms [61]. As cellulitis is usually unilateral, a bilateral presentation is more suggestive of venous insufficiency or stasis dermatitis [57, 58]. Chronic findings lasting over several months with waxing and waning, and a lack of systemic symptoms, likely refute a diagnosis of cellulitis [31, 57]. Nonetheless, a key symptom is pruritus—venous stasis dermatitis is more likely, especially if the itch eclipses the pain [58].

4.2 Lymphedema

Within the dermis, lymph capillaries drain fluid from the interstitium into the lymphatic vessels and ultimately back into the venous circulation via the thoracic duct [62]. Impairment of this system causes an accumulation of this protein-rich fluid within the tissue spaces [63–65]. Lymphedema is a chronic condition that can be managed but, as of now, is incurable [62, 66]. The clinical presentation includes chronic unilateral or bilateral edema, localized pain, atrophic skin changes, and in some cases, secondary infections [67, 68]. Lymphedema of the lower extremity usually arises distally and then progresses proximally. Edema of the dorsal foot and digits often gives a characteristic blunt "squared-off" appearance [69]. In advanced stages, overlying skin may appear orange peel-like (peau d'orange), verrucous, or lichenified (thickened and leathery) [70]. The swelling may produce the Kaposi-Stemmer sign, where the skin on the second digit's dorsal aspect cannot be pinched and folded. In more advanced stages, the skin over these areas has been described as "hyperkeratotic with verrucous cobblestone-like papules, plaques, and nodules with underlying woody fibrosis" [69].

Lymphedema can be attributed to primary or secondary etiologies. Primary lymphedema is due to developmental abnormalities of the lymphatic system, including the vessels (hypoplasia or hyperplasia), the valves, or the lymph nodes [71–73]. It may develop and present throughout the lifespan: congenital, praecox (around puberty), or tarda (as an adult) [64]. Secondary lymphedema involves impairment of the lymphatic system resulting from acquired conditions, including infection, trauma, tumor, radiation, or surgery. Infections of the lymphatic system are more common in developing countries. In developed countries, the most common cause is after the surgical removal of lymph nodes for cancer treatment [62].

The risk factors for lymphedema are common and often concomitant in the geriatric population: advanced age, obesity, sedentary lifestyle, lack of mobility, reduced joint range of motion, and muscle weakness lead to a higher risk of this disease and its progression. Older adults may develop lymphedema following lower extremity surgeries, such as total hip or knee replacement procedures, gynecological in women, and prostate surgery in men. If the patient also has CVI, prior to surgery, a fully functional lymphatic system helps compensate for the venous insufficiency effects. However, following the trauma of surgery and possibly radiation, the lymphatic system cannot keep up with the increased interstitial fluid load.

At this point, secondary lymphedema accelerates due to the combined lymphatic issues and the presence of the prior CVI [66]. Consequently, differentiating between lower extremity lymphedema and CVI may be difficult. Lymphedema can present with unilateral or bilateral edema, but swelling will typically be painless in the early stages, but is unlike CVI, which tends to be bilateral and improves overnight and with elevation [70].

Lymphedema is diagnosed through clinical assessment, although quantitative measures based on bioimpedance spectroscopy [74–76] and localized measures of tissue water using tissue dielectric constant values [77–82] are now available. In the geriatric population, a combination of lymphatic and non-lymphatic edema may exist. Non-lymphatic edema can be caused by renal insufficiency, cardiac conditions (congestive heart failure), hepatic diseases (increased intraportal pressure), thyroid disorders, hypoproteinemia (protein malnutrition), Cushing's syndrome (hyperadrenocorticism), and pulmonary conditions [63]. Thus, a thorough past medical history, palpation of the extremity, and ruling out of other causes are vital due to the added uncertainty and numerous compounding factors. A comprehensive medical history usually indicates past damage to the lymphatic system, such as lymph node dissection [68]. TDC values can be used to assess localized edema at almost any site due to any cause. A probe is shown in Fig. 3 measuring TDC in a 77-year-old woman with unilateral lymphedema caused by gynecologic cancer treatment. Her affected left leg volume was 1370 ml greater than the right. Figure 4 illustrates two bilateral cases, one in an 89-year-old woman with CVI and one of a 71-year-old-woman with bilateral lymphedema.

4.3 Thyroid Dermatopathy

A potential source of leg edema is thyroid dysfunction, common in the geriatric age group, ranging from clinical to subclinical hypothyroidism and hyperthyroidism [83]. Hypothyroidism is more common in the geriatric population, in

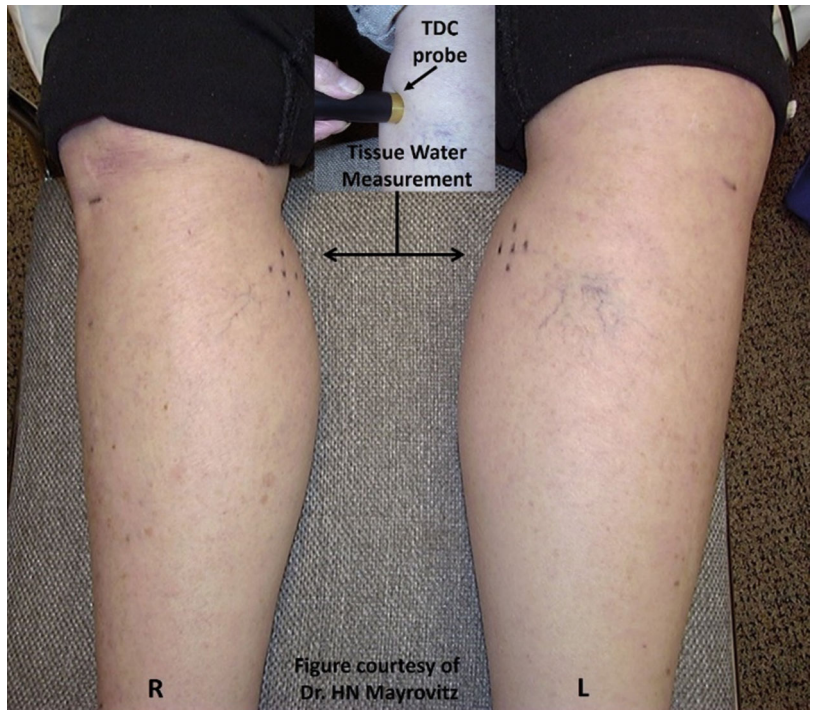

Fig. 3 Measuring tissue water via tissue dielectric constant (TDC) values. TDC, as an index of skin-to-fat tissue water, is shown in the inset being measured on the left leg of a 77-year-old female with unilateral lymphedema 5 years after surgery for gynecological cancer. The TDC value on the lymphedematous left leg at the site with the five dots was recorded as 48.7 in comparison with a value of 24.2 on the non-affected right leg

