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Management of venous ulcers in chronic venous insufficiency: a comprehensive review of etiopathogenesis, biomechanical failure, and evidence-based therapeutic strategies

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ABSTRACT

Chronic venous insufficiency (CVI)-induced venous leg ulcers (VLUs) represent the most advanced stage of venous disease (CEAP C6) and pose a significant clinical and socioeconomic burden. This review aims to evaluate the biomechanical failure of the calf muscle pump, the impact of environmental and systemic factors, and current evidence-based interventional strategies in VLU management. The pathophysiology of VLU is driven by ambulatory venous hypertension. Factors such as prolonged heat exposure, static occupational loading, and systemic comorbidities like diabetes mellitus and smoking further impair the healing process by promoting chronic inflammation and biofilm formation. A comprehensive literature review was conducted, focusing on key clinical trials including EVRA, ESCHAR, and RECOVERY, to analyze the efficacy of endovenous thermal ablations (EVLA, RFA), foam sclerotherapy, and advanced wound care modalities. Evidence confirms that early interventional treatment (ablation within 2 weeks of ulcer presentation) significantly accelerates healing rates. Biomechanical support via graduated compression therapy remains the gold standard for preventing recurrence. Furthermore, the role of "cell-friendly" wound cleansing using clean water, rather than cytotoxic antiseptics, is emphasized for maintaining the integrity of fibroblasts and keratinocytes. Successful VLU management requires a multidisciplinary approach focusing on three pillars: reducing venous pressure through early intervention and compression, activating the calf muscle pump through mobilization, and controlling systemic risk factors.

Keywords: Chronic venous insufficiency, venous leg ulcer, calf muscle pump, endovenous laser ablation, radiofrequency ablation, biofilm

INTRODUCTION

Chronic venous insufficiency (CVI) is a chronic and progressive disease characterized by the pooling of blood distally due to gravity, resulting from the loss of function in the valves of the lower extremity venous system or structural deterioration of the venous wall. Venous ulcers (VU) represent the most advanced clinical stage of this pathophysiological process and correspond to stage C6 in the CEAP classification.

Approximately 10% of the general population develops a chronic leg ulcer during their lifetime, the most common of which is venous pathology.[1] It is estimated that the prevalence of C2 disease ranges from 20% to 64%, and 5% of the general population presents with C3 to C6 disease. C5 and C6 disease are prevalent in 1% to 1.5% of the population. The prevalence of the disease increases with age, and approximately 4% of patients over the age of 65 have venous ulcerative disease.[2]

Epidemiological studies indicate that 70-80% of lower extremity ulcers are of venous origin. While the prevalence in the general population of Western societies is around 1-2%, this rate rises to 5% in individuals over 65 years of age.[3] More importantly, the recurrence rate of venous ulcers is remarkably high, at approximately 70%. This situation clearly demonstrates that treatment cannot be limited solely to local wound care; the underlying fundamental issue, venous hypertension, must be corrected (**Figure**).



Figure. Venous ulcer CEAP C6

CEAP C6: The most advanced stage of venous disease

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ETIOLOGY AND PATHOPHYSIOLOGY

The development of venous ulcers cannot be explained solely by anatomical valvular insufficiency. Genetic predisposition, environmental exposures, occupational habits, and biomechanical factors are major determinants accompanying this process.

The Calf Muscle Pump

The "peripheral heart" Contrary to popular belief, venous return from the lower extremities to the heart is dependent on the rhythmic function of the calf muscles rather than cardiac suction power. The gastrocnemius and soleus muscles are, therefore, frequently described as the "peripheral heart." Venous leg ulcers (VLU) are the most common and difficult-to-manage leg ulcers. They result from venous congestion caused by the insufficiency of the calf pump or dysfunctional venous valves.[4]

Systolic phase: The increase in pressure applied to the deep veins during muscle contraction ensures that blood is propelled proximally. During this phase, the valves of the perforating veins close, preventing reflux into the superficial system.

Diastolic phase: The negative pressure formed in the deep veins when the muscle relaxes draws blood from the superficial system into the deep system through the perforating veins, preparing for the next pump cycle.

