

DEFINITION

The 2004 revised CEAP classification defines a chronic venous ulcer as a full-thickness defect of skin, present for > 30 days, that fails to heal spontaneously and is sustained by chronic venous disease. The ulcer is most commonly in the ankle area.

EPIDEMIOLOGY

Lower limb ulceration affects between 2-4% of people over 65 years causing significant morbidity and reducing the quality of life. Although more common in the elderly, 20% of patients are < 40 years old. Ulcers occur more commonly in females (2-3:1).

The ulcers tend to have a long history with one study showing 50% being present for > 9 months, 20% > 2 years and 8% > 5 years.

Recurrent venous ulceration occurs in up to 70% of those at risk.

PATHOPHYSIOLOGY

Venous leg ulceration is due to sustained **venous hypertension**, which results from chronic venous insufficiency. In the normal venous system, pressure decreases with exercise as a result of the action of the calf muscle pump. When the muscles relax, the valves in the perforating veins connecting the superficial to the deep venous circulation prevent reflux and the pressure remains low. The venous pressure remains high, however, when the valves are incompetent.

Although the definitive terminal process causing skin necrosis remains unknown, a number of pathophysiological mechanisms have

been proposed on how this skin damage occurs at a microcirculatory level. These include activation of leukocytes with release of inflammatory mediators and abnormalities in the transforming growth factor β -signaling pathway.

DIFFERENTIAL DIAGNOSIS

Venous stasis ulcers

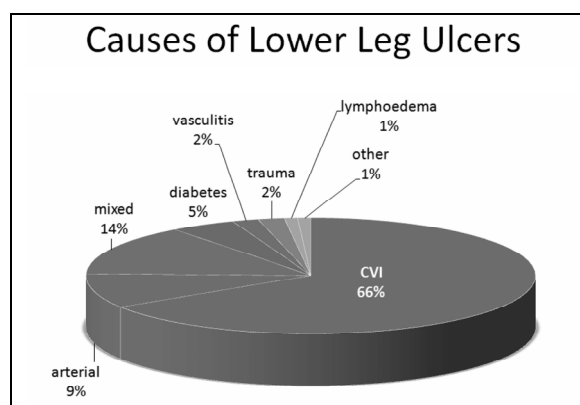


Fig 1: Causes of lower leg ulcers

Venous stasis ulcers account for 60-70% of lower limb ulcers. Classical features include:

- **Location:** The majority occurs in the area from the mid-calf to the ankle, known as the gaiter area. Most are located medially.

Ulcers presenting on the foot or above the mid-calf are less likely to be venous in aetiology and should be investigated for other possible causes.

- **Appearance:** Venous ulcers may be single or multiple and can range in size from small to circumferential. They tend to be irregularly shaped, are generally shallow, and rarely extend to muscle, fascia or bone.

There may be red granulation tissue or yellow fibrinous exudates on the ulcer bed; black necrotic tissue is rarely seen.

- **Hyperpigmentation:** extravasation of red blood cells into the soft tissue results in deposition of haemosiderin in macrophages that stimulate melanin.
- **Lipodermatosclerosis:** this is a chronic fibrosing process of the dermis
- Subcutaneous tissue related to venous insufficiency, resulting in firm and very indurated skin. In its late stages, chronic lipodermatosclerosis alters the shape of the leg, making it look like an inverted champagne bottle—where the proximal leg swells and the distal leg constricts due to loss of subcutaneous fat and fibrosis.
- **Venous eczema** (erythema, scaling, weeping, itching) is common.
- **Pitting oedema** is often present and may pre-date the ulcer
- **Varicose veins**

	Non venous	Venous
calf	8 %	5 %
gaiter	43 %	87 % { 70% Medial 30% Lateral
foot	49 %	8 %

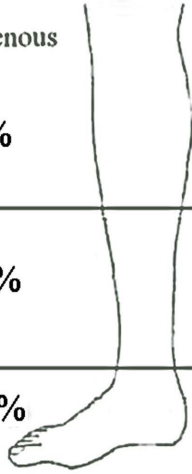


Fig 2: Distribution of Ulcers

Ischaemic ulcers

Purely ischaemic ulcers account for about 10% of lower limb ulcers. Ulceration typically occurs over the toes, heels, and bony prominences of the foot. The ulcers may be very painful, typically occurring at rest and may be alleviated by hanging the foot over the side of the bed or sleeping in a chair.

The ulcer appears “punched out,” with well demarcated edges and a pale, non-granulating, often necrotic base. The surrounding skin may exhibit dusky erythema and may be cool to touch, hairless, thin, and brittle, with a shiny texture. The toenails thicken and become opaque and may be lost. Gangrene of the extremities may also occur.

Examination of the arterial system may show a decreased or absent pulse in the dorsalis pedis and posterior tibial arteries. Capillary refill will be delayed.