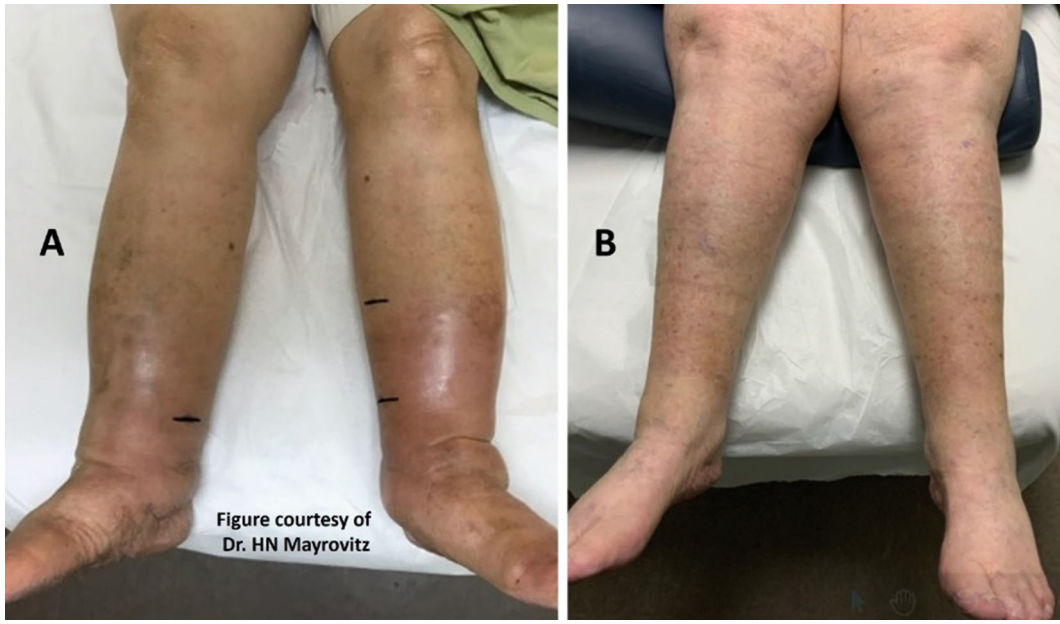

Fig. 4 Bilateral chronic venous insufficiency (CVI) and lymphedema. (**a**) A 71-year-old female with CVI that has impacted both legs. Measurements of TDC at 8 cm proximal to the medial malleolus show a TDC value (as an index of tissue water) of 67.3 compared to a non-edematous forearm site of 25.0. (**b**) A 71-year-old female with long-standing bilateral lymphedema. Measurements of TDC at 8 cm proximal to the medial malleolus show a TDC value of 51.2 compared to a non-edematous forearm site of 25.2

contrast to hyperthyroidism, which is more prevalent in younger individuals [84]. Hashimoto's thyroiditis is the most common cause of primary hypothyroidism in older people [85].

Pretibial myxedema, also known as thyroid dermopathy, is a rare manifestation of autoimmune thyroid diseases: It is seen in Graves' disease and occasionally in hypothyroidism (Hashimoto's thyroiditis) [86–89]. Myxedema is a non-pitting edema due to increased deposition of glycosaminoglycans—hyaluronic acid and chondroitin sulfate—that accumulate due to reduced metabolism of glycosaminoglycans in interstitial tissues [86, 90]. The increased deposition of glycosaminoglycans first occurs in the papillary dermis, around the hair follicles and vessels. As they accumulate, they separate collagen bundles and may lead to some secondary degeneration [86]. However, histological findings show an extensive deposition of glycosaminoglycans in the reticular dermis compared to the superficial papillary dermis. In the papillary dermis, some stellate fibroblast and collagen fibers are fragmented [91–93].

In differentiating thyroid dermatopathy from other edematous conditions or non-thyroid disorders with similar pretibial glycosaminoglycan deposition, a history of thyroid disease, physical examination, and histology is useful [91]. However, since the clinical features of thyroid dysfunction often mimic signs and symptoms of aging [83, 94], thyroid dysfunction may not be identified in the old and perceived as part of normal aging. A thyroid-stimulating hormone (TSH) level should be obtained in patients with non-pitting edema of the lower extremities [68] to assist in diagnosis of thyroid illness related dermatopathy. Further diagnostic issues arise since the thyroid gland gradually atrophies with fibrosis during normal aging decreasing gland size [95], and the gland may be difficult to palpate [83, 96]. Histological findings distinctive to thyroid dermatopathy include glycosaminoglycan deposition in the reticular dermis, spared collagen in the superficial papillary dermis, lack of angioplasia, and the absence of hemosiderin [91].

4.4 Edema from Volume Overload

A disruption of the Starling forces that maintains water balance between intravascular and extravascular compartments can lead to edema from volume overload. The hydrostatic pressure gradient between the two compartments, differences in oncotic pressures within the interstitial space and plasma, and the vessel wall hydraulic permeability determine the Starling forces. Proteins, capillary protein permeability, and the rate of lymphatic clearance influence the oncotic pressure [97, 98]. This type of generalized non-lymphatic edema can be attributed to chronic disease processes, including renal insufficiency, cardiac conditions (heart failure), cirrhosis, protein malnutrition (hypoproteinemia), Cushing's syndrome, and pulmonary hypertension [63]. Volume overload edema caused by heart failure, hepatic disease, and renal disease most often is bilateral, pitting, and non-tender without skin changes [99]. Patients with CVI have dependent edema that often improves with elevation and also have brawny, reddish skin changes due to hemosiderin deposition [100, 101]. In comparison, edema due to low plasma oncotic pressure (such as liver failure, nephrotic syndrome, and protein malabsorption) will present with edema that does not improve with positional changes [101]. Further related details on these conditions follow.

In *heart failure*, ventricular dysfunction causes an elevation in venous pressure propagated as increased hydrostatic pressure in the capillary bed, with right ventricular dysfunction or failure leading to peripheral edema [63]. In the old, heart failure is most often attributed to coronary artery disease and hypertension, which are common and often coexist. In developing countries, arrhythmias, endocrinopathies, cardiomyopathy, infection, and alcohol are common causes of heart failure. Constrictive pericarditis or restrictive cardiomyopathy can cause peripheral edema from the decreased output and venous congestion. Patients will present with symptoms of right heart failure—elevated jugular venous pressure (JVP), hepatic congestion, and ascites [63]. Additionally, age is an independent risk factor for developing heart failure after acute myocardial infarction [102]. The cardinal symptoms of heart failure include exertional dyspnea, orthopnea, lower extremity edema, and reduced exercise tolerance [103]. Especially after the age of 80, atypical symptoms are more common manifestations of heart failure, such as confusion, memory deficits, sleepiness, episodes of delirium, irritability, syncope, fatigue, anorexia, and reduced activity levels. Fatigue in the geriatric population is a complex symptom related to "low cardiac output, peripheral hypoperfusion, and skeletal muscle deconditioning" [104]. An example of two geriatric patients with CHF is shown in Fig. 5. Both patients had their edema extent assessed on the foot dorsum using TDC measurements that showed significant excess tissue water in both cases.

