Venous ulceration

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, in a system where the valves are incompetent.

Up to 10% of the population in Europe and North America has valvular incompetence, with 0.2% developing venous ulceration. Forty to fifty per cent of venous ulcers are due to superficial venous insufficiency and/or perforating vein incompetence alone with a normal deep venous system. There are many risk factors for venous ulceration. Recurrent venous ulceration occurs in up to 70% of those at risk. Many venous ulcers are painful, so appropriate pain relief and advice should be given.

Examination

Ninety five per cent of venous ulceration is in the gaiter area of the leg, characteristically around the malleoli. Ulceration may be discrete or circumferential. The ulcer bed is often covered with a fibrinous layer mixed with granulation tissue, surrounded by an irregular, gently sloping edge. Ulcers occurring above the mid-calf or on the foot are likely to have other origins.

Pitting oedema is often present and may predate the ulcer. It is often worse towards the end of the day. Extravasation of erythrocytes into the skin occurs, resulting in the deposition of haemosiderin within macrophages, which stimulates melanin production, pigmenting the skin brown.

In long term venous insufficiency, lipodermatosclerosis occurs. This is characterised by the dermis and subcutaneous tissue becoming indurated and fibrosed with the lack of pitting oedema; the skin also becomes atrophic, loses sweat glands and hair follicles, and becomes variably pigmented (ranging from hypopigmented to hyperpigmented). Severe lipodermatosclerosis may lead to atrophic blanche—white fibrotic areas with low blood flow. Lipodermatosclerosis often precedes venous ulceration.

As a result of lipodermatosclerosis, a rigid woody hardness often develops, which at its worst may result in the leg resembling an “inverted champagne bottle.” Venous eczema (erythema, scaling, weeping, and itching) is also common and is distinct from cellulitis.
Management

Compression is the mainstay of venous ulcer management (see also 11th article in this series). Graded compression, with greatest pressure (about 40 mm Hg) at the ankle, tapering off to lower pressure (about 18 mm Hg) below the knee, increases the limb hydrostatic pressure and concomitantly reduces the superficial venous pressure. Various compression bandage systems are used. These include the single and multilayer elastic bandage system, short stretch bandage, and elasticated tubular bandages (for example, Tubigrip). Compression with pneumatic devices (for example, Flowtron) has been used to promote healing of venous ulcers in patients with oedematous legs. 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 in an attempt to prevent ulcers from recurring. Shave therapy (excision of the whole ulcer) followed by skin grafting, or skin grafting alone, may be useful in patients where other treatments have failed.

Venous leg ulcers often become infected (see 10th article in this series for how to detect signs of infection). The most common organisms include Staphylococcus aureus, Pseudomonas aeruginosa, and \( \beta \)-haemolytic streptococci. Initially, these should be treated empirically (with broad-spectrum penicillin or macrolide or quinolone antibiotics) until definitive culture and sensitivities are available. Infection should be treated with a two week course of antibiotics. 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, calf exercises, and adopting a suitable diet. The reported annual recurrence rate of venous ulcers (20%) is strongly influenced by patient adherence. Local “leg clubs” (www.legclub.org) may help to reduce this rate.

Arterial ulceration

Arterial ulceration is due to a reduced arterial blood supply to the lower limb. The most common cause is atherosclerotic disease of the medium and large sized arteries. Other causes include diabetes, thromboangiitis, vasculitis, pyoderma gangrenosum, thalassaemia, and sickle cell disease, some of which may predispose to the formation of atheroma. Further damage to the arterial system occurs with concurrent hypertension through damage of the intimal layer of the artery. The reduction in arterial blood supply results in tissue hypoxia and tissue damage. Thrombotic and atheroembolic episodes may contribute to tissue damage and ulcer formation.

Peripheral vascular disease is most common in men older than 45 and women older than 55, and patients may have a family history of premature atherosclerotic disease. Modifiable risk factors for peripheral vascular disease include smoking, hyperlipidaemia, hypertension, diabetes, and obesity, with associated decreased activity. Patients may also have a history of generalised vascular problems, such as myocardial infarction, angina, stroke, and intermittent claudication.

Examination

Arterial ulceration typically occurs over the toes, heels, and bony prominences of the foot. 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. There may be bruits in the proximal leg arteries, indicating the presence of atherosclerosis.

Patients with arterial ulcers have a reduced capillary refill time. With normal capillary refill, after compression of the great toe or dorsum of the foot for a few seconds, the skin colour should return to normal in less than two to three seconds. Delay in return of the normal colour is indicative of vascular compromise. A delay of more than 10 to 15 seconds in return of colour after raising an ischaemic leg to 45 degrees for one minute (Buerger’s test) indicates vascular compromise. The ankle brachial pressure index is helpful in identifying peripheral vascular disease in the absence of non-compressible vessels resulting from vessel calcification (for example, diabetes) or tissue oedema. A duplex ultrasound scan will give further information on arterial occlusion, stenosis, and areas of diffuse and continuous atheromatous disease. Arteriography is the ideal investigation in preoperative planning, allowing direct assessment of the vascular anatomy of the lower limbs.

