1 Introduction

Beseech you, tenderly apply
Some remedies for life.
(A Winter's Tale *III, ii, William Shakespeare)*

In this second edition, we still emphasize the importance of the simple staging system (fully described below), as a fundamental part of our approach to the management of the diabetic foot. By applying the staging process to all diabetic foot patients it is possible, through one simple assessment, to detect the conditions that need addressing and plan their treatment using simple but fundamental principles.

- Where there are high plantar pressures in the neuropathic foot leading to callus, or abnormal mechanical forces on the margins of the neuroischaemic foot, then the pressure must be relieved
- The ulcer is a pivotal event on the road to amputation and needs specialist treatment
- Ischaemia and infection are the greatest dangers faced by the diabetic foot patient
- When significant ischaemia is found in conjunction with a foot ulcer then urgent vascular intervention should be sought
- If infection is present it must be eradicated
- Good control of diabetes helps to prevent complications and heal foot problems
- Patients and health-care professionals who have been educated effectively are safer than patients and health-care professionals who are ignorant.

The importance of all the above points is reflected in the structure of the simple staging system.

We prefer to make the assessment simple, and *treatment-based*. We omit any non-essential aspects, which sometimes form a part of other assessment systems and make them unnecessarily complicated. For example, when examining ulcers we feel that the differences between a superficial ulcer and a deep ulcer do not really change the treatment and small shallow ulcers which go wrong can destroy the foot.

The simple staging system has therefore been developed as a practical, treatment-based tool that provides a framework for diagnosing and managing the diabetic foot, and this chapter describes the application of this system through:

- Practical assessment, which consists of history-taking, examination and investigations
- Classification of the diabetic foot into the neuropathic and neuroischaemic foot
- Staging the foot. The six specific stages in the natural history of the diabetic foot (Table 1.1) are described in this chapter
- Management plan for each stage.

The clinical assessment we describe is quick and simple, but will enable the practitioner to make a basic classification and staging and the foot can then be placed into the correct stage, which has specific implications for treatment. Investigations will be needed to assess severity so as to determine treatment.

Following this system will enable the practitioner to make rapid, effective decisions that will detect problems early, organize rapid treatment and prevent deterioration and progression.

Table 1.1 Stages of the diabetic foot

Stage l	Normal foot
Stage 2	High-risk foot
Stage 3	Ulcerated foot
Stage 4	Infected foot
Stage 5	Necrotic foot
Stage 6	Unsalvageable foot

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PRACTICAL ASSESSMENT

This can be divided into three parts:
- History-taking
- Examination
- Investigations.

History

Patients should be encouraged to be open and non-defensive. The history can be divided into the following sections:
- Presenting complaint
- Past foot history
- Diabetic history
- Past medical history
- Drug history
- Family history
- Psychosocial history.

Presenting complaint

Be aware that some patients may be asymptomatic due to neuropathy.

The presenting complaint is usually one or more of the following:
- Skin breakdown
- Oedema
- Colour change
- Pain, discomfort and abnormal sensations.

For skin breakdown, oedema and colour change or any other presenting complaints, the following questions may be helpful:
- Where is the problem?
- When did it start?
- How did it start?
- What makes it better?
- What makes it worse?
- How has it been treated?

As regards pain, this may be a specific complaint alone or it may accompany the above problems. Pain is always a significant symptom in the diabetic foot and should be taken very seriously. Pain may arise locally or it may be diffuse. Local sources may originate from bone, joint and soft tissue including skin and subcutaneous tissue. Generalized pain in both feet suggests neuropathy. Diffuse pain in a single foot suggests ischaemia. However, pain in the ischaemic foot should not always be blamed on reduced arterial perfusion because it may be caused by infection, and infection in the ischaemic patient will rapidly destroy the foot unless quickly treated.

In the neuropathic foot, severe infections can cause pain or throbbing. Pain around an ulcer suggests infection or ischaemia. Throbbing suggests infection. The following questions should be asked about pain:
- When did it start?
- How did it start?
- Was there an injury? Neuropathic patients are often unaware of injury
- Where is the pain?
- What is its nature?
- What aggravates the pain?
- What relieves the pain?
- When does it occur?
- Is it related to time of day or activity?
- What treatments have been given so far?

Clinical tips to diagnose pain due to neuropathy and ischaemia are shown in Table 1.2.

Patients may not complain of pain itself but of other abnormal sensations which would suggest neuropathy:
- Pins and needles (paraesthesiae)
- Unpleasant tingling (dysaesthesiae)
- Tightness (as if a constricting band is around the foot)
- A subjective feeling of coldness (which the patient may erroneously attribute to ischaemia) even though the temperature of the feet is warm
- A burning sensation
- Heaviness
- Numbness ('my feet feel as if they don't belong to me').