Liver disease both increases intravenous hydrostatic pressure (through portal vein congestion) and decreases the oncotic plasma pressure through decreased albumin synthesis [101]. In chronic heart failure and cirrhosis, the decrease in "effective intravascular volume initiates a neurohumoral cascade that attempts to maintain adequate circulating volume," thus, reducing glomerular filtration rate via renal vasoconstriction and increasing sodium and water reabsorption. The homeostatic fluid balance is shifted from excretion to retention, consequently promoting edema development [97]. In end-stage liver disease, this profound salt and water retention often leads to ascites, but in later stages may manifest as more prominent lower extremity peripheral edema, especially when there is severe hypoalbuminemia. As albumin is a significant contributor to oncotic plasma pressure, a plasma level below 2 g/dL may result in edema.

Renal disease demonstrates a similar mechanism causing edema by decreasing the oncotic plasma pressure through protein loss and increasing plasma volume due to renal water retention [101]. Thus, hypoalbuminemia and edema are also characteristic signs of nephrotic syndrome, which may also include severe proteinuria and hyperlipidemia [63]. Patients with possible nephrotic syndrome should have serum lipids measured in addition to the basic laboratory studies [105]. Other than nephrotic syndrome,

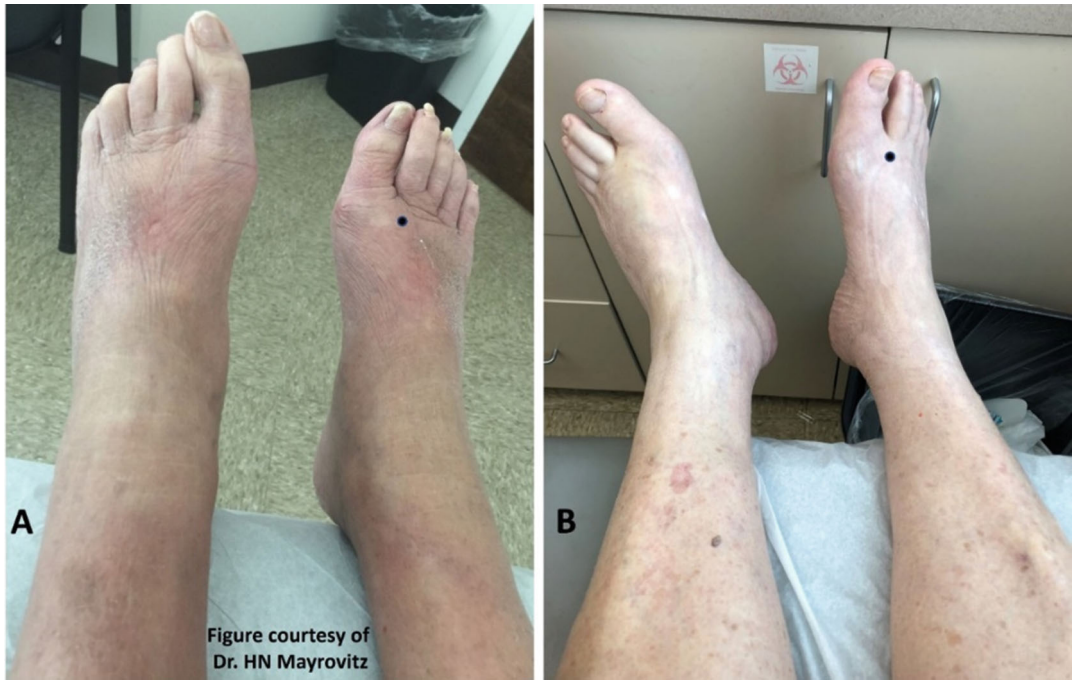

Fig. 5 Lower extremity edema in congestive heart failure. (**a**) A 75-year-old male who has class II heart failure with preserved ejection fraction (HFpEF). (**b**) An 89-year-old man who also has class II heart failure but with reduced ejection fraction (HFrEF). The black dots on the foot dorsum are sites at which TDC was measured to assess tissue edema. For patient A, a TDC value of 47.0 was measured vs. a value of 24.3 in non-edematous tissue. For patient B, a TDC value of 40.8 was measured vs. a value of 24.7 in non-edematous tissue

hypoproteinemia can occur in other conditions leading to peripheral edema; they include severe nutritional deficiency (Kwashiorkor), protein-losing enteropathies, and impaired hepatic function [63].

As with thyroid dermatopathy, the associated symptoms of the above disorders may be confused with symptoms of aging, and the comorbidities may add to the uncertainty of the diagnosis.

4.5 Lipoedema

Lipoedema is a chronic progressive disorder of adipose tissue accumulation, which may be mistaken for other pathologies that involve enlargement of the lower extremities [64, 106, 107]. The pathophysiology of this condition is not fully understood, but hypotheses have included microangiopathy, adipogenesis, genetic mutations [107], and hormonal or lymphatic disorders [108]. Accordingly, it has been proposed that persistent microcirculatory dysfunction due to lymphangiopathy or microvascular dysfunction may result in permanent changes and eventual increase in the adipose tissue [108].

The lower leg edema of lipoedema manifests as bilateral, symmetrical [73, 109] and is disproportionate to the upper body—appearing as "riding breeches" or "saddlebag" legs [110]. The fatty depositions may extend from the hips to the ankles and distinctively stops abruptly at the malleoli [64, 110]. Rarely, it may affect the arms but characteristically spares the trunk, feet, and hands [64, 111]. The swelling often worsens upon standing and is associated with pain, tenderness, and easy bruising [109, 112–114]. Leg elevation marginally improves the swelling [110]. Bruising may occur with minimal trauma due to the fragility of capillaries within the adipose tissue [115], ranging from minor bruises to large hematomas.

Thus, a positive tourniquet test (petechiae developing on the arm after a blood pressure measurement) may be observed, and the Kaposi-Stemmer's sign (the inability to pinch a fold of skin at the base of the second toe) is negative [64, 110, 116]. The skin is usually hypothermic, spongy without pitting, and may eventually develop telangiectasias [110, 117].

