Foot loss is one of the most feared complications of diabetes. The prevalence of foot ulceration in the diabetic population is high, with up to 10% of diabetic subjects developing a foot ulcer within their lifetime. Ulceration precedes more than 80% of all amputations. Acute foot complications of diabetes will frequently present to a physician. Timely and appropriate management will prevent more catastrophic problems.

**Aetiology of diabetic foot ulceration**

Diabetic foot disease has many different causes and presentations. Ulcers can be classified by aetiology as neuropathic or neuro-ischaemic, the clinical distinguishing features of which are summarised in Table 1, and by grade (Table 2). The neuro-ischaemic and neuropathic factors implicated in the aetiopathogenesis of foot ulceration are listed in Table 3.

Peripheral vascular disease is the key factor in determining clinical outcome and is the major factor in the development of ischaemic ulceration, critical ischaemia and subsequent gangrene, and hence the need for amputation. Atherosclerosis is related to long-term hyperglycaemia and the presence of conventional modifiable risk factors, including dyslipidaemia, smoking and hypertension. As in non-diabetic patients, there may be significant aortoiliac and superficial femoral arterial disease. In addition, there is a higher incidence of distal infrapopliteal arterial disease.

The majority of cases of diabetic foot ulceration are classified as neuro-ischaemic. It is the combination of the two fundamental factors of neuropathy and peripheral vascular disease, rather than either factor alone, which contributes to the clinical problem. This directly implicates the interaction of the peripheral and autonomic nervous systems with the defective diabetic microcirculation.

Other factors contribute to ulcer formation (Table 3). The presence of oedema is an ominous sign, enhancing the risk of infection, increasing the risk of shoe trauma and impairing local blood flow. Oedema may be a manifestation of coexistent cardiac or renal disease, but it is also related to the abnormal microcirculatory and endothelial function in the diabetic population.

**Prevention of foot ulceration**

Current understanding of the complex and multifactorial aetiology of foot ulceration suggests numerous social, organisational and medical pathways for prevention, which may be targeted at the different stages in the development of diabetic foot disease. These stages are:

- **Primary prevention**: a foot without clinical evidence of neuropathy or peripheral vascular disease
- **Secondary prevention**: a foot which is at risk, with features of neuropathy and/or vascular disease
- **Treatment of the acute foot**: a foot which has already developed ulceration and/or gangrene.

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**Table 1. Distinguishing clinical features of neuropathic and neuro-ischaemic diabetic foot ulcers.**

| Feature               | Neuropathic | Neuro-ischaemic |
|-----------------------|-------------|-----------------|
| Ulcer site            | Plantar aspect | Borders        |
| Callus                | Abundant    | Absent          |
| Foot pulses           | Present, may be strong | Absent        |
| Foot temperature      | Warm        | Cool            |

**Table 2. Wagner classification of diabetic ulceration by grade.**

| Grade | Description                                      |
|-------|--------------------------------------------------|
| 0     | High risk, no ulcer                             |
| 1     | Superficial ulcer, not clinically infected       |
| 2     | Deeper ulcer ± cellulitis, no abscess/bone infection |
| 3     | Deep ulcer with abscess/bone infection          |
| 4     | Localised gangrene (heel, toe or forefoot)      |
| 5     | Gangrene of whole foot                          |

**Table 3. Factors contributing to diabetic foot ulceration.**

| Aetiological classification: |
|-------------------------------|
| Peripheral vascular disease   |
| Peripheral neuropathy:        |
| autonomic                     |
| peripheral                   |
| Foot deformity                |
| Abnormal pressure loading and callus |
| Defective microcirculatory function: reduced tissue perfusion |
| Limited joint mobility        |
| Oedema                        |
| Other:                        |
| Socio-economic                |
| Poor vision                   |
| Previous amputation           |
| Inability to reach/examine feet|
| Poor understanding of diabetes|
| Inappropriate professional management|
| Poorly fitted shoes           |
Primary prevention

A key factor in the primary prevention of foot disease is education and training in foot care; this requires regular repetition and reinforcement.

A regular and structured risk assessment is normally carried out in primary care, and physicians should assess risk status at any new medical presentation. The aim of risk assessment is to define patients as:

- 'high risk' (intensive education and regular, expert chiropody needed)
- 'low risk' (standard advice and annual review assessment needed).

Risk assessment includes the removal of shoes and socks to inspect for foot deformity and the presence of excessive callus and ulceration, and a rapid clinical assessment of peripheral neuropathy by testing of ankle and knee reflexes, and measuring pin-prick, and vibration sense using a C128 Hz tuning fork. If abnormalities are present, it may be appropriate for the patient to enter a screening programme in which more sensitive tools, such as a Semmes Weinstein nylon monofilament4 or biothesiometry may be used to assess the presence of neuropathy. The peripheral circulation can be assessed by feeling the peripheral pulses. The absence of two or more pulses is an indicator of vascular disease.

The high-risk patient should be further assessed by the appropriate specialist (who may include the diabetic physician, vascular physician, and specialist chiropodist) or be referred to a multidisciplinary diabetic foot clinic.

The physician should be aware that there is good evidence that long-term blood glucose control is important in the prevention of neuropathy as an antecedent to the development of foot ulceration5. Peripheral vascular disease can be modified by the aggressive treatment of hypertension, lipid abnormalities and smoking. Aspirin treatment may be indicated.