After discussing the presenting complaint, the rest of

Table 1.2 Clinical tips on pain

Pain due to neuropathy
- Burning pain with contact discomfort in both feet and lower legs which may also involve the thighs
- Sharp shooting (lancinating) or lightning pains like electric shocks, lasting a few seconds
- Pain relieved by cold
- Pain worse during periods of rest
- Unilateral burning pain in the leg with muscle wasting suggests a focal neuropathy, commonly a femoral neuropathy

Pain due to ischaemia
- Persistent pain, worse on elevation and relieved by dependency (hanging the leg out of bed)
- Pain in the calf on exercise relieved by rest (claudication). However, claudication is often absent in ischaemia because of concurrent neuropathy and the distal distribution of the arterial disease
- Feet with severe ischaemia may have little pain because of neuropathy

the history-taking is devoted to gathering important relevant information about the patient to aid diagnosis and management. This information can be acquired from various sources including direct questioning of the patient or his family, the patient's medical notes and the referral letter.

Past foot history
- Previous ulcers and treatment; other foot problems such as Charcot's osteoarthropathy, etc.
- Amputations:
  Major
  Minor
- Peripheral angioplasties
- Peripheral arterial bypasses.

Diabetic history
- Type of diabetes
- Duration of diabetes
- Treatment of diabetes:
  Insulin
  Oral hypoglycaemics.

Complications of diabetes

Retinopathy
- Background
- Proliferative
- Previous laser therapy
- Vitrectomy
- Cataract.

Nephropathy
- Proteinuria
- Severe renal impairment (estimated glomerular filtration rate [eGFR] ≤ 30 mL/min)
- Renal replacement therapy:
  Continuous ambulatory peritoneal dialysis (CAPD)
  Haemodialysis
  Renal transplant.

Cardiovascular
- Angina
- Heart failure
- Myocardial infarction
- Coronary artery angioplasty
- Coronary artery bypass.

Cerebrovascular
- Transient ischaemic attack
- Stroke.

Past medical history
- Serious illness (e.g. cancer, rheumatoid arthritis, etc.)
- Accidents
- Injuries
- Hospital admissions
- Operations.

Drug history
- Present medication
- Known allergies.

Family history
- Diabetes
- Other serious illness
- Cause of death of near relatives.

Psychosocial history
- Occupation
- Number of cigarettes smoked per day
- Number of units of alcohol per day
- Psychiatric illness; drug or alcohol dependency, etc.
- Home circumstances:
  Type of accommodation
  Lives alone
  Lives with friends or relatives.

Examination

There is a need for sensitivity on the part of the examiner. Many elderly patients find it difficult to describe their symptoms accurately and extreme patience is needed to draw out the required information. Many people will be fearful and anxious at their first visit. If, rarely, they have ischaemia but no neuropathy, or they have a severely infected foot, then they will be afraid that the examination will be painful. Other patients may be embarrassed about their feet, or may have very sensitive and ticklish feet. Before the feet are handled the patient should be reassured that the examination will not be painful and that everything will be explained. The feet should be grasped gently but firmly, and poking, prodding and tickling should be avoided. The toes should be separated gently: if they are pulled apart violently the skin between the toes may split.

The examination should be performed systematically. It consists of five parts:
- Inspection
- Palpation
- Neurological assessment
- Footwear assessment
- General examination.

Inspection

The feet should be fully examined in a systematic fashion: first the right and then the left, including dorsum, sole, medial border, lateral border, back of the heel, malleoli and interdigital areas, with a full assessment of the following:

- Skin
- Corns and callus
- Nails
- Oedema
- Deformity
- Limited joint mobility
- Colour
- Necrosis.

Skin

The general features of the skin should be assessed, especially looking for signs of skin breakdown.

In the neuropathic foot, the skin is dry and fissured and prominent dilated veins secondary to autonomic neuropathy may be visible. Hair loss can be a sign of neuropathy as well as ischaemia. Atrophy of the subcutaneous layer with a thin, shiny, wrinkled skin may indicate ischaemia.

The classical sign of skin breakdown is the foot ulcer. Ulcer assessment is described in Chapter 4. Abrasions, bullae and fissures also represent breakdown of the skin. Bullae are often the first sign of skin breakdown in the ischaemic foot. They are also a feature of fungal skin infections (tinea pedis), as is webspace maceration. Dry skin around the heel will form deep fissures unless an emollient is applied regularly (Fig. 1.1).

