

# Venous Stasis Leg Ulcers: A Review

Gaus, D. Venous Stasis Leg Ulcers: A Review. *Práctica Familiar Rural*. 2020 marzo; 5(1).

David Gaus

Andean Health and DevelopmentUSA..

Orcid: <https://orcid.org/0000-0003-4166-3815>

DOI <https://doi.org/10.23936/pfr.v5i1.139>

Recibido: 12/11/2019 (En revisión)

## Abstract

Chronic venous stasis ulcers (CVSU) of the lower extremity affect up to 5% of the population over 65 years and 1.5% of the general population. CVSU is caused by chronic venous disease produced by venous hypertension. Venous hypertension results from valvular incompetence within the deep venous system, or by the obstruction of venous outflow. Both of these mechanisms produce poor venous return. Additionally, poor mobility and decreased calf muscle pump function are thought to be contributing factors. Life-long use of compressive therapy is indicated in patients with chronic venous disease in lower extremities. It reduces ambulatory venous pressure. These include bandaging systems, garments (stockings), or devices.

**Keywords:** venous stasis leg ulcers, Up to date, review

Úlceras venosas en extremidades inferiores por estasis venoso. Revisión

## Resumen

Las úlceras por estasis venoso crónico de las extremidades inferiores afectan hasta el 5% de la población mayor de 65 años y el 1,5% de la población general. Las úlceras venosas son causadas por una enfermedad venosa crónica producida por hipertensión venosa. La hipertensión venosa es el resultado de la incompetencia valvular dentro del sistema venoso profundo o por la obstrucción del flujo venoso. Ambos mecanismos producen un pobre retorno venoso. Además, se cree que la escasa movilidad y la disminución de la función de la bomba muscular de la pantorrilla son factores contribuyentes. El uso de terapia compresiva durante toda la vida está indicado en pacientes con enfermedad venosa crónica en las extremidades inferiores. Reduce la presión venosa ambulatoria. Estos incluyen sistemas de vendaje, prendas (medias) o dispositivos.

Palabras clave: úlceras venosas por estasis venosa, actualización, revisión bibliográfica

## Epidemiology

Chronic venous stasis ulcers (CVSU) of the lower extremity affect up to 5% of the population over 65 years and 1.5% of the general population. [\(1\)](#) Venous Thromboembolism (VTE) is a principal cause of chronic venous stasis ulcers. The

overall annual global incidence of VTE is reported to be 0.75 to 2.69 per 1000 population, demonstrating 6 million cases among 5.5 billion population in low and middle-income countries. [\(2\)](#) Underdiagnosed VTE in low and middle-income countries likely contributes significantly to the development of CVSU, particularly as the population ages. Appropriate care for venous leg ulcers can be inadequate



in up to half of patients and the lack of evidence-based treatment results in CVSU

## Pathophysiology

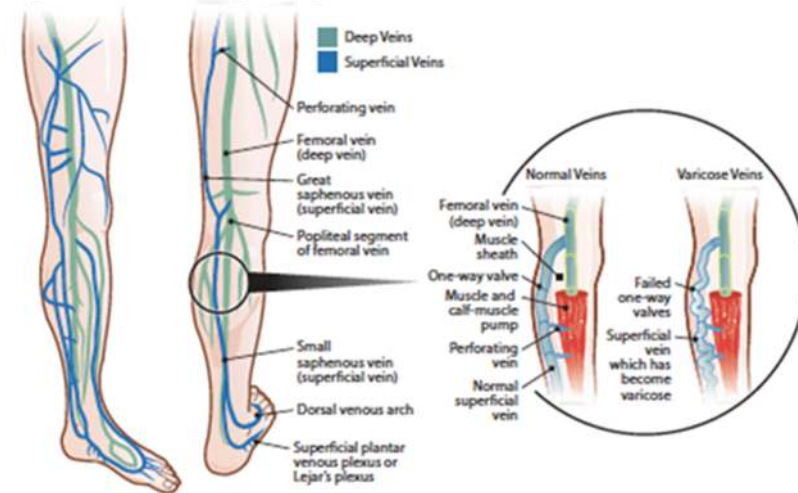
### Venous Hypertension

CVSU is caused by chronic venous disease produced by venous hypertension. Venous hypertension results from valvular incompetence within the deep venous system, or by the obstruction of venous outflow. Both of these mechanisms produce poor venous return.<sup>(4)</sup> Additionally, poor mobility and decreased calf muscle pump function are thought to be contributing factors.<sup>(5)</sup>

