Topical Questions, Sound Answers

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Chronic Leg Ulcers



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The management of lower limb ulcerations can pose a daunting challenge to GPs. In fact, a recent study of Canadian FPs suggested that a large majority were not confident in their ability to appropriately treat such skin conditions.¹ While at first glance large leg ulcers may appear to be something not readily handled in the office, a careful approach guided by existing evidence can greatly facilitate their management without the need for specialist intervention. The goal of this article is to outline a practical approach to the diagnosis, management and prevention of chronic leg ulcers.

What are the causes and consequences of ulcers?

Ulcerations of the lower limb may be broadly defined as breaks in the layers of the skin below the knee that require prolonged periods of healing time. The most common cause of leg ulcers is chronic venous insufficiency (CVI). Other common causes include peripheral arterial disease (PAD) and neuropathic etiologies such as diabetes mellitus. Leg ulcerations due to CVI, PAD and diabetes are chronic conditions that require long-term treatment plans. In the face of inadequate management, lower limb ulcer recurrence rates can be as high as 80%.² Needless to say, the burden of such a disease has

significant impact on both healthcare provider and medical system costs; however, the debilitating effect on a patient's quality of life cannot be overstated and should be of foremost concern.

What are the approaches to diagnosis and treatment?

When a patient presents with a leg ulcer, the
 first principle of management is to uncover and
 treat the underlying cause. The patient interview
 therefore plays an important role in the direction of further management. A sense of ulcer
 etiology can often be gleaned by key historical
 data (Table 1). A general physical exam should
 include evaluation of the location and morphology of the ulcer, as well as surrounding skin with
 evidence of peripheral neuropathy and an assessment of peripheral pulses including an anklebrachial index (ABI) or systolic toe pressure.

Ulcer characteristics should be documented carefully in order to track the progress of the wound over time.

Initial laboratory investigations should be aimed at assessing the general status of the patient and can include a complete blood count with differential and a prealbumin. In patients < 50-years-of-age presenting with chronic

Table 1

Key historical data in discovering the underlying etiology of chronic leg ulcers

- Onset, localization, duration, course and recurrence
- Associated symptoms such as pain, edema, anesthesia, paresthesia, or claudication
- Exacerbating or relieving factors
- Previous history of deep vein thrombosis, surgery, or trauma
- Personal or family history of diabetes, obesity, or varicose veins
- Current medication profile
- Social habits such as smoking and alcohol consumption

ulceration of the lower limb, a coagulation workup can be considered including protein C, protein S, antithrombin III, Factor V Leiden and coagulation times. Routine bacterial swabs should not be performed unless clinical evidence of invasive infection exists.

Do not be afraid to debride. If the ulcer is not ischemic, the sharp debridement of any eschar or slough is beneficial to the healing process. Application of idocaine/prilocaine cream three hours prior to the debridement procedure is recommended. Following curettage, a simple antimicrobial dressing can be applied.

Should chronic leg ulcers be treated with systemic antibiotic therapy?

Routine swabs of ulcer sites often demonstrate growth of a variety of microorganisms. However, these results are usually due to surface contamination or site colonization—two conditions which do not impact the wound healing process. Current evidence suggests that



Figure 1. Venous ulcer on the medial malleolus with atrophie blanche.

non-specific treatment with systemic antibiotics does not facilitate the healing of chronic leg ulcers.³

The clinical features of common leg ulcer types are summarized in (Table 2). Venous ulcers are usually secondary to venous reflux, venous hypertension and calf muscle pump dysfunction. Common clinical findings include varicosities, pitting edema, woody fibrosis of the skin and porcelain-like atrophic changes (Figure 1). Aside from the general treatment principles mentioned above, the use of compression bandages and stockings greatly benefits venous ulcer healing and should be ideally worn for life to minimize recurrences provided that the patient's ABI is > 0.6. Choice of wound dressing should take into account factors such as wound exudates, quality of granulation tissue and undermining of the ulcer.

What medication can be used to promote the healing of venous ulcers?

Some venous ulcers remain unhealed despite proper compression therapy and some patients

Table 2 Common types of ulcers			
Venous	Medial malleolus	 Irregular shape Wet, shallow, minimal necrotic 	 Firm brawny edema Hemosiderin staining, dilated veins "Inverted champagne bottle leg" Atrophie blanche Eczema Palpable pulses, minimal pain, relieved when elevated
Aterial	Bony prominences (toes, ankles)	 Well-demarcated edges Necrotic tissue Deep, pale base 	 Poor peripheral pulses Very painful Pallor increased upon elevation
Diabetic	Pressure sites	 Punched-out appearance with thick rim of callus surrounding the ulcer 	 Evidence of peripheral neuropathy Often asymptomatic but many present with numbness or paresthesia

are not suitable candidates for compression. In these cases, oral pentoxifylline has also been shown to promote the healing process.⁴ Pentoxifylline acts by aiding blood flow to ulcer sites. The most common side-effect with pentoxifylline is GI upset. Dosing should be 400 mg Arterial ulcers are the product of insufficient arterial blood supply to the limb which results in tissue ischemia and necrosis. This is often secondary to atherosclerotic PAD. In contrast to venous ulcers, arterial ulcers present with well-demarcated edges, extreme pain and evidence of necrotic tissue (Figure 2). Specific management of arterial ulcers includes lifestyle modifications such as smoking cessation and regular exercise.

Otherwise, angioplasty, sympathectomy, or bypass surgery can be considered. In advanced cases, amputation may be necessary.



Figure 2. Well-demarcated arterial ulcer.

Diabetic ulcers are the most common neuropathic ulcers in Canada and the US. They are usually the result of loss of protective sensations (LOPS) and small arterial vessel disease. These



Figure 3. Diabetic ulcer on the plantar surface of the great toe.

types of ulcers should be suspected if the ulcer has a callous at the margin and the patient presents with numbness (Figure 3). An ABI measurement may be unreliable in a diabetic patient due to arterial calcification. Hence, systolic toe pressure should be measured. A systolic toe pressure of < 60 mmHg indicates that the ulcer is unlikely to heal without vascular surgery.

Educating patients in proper foot care can decrease the recurrence of diabetic foot ulceration.⁵

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Take-home message

- FPs can offer excellent management of chronic leg ulcers by following a practical approach
- 2. Principles of management include correction of the underlying etiology, promotion of the circulation, optimization of the local healing environment and prevention of recurrence
- Compression is the mainstay of therapy for venous ulcers
- Antibiotic therapy is not indicated in the treatment of chronic leg ulcers unless there is clinical evidence of invasive infection

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