Lipoedema occurs almost exclusively in women and is associated with hormonal changes, often arising within a few years after puberty but rarely during pregnancy or menopause [112–114]. The onset is usually insidious, and the disease progresses gradually [64, 110]. Consequently, **older patients are** more likely to have a longstanding disease, increasing the chances of comorbidities related to age, severity, and disease duration. Severe impairments resulting from pain may further reduce physical mobility in this population [116]. Advanced stages of the disease may be associated with secondary lymphatic insufficiency referred to as lipolymphedema [113, 115] or deterioration of the venous system referred to as venolipoedema [118]. These complications may not only add difficulty in the diagnosis but also increase the risk of recurrent infection and ulceration [118]. Lastly, as in CVI, stasis dermatitis and skin discoloration may occur in advanced stages of lipoedema [110]. The role of liposuction in the treatment of this condition has recently been discussed [119].

5 Disease Burden

5.1 Risk Factors

Various risk factors have been identified as contributing to CVI including advanced age, female gender, obesity, and prolonged standing or immobility [17, 18, 20, 120]. In geriatric patients, advanced age is the most prevalent risk factor for CVI [17, 19, 121]. This is in part due to a correlation between advanced age and increased vessel wall deterioration and increased venous pressure due to weakened calf muscles as the patient ages [17]. A case-control study has revealed that for each 1-year increase in age, an individual's risk of CVI increased by 6% [122]. Although advanced age is the biggest risk factor that contributes to geriatric patients developing CVI, there are other risk factors such as gender, obesity, and prolonged standing, which may have been present earlier in their life leading to the development of CVI as they age.

Studies suggest that being overweight or obese are risk factors for CVI [16, 18, 20, 121, 123, 124] with a BMI > 30 Kg/m^2 being particularly risky [121]. Lack of adequate physical activity in this population may contribute to CVI due to restriction of venous emptying in the lower extremities [123, 125]. Thus, geriatric adults with a high BMI may be more likely to have a predisposition to CVI than their counterparts with a low BMI.

CVI is also more prevalent in persons with occupations that require prolonged standing [123]. Multiple studies support the claim that prolonged sitting or standing contributes to CVI [126–128]. This is due to the increased pressure that results from standing or sitting to long, leading to the development of CVI [127]. As a result, those employed in occupations that required an extended amount of time sitting or standing could have a predisposition to CVI. In addition, persons that are not physically active and spend an increased amount of time sitting are also prone to developing CVI.

5.2 Psychosocial Effects

The quality of life for patients with CVI is impacted by their symptoms, treatment options, and frequent medical appointments. As already noted, CVI can cause lower extremity pain, swelling, skin changes and lead to infection and ulcer development. These and other symptoms manifest in psychosocial effects such as anxiety, depression, low self-esteem, and social deprivation [19, 129, 130]. The evolution of CVI to venous ulcerations has amplified psychosocial effects in relation to ulcer odors and excessive exudate which affect the patient's quality of life [130]. Pain, another major CVI complication, can also negatively impact one's quality of life [122]. These ulcer-related symptoms contribute to the feelings

of disgust, self-loathing, and low self-esteem [130] and consequent social isolation and depression; the presence of venous leg ulcers in older patients worsen quality of life [25]. Based on patient interviews, altered appearance, loss of sleep, functional limitations, and disappointment with treatment were cited. Geriatric patients with CVI or its complications are often dependent on caregivers to help care for their symptoms thereby creating approximately equal emotional and social burdens for both patient and caregivers [131]. CVI symptoms and its complications also affect a patients work and leisure activities with 42% of patients who developed venous ulcers indicating interference with work and leisure activities [132]. Overall, whether the CVI manifestations are mild, moderate, or severe, research has proven the effects the disease has on patients and their caregivers. Thus, psychosocial effects as well as the quality of life of the patient should be taken into consideration when managing CVI.

5.3 Predisposition to Deep Vein Thrombosis and Thromboembolism

CVI results in venous hemodynamics that predisposes to thrombotic events such as a superficial venous thrombosis or DVT in lower extremity veins [133]. The patterns of venous insufficiency that occur after an acute deep vein thrombosis has been evaluated in 70 limbs in 67 patients with multi-segment DVTs found to have a higher prevalence of deep vein insufficiency compared to single segment DVTs [134]. Venous thrombosis is more common in geriatric patients, especially in those aged greater than 70 with the risk of venous thrombosis greater with increasing venous insufficiency severity [135]. Formation of these DVT present a significant risk of pulmonary embolisms in which fragments of the DVT and lodge in the lung [136]. Pulmonary embolism is the third most common cardiovascular cause of death, and in older people, pulmonary embolism is an immediate threat to their life [137].

6 CVI and Linkage to Venous Ulcers

Throughout this chapter, the potential linkage between CVI and venous ulcer development has been mentioned and is here further detailed. A study of over 600 health care workers, mostly women, with a median age of 42 years, demonstrated that even in this young group, clinical CVI and the presence of venous reflux was relatively high, with clinical evidence of CVI in at least one leg reported as 69.1% [138]. In the age range of 65–74 years, venous disease with or without ulceration was the main diagnosis in about 5% of patients admitted to hospital [139]. Further, of 141 patients over the age of 75 admitted to hospital for chronic peripheral edema without dyspnea, 69% were reported to have CVI [140]. It has also been reported that venous ulcers are a complication of CVI in up to 18% of patients with CVI, with an increase in incidence in the geriatric age group, 65 and above [141]. Treatment of these leg ulcers in the older population is complex to achieve wound healing [142], with significant socioeconomic and life functioning impact [129, 143, 144]. It is evident therefore that preventing the transition of CVI to venous ulcers is a fundamental treatment goal [145]. A potential correlation between CVI, venous ulcer presence, and contact allergic reactions has also been reported, a finding that further complicates dermatological care in the geriatric patient [146].

As noted, a main impact of CVI on skin integrity relates to its connection to skin breakdown and venous ulcers. An understanding of factors involved in the pathophysiology of the CVI-venous ulcer connection is well studied but incomplete [147–151]. Altered aspects of the microcirculation have been studied and are likely involved in this transitional process [152–158]. Exercise-related treatments have been reported to improve healing in part accompanied by improvements in microcirculation [159, 160].

7 Physiological Considerations of CVI and Its Pathological Impacts

7.1 Valve Dysfunction

Venous valve dysfunction has major impacts on venous hemodynamics and contributes to venous insufficiency [161–163]. With legs in a gravity dependent and relaxed position, the presence of normally functioning lower extremity valves will segment the hydrostatic pressure column and thereby reduce gravity-dependent pressure. Calf contraction acts as a pump by compressing veins; flow is directed centrally via valve presence. Walking versus standing reduces dependent vein pressure by displacing volume centrally. Adequate function depends on normal valves, so valve incompetence, as depicted in Fig. 6, causes altered patterns of venous flow exposing superficial veins to high impulse pressures and an elevation in average venous ambulatory pressure. The associated venous hypertension is a major factor that leads to the development of skin breakdown and ulceration [164].