Venous hypertension from the deep venous system is transferred to the superficial venous system (fig. 1) through perforating veins, causing a chronic inflammatory process that is not well understood. White blood cells are trapped in capillaries or post-capillary venules. These white cells are then activated, releasing inflammatory mediators that result in tissue injury, inadequate healing, and, finally, necrosis. This "fibrin cuff theory" suggests that the fibrin deposited around these capillaries results in skin hypoxia.<sup>(6)</sup>

representing a significant public health challenge. <sup>(3)</sup>

**Figure 1. Anatomy of Superficial and Deep Venous System in Lower Extremity.**

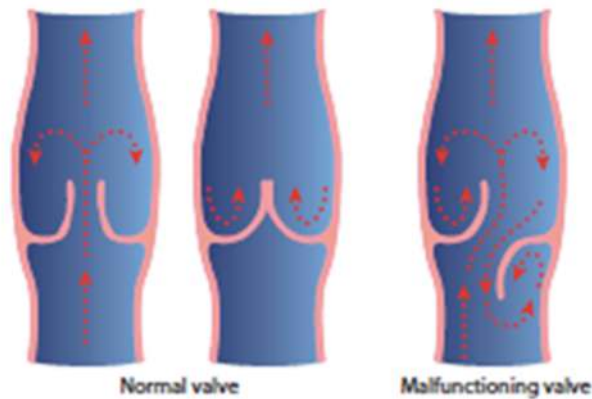


Source: Evans R, Kuhnke J, Burrows C, et al. Best practice recommendations for the prevention and management of venous leg ulcers. 2019 Canadian Association of Wound Care.

### Post-thrombotic Syndrome

Acute deep venous thrombosis (DVT) causes obstruction of venous flow. The thrombus leads to vein wall remodeling and inflammation, causing venous valvular damage.<sup>(7)</sup> (fig. 2) Incompetent valves leads to venous hypertension and resulting CVSU. Early identification and treatment of DVT is thought to reduce the risk of valvular damage.

**Figure 2. Normal and malfunctioning venous valves that lead to pooling and venous hypertension.**



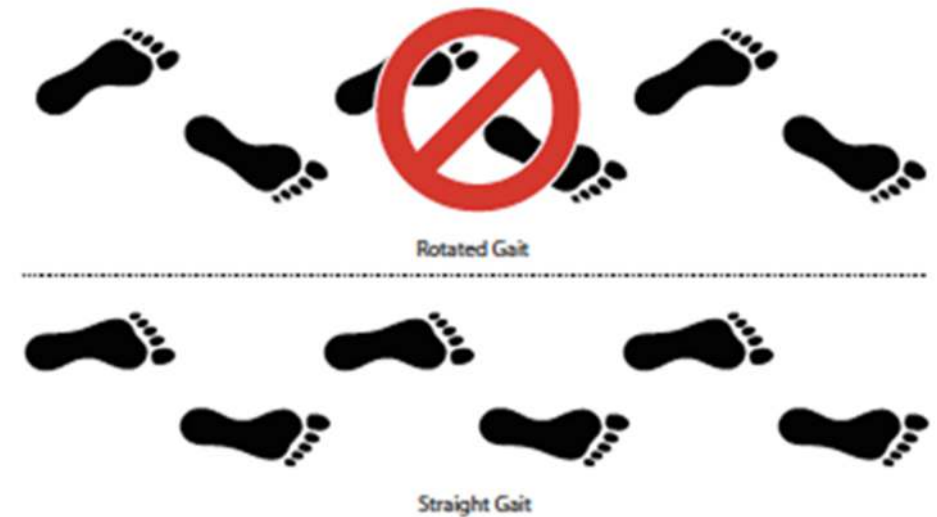
Source: Evans R, Kuhnke J, Burrows C, et al. Best practice recommendations for the prevention and management of venous leg ulcers. 2019 Canadian Association of Wound Care.