Look for other skin lesions, on the leg as well as the foot, including:

- Necrobiosis lipoidica diabeticorum
- Shin spots (diabetic dermopathy).

Necrobiosis lipoidica diabeticorum (NLD) is characterized as well-circumscribed red papules that extend radially with waxy atrophic telangiectatic centres (Fig. 1.2a,b). NLD evolves to ulceration in about one-third of cases and a case has been reported of association with malignancy.

The round or oval macular hyperpigmented lesions of diabetic dermopathy (Fig. 1.3) are found in the anterior tibial region.

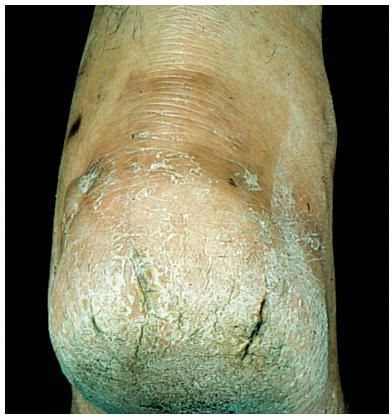
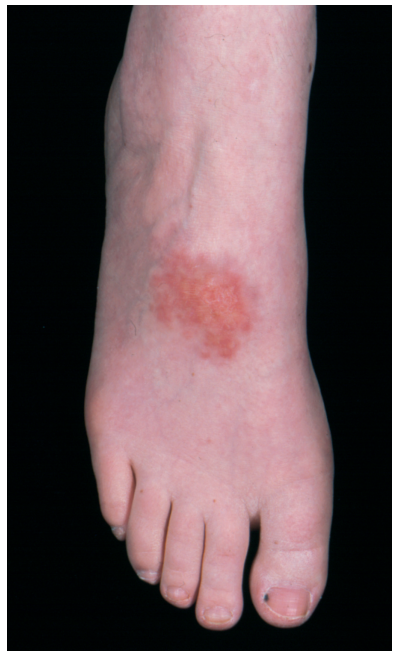
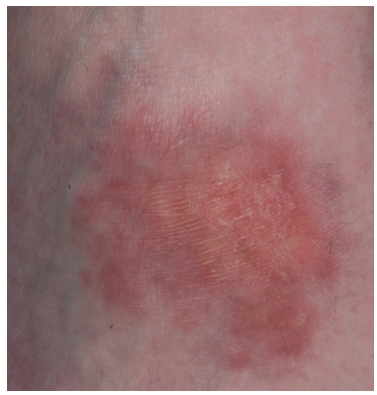


Fig. 1.1 Fissures are a portal of entry for infection and can lead to severe ulceration.



(a)



(b)

Fig. 1.2 (a) Necrobiosis lipoidica diabeticorum (NLD) on dorsum of foot. (b) Close-up of NLD.

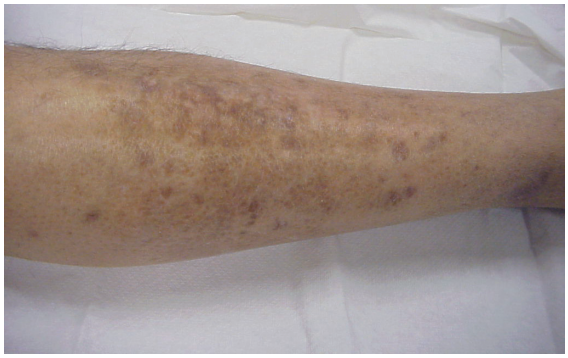


Fig. 1.3 Diabetic dermopathy.

As well as skin lesions specific to the diabetic foot, it is important to recognize inflammatory skin disease such as psoriasis, eczema and dermatitis, which also occur in non-diabetic patients but may complicate the diabetic foot and leg.

Corns and callus

These are thickened areas of keratosis which develop at sites of high pressure and friction (Fig. 1.4). Corns are discrete areas, usually not more than 1 cm in diameter, and can extend to a depth of several millimetres. Callus forms diffuse plaques. Neither should be allowed to become excessive as this is a common forerunner of ulceration (usually in the presence of neuropathy) and urgent action should be taken to deflect pressure from the area. Haemorrhage within callus is an important precursor of ulceration.

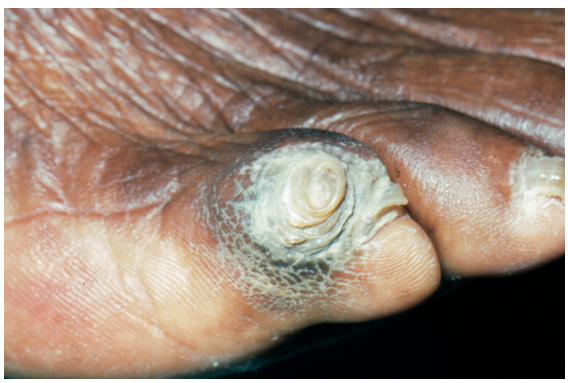


Fig. 1.4 Corn on the 5th toe.

