# The diabetic foot: recognition and principles of management

*Complications associated with the diabetic foot are the most common reason for admitting diabetics.* 

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Diabetes mellitus is increasing globally at an alarming rate. The disabling complications of the disease are draining the health care resources of both developed and developing nations. Admissions for diabetic foot-related complications to hospitals and clinics worldwide are the most common reason for hospital bed occupancy in this patient population and, in fact, 15% of the annual health care budget is used on treating the diabetic foot.

The World Health Organization has predicted that there will be 380 million diagnosed diabetics worldwide by 2025. Africa will face the second highest increase in prevalence of the disease – an expected increase of 160%, second only to the increased prevalence expected in the Middle East. This predicted increase equates to 760 million feet at risk of possible ulceration!

# Foot screening programmes that identify those feet at risk need to be taught to all health care professionals involved in the care of the diabetic patient.

The lifetime risk for foot ulcers in people with diabetes is estimated to be as high as 25%. Most of these ulcers are entirely avoidable. Knowing that 85% of all amputations done in diabetics are preceded by ulceration, it is of paramount importance that diabetic foot care protocols are developed to prevent ulceration and that they are implemented immediately. Independent risk factors for the development of a foot ulcer in the diabetic patient include long diabetes duration, the presence of peripheral neuropathy, peripheral vascular disease, a history of any prior foot ulcer, and prior amputation of either part of the ipsilateral foot or of the contralateral limb.

Foot screening programmes that identify those feet at risk need to be taught to all health care professionals involved in the care of the diabetic patient. Fig. 1 shows the typical neuro-ischaemic foot with high arch, claw toes and fatty pad thinning.



*Fig. 1. Typical neuro-ischaemic foot with high arch, claw toes and fatty pad thinning.* 

# Clinical examination of the diabetic foot and identification of the 'at risk' patient

It is crucial to note that not only those patients with longstanding diabetes, but any person with newly diagnosed diabetes, is at risk of developing foot complications, i.e. a diabetic foot.

The diabetic foot can be divided into two distinct entities – the neuropathic foot and the neuroischaemic foot. When in doubt, it is better to classify the foot as neuroischaemic.

The calamitous aetiological triad of diabetic foot ulceration, i.e. peripheral neuropathy, ischaemia and infection, is often accompanied by deformity of the foot and a history of either acute or chronic trauma, mostly the trauma caused by ill-fitting footwear.

Of the three above-mentioned factors, peripheral neuropathy remains the leading cause of initial foot ulcer formation in 78% of patients. However, the subsequent deterioration of the wound and its progression to gangrene, with secondary infection, is largely due to poor blood flow in 65% of those presenting with a non-healing ulcer (Fig. 2).

Fig. 2. A late presentation of forefoot gangrene due to decreased blood flow. This patient had no palpable pulses and both the ankle pressures and toe pressures were decreased.

The clinical examination of the diabetic foot consists of

- inspection
- palpation
- neurological assessment
- inspection of footwear.

### Inspection

The dorsum, sole, medial and lateral borders of both feet, as well as the inter-digital areas and back of the heel over the Achilles tendon, should be carefully inspected and assessed for callus, swelling, deformity and skin breakdown.

Corns and calluses, both of which develop at sites of high pressure and friction, may become the forerunner of ulceration in the neuropathic foot.

In-growing toenails, due to incorrect nail cutting and poorly fitting shoes, and fungal nail infections are one of the most common causes of inflammation and secondary infection in the diabetic foot, and must be identified promptly.

It is extremely important as well to distinguish the causes of either bilateral or unilateral swelling of the feet. Cardiac failure and renal impairment are commonly associated with bilateral swelling, while a unilateral hot, red, swollen foot is a Charcot foot until otherwise proven. Charcot foot, which involves inflammatory damage to the joints and bones of the foot in the acute phase, is a medical emergency. If the patient is not correctly diagnosed in this phase, and does not offload weight bearing from the affected foot, fracture and dislocation of the bones of the foot with permanent deformity will occur. Fibrofatty pad reduction over the heads of the metatarsal bones is a common sign of diabetic neuropathy, and this pad thinning reduces the ability of the diabetic foot to absorb increased plantar pressure and increases the risk of ulceration in this area.