#### Calf-muscle pump

With the activation of the calf-muscle pump during normal ambulation, leg veins are compressed, decreasing venous pressures and pushing blood back towards the heart. For the pump to function properly, three requirements must be met:[\(8\)](#)

- Adequate range of motion into plantar and dorsiflexion of the ankle joint
- Adequate strength of the gastrocnemius and soleus muscles
- Appropriate heel-toe gait (fig. 3)

**Figure 3. Appropriate heel to toe gait to activate calf-muscle pump**



Source: Evans R, Kuhnke J, Burrows C, et al. Best practice recommendations for the prevention and management of venous leg ulcers. 2019 Canadian Association of Wound Care.

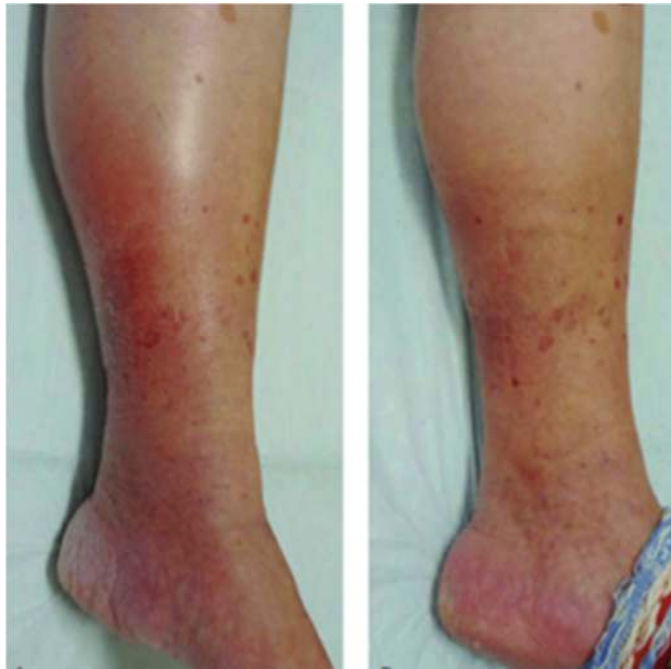
#### Clinical Presentation

Symptoms of chronic venous disease frequently develop before leg ulcers appear. These symptoms/signs include:

- pruritus
- varicosities
- heaviness
- edema
- eczema
- lipodermatosclerosis: an inflammation of subcutaneous tissue with brownish-red pigmentation (hemosiderin) and induration. (fig.4)

Symptoms tend to be worse at the end of the day, and can improve with leg elevation.

**Figure 4. Lipodermatosclerosis**



**Ulcer Characteristics**

Ulcer location is variable, but are commonly found in the "gaiter" area, particularly the



medial aspect, where perforating veins from the superficial system connect to the deep system and venous pressure might be highest. (fig. 5) Trauma may be associated with the development of the venous ulcer. At the time of presentation, the ulcer may be secondarily infected, with increased pain, exudate and malodorous.

**Figure 5. Chronic Venous Stasis Ulcer with chronic lipodermatosclerosis**



Up to 25% of venous ulcers are mixed venous/arterial. In these patients, cardiovascular risk factors should be assessed, along with symptoms of claudication. In areas of endemic Leishmaniasis transmission, other signs of venous stasis can help to clarify the etiology of the ulcer. (fig 6) Ankle Brachial Index (ABI) can also assist to distinguish arterial disease.

**Figure 6. Venous stasis ulcer potentially confusing for Leishmaniasis**

## Laboratory Analysis and Imaging

Complete blood count is useful to rule out anemia, polycythemia, and leukocytosis. Diabetes, dyslipidemia, and chronic renal disease should be excluded as well. Wound culture is only required in the presence of infection or cellulitis. Ultrasound study of the lower extremity can assess for venous incompetence and exclude ongoing venous thrombus. If arterial ulcers are suspected, Ankle Brachial Pressure Index (ABPI) can be measured. If the ABPI is less than 0.6, peripheral artery disease is likely and compression devices are contraindicated.

