All about compression: A literature review

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Lower extremity ulcers represent a significant public health problem as they frequently progress to chronicity, significantly impact daily activities and comfort, and represent a huge financial burden to the patient and the health system. The aim of this review was to discuss the best approach for venous leg ulcers (VLUs). Online searches were conducted in Ovid MEDLINE, Ovid EMBASE, EBSCO CINAHL, and reference lists and official guidelines. Keywords considered for this review were VLU, leg ulcer, varicose ulcer, compressive therapy, compression, and stocking. A complete assessment of the patient's overall health should be performed by a trained practitioner, focusing on history of diabetes mellitus, hypertension, dietetic habits, medications, and practice of physical exercises, followed by a thorough assessment of both legs. Compressive therapy is the gold standard treatment for VLUs, and the ankle-brachial index should be measured in all patients before compression application. (J Vasc Nurs 2016;34:47-53)

Lower extremity ulcers represent a significant public health problem as they frequently progress to chronicity, significantly impact daily activities and comfort, and represent a huge financial burden to the patient and the health system. According to Rahman et al,[19] a leg ulcer is considered chronic when there is no healing of the wound within 6 months. In the United States, 65,000 people are affected by chronic wounds, with overall treatment expenses up to $25 billion/year, as pointed by Sen et al.[22] Agale[2] emphasizes that venous leg ulcers (VLUs) are the most common chronic wounds, representing more than 70% of all types of leg ulcers. Treatment of VLU can cost the US health system around $1.5–$3.5 billion and affect about 600,000 citizens yearly according to Hankin et al.[9] Sen et al[22] and Raffetto[20] disclosed that several patients with VLU failed to reconcile the treatment of the wound with their jobs, with consequent negative impact in income and increasing early retirement. Moreover, patients with VLU suffer with significant changes in their daily lives, overwhelming pain associated with dressing changes and wound surface, daily discomfort related to edema and odor, mobility difficulties, as well as social and family isolation.[6,8]

Studies throughout the world have been conducted to determine the best approach to the management of VLU, considering the cost and duration of therapy, topic products, technique of debridement, types of dressings and bandages, speed of healing time, recurrence prevention, and improvement of patients' quality of life.[7,20]

The pathophysiology of VLU remains unclear, yet, some theories have been proposed in an attempt to explain the pathogenesis of the ulcer. The most commonly accepted theory is that VLU is secondary to venous insufficiency. The superficial and/or deep venous systems are damaged, with valvular dysfunction leading to venous hypertension and consequent tissue hypoxia.[16] According to Casey[4] and Agale,[2] the main mechanism of valvular dysfunction is an increase in hydrostatic pressure, with consequent vein dilation, less functional valves, and considerable blood back flow. Deep vein thrombosis, pregnancy, leg fractures, phlebitis or congenital weakness of venous walls can lead to permanent damage of the superficial and/or deep venous systems, increasing hydrostatic pressure. It is also theorized that water and sodium, fibrinogen, and white and red blood cells may transudate from the vessel lumen in the presence of venous hypertension with consequential damage to the skin, clinically represented as edema, fibrin cuff formation, eczema, and hyperpigmentation of the tissue.[2] Cells might become ischemic and inactive, with the development of an extremely fragile skin tissue, susceptible to breakdown with minimal trauma. Finally, with associated impaired healing mechanisms, a chronic ulcer can develop in the long term.[4]

The most efficient way to manage venous ulcers is to address venous insufficiency in the first place, with a strong recommendation for the use of compressive therapy as the gold standard treatment for VLU. However, there is no consensus regarding the most effective compressive therapy technique.

The aim of this article was to discuss the best approach for VLU.

METHODS

A systematic search was conducted in Ovid MEDLINE, Ovid EMBASE, EBSCO CINAHL, and reference lists and official guidelines in October 2015. Keywords used for this review were venous leg ulcer, leg ulcer, varicose ulcer, compressive therapy, compression, and stocking. Documents published in English, Portuguese, or Spanish were considered.

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TABLE 1

ASSESSMENT OF THE PATIENT WITH VLU

Comorbid conditions	*Congestive heart failure, lymphedema, diabetes, thrombosis, hypertension*
Wound bed tissue	Base: ruddy red, granulation tissue present, yellow adherent or loose slough may be present. Size: variable, can be large. Depth: usually shallow. Margins: irregular, undermining or tunneling are uncommon. Exudate: moderate to heavy. Infection: not common.
Surrounding skin	Edema: Pitting or nonpitting, worsens with prolonged standing or sitting with legs dependent. Ankle flare, varicose veins, hemosiderosis (ie, brown staining), lipodermatosclerosis, atrophie blanche, maceration, temperature: normally warm to touch.
Complications	Venous dermatitis, infection/cellulitis, variceal bleeding.
Pain	Dull aching, itchy, sore, tender; severe sharp or throbbing. Note: Elevation of the limb relieves pain.
Perfusion	Capillary refill: normal (less than 3 seconds). Venous refill time: shortened (less than 20 seconds). Ankle-brachial index: within normal limits (1.0–1.3).

VLU = venous leg ulcers.
Adapted from the Wound, Ostomy and Continence Nurse Society Wound Committee.[28]

TABLE 2

ABI AND SEVERITY OF THE DISEASE

Index (mm Hg)	*Severity of the disease*
1.0 >1.3	Lower-extremity venous disease (LEVD) with no arterial disease involved
>0.8–0.95	LEVD with mild arterial disease
>0.6–0.8	Borderline for LEAD
<0.5	Severe ischemia
<0.4	Critical ischemia
Abnormally high ABI (greater than 1.3)	Vessel calcification. Toe pressure may be more useful

ABI = ankle-brachial index; LEVD = lower-extremity venous disease.
Adapted from Agale[2] and Wound, Ostomy and Continence Nurse Society Wound Committee.[28]

RESULTS AND DISCUSSION

Assessment of the patient with VLU

A complete assessment of the patient's overall health should be performed by a trained health care provider. Great emphasizes should be given to the patient's medical history including history of prior deep venous thrombosis, diabetes mellitus, hypertension, peripheral arterial disease, malnutrition, medications in use, and mobility.[23,16]

Both legs should be thoroughly assessed for signs of venous disease. Particularly, the presence of varicose veins, venous dermatitis, atrophie blanche, hemosiderin deposition, and lipodermatosclerosis.[23]

The ankle-brachial index (ABI) must be checked before the application of compressive therapy, to exclude any arterial component that could affect the healing process.[2] ABI can be obtained by measuring the systolic pressure of the affected leg (using a hand-held continuous wave Doppler ultrasound) divided by the brachial systolic pressure (the highest systolic value of both arms). The Wound, Ostomy & Continence Nurse Society (WOCN) established that all patients with VLU should be assessed thoroughly regarding comorbid conditions, wound bed tissue, surrounding skin, associated complications, pain, and limb perfusion[28] (see Table 1).

The ulcer should be re-evaluated over time, and the characteristics should be clearly described and carefully documented,