7.2 Compression in Relation to Function

Differences in garment or bandage materials produce functional differences [165–167]. For example, material that has a high percentage of elastic fibers that forms the compression bandage is called a "long-stretch" bandage [168]. These act similarly to springs; stretch it more and the recoil force increases. Such bandages stretch up to three times their zero-tension length on the leg and produce a sub-bandage pressure (SBP) that results in increases in tissue, interstitial, and muscle pressures. It also alters the transmural pressure of blood vessels subjected to the compression pressure as shown in Fig. 7. This SBP is directed radially inward and may be further distinguished as "resting pressure" or "working pressure" which distinguishes between a muscularly relaxed limb

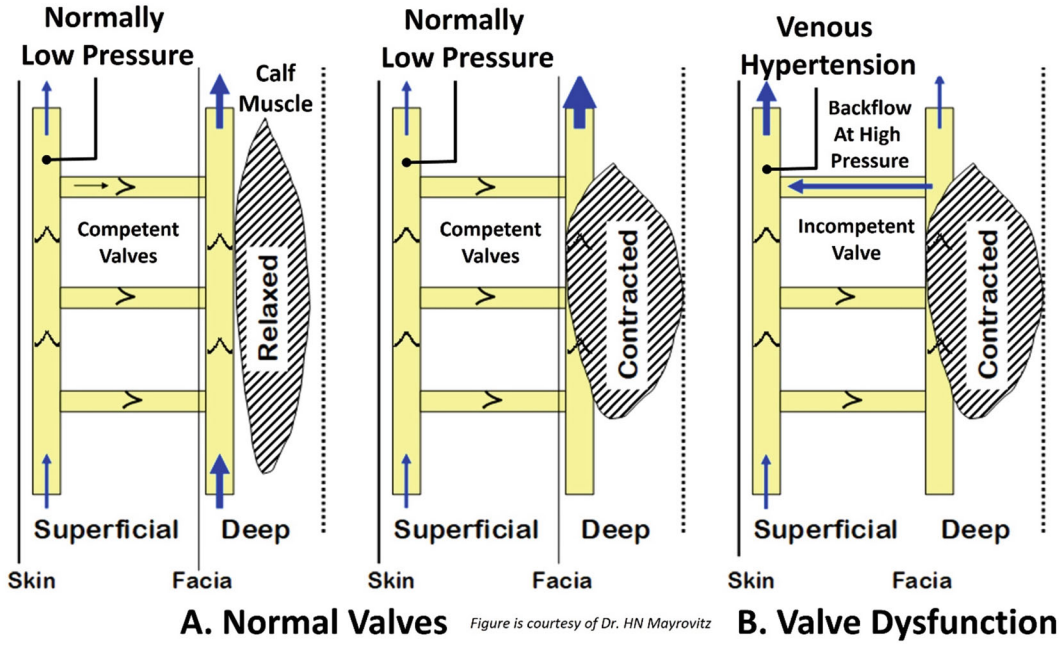

Fig. 6 Illustration of the impact and hemodynamics of incompetent venous valves. The normally low pressure experienced by superficial veins is exposed to high pressures when there is valve incompetency as shown in (**b**). This elevated pressure is not well tolerated and causes venous injury that triggers a sequence of events that may cause a venous ulcer. (Figure is provided as a courtesy of Dr. HN Mayrovitz)

illustrated in Fig. 2a from one undergoing muscular contraction as illustrated in Fig. 2c. A so-called "short-stretch" bandage has few if any elastic fibers and under resting conditions produces less recoil force on the leg resulting in a lower resting pressure but produces a greater working pressure as shown in Fig. 8. Under resting conditions, a portion of the bandage-related SBP is transmitted interiorly and increases interstitial tissue pressures (P_T), which reduces vascular transmural pressures and helps reduce transcapillary filtration into tissue. Under dynamic working conditions, internal pressures assume much greater values when "no stretch" bandages are used since the effective dynamic compliance of the limb to volume expansion is reduced by the relatively more rigid bandage. These greater dynamic pressures are important in controlling edema/lymphedema via their favorable effects on interstitial fluid movement, which, together with lymphatic activation, helps reduce localized tissue edema [169–171].

7.3 Compressive Therapy as a Therapeutic Intervention

A mainstay of the noninvasive treatment of CVI depends on properly used compression therapy in

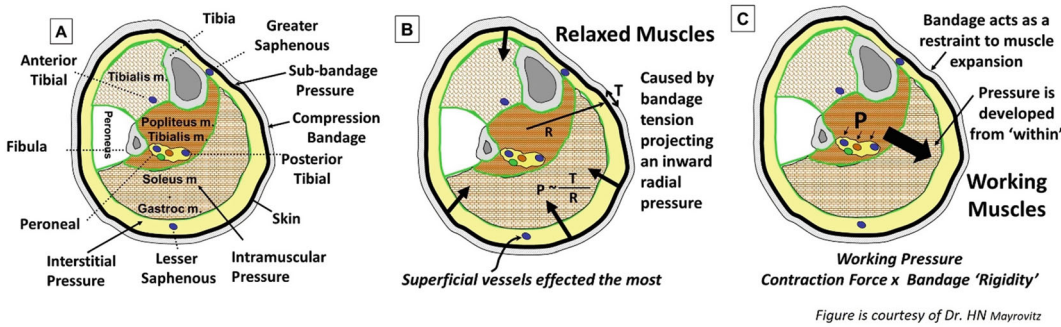

Fig. 7 Illustration of main pressure effects of leg compression bandaging. There is an inward directed pressure caused by the tension produced by the bandage similar to that caused by Laplace's law as shown in (**b**). When the muscle contracts indicated in (**c**) as a working muscle, there is a pressure developed within, the working pressure, that depends on the magnitude of the contraction force and the relative rigidity of the bandage material. An indication of the pressures developed is illustrated in Fig. 3