## General Care

### Leg Elevation

Limited evidence exists to support the use of leg elevation as a modality to prevent ulcer recurrence, as leg elevation with compression did not improve ulcer healing.<sup>(11)</sup> One study demonstrated a reduction in venous ulcer recurrence when leg elevation daily for at least one hour for at least 6 weeks was coupled with compression.<sup>(12)</sup>

### Exercise

Addressing calf-muscle pump failure is of significant therapeutic importance, as one study demonstrated a 60% prevalence among patients with venous ulcers.<sup>(13)</sup> Focus on calf muscle strengthening and stretching, particularly with compression, can improve pump function.<sup>(14)</sup> Progressive resistance exercise with prescribed physical activity can result in 9-45 additional venous ulcers healed per 100 patients. <sup>(15)</sup>

### Compression Therapy

Life-long use of compressive therapy is indicated in patients with chronic venous disease in lower extremities. It reduces ambulatory venous pressure. These include bandaging systems, garments (stockings), or devices. Before applying compression, the physician must be aware of contraindications to compression, which include:<sup>(16)</sup>

- peripheral arterial disease
- decompensated heart failure
- Ankle Brachial Pressure Index of less than 0.6

## Bandages

Compression bandages are divided into elastic and inelastic. Difficulty in applying the bandages, physical impairment, excessive wound drainage, and pain can be significant barriers to their use.

The Unna boot is the most common inelastic compression bandage. The boot is impregnated with zinc-oxide, which also provides relief from venous stasis dermatitis. It has limited utility due to incapacity to absorb highly exudative ulcers, and is indicated only in ambulatory patients with small ulcers.<sup>(17)</sup>

Elastic compression bandages are fitted to leg dimensions and are effective at rest and during ambulation. They require changing on an average of once per week.<sup>(18)</sup> Multiple layer systems (Fig 7) are superior to single layer for ulcer healing.<sup>(19)</sup>

Stockings & Devices  
Compression stockings demonstrate utility for ulcer healing and prevention.<sup>(20)</sup> They can be knee high, or thigh high, and are removed at night. One suggestion is to don them first thing in the morning before even getting out of bed, as venous pooling can begin as legs are swung to a sitting position at the bedside. They should be replaced approximately every 6 months due to normal wear. Intermittent Pneumatic Compression devices have not demonstrated effectiveness compared to other forms of compression and can be costly.

**Figure 7. Multiple Layer Compression Bandage**





## Dressings

Dressings can promote wound healing, and occupy a supporting role in compression. They come in many varieties, and are chosen based on protection, absorption, pain reduction, infection, odor control, and patient preference. Their is no robust evidence that one is superior to another when used with appropriate compression therapy.[\(21\)](#), [\(22\)](#), [\(23\)](#) The presence of matrix embedded with bacteria, or biofilm, on a wound such as a venous ulcer can impede healing. Up to 60% of non-healing wounds in one study demonstrated the presence of biofilm.[\(24\)](#) Certain dressings appear to be more effective at disrupting biofilm, such as cadexomer iodine and time-release silver gel.[\(25\)](#)

## Pharmacologic Therapy

### Pentoxifylline and Flavonoid

A hemorrheologic agent that affects blood viscosity and red cell deformability, pentoxifylline has demonstrated improved venous ulcer healing, alone, or with compression therapy.[\(26\)](#) Micronized purified flavonoid fraction (MPFF) is a vasotonic agent that can reduce venous distension and improve lymphatic drainage.[\(27\)](#) However, there was no difference in venous healing when compared with placebo.[\(28\)](#)

### Aspirin & Statins

A platelet inhibitor, and anti-inflammatory agent, aspirin, when coupled with compression therapy, promote faster healing of venous ulcers, but long term effectiveness remains unclear.[\(29\)](#) In one study Simvastatin demonstrated a higher rate of ulcer healing than matched placebo.[\(30\)](#)

## Debridement

There is limited evidence for the benefit of debridement. A review of 10 RCTs showed no benefit, but did not include sharp surgical debridement.[\(31\)](#) Two other studies of sharp surgical debridement as an office procedure showed significant reduction in wound size compared with no sharp surgical debridement.[\(32\)](#) The theoretical benefits of debridement include preparation of the wound bed, removal of necrotic tissue, biofilm disruption, and the identification of the extent

of the wound. Other forms of debridement such as collagenase and maggot therapy, offer no advantages over sharp surgical debridement.[\(33\)](#), [\(34\)](#)

## Surgery

Surgery can remove the incompetent superficial vein, diverting flow to the deep system. This reverses the effect of venous hypertension on the ulcerated skin. Procedures include vein ligation, stripping, laser ablation, and foam injection. The ESCHAR study, comparing surgery/compression versus compression alone, demonstrated similar healing rates, but lower recurrence in the surgical group.[\(35\)](#)

## Infected Venous Ulcers

Infection can impede wound healing. If local infection is evident, topical antibiotics can be used. If systemic infection is apparent, systemic antibiotics are indicated.[\(36\)](#) Routine cultures of wounds are not universally recommended, as colonization without infection might result in inappropriate antibiotic use. There is no evidence for the routine use of antibiotics to promote healing of venous leg ulcers.[\(37\)](#) When infected, polymicrobial coverage is frequently warranted.