The disruption of this physiological mechanism forms the fundamental biomechanical basis for venous hypertension and, ultimately, ulceration.

Heat Exposure and Static Occupational Loading

In individuals working at high temperatures (such as bakers, cooks, and foundry workers), vasodilation in the venous walls reaches maximum levels. Heat impairs the coaptation of the valves by reducing venous tone. Thermal conditions, in particular, appear to regulate symptom severity; many patients report that heat exacerbates discomfort, while cold alleviates it.[5] In static occupations requiring prolonged standing without movement, the calf muscle pump cannot be adequately activated. Consequently, blood pools distally, and the increased pressure in the capillary bed leads to tissue destruction that initiates an inflammatory vicious cycle.

SYSTEMIC AND LOCAL FACTORS HINDERING HEALING

Molecular Effects of Diabetes and Smoking

Venous ulcers transform into a classic "non-healing wound" model in the presence of systemic risk factors.

Diabetes mellitus (DM): Concerns regarding poorer treatment adherence are increasing, particularly among diabetics, as adherence to diabetes treatment and lifestyle modifications is generally lower. Furthermore, the risk of CVD (chronic venous disease) and peripheral arterial disease is higher in diabetic patients. Patients with CVD should always be screened for the presence of diabetes, as its presence impacts CVD symptoms, diagnostic methods, and treatment strategies.[6]

Advanced glycation end products (AGEs) bind to collagen and elastin fibers, reducing tissue elasticity and preventing wound contraction.

Hyperglycemia creates an ideal culture medium for bacteria while impairing leukocyte chemotaxis and phagocytic capacity.

Increased matrix metalloproteinase (MMP) activity causes the degradation of newly formed granulation tissue before it can mature.

Diabetic neuropathy and autonomic dysfunction lead to skin dryness and fissures, increasing the risk of secondary infections.

Smoking: While nicotine causes tissue hypoxia by producing vasoconstriction in the microcirculation, carbon monoxide binds to hemoglobin, reducing the effective amount of oxygen transported to the tissues. Therefore, the wound healing rate is significantly lower in patients who smoke.

Wound Microbiota and the Biofilm Barrier

Different types of biofilms can be produced depending on the ulcer environment. Its formation begins with the attachment of a bacterium in planktonic form to an exposed surface, which can be biotic or abiotic. Subsequently, microorganisms establish connections among themselves and attach irreversibly. Then, they begin to multiply and activate quorum sensing. The biofilm grows and differentiates into a mature biofilm community, exhibiting structural characteristics. Subsequently, a matrix consisting of polymeric substances such as water, polysaccharides, proteins, and extracellular DNA is formed. Finally, the mature biofilm initiates a dispersal process through the detachment of cells or microcolonies. Dispersal can lead to the colonization of new niches and thus new infection sites by the biofilm.[7]

The most significant microbiological issue in venous ulcers is biofilm formation. The polysaccharide matrix secreted by bacteria protects them from both the immune system and antibiotics. In the absence of active purulent infection, the use of aggressive antiseptics exerts a cytotoxic effect on fibroblasts and keratinocytes. Therefore, gentle mechanical cleaning performed with clean water of drinking quality or saline is the most scientific approach to removing the biofilm layer while preserving viable tissue.

Evaluation of Arterial Circulation

In mixed ulcers, peripheral arterial circulation must be evaluated in every patient with a venous ulcer. Concomitant arterial insufficiency is present in approximately 15-20% of cases. The negative impact of arterial and venous diseases on each other should be examined in detail, and separate strategies should be developed for each.[8] Ankle-Brachial Index (ABI) measurement is, therefore, of critical importance. In patients with an ABI <0.5, high-pressure compression may impair tissue perfusion, leading to serious complications that can progress to gangrene.

DIAGNOSTIC STEPS AND ALGORITHM

Diagnosis is established through careful clinical evaluation and radiological confirmation.

Physical examination: Generally, shallow and exudative ulcers with irregular borders are observed around the medial malleolus. Evaluation of peripheral pulses is mandatory.