Management

Increasing the peripheral blood flow by, for example, reconstructive surgery (for diffuse disease) or angioplasty (for localised stenosis) is the intervention most likely to affect the healing process in arterial ulceration. Operative indications for chronic ischaemia include non-healing ulceration, gangrene, rest pain, and progression of disabling claudication.

The patient should stop smoking, and control of diabetes, hypertension, and hyperlipidaemia should be optimised. Patients may find benefits from sleeping in a bed raised at the head end. Patients should follow simple advice on foot and leg care. Walking is beneficial.

Infection can cause rapid deterioration in an arterial ulcer, and treatment with systemic antibiotics (along the lines for venous ulceration outlined above) should be started. Patients with rest pain or worsening claudication, or both, and a non-healing ulcer should be referred to a vascular surgeon; opioid analgesia may be necessary during the wait for surgery.

It is not appropriate to debride arterial ulcers as this may promote further ischaemia and lead to the formation of a larger ulcer.

Choice of wound dressings will be dictated by the nature of the wound. Vasoconstrictive drugs such as non-selective β blockers should be avoided. (See 11th article in this series for more information on drug treatment.)

Ulceration of mixed aetiology is not uncommon: patients may have a combination of venous and arterial diseases, resulting in ulcers of mixed aetiologies, which will limit the degree of compression (if any) that can be used.

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Severe eczema

- Very potent corticosteroid for 3-4 weeks (such as clobetasol propionate); also emollient*

Infected eczema

- Combination of highly potent corticosteroid, antiseptic and astringent agent such as potassium permanganate (1 in 10 000); and oral antibiotics

Mild eczema

- Moderately potent corticosteroid for 3-4 weeks (such as clobetasone butyrate); also emollient*

Weeping eczema

- As for infected eczema, but without oral antibiotics

No eczema

- Daily emollient*

*Such as aqueous cream or liquid and white soft paraffin (50/50)
Introducing ankle brachial pressure index

<table>
<thead>
<tr>
<th>Index</th>
<th>Signs and symptoms</th>
<th>Severity of disease</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 0.7-1</td>
<td>Mild intermittent claudication or no symptoms</td>
<td>Mild arterial disease</td>
<td>Reduce risk factors and change lifestyle; stop smoking, maintain weight, exercise regularly, consider antiplatelet agent</td>
</tr>
<tr>
<td>0.7-0.5</td>
<td>Varying degrees of intermittent claudication</td>
<td>Mild to moderate arterial disease</td>
<td>As for index ≥ 0.7-1, plus referral to outpatient vascular specialist and possible arterial imaging (duplex scan and/or angiogram)</td>
</tr>
<tr>
<td>0.5-0.3</td>
<td>Severe intermittent claudication and rest pain</td>
<td>Severe arterial disease</td>
<td>As for index ≥ 0.7-1, plus urgent referral to vascular specialist and possible arterial imaging (duplex scan and/or angiogram)</td>
</tr>
<tr>
<td>&lt; 0.3 or ankle systolic pressure &lt; 50 mm Hg</td>
<td>Critical ischaemia (rest pain &gt; 2 weeks) with or without tissue loss (ulcer, gangrene)</td>
<td>Severe arterial disease; risk of losing limb</td>
<td>Urgent referral to vascular emergency on-call team and possible surgical or radiological intervention</td>
</tr>
</tbody>
</table>

An index of 1-1.1 is considered to be normal. The data in the table should be used as an adjunct to the clinical findings. Erroneous readings may be the result of incompressible arteries secondary to presence of calciﬁcation or presence of tissue oedema. Patients may present with an arterial ulcer even with a normal index. Patients may present with an acutely ischaemic limb either due to an embolus or a thrombus (“acute on chronic” ischaemia) and should be referred as an emergency to a vascular specialist or emergency department for urgent intervention to prevent imminent limb loss.

Arterial ulceration often occurs after seemingly trivial trauma or as the result of localised pressure.

Guidelines for patients on protecting lower limbs and feet

- Examine the feet daily for broken skin, blisters, swelling, or redness
- Report worsening symptoms—for example, decreasing walking distance, pain at rest, pain at night, changes in skin colour
- Keep the skin moist with, for example, 50/50 white soft paraffin and liquid paraffin mix
- Never walk barefoot
- Ensure shoes are well fitting and free of friction and pressure points; check them for foreign objects (such as stones) before wearing; and avoid open toed sandals and pointed shoes
- Give up smoking
- Take regular exercise within limits of pain and tolerance

Further reading


Stuart Enoch research fellow of the Royal College of Surgeons of England, Keith G Harding director and professor of rehabilitation medicine (wound healing) at Cardiff and Vale NHS Trust, Wound Healing Research Unit, Cardiff University Competing interests: KGH’s unit receives income from many commercial companies for research and education, and for advice. It does not support one company’s products over another.

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