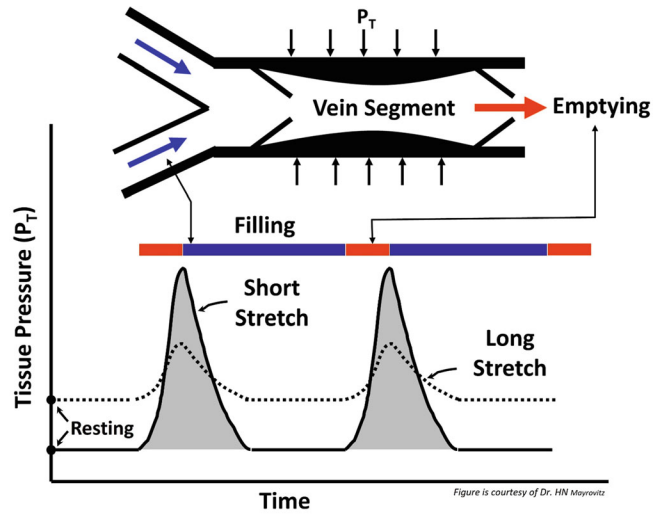

Fig. 8 Tissue resting and working pressures with short- and long-stretch bandaging. Short-stretch materials are stiffer so that during calf muscle contraction, there is an associated greater working pressure developed. During ambulation, this is more effective in moving venous blood centrally thereby reducing venous pressure. The lower resting pressure of the short stretch is because it does not need to be tightly wrapped to achieve its function

the form of compression garments and bandages and in some cases intermittent pneumatic compression (IPC). Compression therapy can be used to help minimize or reduce lower extremity edema or lymphedema in the presence or absence of skin ulceration and thereby assist in preventing or healing CVI-related leg ulcers. A recent meta-review has reported that the primary difference in treatment and healing outcomes is between the use of compression versus the nonuse of compression with the former yielding significantly greater healing [172]. However, because compression by design may impact both tissue and vascular aspects of treated limbs in any patient, its non-therapeutic side effects may have greater value in the geriatric population.

Of relevance to this issue was a study of 102 patients with venous ulcers that were divided by age above and below 65 years [173]. Of the 51 geriatric patients, 25 were treated with moderate compression pressures ranging from 37.1 to 46.3 mmHg and 26 patients were treated with higher pressures ranging from 61.1 to 72.4 mmHg. Results showed greater healing with higher compression pressures in both younger and older groups, but local complications such as superficial skin necrosis and skin discoloration trended greater in the geriatric group. It is hence important to consider the various aspects of compression therapy; they include the functional dependence on compression materials, the rationale for compression in wound healing, the mechanism of action of compression, and its impacts on limb pressures and blood flow.

8 CVI and Compression in Relation to Venous Ulceration

8.1 Overview of Issues

A goal of compression for preventing or treating venous ulcers is to try to normalize altered venous and microcirculatory hemodynamics that contribute to ulcer development and prolongation. Normally, leg venous hemodynamics and volumes rely on valve competency of superficial, perforating, and deep venous systems to protect against gravitational and muscle pump pressures. Normal venous return for muscle is via the deep system, and via the superficial system for skin and subcutaneous structures. Properly functioning valves in perforating veins, as illustrated in Fig. 6, prevent exposure of superficial veins to relatively high pressures in deep veins when they compress against fascia during calf muscle contraction. The properly functioning valves also permit unidirectional flow from superficial-to-deep veins during relaxation that results in an adequate blood volume ejection fraction to keep venous and leg volumes at normal levels.

Valve dysfunction alters this situation. If perforator vein valves are dysfunctional, some deep vein volume at high pressure is transmitted to the superficial system with each calf contraction as pictured in Fig. 6b. As a consequence, effective ejection fraction for venous return from the deep system is reduced, excessive pressures in the superficial system further compromise valve competency, and the sustained increase in venous volume effects microcirculation causing endothelial cell changes and increased outward flux of fluids and materials from capillaries and venules. Changes in capillary hemodynamics, nutritional blood flow, and interstitium content and volume then follow.

Although the precise sequence whereby initiating hemodynamic changes end in skin ulceration are not fully worked out, there is evidence implicating reduced nutritional capillary density and degradation of capillary function [174, 175]. These changes may be due to retrograde dynamic pressures that are transmitted to nutritive capillaries [156], likely causing trauma and inflammatory-like responses [176]. The venous hypertension may result in vessel rarefaction in a manner akin to that seen in systemic hypertension. Surprisingly, in spite of increased leg blood flow in the ulcer region [177] and in peri-ulcer subcutaneous microcirculation [158], transcutaneous oxygen is reduced. In addition to microcirculatory effects, limb compression augments arterial flow pulsatility [178], which likely stimulates interstitial fluid and lymphatic dynamics and ulcer healing. Thus, appropriate compression therapy

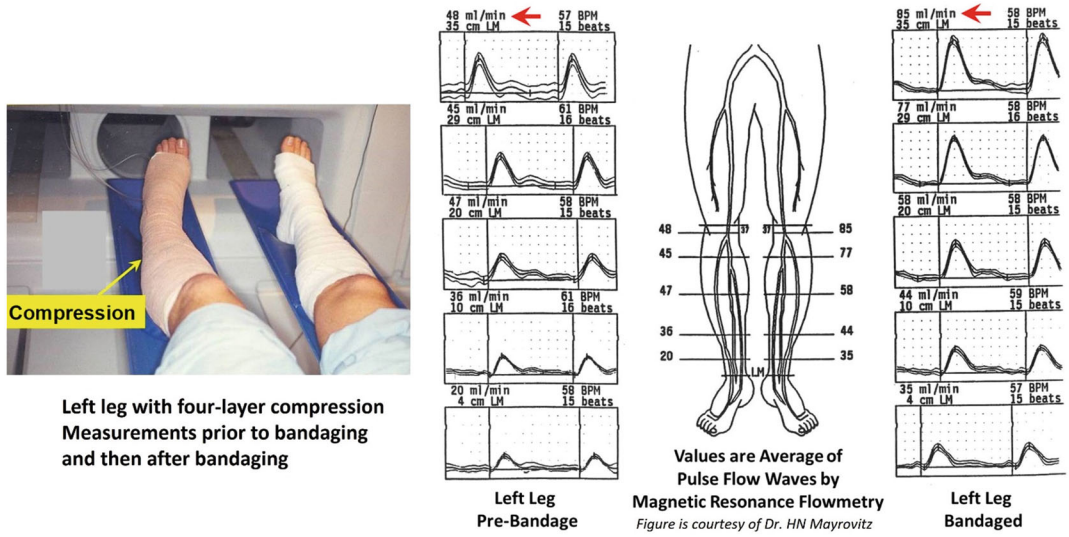

Fig. 9 Impact of compression on leg pulsatile blood flow. Right-hand panel shows pulsatile blood flow of the left leg prior to bandaging and then after bandaging. The vertical flow waveforms (ml/min) are obtained at varying distances from the lateral malleolus (LM). The main point is that the pulsatile flow is greater in the presence of the compression at every location

may preempt ulcer formation in cases of CVI and significantly aid in healing of ulcers in part due to combined hemodynamic effects. The potential impact of compression bandaging on arterial pulses is visualized in Fig. 9 that shows the effect on leg pulsatile blood flow measured using a nuclear magnetic resonance method. Pulsatile blood flow is seen to be substantially elevated at each longitudinal section where it was measured. The measurement locations are designated as cm proximal to the lateral malleolus (LM).

