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Techniques in
Vascular and
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Radiology

Management of Venous Ulcers

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Chronic venous insufficiency (CVI) results from venous hypertension secondary to superficial or deep venous valvular reflux, as well as venous obstruction. The most severe clinical manifestation of CVI is venous leg ulceration that can result in significant morbidity, including venous gangrene and amputation, albeit rare. Treatment modalities are aimed at reducing venous hypertension. Diuretic therapy, although widely used, only provides short-term improvement of the edema but provides no long-term benefit. Compression therapy is the cornerstone in the management of CVI. Compression can be achieved using compression bandaging, compression pumps, or graduated compression stockings. Topical steroid creams may reduce inflammation, venous eczema, and pain in the short term, but they can be detrimental in the long run. Apligraf (a living, bilayered, cell-based product) in conjunction with compression therapy was noted to be more effective in healing venous leg ulcerations, when compared with treatment with compression therapy and zinc paste. Endovascular and surgical techniques that minimize valvular reflux and relieve venous obstruction improve venous hemodynamics, promoting wound healing.
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Introduction

Venous ulcers have been treated by bandaging and topical applications for thousands of years. The first well-documented case of venous leg ulceration (VLU) was in 1271. Raoul, the cobbler, developed unilateral edema after a deep vein thrombosis (DVT), and then a venous ulceration that was treated with dust from the tomb of King St. Louis.[1] These bandaging techniques, although transformed into contemporary brands and materials, remain vital in the management of venous ulcerations. VLUs account for approximately 80% of all lower-extremity ulcerations. The overall prevalence of venous ulcers in the United States is approximately 1% and is more common in women and the elderly. With recurrence rates reported as high as 78%, these ulcers can result in significant health care expenditure, socioeconomic burden from the related morbidity, poor quality of life, and work-loss days.[2,3] Venous specialists must understand the underlying pathophysiological mechanisms leading to the development of a VLU.

Pathophysiology

Venous incompetence and associated venous hypertension are the primary mechanisms for ulcer formation. Well-described risk factors for valvular incompetence and chronic venous insufficiency (CVI) include family history, advanced age, female gender, venous thrombosis, obesity, pregnancy, trauma, and occupation requiring prolonged standing. Venous hypertension transmitted into the microcirculation results in dilation of capillaries, thickening of basement membranes, increased collagen and elastic fibers, and endothelial damage. Leukocyte activation and trapping, endothelial damage, as well as platelet aggregation, have been implicated in the release of inflammatory mediators and proteolytic enzymes, which eventually lead to increased permeability and intracellular edema, leading to venous ulcer development. In addition to venous system damage, destruction of microlymphatics and local nerve fibers lead to impaired fluid drainage and altered regulatory mechanisms, resulting in impairment of wound healing.[4]

Clinical Assessment

Chronic venous disease commonly presents as an ache, pain, or discomfort in the lower extremities along with