Color Doppler ultrasonography: Reflux duration (>0.5 sec) at the saphenofemoral and saphenopopliteal junctions and sequelae of deep vein thrombosis are investigated. It is the gold standard.

Ankle-Brachial Index (ABI): It must be measured to exclude arterial insufficiency before compression therapy.

PHARMACOLOGY AND COMPRESSION: FUNDAMENTAL SUPPORT

Venotonic Medications

Realistic expectations: It has been reported that sulexollide treatment shows a better potential probability (48%) compared to pentoxifylline (37%) and micronized purified flavonoid fraction (MPFF) (16%). While MPFF is a more effective treatment in reducing lower leg volume, CIVIQ-20 score, and pain VAS scale, calcium dobesilate has been evaluated as more effective in reducing foot volume and ankle circumference.[9]

MPFF and similar venotonics do not correct valvular insufficiency. Their effects involve reducing leukocyte adhesion, suppressing inflammation, and providing edema control. The literature supports the use of these agents as adjuvants to surgical or compression therapy, particularly for 3-6 months during the active healing period. Lifetime use is not required.

Compression Therapy

The gold standard no advanced technology can replace a correctly applied compression stocking. Graduated pressure, which is highest at the ankle and decreases proximally, increases valve coaptation by narrowing the vein diameter and activates the calf muscle pump. A pressure of 30-40 mmHg (class III) is generally preferred in patients with venous ulcers. Compression therapy should be maintained for life to prevent recurrence even after the wound has healed.

A fairly recent meta-analysis comparison of seven studies reported that there is moderate evidence that compression therapy heals venous ulcers more easily, regardless of the type of compression system, as long as it can provide consistent graduated compression.[10]

INTERVENTIONAL AND SURGICAL TREATMENT STRATEGIES

Endovenous Thermal Ablation (EVLA and RFA)

In modern approaches, endovenous ablation techniques have replaced the classic stripping method. The long-term anatomical success rates of EVLA and RFA are over 90%. The EVRA study demonstrated that early intervention performed as soon as the ulcer is diagnosed significantly shortens the healing time.

Foam Sclerotherapy and Perforating Vein Treatment

Closure of insufficient perforating veins, particularly those beneath the ulcer bed, accelerates healing by directly reducing local venous pressure. While the recurrence rate is high when applied alone, it is highly effective when combined with ablation methods.

Surgery and the ESCHAR Study

The ESCHAR study revealed that the addition of superficial vein surgery to compression therapy significantly reduces recurrence rates rather than the rate of wound healing. The primary role of surgery is to prevent the reopened wound from recurring.

It provides evidence that polidocanol foam is another useful intervention that can be included in the toolbox used to address venous abnormalities in patients with leg ulceration due to chronic venous insufficiency. The data presented here, along with ESCHAR, EVRA, and other studies, support the concept of aggressively treating all sources of abnormal venous flow in patients with VLU, especially those who do not respond to compression protocols.[11]

MODERN WOUND CARE APPROACHES

Considering the cytotoxic effects of aggressive antiseptics, the goal in modern wound care is to provide a moist, clean, and cell-friendly wound environment.

- **High exudate wounds:** Alginates and hydrofibers
- **Moderate exudate:** Polyurethane foam dressings
- **Epithelialization phase:** Hydrocolloids
- **Signs of infection:** Short-term silver-containing dressings

The choice of dressing should be continuously re-evaluated based on the dynamic characteristics of the wound bed. Many international guidelines for leg ulcers recommend cleaning both the leg and the ulcer, and most advise cleaning with drinking water.[12]

The hidden danger of antiseptics: "Cytotoxicity" and the clean water miracle while harsh disinfectants kill bacteria, they also kill living cells such as "fibroblasts" and "keratinocytes" that work hard for wound healing.

Cellular toxicity: In vitro studies have shown that even a 1% povidone-iodine solution kills human fibroblasts within seconds. If fibroblasts die, the wound cannot close.