Nails

It is important to inspect the nails closely as the nail bed and periungual tissues may become the site of ulceration. (Problems with nails are discussed fully in Chapter 2). The following should be assessed:

- Structure of the nails
- Colour of the nail bed
- Abnormalities under the nail
- Signs of nail infections.

Structure of the nails

Thickened nails are common in the population at large. If the shoes press on thickened nails they may cause bleeding under the nail. Eventually this may lead to ulceration. Atrophic nails may be present in patients with neuropathy and ischaemia.

Ingrowing toe nail (onychocryptosis) arises when the nail plate is excessively wide and thin, or develops a convex deformity, putting pressure on the tissues at the nail edge. Callus builds up in response to pressure and inflammation. Eventually, usually after incorrect nail cutting (when the nail is cut incompletely leaving behind a splinter of nail at the side which grows into the sulcus) or trauma, the nail penetrates the flesh.

Colour of nail bed

Red, brown or black discolouration of the nails may indicate subungual haematoma. The cause may be acute trauma or chronic trauma such as pressure from ill-fitting shoes (Fig. 1.5a).

In acute ischaemia the nail beds are very pale (Fig. 1.5b).

Abnormalities under the nail

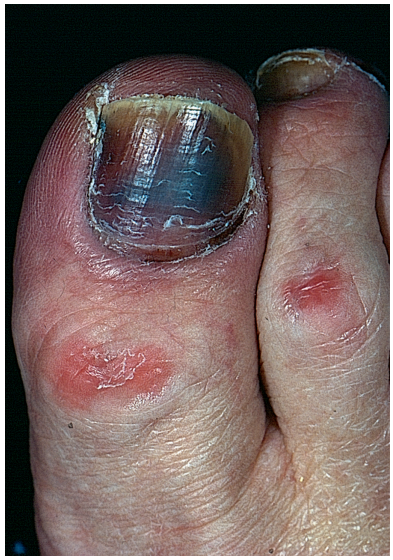
Discharge of fluid from beneath or around the nail, and any maceration or softness of the nail plate, may reveal the presence of a subungual ulcer or infection.

Nail infections

Fungal infection of the nail usually invades the nail plate dorsally causing onycholysis, where the nail comes away from the nail bed and is often thickened. The hallux nail is most commonly affected. Infection starts in one corner and over a period of years it spreads to involve the entire toe nail and may affect other nails.

Paronychia is associated with a nail that has a convex nail bed with tendency to incurve in the corners. Repetitive pressure in the insensitive foot can cause microtrauma in the nail groove, causing the nail to act as a foreign body thus creating a foreign body inflammatory response with secondary inflammation and localized infection (Fig. 1.6).

(a)



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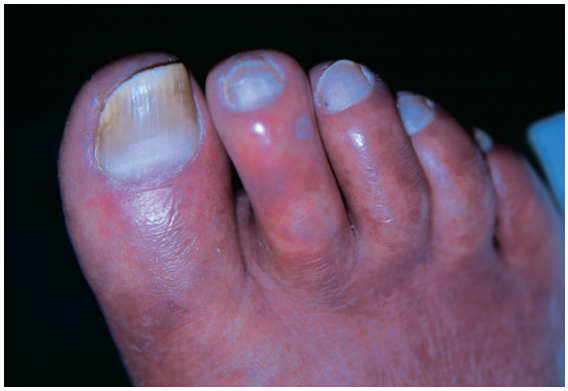


Fig. 1.5 (a) Subungual haematoma and red marks on toes resulting from wearing tight shoes. (b) Acute ischaemia—pale nail beds.

Oedema

Oedema is a major factor in predisposing patients to ulceration and delaying healing, and often exacerbates a tight fit inside poorly fitting shoes. The cause of oedema in the foot must be vigorously pursued and treated appropriately.

In patients on dialysis, oedema often fluctuates and becomes most marked just before dialysis. Appropriate arrangements need to be made for provision of adjustable footwear.

Oedema also impedes healing of established ulcers. It may be bilateral or unilateral and may involve the foot or be limited to the toes.