Neuropathic ulcers

These ulcers are deep, punched out and situated over pressure points, particularly the heads of the metatarsals. Sensory, motor and autonomic nerves may be involved. The ulcers are usually painless, associated with foot deformities like clawing of the toes and a high arched foot and with dry skin due to decreased sweating.

These three groups account for 95% of lower limb ulcers.

Other

The less common causes are more difficult to characterize. Ulcers associated with diastolic hypertension (Martorell's ulcer) and vasculitis is caused by areas of infarction of the skin and are extremely painful without evidence of peripheral ischaemia. Rheumatoid arthritis is a common

cause. Polycythaemia and sickle cell anaemia are other rare causes.

HISTORY AND EXAMINATION

The aim is to accurately categorize the ulcer. This requires determination of the duration of ulceration, the nature of the pain, of any associated conditions such as diabetes, hypertension, rheumatoid arthritis, previous deep vein thrombosis, leg injuries and decreased mobility.

Examination must include assessment of both arterial and venous circulation noting the peripheral pulses, capillary and venous filling times, varicose veins, hyperpigmentation, eczema and lipodermatosclerosis. Sensation, proprioception, reflexes and joint mobility must be assessed. Signs of inflammation and infection must be noted.

INVESTIGATION

Investigations are directed towards confirming the diagnosis. It is very important to exclude underlying ischaemia requiring prompt intervention. Diabetic ulcers associated with infection may also require urgent treatment as the extent of the infection frequently is more advanced than is superficially apparent with underlying osteomyelitis frequently present.

Venous stasis ulcers require an assessment of the underlying cause. The degree to which deep or superficial venous incompetence contributes to the problem needs to be quantified.

As a very minimum, these patients require a duplex doppler examination to look for deep and superficial venous incompetence and/or evidence of obstruction.

The gold standard for assessing the venous function in CVI is the ***ambulatory venous pressure***. The test is performed standing, with and without walking on the spot (or

tiptoeing), with and without manual or tourniquet compression of the superficial veins to simulate superficial vein exclusion. The venous pressure, which is normally reduced during ambulation, and the time from stop of walking until pressure reaches its pre-walking level (recovery time) are the diagnostic criteria.

Plethysmography is an investigation that detects changes in the volume of the extremity. Different recording techniques are available, including mercury strain gauge (SGP), air-filled, water filled, impedance or photo (PPG) plethysmographs. The assessment of maximal venous outflow by using these techniques (except PPG) provides objective information on the presence and amount of venous obstructions. Plethysmography is also used in conjunction with specific manoeuvres to evaluate the vein muscle pump system and to detect venous reflux. PPG has been used extensively to mimic vein pressure recordings, especially the recovery time.

MANAGEMENT

Venous stasis ulcers

Compression is the mainstay of venous ulcer management. Graduated compression, with about 40 mm Hg at the ankle, tapering off to about 18 mm Hg below the knee, decreases the oedema and reduces the superficial venous pressure. Various compression bandage systems are used. The most effective systems usually comprise multiple layers with both elastic and non-elastic components.

The ankle-brachial index must be >0.8 for compression bandaging to be applied safely. The bandages can usually be changed once a week though initially this may be required twice weekly. The skin of the lower leg should be treated weekly with lanolin or aqueous cream. With careful

attention to detail about 70% of ulcers can be healed within 6 months.

Patients should be warned to remove the compression if they notice any side effects (such as numbness, tingling, pain, and dusky toes) and seek advice.

Sharp debridement of non-viable tissue may expedite healing of venous ulcers and can be done in the primary care setting. Surgery is normally indicated to correct superficial venous disease. Superficial venous surgery does not affect the rate of healing but does decrease the recurrence rate of ulcers.

Topical antibiotics should be avoided owing to the risk of increasing bacterial resistance and contact dermatitis. Associated venous eczema should be treated with topical steroids and emollients. The eczema may be secondarily infected and require systemic antibiotic therapy.

Once the venous ulcer has healed, it is essential that patients follow simple advice aimed at preventing the recurrence of the ulcer: this includes wearing compression stockings, skin care, leg elevation and calf exercises. The reported annual recurrence rate of venous ulcers (20%) is strongly influenced by patient adherence.

Arterial ulcers

The underlying ischaemic abnormality must be corrected. This requires assessment by colour flow duplex or arteriography to assess the possibility of re-vascularisation. Endovascular methods are attractive as bypass grafting in the presence of ulceration and infection carries an increased risk. Saphenous vein is preferable to prosthetic material in this situation.

Neuropathic ulcers

Diabetes is the commonest cause. Associated ischaemia and infection

complicate the management. Infection must be treated, blood supply improved (often with bypass grafting or angioplasty) and dead tissue debrided. Very careful attention must be paid to the pressure points and once healing has taken place footwear carefully designed to protect and reduce trauma is important.



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