Secondary prevention

The identification of the at-risk foot should initiate regular intensive foot care, education and chiropody, with provision of suitable footwear. This may take place in the specialist diabetic chiropody clinic, community clinic or hospital multidisciplinary diabetic foot clinic. Such a programme6, or as little as one hour of education,7, can reduce hospitalisation and amputation by 50–80%.

Treatment of the acute diabetic foot

The principles of treatment of the acute diabetic foot include the management of infection, debridement to remove necrotic and devitalised tissue, non-weight bearing, foot elevation to reduce oedema, and vascular assessment. The physician should perform a full systemic review, including investigation of the cardiovascular system with ECG, renal and fluid status. In the presence of infection, the patient may have uncontrolled diabetes and may require intensive insulin therapy.

Infection. The ulcer should be carefully examined for infection, with a search for evidence of systemic spread, which should be suspected even when clinical signs are absent. A routine culture of a dry surface swab may not indicate the organism responsible for cellulitis, but a culture of an exudate or deep tissue in the wound is helpful. In the antibiotic naïve patient, a single organism may be responsible for infection. With deep tissue infections, the presence of tissue necrosis, or previous antibiotic treatment, multiple organisms may be implicated, including Gram-positive cocci, Gram-negative bacilli, and anaerobic organisms which may be gas-forming. Antibiotic therapy should be directed according to local policy, but initial treatment will include flucloxacillin, amoxycillin and metronidazole.

Probing of ulcer. The wound or ulcer should be carefully probed. The ability to probe to bone is a strong pointer to the presence of osteomyelitis in about 85% of cases9. Formal diagnosis of osteomyelitis may be difficult because it can take three weeks for radiological changes to appear on plain X-rays. Standard 99Tc bone scans have a high sensitivity and low specificity, with false-positive scans indicating the presence of inflammation and increased blood flow, rather than infection. 111Indium-labelled leukocyte scans are more accurate, but again may reflect only soft tissue inflammation. The gold standard is magnetic resonance imaging.

Debridement. Debridement is an important adjunct of treatment of diabetic ulcers to remove necrotic tissue, assess the depth and extent of the ulcer, allow the exposure of undrained exudate and assess the involvement of tendon and bone. Enzymatic debriding agents have little role as primary treatment agents in the acute situation when there is a large amount of necrotic tissue. Patients who require wound debridement in the presence of palpable distal pulses may be referred to a dedicated general or orthopaedic surgeon as an alternative to a vascular surgeon.

Charcot foot. A rare, but serious, complication of the diabetic foot is the Charcot foot. In the initial stages of development, it may be confused with acute infection because of acute inflammation, hyperaemia and erythema. It may be possible to obtain a history consis-

Key Points: Prevention

- Up to 10% of diabetics develop foot ulcers in their lifetime
- Foot inspection is mandatory at each presentation of diabetic patients
- All diabetics should enter a regular programme of risk assessment
- Aggressive management of glycaemic control and correction of cardiovascular risk factors minimise the likelihood of foot ulceration
- Patient and staff education reduces amputation
tent with a spontaneous fracture or preceding trauma. Plain radiology may help to differentiate this from an acute infected foot by demonstrating evidence of bone fragmentation and fracture dislocation. A Charcot foot requires an adequate blood supply and does not occur in an ischaemic foot.

Vascular assessment. The vascular status of the foot may initially be assessed by the presence of foot pulses. Local factors may make this difficult, and all patients with foot ulcers should have non-invasive vascular testing. Measurement of the lower extremity arterial pressure, using ankle cuff and Doppler probe, enables the calculation of the ankle/brachial index (ABI) (normal value 0.9–1.1). Rest pain will occur with an ABI of 0.4, and the tissue is imminently in danger of necrosis below this value. Non-invasive laboratory testing frequently underestimates the severity of arterial disease in patients with diabetes, many of whom have a falsely elevated ABI as a result of a partially non-compressible arterial wall. An alternative assessment of blood flow may be useful; this can include the measurement of toe pressure, Doppler wave form analysis and measurement of transcutaneous PO₂ in an experienced vascular laboratory. If there is evidence of, or doubt about, arterial insufficiency, the patient should be urgently referred to a vascular service with a full range of modern diagnostic and therapeutic vascular techniques. These include specialist radiological services for arteriography, Doppler and duplex scanning, an interventional radiological service, and a vascular surgeon capable of performing distal revascularisation procedures. Diabetic patients do as well as non-diabetic patients after both angioplasty and vascular reconstruction, both of which reduce the need for amputation. New and established treatments and wound dressings have recently been systematically reviewed, the evidence for the management of diabetic foot disease is described in this review, and clinical guidelines are available.

Key Points: The acute foot

- Immediate referral to a specialist centre
- Full clinical review of patient
- Control diabetes with intravenous insulin
- Systemic infection may be silent
- Superficial swab may not include infective agent
- Culture exudate should be taken on deep tissue sample
- Surgical debridement
- A high probability of osteomyelitis if it is possible to probe to bone
- Assess vascular state
- The ankle/brachial pressure index may be falsely elevated and underestimate peripheral vascular disease

Summary

Diabetic foot ulceration is both preventable and treatable. The management of diabetic foot disease is best achieved through implementation of local protocols involving the primary care team, community care and the multidisciplinary diabetic team. It is important that the feet are assessed as part of the overall assessment of a diabetic patient at any clinical presentation. The initial management of the acute diabetic foot may present to a general practitioner or general physician, and involves the urgent assessment and treatment of infection, foot elevation, wound debridement and, where appropriate, referral for urgent vascular assessment in a specialist centre.

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