Web-space maceration and skin blistering are often a sign of fungal infection, while fissuring and ulceration of the skin on the sole of the foot increases the risk of infection and amputation.

### Palpation

'Every diabetic foot needs a pulse.' In the Trans-Atlantic Inter-Society Consensus (TASC) document on the management of peripheral arterial disease, absent foot pulses in a diabetic equate to an acutely ischaemic limb until otherwise proven.

Peripheral vascular disease is on the increase in the general population, and with a four times higher incidence of peripheral atherosclerosis in diabetics compared with non-diabetics, revascularisation is required much more consistently in order to promote ulcer healing. The presence of peripheral arterial disease in the diabetic population also increases the risk of amputation tenfold.

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Palpation of foot pulses is crucial in the diabetic foot. The assessment of the femoral and popliteal pulses is equally important, because without sufficient inflow into the leg neuro-ischaemic ulceration remains a high risk.

If either the dorsalis pedis pulse or the posterior tibial pulse can be felt, then significant ischaemia of the foot is unlikely.

Checking both feet for increased temperature is also important. Warm or hot areas may indicate infection, a fracture or acute Charcot foot. The presence of oedema and/or crepitus indicates a foot at high risk for severe infection, and immediate referral to a specialised unit is required.

### Neurological assessment

Motor and autonomic neuropathy are usually easily diagnosed on inspection of the foot. The classic signs of motor neuropathy are a high medial arch, claw toes and metatarsal head prominence with fatty pad thinning. Dry skin and fissuring are the cardinal signs of autonomic neuropathy.

Sensory neuropathy is easily diagnosed in any patient who walks into the room in spite of extensive plantar ulceration!

Detecting lack of sensation is carried out using a 10 g monofilament which is applied perpendicularly to the skin surface of the foot for 2 seconds in various sites. These include the plantar aspects of the first toe, the first, third and fifth metatarsal heads, the skin of the heel and the dorsum of the foot. Obviously, all calluses should be removed prior to testing.

Fig. 3 shows extreme plantar callus formation with nail thickening and deformity due to fungal infection.



Fig. 3. Extreme plantar callus formation with nail thickening and deformity due to fungal infection.

Vibratory sensation using a 128 Hz tuning fork placed on the first toe and over the medial malleolus of each foot is a simple assessment to carry out if a monofilament is not available.

## Inspection of footwear

Both the patient's shoes and socks should be assessed. Patients with severe peripheral diabetic neuropathy usually wear shoes that are too small as well as too narrow. This increases the risk of increased friction and ulceration of the foot. The toe box of the shoe must be wide and deep, and the sole of the shoe should be thick enough to provide protection from penetration by any sharp objects.

Check that there are no foreign bodies in the shoes. Diabetic patients with sensory neuropathy are notorious for walking around on car keys, marbles, etc.

# The diabetic foot

### The diabetic foot

Socks should be absorbent, without prominent seams and must not be too thick, taking up space in the shoe.

Fig. 4 shows a Charcot foot deformity with heater burn wounds as a result of sensory neuropathy.



*Fig. 4. A Charcot foot deformity with heater burn wounds as a result of sensory neuropathy.* 

# special investigations

Special investigations to assist in the treatment of diabetic foot include:

- vascular investigations
- radiological investigations
- neurological investigations
- foot pressure assessment
- laboratory tests
- skin temperature assessment.

### Vascular investigations

The ankle brachial index (ABI), using a hand-held Doppler and blood pressure cuff, enables the clinician to calculate the extent of ischaemia and/or calcification present.

# Palpation of foot pulses is crucial in the diabetic foot.

In normal subjects the ratio of ankle systolic pressure to brachial systolic pressure is usually between 0.9 and 1.15. In the ischaemic foot the ABI is usually <0.9.

Systolic ankle pressures of <50 - 70 mmHg denote critical limb ischaemia and this, together with absent pulses, confirms the diagnosis of the high-risk poorly perfused foot.

It is important to note that in the case of extreme calcification of the arteries, a falsely elevated ABI is often still indicative of ischaemia.