## Bibliography

- [1](#) Etufugh CN, Phillips TJ. Venous ulcers. Clin Dermat. 2007. 25: 121-130
- [2](#) Jha AK, Larizgoitia I, Audera-Lopez C, et al. The global burden of unsafe medical care: analytic modelling of observational studies. BMJ Quyal Saf 2013;22:809-15
- [3](#) Guest JF, Fuller GW, Vowden P. Venous leg ulcer management in clinical practice in the UK: costs and outcomes. Int Wound J 2018;15:29-37. 10.1111/iwj.12814 29243398
- [4](#) Vivas A, Lev-Tov H, Kirsner RS. Venous leg ulcers. Ann Intern Med 2016;165:ITC17-32. 10.7326/AITC201608020 27479227
- [5](#) Comerota A, Lurie F. Pathogenesis of venous ulcer. Semin Vasc Surg 2015;28:6-14. 10.1053/j.semvascsurg.2015.07.003 26358304
- [6](#) Crawford JM, Lal BK, Durán WN, Pappas PJ. Pathophysiology of venous ulceration. J Vasc Surg Venous Lymphat Disord 2017;5:596-605. 10.1016/j.jvs.2017.03.015 28624002

- [7](#) Shbaklo H, Holcroft CA, Kahn SR. Levels of inflammatory markers and the development of the post-thrombotic syndrome. *Thromb Haemost* 2009; 101:505.
- [8](#) Shiman MI, Pieper B, Templin TN, Birk TJ, Patel AR, Kirsner RS. Venous ulcers: A reappraisal analyzing the effects of neuropathy, muscle involvement, and range of motion upon gait and calf muscle function. *Wound Repair Regen*. 2009;17(2):147–152.
- [9](#) Vivas A, Lev-Tov H, Kirsner RS. Venous leg ulcers. *Ann Intern Med* 2016;165:ITC17-32. 10.7326/AITC201608020 27479227
- [10](#) Hedayati N, Carson JG, Chi YW, Link D. Management of mixed arterial venous lower extremity ulceration: A review. *Vasc Med*. 2015;20(5):479–486.
- [11](#) Dix FP, Reilly B, David MC, et al. Effect of leg elevation on healing, venous velocity and ambulatory venous pressure in venous ulceration. *Phlebology*. 2005;20(2):87-94.
- [12](#) Finlayson K, Edwards H, Courtney M. Relationships between preventive activities, psychosocial factors and recurrence of venous leg ulcers: a prospective study. *J Adv Nurs*. 2011;67(10):2180-2190.
- [13](#) Gross EA, Wood CR, Lazarus GS, Margolis DJ. Venous leg ulcers: An analysis of underlying venous disease. *Br J Dermatol*. 1993;129(3):270–274
- [14](#) Orr L, Klement KA, McCrossin L, O’Sullivan D, Houghton PE, Spaulding S, Burke S. A systematic review and meta-analysis of exercise intervention for the treatment of calf muscle pump impairment in individuals with chronic venous insufficiency. *Ostomy Wound Manag*. 2017;63(8):30–43.
- [15](#) Jull A, Slark J, Parsons J. Prescribed exercise with compression vs compression alone in treating patients with venous leg ulcers: a systematic review and meta-analysis. *JAMA Dermatol*. 2018;154(11):1304-1311.
- [16](#) Andreissen A, Apelqvist J, Mosti G, Partsch H, Gonska C, Abel M. Compression therapy for venous leg ulcers: Risk factors for adverse events and complications, contraindications: A review of present guidelines. *J Eur Acad Dermatol Venereol*. 2017;31(9):1562–68.
- [17](#) Dolibog P, Franek A, Taradaj J, et al. A comparative clinical study on five types of compression therapy in patients with venous leg ulcers. *Int J Med Sci*. 2013;11(1):34-43.
- [18](#) Vivas A, Lev-Tov H, Kirsner RS. Venous leg ulcers. *Ann Intern Med*. 2016; 165(3):ITC17-ITC32.
