Chronic ulceration of the leg

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Abstract

The assessment and treatment of patients with chronic leg ulcers represents an enormous clinical and financial burden to community and secondary care services in the United Kingdom. Most leg ulcers are due to chronic venous hypertension, although arterial compromise, malignancy and vasculitis are also recognized causes. The assessment of patients with chronic leg ulceration should include a detailed history and clinical examination, supported by relevant investigations. Ankle brachial pressure index measurement and non-invasive venous (±arterial) imaging using colour duplex are the principal investigations. The mainstay of treatment for patients with chronic venous ulceration is multilayer compression bandaging, applied by trained staff working within a specialist service bridging primary and secondary care settings. Endovenous treatment of superficial venous reflux may also play an important role in improving outcomes. Patients with arterial compromise may require endovascular or surgical revascularization to promote healing. In this article, the causes, assessment and management of patients with chronic leg ulceration are discussed.

Keywords Arterial ulcer; compression bandaging; superficial venous surgery; vascular surgery; venous ulcer

Definition

Chronic leg ulceration may be defined as:

- a breach in the epithelial integrity of the skin
- occurring between the knee and malleoli
- of greater than 6 weeks' duration.

Aetiology

The vast majority (>90%) of chronic leg ulcers have a vascular aetiology. Chronic venous hypertension is thought to be the primary cause of around 70% of leg ulcers and a significant contributory factor in a further 15%[1] (Figure 1). In addition to the primary aetiology, other factors may also delay wound healing and should be considered and treated where possible. These include dependent oedema (which is often poorly controlled), medical comorbidities, poor nutrition and medications (including steroids or immunosuppressive drugs). Arterial ulceration is usually due to multilevel occlusive peripheral arterial disease causing critical leg ischaemia. Other causes of leg ulceration include vasculitis and malignancy.

Epidemiology

Chronic leg ulceration is generally considered to be a disease of the Western world, with an overall prevalence of ulceration of around 0.5–1% in the adult population,[2] increasing to over 3% in patients over 80 years.[3] The female preponderance is more apparent in patients >60 years. Studies have suggested that the development of venous ulcers is related to severity of varicose veins, or previous deep vein thrombosis (DVT). It is certainly the case that many patients with venous ulcers do have evidence of venous disease (usually reflux, sometimes deep venous occlusion). However, as many patients with varicose veins or previous DVT do not develop ulcers, these relationships are not fully understood. Ulcers due to arterial disease are more common in men and are rare in patients <50 years.

Pathology and pathogenesis

Venous ulceration

Patients who develop venous ulcers have longstanding high pressure in the veins of the leg.[4] This 'chronic venous hypertension' is usually a result of incompetence (or reflux) in superficial or deep veins due to faulty valves, but may also occur in patients with venous occlusion or recanalization after DVT. Other factors such as immobility, obesity, ankle stiffness, leg dependency and poor calf muscle pump function may also contribute to venous hypertension. In the initial stages, patients with venous hypertension may be asymptomatic, but over time the patient may develop varicose veins, the skin may become inflamed (venous eczema), pigmented (haemosiderinosis) or thickened and scaly (lipodermatosclerosis) and eventually break down resulting in ulceration. The Clinical, Etiologic, Anatomic, Pathophysiological (CEAP) classification is used to describe the severity of venous disease[5] (Table 1). Although the sequence of skin changes is well described, the precise pathogenesis of ulceration due to venous hypertension is poorly understood. Proposed theories include the *fibrin cuff theory* (observation of peri-capillary fibrin cuffs which may reduce local oxygenation),[6] the *white cell trapping hypothesis* (trapped white blood cells may become activated resulting in cytokine release and local tissue damage)[7] and the *growth factor trapping theory* (growth factors important for healing are inhibited by large molecules which have leaked out of capillaries due to venous hypertension).[8] The typical location for a venous ulcer is the medial gaiter area of the leg, although lateral leg and foot ulceration (rare) may occur. Other venous skin changes are commonly seen with the ulcer (Figure 2).

Arterial ulceration

Risk factors for occlusive peripheral vascular disease include age, male sex, smoking, dyslipidaemia, diabetes and hypertension. Multilevel arterial stenosis or occlusion may result in critical limb ischaemia and tissue loss. Although arterial ulceration is often seen on the foot (or involving the toes), more proximal leg ulcers may also be seen (Figure 3). Patients with a combination of arterial and venous disease present a unique challenge, as identification of the primary ulcer cause (and main therapeutic target) may be difficult. A combination of clinical, anatomical