Duplex Doppler with arterial waveform and blood vessel visualisation can be of help in deciding which patients will benefit from an open surgical or endovascular intervention. Further vascular investigations include toe systolic pressures. Although diabetics present with more severe infra-popliteal atherosclerotic disease than normal subjects, the smaller blood vessels in the forefoot and toes are often spared. This makes the toe brachial index a more accurate assessment of perfusion to the foot. Toe pressures of <30 - 50 mmHg are indicative of critical ischaemia.

Transcutaneous oxygen tension measurement,  $(TcPO_2)$  is also available to assess the degree of ischaemia more distally in the foot. Using heated electrodes placed on the skin, the level of oxygen that has diffused through the epidermis can be measured. If the TcPO\_2 is greater than 30 mmHg, wound healing can be anticipated.

### **Radiological investigations**

The extent of radiological investigation is always determined by the presenting abnormality detected clinically. A foot X-ray is usually sufficient to detect a fracture, gas in soft tissue, a foreign body, or a Charcot foot. Osteomyelitis can usually be diagnosed on MR investigation.

CT angiograms are often used to assess the extent of pathology in the blood vessels.

Arteriography is usually reserved for those patients who will be undergoing either open surgery or a minimally invasive endovascular procedure.

### Neurological investigations

A neurothesiometer can quantify the degree of neuropathy present. A vibratory stimulus is applied to the foot, and this increases as the voltage is raised. Any patient unable to feel a vibratory stimulus of 25 volts is at risk of ulceration.

### Foot pressure assessment

An assessment of the distribution of plantar pressure on the sole of the foot identifies the patient at risk for neuropathic ulceration. In-shoe foot pressures give a basis for the implementation of footwear adjustments.

### Laboratory tests

• Full blood count for the exclusion of anaemia.

- Electrolytes, urea and creatinine to assess baseline renal function
- Blood glucose and  $HbA_{1c}$
- Lipogram
- C-reactive protein.

The multidisciplinary foot care team is the most effective way to provide patient education, manage foot ulceration, infection and deformity.

### Skin temperature assessment

A digital skin thermometer enables the comparison between similar areas on each foot. This is very useful when managing the acute Charcot foot.

# Principles of management

The cornerstone of management of the diabetic foot is regular inspection and examination of the foot. Risk categorisation as set out by the International Working Group on the Diabetic Foot guides the frequency of visits and correlates well with ulcer incidence (Table I).

Treatment of the high-risk foot may be either non-surgical or surgical, but in both instances requires a team approach from a variety of specialists working together to salvage the foot at risk. At the same time, education of the patient and family members is crucial and should be simple, relevant and consistent.

The steps of successful management include the following:

- prompt detection and intervention of the high-risk foot
- medical management of diabetes and comorbid conditions
- antibiotic coverage

# Table I. Risk categorisation system

| Category | Risk profile                                                                        | Check-up frequency      |
|----------|-------------------------------------------------------------------------------------|-------------------------|
| 0        | No sensory neuropathy                                                               | Once a year             |
| 1        | Sensory neuropathy                                                                  | Once every 6 months     |
| 2        | Sensory neuropathy and signs of peripheral vascular disease and/or foot deformities | Once every 3 months     |
| 3        | Previous ulcer                                                                      | Once every 1 - 3 months |

- vascular work-up
- consultations with
  - · diabetologist
  - · infectious disease specialist
  - · foot and ankle surgeon
  - vascular surgeon
  - podiatrist
  - prosthetist/orthotist
  - physiotherapist
  - wound care sister
- post-surgical surveillance/wound care
- lifelong multidisciplinary clinic attendance.

Increased plantar foot pressure with callus formation remains a risk factor for ulceration. The removal of callus by a podiatrist, or a trained professional who is knowledgeable about the pathology of the diabetic foot, can reduce plantar pressure by up to 30%.

Both neuropathic and ischaemic ulcers are frequently complicated by infection. All non-viable tissue must be extensively debrided and any abscesses from the deep compartments of the foot must be drained. It is extremely important to send tissue specimens for culture, and in the case of suspected osteomyelitis, a bone biopsy is essential for diagnosis.

Immediate revascularisation must follow after debridement of foot ulcers in the ischaemic foot if wound healing is to take place.

Surgical procedures involve either foot salvage surgery or amputation with rehabilitation whenever possible. Surgery can be divided into elective surgical procedures, prophylactic surgical procedures or emergency surgical procedures. All these procedures can also be done in a minimally invasive way by means of arteriography with percutaneous transluminal angioplasty and/ or stenting.