- [19](#) Dolibog P, Franek A, Taradaj J, et al. A comparative clinical study on five types of compression therapy in patients with venous leg ulcers. *Int J Med Sci*. 2013;11(1):34-43.
- [20](#) Nelson EA, Bell-Syer SE. Compression for preventing recurrence of venous ulcers. *Cochrane Database Syst Rev*. 2014;(9):CD002303.
- [21](#) O’Meara S, Al-Kurdi D, Ologun Y, et al. Antibiotics and antiseptics for venous leg ulcers. *Cochrane Database Syst Rev*. 2014;(1):CD003557.
- [22](#) Tate S, Price A, Harding K. Dressings for venous leg ulcers. *BMJ*. 2018; 361:k1604.
- [23](#) Chapman S. Venous leg ulcer: An evidence review. *Br J Community Nurs*. 2017;22(Suppl 9):S6–S9.
- [24](#) James GA, Swogger E, Wolcott R, Pulcini ED, Secor P, Sestrich J, et al. Biofilms in chronic wounds. *Wound Repair Regen*. 2008;16(1):37–44.
- [25](#) Phillips PL, Yang Q, Davis S, Sampson EM, Azeke JI, Hamad A, et al. Antimicrobial dressing efficacy against mature *Pseudomonas aeruginosa* biofilm on porcine skin explants. *Int Wound J*. 2015;4(12):483–469.
- [26](#) Jull AB, Arroll B, Parag V, et al. Pentoxifylline for treating venous leg ulcers. *Cochrane Database Syst Rev*. 2012;(12):CD001733.
- [27](#) Scallon C, Bell-Syer SEM, Aziz Z. Flavonoids for treating venous leg ulcers. *Cochrane Database Syst Rev*. 2013;(5):1–41.
- [28](#) Martinez-Zapata MJ, Vernooij RW, Uriona Tuma SM, et al. Phlebotonics for venous insufficiency. *Cochrane Database Syst Rev*. 2016;(4): CD003229.
- [29](#) Del Rio Sola ML, Antonio J, Fajardo G, Vaquero Pureta C. Influence of aspirin therapy in the ulcer associated with chronic venous insufficiency. *Ann Vasc Surg*. 2012;26(5):620–629.
- [30](#) Evangelista MT, Casintahan MF, Villafuerte LL. Simvastatin as a novel therapeutic agent for venous ulcers: a randomized, double-blind, placebo-controlled trial. *Br J Dermatol*. 2014;170(5):1151-1157.
- [31](#) Gethin G, Cowman S, Kolbach DN. Debridement for venous leg ulcers. *Cochrane Database Syst Rev*. 2015;(9):1–63.
- [32](#) Cardinal M, Eisenbud DE, Armstrong DG, et al. Serial surgical debridement: a retrospective study on clinical outcomes in chronic lower extremity wounds. *Wound Repair Regen*. 2009;17(3):306-311.

[33](#) Falanga V. Wound bed preparation and the role of enzymes: a case for multiple actions of the therapeutic agents. *Wounds*. 2002;14(2): 47-57.

[34](#) Dumville JC, Worthy G, Bland JM, et al. Larval therapy for leg ulcers (VenUS II): randomised controlled trial. *BMJ*. 2009;338:b773.

[35](#) Barwell JR, Davies CE, Deacon J, Harvey K, Minor J, Sassano A, et al. Comparison of surgery and compression with compression alone in chronic venous ulceration (ESCHAR study): Randomised controlled trial. *Lancet*. 2004;363(9424):1854–59.

[36](#) International Wound Infection Institute (IWII). Wound Infection in Clinical Practice: Principles of Best Practice. Wounds International. 2016. Retrieved from: [www.woundinfection](http://www.woundinfection)

[37](#) O'Meara S, Al-Kurdi D, Ologun Y, Ovington LG, Martyn-St James M, Richardson R. Antibiotics and antiseptics for venous leg ulcers. *Cochrane Database Syst Rev*. 2014;(1):1–190.