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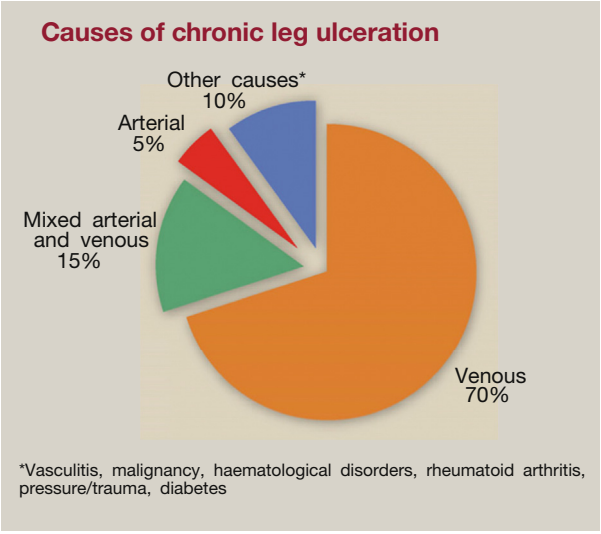

Figure 1

and haemodynamic assessments can be used to guide patient management.

Malignancy

Skin malignancies may present as chronic leg ulcers and should be considered in all ulcers with suspicious features or poor healing.[9] Basal cell (60%) and squamous cell carcinomas (40%) account for the vast majority. Malignant transformation (squamous cell carcinoma) of chronic venous ulcers is an occasional, but rare, occurrence (Marjolin's ulcer).

Other causes

Vasculitis, diabetes, rheumatoid arthritis and other systemic conditions (such as sickle cell disease or sarcoidosis) can cause leg ulceration and may be difficult to distinguish from venous ulcers. Biopsy of the ulcer edge for histological examination will usually provide accurate diagnosis of uncommon causes for leg ulceration.

Diagnosis

Clinical history and examination

The specific aims of patient assessment are to ascertain:

- the aetiology of the ulceration
- other contributing factors that may delay healing
- the impact of the ulcer on the patient
- patient expectations of treatment.

A detailed history may provide key clues to the aetiology of the ulcer and should provide strong clinical suspicions to be confirmed by physical examination and appropriate investigations. A history of DVT or varicose veins may indicate chronic venous hypertension, whereas a history of smoking, diabetes, other risk factors or symptoms of peripheral vascular disease may be more suggestive of an arterial component. Advanced patient age and, large ulcer size and long ulcer chronicity have been shown to be independent risk factors for delayed ulcer healing and may be useful prognostic indicators.[10] Moreover, a detailed knowledge of previous surgery, co-existing illnesses, medications and patient occupation may be important in planning treatment.

General clinical examination may reveal systemic illness or nutritional deficiencies contributing to poor wound healing. The ulcerated skin should be examined to identify the ulcer location, size, edges, signs of granulation and surrounding skin. Evidence of typical venous skin changes and a granulating ulcer in the medial gaiter area would be consistent with a chronic venous ulcer, whereas a painful punched-out ulcer in a limb with absent pulses may indicate an arterial cause. In order to monitor ulcer progression and response to treatment, ulcer assessment should include details of ulcer morphology, precise location and size, facilitated by use of wound tracings or photography.

Clinical, Etiological, Anatomical and Pathophysiological (CEAP) classification of chronic venous disease

CEAP clinical stage	Description
C0	Absence of any signs of venous disease
C1	Reticular veins
C2	Truncal varicose veins
C3	Oedema
C4	Skin changes (pigmentation, lipodermatosclerosis)
C5	Healed ulceration
C6	Open ulceration

Table 1

Investigations

Ankle brachial pressure index (ABPI): all patients with chronic leg ulceration should undergo ABPI assessment to identify underlying arterial compromise (Figure 4). Significant arterial disease may be excluded if ABPI >0.85, although falsely raised ankle pressures may be present due to calcification in the vessel wall. Patients with ABPI <0.85 should be considered to have arterial compromise and may require further specialist investigation or treatment.

Colour venous duplex scan: colour venous duplex uses a combination of Doppler and B-mode ultrasound to accurately map superficial and deep venous patency and competence in the ulcerated leg. Duplex is widely accepted as the investigation of choice to identify superficial venous reflux potentially amenable to surgery or endovenous intervention. Colour duplex scanning can also identify post-thrombotic scarring and occlusions in iliac and femoral veins.

Ulcer biopsy: as basal cell or squamous cell carcinoma may be a feature in up to 1% or more of chronic leg ulcers, wounds with a suspicious appearance or no evidence of healing after 3 months should be biopsied.[9] Ulcer edge biopsy may also help identify ulcers due to vasculitis or diabetes, which may be difficult to distinguish from venous or arterial ulcers.

Other investigations: tests of venous haemodynamic function are generally limited to research studies, but may be of benefit in some cases. Preoperative digital photoplethysmography (PPG) using a tourniquet has been shown to predict improvements after surgery and may identify good candidates for superficial venous intervention in patients with mixed superficial and deep venous reflux.[11] Further arterial investigations such as colour duplex or