# Conclusion

It has been shown in numerous studies in both the UK and the USA that healing foot ulcers in the diabetic population costs the health care system of any country much less overall, in the long term, than amputating the affected foot. Amputation rates will be significantly reduced if the following protocol is implemented:

- inspection of feet and footwear during patient's regular visits
- use of preventive foot and shoe care in high-risk feet (e.g. podiatry, protective shoes, education)
- implementation of a multifactorial and multidisciplinary approach to care for established foot ulcers
- early diagnosis of peripheral vascular disease and vascular intervention if required
- continuous follow-up of patients with previous foot ulcers
- registration of amputations and foot ulcers.

The role of the multidisciplinary foot clinic in reducing the amputation rate has proven itself worldwide in a variety of circumstances and economic resources. The multidisciplinary foot care team is the most effective way to provide patient education and manage foot ulceration, infection and deformity. The evidence continues to show that to achieve limb salvage rates of 95% or greater, no other more effective measure exists.

The ongoing wound care treatment required to close diabetic foot wounds has become an extremely important aspect of the multidisciplinary approach. Wound care *per se* is in fact a science that requires both experience and good judgement if the eventual outcome is going to be beneficial to the patient.

The absolute goal of treatment of the diabetic foot is the prevention of ulceration, the prevention of the recurrence of ulceration and, ultimately, the reduction of amputation. Every 30 seconds a limb is lost somewhere in the world because of diabetes. There has to be a radical change in the way we assess and treat these patients, and knowledge of current guidelines and protocols is essential if we are to achieve this.

### Further reading

Armstrong D, Lavery L. Diabetic Foot Study Consortium. Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomized controlled trial. *Lancet* 2005; 366(9498):1704-1710.

Boulton AJM. The pathway to ulceration: aetiopathogenesis. In: Boulton AJM, Cavanagh PR, Rayman G, eds. *The Foot in Diabetes*. 4th ed. Hoboken,NJ: Wiley & Sons, 2006: 51-67.

Boulton AJ, Vileikyte L, Ragnarson-Tennvall G, Apelqvist J. The global burden of diabetic foot disease. *Lancet* 2005; 366 (9498).

Edmonds ME, Foster AVM, Sanders LJ. A Practical

Manual of Diabetic Foot Care. Massachusetts: Blackwell Publishing, 2004.

'Facts & Figures: Did You Know' International Diabetes Federation. Available at http://www.idf. org

International Working Group on the Diabetic Foot. *International Consensus on the Diabetic Foot.* Maastricht: 1999 (www.idf.org/bookshop).

Levin ME, O'Neil LW, Bowker JH, Pfeifer MA, eds. *The Diabetic Foot.* 7th ed. Philadelphia: Mosby Elsevier, 2008.

Palumbo PJ, Melton LJI. Peripheral vascular disease and diabetes. In: National Diabetes Data Group, eds. *Diabetes in America*, 2nd ed (NIH publ. no 495-1468). Washington, DC: US Government Printing Office, 1995, pp 401-408.

# In a nutshell

### Neuropathic foot

- Is warm and well perfused with bounding pulses and distended veins due to arteriovenous shunting.
- Skin is dry with callus formation and heel fissures.
- Ulceration due to callus and high plantar pressures is common on the sole of the foot.
- Bone and joint deformity, called Charcot's osteoarthropathy, is common in longstanding diabetes.
- Toes may be clawed, and fat pad thinning is present over the metatarsal heads.
- Necrosis and gangrene develop secondary to infection in spite of good blood flow.

### Neuroischaemic foot

- Is cool and pulseless with delayed capillary filling.
- Dilatation of superficial capillaries can lead to a deceptively pink or red colour of the foot in spite of severe ischaemia.
- Ischaemic ulceration is more common around the edges of the foot at the tips of the toes and the back of the heels, associated with trauma, mostly from poorly fitting shoes.
- Even in the presence of neuropathy, heavy callus formation and thus ulcer formation on the sole of the foot is rare, as callus requires good blood flow.
- Necrosis and gangrene develop due to diminished tissue perfusion and secondary infection.