Modern approach: If there is no active, purulent infection in the wound, washing with water of drinking quality (or saline) and a mild soap is sufficient. This method removes the biofilm layer mechanically without damaging the healing tissue. Regular mechanical cleaning with clean water provides faster granulation (new tissue formation) than aggressive chemical use.

Dressing selection: Alginates should be preferred in exudative wounds, and foam dressings in the granulation phase. Silver-containing dressings should only be used for a short term for infection control.

CONCLUSION

Venous ulcers are a condition that dramatically reduces the patient's quality of life due to pain, odor, exudate, and social isolation. The need for long-term dressing changes and loss of workforce creates a serious economic burden. A successful treatment strategy is based on these three fundamental principles:

- **Reduce the pressure:** Compression and appropriate surgical intervention
- **Clean the wound:** Clean water and gentle debridement
- **Activate the muscle pump:** Walking and exercise

Permanent healing is not possible without smoking cessation, diabetes control, and early interventional treatment. While current cellular and biosynthetic treatments are promising, the gold standard today is the triangle of correct diagnosis, early intervention, and disciplined compression.

ETHICAL DECLARATIONS

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The author declares no conflicts of interest related to this study.

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Author Contributions

The author is solely responsible for the entirety of conception, execution, analysis, and writing of the manuscript.

REFERENCES

1. Chatterjee SS. Venous ulcers of the lower limb: where do we stand? *Indian J Plast Surg.* 2012;45(2):266-274. doi:10.4103/0970-0358.101294
2. Crawford JM, Lal BK, Durán WN, Pappas PJ. Pathophysiology of venous ulceration. *J Vasc Surg Venous Lymphat Disord.* 2017;5(4):596-605. doi:10.1016/j.jvsv.2017.03.015
3. Agale SV. Chronic leg ulcers: epidemiology, aetiopathogenesis, and management. *Ulcers.* 2013;2013:413604. doi:10.1155/2013/413604
4. Jindal R, Chaudhary P, Gupta B, Kaur T, Dhillon S. Venous ulcers: review article. *Indian J Surg.* 2023;85(suppl 1):121-132. doi:10.1007/s12262-021-03064-x
5. Serra R, Grande R, Butrico L, Fugetto F, De Franciscis S. Epidemiology, diagnosis and treatment of chronic venous disease: a systematic review. *Chirurgia.* 2016;29(2):34-45.
6. Jarošíková R, Roztočil K, Husáková J, et al. Chronic venous disease and its intersections with diabetes mellitus. *Physiol Res.* 2023;72(3):280-286. doi:10.33549/physiolres.935033
7. Goswami AG, Basu S, Banerjee T, Shukla VK. Biofilm and wound healing: from bench to bedside. *Eur J Med Res.* 2023;28(1):157. doi:10.1186/s40001-023-01121-7
8. Wang SH, Shyu VBH, Chiu WK, Huang RW, Lai BR, Tsai CH. An overview of clinical examinations in the evaluation and assessment of arterial and venous insufficiency wounds. *Diagnostics (Basel).* 2023;13(15):2494. doi:10.3390/diagnostics13152494
9. Pompilio G, Nicolaides A, Kakkos SK, Integlia D. Systematic literature review and network meta-analysis of sulodexide and other drugs in chronic venous disease. *Phlebology.* 2021;36(9):695-709. doi:10.1177/02683555211015020
10. Patton D, Avsar P, Sayeh A, et al. A meta-review of the impact of compression therapy on venous leg ulcer healing. *Int Wound J.* 2023;20(2):430-447. doi:10.1111/iwj.13891
11. Marston WA. Polidocanol sclerotherapy for venous insufficiency in patients with recalcitrant leg ulcers: more treatment is better. *J Vasc Surg Venous Lymphat Disord.* 2023;11(5):1082-1083. doi:10.1016/j.jvsv.2023.05.001
12. McLain NEM, Moore ZEH, Avsar P. Wound cleansing for treating venous leg ulcers. *Cochrane Database Syst Rev.* 2021;3(3):CD011675. doi:10.1002/14651858.CD011675.pub2