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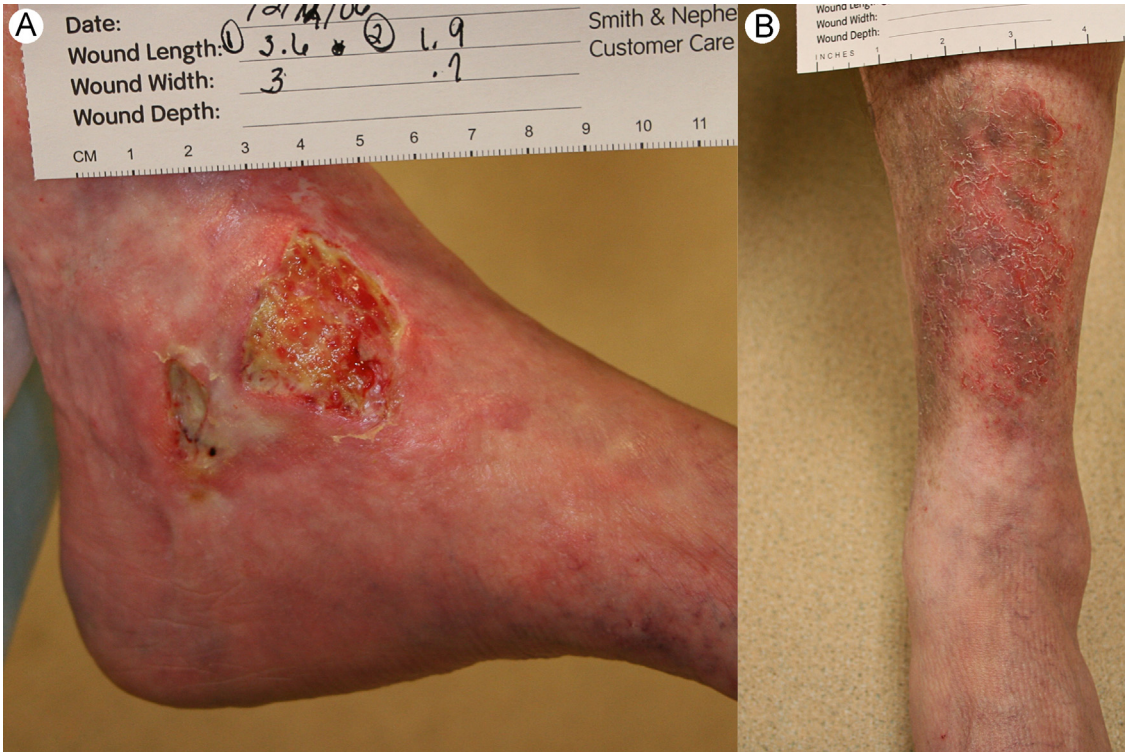

Figure 1 (A) VLU. The irregular margins and wound base with pale slough mixed with healthy granulation tissue surrounded by circumferential atrophie blanche area can be noted. (B) Venous eczema. The speckled, scaly, inflamed and hyperpigmentation can be noted. (Color version of figure is available online.)

swelling that is worse with prolonged standing and subsides with elevation. Venous ulcers occur predominantly in the gaiter area (over the medial malleolus). But they may occur on the lateral malleolus, calf, or less frequently on the feet. On several occasions, patients provide history of trauma as the inciting event, but poor wound healing ensues owing to the underlying venous hypertension due to underlying reflux or obstruction or both.

On physical examination, venous ulcers are generally irregular and shallow with pale slough mixed with granulation tissue and fibrin in the ulcer base (Fig. 1A). Other findings in the lower extremity include varicose veins, stasis dermatitis, hyperpigmentation, lipodermatosclerosis, atrophie blanche areas, and edema. Patients may have varicose veins or spider veins. Some also present with surrounding stasis eczema or venous eczema of the lower extremities (Fig. 1B). Lipodermatosclerosis (thickening of the skin) and atrophie blanche areas are high risk for new and recurrent VLUs. Although venous ulcers are the most common type of lower-extremity ulcer, it is important to rule out other potential etiologies that may result in lower-extremity ulcerations.

Investigations

Every patient with VLU and CVI must undergo workup to rule out other causes of edema (Tables 1 and 2). Ruling out concomitant peripheral arterial disease (PAD) is crucial in the management of venous disease, as it needs to be addressed before the treatment of venous disease. Venous duplex to assess valvular incompetence (venous incompetence test or venous reflux test) is the most important investigation in these patients (grade 1A

Table 1 Differential Diagnosis of Edema

Causes of Edema	
Cardiac	Venous insufficiency
Biventricular failure	Pelvic tumor or adenopathy causing extrinsic venous obstruction
Right-sided heart failure	May-Thurner syndrome
Hepatic	Idiopathic edema
Results in decreased protein synthesis	Medications
Renal	CCBs
Results in increased protein losses resulting in edema	Glitizones
Endocrine	Estrogens or steroids
Thyroid (myxedema)	NSAIDs
Cushing syndrome	Other
Lymphedema	Infectious or inflammatory
	Trauma or injury
	Premenstrual edema
	Pre-eclampsia
	Pregnancy

CCBs, calcium channel blockers; NSAIDs, nonsteroidal anti-inflammatory drugs.