8.2 Further Considerations Regarding Compression for CVI and Venous Ulcers

Compression bandaging causes a "counter-pressure" that is directed in such a fashion so as to reduce abnormally elevated transmural pressures of veins that may be caused by combined valve incompetence, gravitational forces, and muscular dynamics already described. Also as already noted, the amount of counter-pressure that is functionally effective depends on the type of bandage material that is used (highly extensible or relatively inelastic), the manner of wrapping (tightness of wrap), and whether the limb is relaxed (static conditions) or muscle is contracting (dynamic conditions). For a given set of conditions, the counter-pressure needs to be sufficient to reduce abnormally elevated superficial venous volume by reducing overload volume entering the superficial system during muscular contraction and to maintain a lessened volume during static conditions. The resultant lessened average venous pressure tends to reduce excessive transcapillary outward filtration into interstitial spaces, which in turn reduces tissue edema. Simultaneously, a greater dynamic tissue pressure promotes greater tissue fluid movement and its uptake and removal by the lymphatic system, which adds to the edema lessening process. Deep vein volume is also reduced because its effective ejection fraction is returned toward normal concomitant with the reduction in venous reflux.

The literature is replete with reports demonstrating the efficacy of compression bandaging as a main component in the treatment of venous ulcers [179–181]. Most small ulcers, present for short durations (<6–9 months), tend to heal with good standard wound care combined with

compression bandaging [182–184]. However, questions still remain about the optimum approach to compression bandaging and the precise mode of action. One issue concerns the relative efficacies of long-stretch, short-stretch, or multilayer bandaging systems [185]. A benefit has been suggested for multilayer as compared to short stretch [186] whereas others have found both to be equally effective [187, 188]. Still others have advocated short-stretch bandaging to achieve therapeutically effective graduated compression [189]. Other questions relate to achieving verifiable and appropriate compression pressures and gradients. Still other questions relate to possible direct effects of the compression on the wound bed [190].

As noted previously, part of the effectiveness of compression therapy depends on an increase in compression-related subcutaneous tissue pressure (P_T) to diminish transcapillary fluid filtration and promote vascular resorption and removal of interstitial fluids. Local or generalized reduction in such edema or micro-edema allows better oxygen and nutrient delivery to and chemical byproduct removal from skin and subcutaneous tissues in cases of CVI and the wound bed in the case of a venous ulcer. It is notable that tissue pressure is already elevated in edematous legs or arms. For example, in untreated arm lymphedema, P_T differences between affected and non-affected arms averaged 4.6 cm H_2O [191] with even larger differences reported for legs [192]. This elevated P_T is reduced if compression therapy reduces fluid volume. However, promotion of fluid resorption by the compression-induced increase in P_T is not as great as would be indicated by the amount of P_T increase because capillary pressure simultaneously increases by about 80% of the P_T increase [193]. There is evidence that sub-bandage pressures of about 20–40 mmHg blunt a normal venoarterial constriction response in dependent limbs [194, 195]. This would tend to maintain capillary pressure higher and detract from the sought-after decrease in capillary filtration.

If compression-induced tissue pressures become too large for too long, there is a negative impact on blood vessels and perfusion and on lymphatic vessels and their lymph flow [196]. Optimal tissue pressures have not been defined, and there is little direct information as to relationships between surface or sub-bandage pressures and associated tissue pressures [197]. Subdermal pressure measurements under compression garments give some idea of surface-to-subsurface radial pressure gradients to be expected at different sites [198]. Absolute pressures are greater at bony prominences, but gradients are larger for soft tissue. For example, measurements at the posterior mid-calf found that a sub-bandage pressure of 66 mmHg resulted in a subdermal pressure of 24 mmHg whereas at medial mid-calf, a sub-bandage pressure of 36 mmHg resulted in a subdermal pressure of 21 mmHg.

8.3 Role of Intermittent Pneumatic Compression

Some small studies suggest that IPC applied to the leg [199] or foot [200] in the form of impulses was useful in treating venous ulcers possibly in relation to coexisting CVI [201–203]. IPC devices deliver controlled sequential pressures to a limb but with considerable variation in pressure magnitude and timing and other IPC-related parameters [204]. For use in the geriatric population, care must be used in assure that pressures developed by a chosen IPC device are not injurious to skin or underlying tissue or vascular structures. The efficacy of IPC in treating venous ulcers associated with CVI is, according to an earlier report, inconclusive [205] with some studies reporting no benefit while others reported some benefit in patients with long-standing, previously intractable ulcerations [203]. The potential therapeutic value of IPC for venous ulcers in immobile patients has been suggested but not established [206].

An aspect not previously considered in this connection is the possible wound healing effects of IPC via nitric oxide pathways or related vasodilatory processes. An early experimental study reported that IPC upregulates nitric oxide synthase (eNOS) mRNA causing vasodilation in non-compressed tissues [207]. Such vasodilatory actions are favorable for wound healing so variability in effectiveness of IPC to induce such

vasodilatory actions may explain varied outcomes. A greater IPC-induced vasodilatory effectiveness has been reported to be associated with an IPC action with a greater inflation velocity [208], so variability in effectiveness may also be due to differences in IPC device parameters. There is also some evidence that IPC-related edema reduction is associated with an increase in peri-wound oxygen tension of venous ulcers [209]. IPC effectiveness for limb volume reduction in early studies was reported to be inversely related to the extent of fibrosis present [210, 211]. Further, with early approaches to lower extremity pump therapy, there were concerns about using it for fear of causing genital lymphedema [212]. A retrospective study of patients with lower extremity critical ischemia and nonhealing ulcers suggest positive benefits in wound healing and limb salvage [213, 214]. These effects may be related to the improvement in arterial blood flow associated with a very rapid rise in compression pressure [215, 216]. Early discussions of IPC mechanisms of action and its various clinical applications may be historically useful [217].

9 Other Treatments and Prevention Strategies Applicable to the Geriatric Population

9.1 Exercise

In Europe, physical therapy is utilized for CVI but has not been widely adopted in North America [218]. As CVI progresses, individuals have decreased ankle range of motion and diminished calf muscle pump function. To investigate this, 31 geriatric patients with advanced CVI were recruited and divided into two groups: control and treatment. Both groups received compression therapy while the treatment group also received physical therapy focused on strengthening calf musculature. After 6 months, the treatment groups' calf muscle pump function and strength increased with the structured exercise. Other studies also support the finding that if one improves calf muscle strength, venous flow also increases and indicates that tiptoe exercise in the standing position and flexing and stretching of the feet in the sitting position also stimulate the calf muscle and improve venous blood pressure, venous residual volume fraction, and ejection fraction [219–221].

9.2 Osteopathic Manipulative Medicine

Osteopathic manipulative treatment is another technique that can help increase lymphatic and venous flow by removing somatic dysfunction that is obstructing lymphatic and venous flow [222], leading to edema reduction if present. Two manipulations are used to improve lymphatic and venous function: lymphatic techniques and myofascial release. Lymphatic treatment addresses somatic dysfunction around body regions restrict flow. In the lower extremity, these areas may be the pelvic diaphragm or the popliteal fossa. Both of these approaches have been evaluated and have shown effectiveness [3], and might be considered as adjuvant therapies that may help geriatric patients with CVI.

9.3 Role for Anticoagulants

CVI is common among geriatric patients due to structural changes that occur in venous valves and walls as the patient ages. CVI has been associated with a threefold elevated risk of venous thrombosis including both deep vein thrombosis and pulmonary embolism [223]. This risk is due to venous stasis that occurs in patients with CVI. The treatment for venous thromboembolism in geriatric patients is complex since these patients have a higher likelihood for both thrombosis and bleeding. To prevent this from occurring, patients are prescribed anticoagulant therapy if there are no contraindications present [223, 224].

9.4 Surgical Therapy

Surgical or endovenous procedures are utilized to treat CVI by removing an incompetent vein or

isolating a reflux source from the rest of the vascular system. Once completed, the patient usually experiences a significant decrease in symptoms and an improvement in quality of life. The classic saphenofemoral ligation and stripping is a standard surgical treatment for CVI [20]. During this surgery, the great saphenous is ligated and dissected from the femoral veins along with its tributaries. The lesser saphenous vein is then dissected from the popliteal vein and ligated close to the junction [20]. One study investigated the role of superficial venous surgery in advanced CVI in 146 legs treated with compression alone and 115 with compression and surgery [225]. At 12 months post-intervention, the compression group had more legs with incompetent perforators compared to baseline. With the surgery group, there were significantly fewer legs that had incompetent calf perforators. In addition, patients in this group developed less new perforator incompetence in comparison to the compression only group. However, surgical treatment is rarely offered for geriatric patients as the risks and benefits must be taken into consideration. In one study in which 28 geriatric patients (70–89 years) with CVI were treated with superficial venous surgery, it was concluded that surgery of the superficial venous system for treatment of CVI is effective and can be utilized for older patients since there is minimal risk [226].

Other approaches include radiofrequency ablation, endovenous laser ablation, and foam sclerotherapy.

Radiofrequency ablation (RFA) uses electromagnetic energy to heat the vein wall, destroying the intima resulting in a fibrotic occlusion of the vein [227]. This is done under local anesthesia in an outpatient setting. RFA was compared to the standard ligation/stripping procedure and reported to have earlier recovery and less postoperative pain [227]. RFA use to treat CVI due to superficial disease was evaluated in young and older patients and reported their results indicated RFA is a safe and effective procedure for geriatric patients with CVI [228].

Endovenous laser ablation (EVLA) delivers laser energy directly into the lumen of the vein causing the blood inside to boil and form steam bubbles which induces local heat injury to the inner wall of the vein [229]. The heat causes the wall of the vein to shrink causing the lumen to reduce. This technique is also done with local anesthesia in an outpatient setting. The effectiveness of RFA and EVLA are reported to be about the same; however, RFA is associated with fewer side effects and a more rapid recovery [20]. The side effects of RFA and EVLA include thrombophlebitis, hyperpigmentation, paresthesia, and bruising [20].

Foam sclerotherapy is another minimally invasive procedure used to ablate the saphenous vein. The foams are a mixture of gas and a liquid solution with surfactant properties such as sodium tetradecyl sulfate or polidocanol [229]. This technique causes endothelial damage which exposes the subendothelial collagen leading to platelet aggregation with subsequent coagulation and fibrosis of the vessel lumen. The most common complication with foam sclerotherapy is superficial thrombophlebitis. However, in experienced hands, foam sclerotherapy is reported safe and serious complications are rarely encountered.

In a study with 152 patients older than 65 years, ultrasound catheter directed foam sclerotherapy was done in patients with great saphenous vein valvular incompetence and saphenofemoral junction incompetence [230]. Complete occlusion rate of the great saphenous vein 12 months following the procedure was 86.4% for these geriatric patients with no major complications reported. It seems that surgical treatment, whether invasive or minimally invasive, is a viable option for patients with severe CVI interested in diminishing their symptoms and improving their quality of life.

10 Key Points

- Encompassing the more severe end of the chronic venous disease spectrum, the main clinical features of CVI are dilated veins, edema, aching pain or discomfort (leg heaviness), and skin changes (stasis dermatitis, hyperpigmentation, conceivably lipodermatosclerosis, and atrophie blanche).

- The gold standard for diagnosing CVI is duplex ultrasonography to visualize reflux or occlusion. Still, other modalities may have benefits in certain situations, such as CT or MRI in assessing pelvic veins or dermoscopy, which can visualize glomerular vessels at high magnification.
- Mimics and masqueraders of CVI comprise of conditions that lead to lower extremity swelling, such as cellulitis, lymphedema, thyroid dermatopathy, volume overload edema, and lipoedema—which may be differentiated clinically based on key features such as symmetry, pain/pruritis, pitting, improvement with elevation, presence of systemic symptoms, and specific skin changes.
- The main risk factors that contribute to CVI are advanced age, female gender, obesity, and prolonged standing or immobility due to the physiologic changes that occur in the venous system in the lower extremity.
- Providers must also treat psychosocial effects associated with CVI as these patients may also suffer from anxiety, depression, low self-esteem, and social deprivation due to symptoms associated with CVI.
- Compounded with age-related factors, CVI increases the risk of severe complications: thrombotic disease (deep vein thrombosis and pulmonary embolism) and venous ulcers, both caused by compromised hemodynamics in the venous system.
- Venous return from the lower extremity depends on the calf muscle pump and functioning valves to overcome gravitational pressures; dysfunctional valves cause an alteration in hemodynamics, and venous hypertension is propagated through the system.
- Compressive therapy, including long-stretch, short-stretch, and multilayer bandaging and intermittent pneumatic compression, can help normalize the altered venous and microcirculatory hemodynamics by increasing subcutaneous tissue pressure and helps prevent ulceration.
- Although compression therapy is the main treatment for CVI, other treatment and prevention strategies, including exercise, osteopathic manipulative medicine, anticoagulants, and surgical procedures (radiofrequency ablation, endovenous laser ablation, and foam sclerotherapy) have proven to be effective at treating CVI in geriatric patients.

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