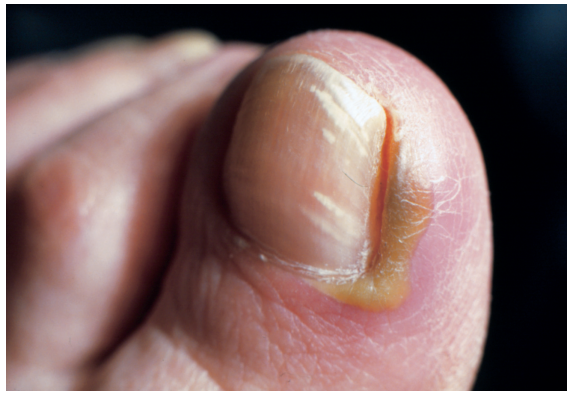


Fig. 1.6 Pressure on the sulcus from a convex nail has resulted in inflammation with secondary infection.

Causes of bilateral foot oedema include:

- Cardiac failure
- Renal impairment secondary to diabetic nephropathy
- Chronic venous insufficiency (sometimes unilateral)
- Rarely, neuropathic oedema secondary to diabetic neuropathy, when it is related to increased arterial blood flow and arteriovenous shunting
- Primary lymphoedema
- Secondary lymphoedema related to previous recurrent bilateral foot infections
- Severe ischaemia associated with dependency (often unilateral).

Causes of unilateral foot oedema are usually associated with local pathology in the foot or leg. These include:

- Infection, when it is usually associated with erythema and a break in the skin
- Charcot foot (a unilateral hot, red, oedematous foot; sometimes the oedema can extend to the knee)
- Gout, which may also present as a painful, hot, red, oedematous foot
- Trauma, fracture, muscle or tendon rupture, often associated with bruising
- Deep vein thrombosis
- Venous insufficiency
- Secondary lymphoedema due to malignancy or previous infection in the lower limb
- Common peroneal nerve palsy
- Localized collection of blood or pus in the foot, which may present as a fluctuant swelling
- In the early postoperative phase of revascularization of a limb.

In the tropics, filaria may be the cause of lymphoedema.

Chronic post-phlebitic limbs develop lymphoedema

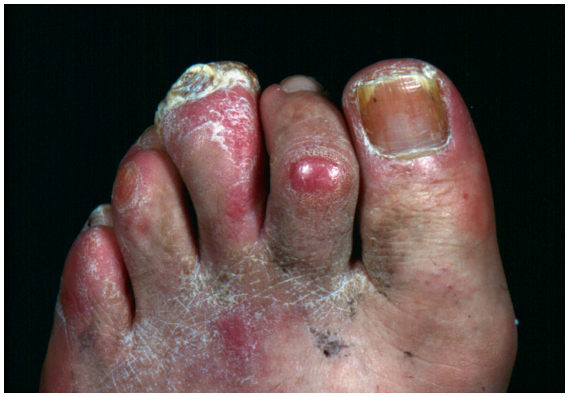


Fig. 1.7 Gout with tophi on second toe.

following repeated deep vein thromboses, with ligneous changes to the soft tissues.

Oedema of the toe can be due to:

- Trauma
- Fracture
- Soft tissue infection
- Osteomyelitis
- Gout (Fig. 1.7)
- Charcot toe.

Deformity

Common deformities include:

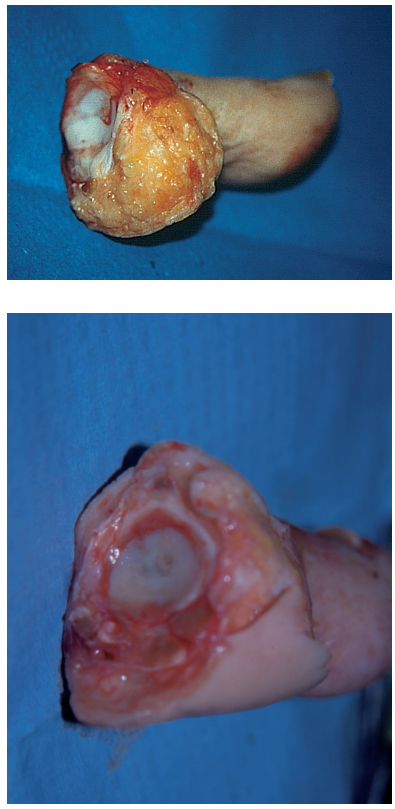
- Pes cavus
- Fibrofatty padding depletion (FFPD) or displacement
- Hammer toes
- Claw toes
- Hallux valgus
- Charcot foot
- Deformities related to previous trauma and surgery.

Pes cavus

Normally the dorsum of the foot is domed due to the medial longitudinal arch, which extends between the first metatarsal head and the calcaneus. When it is abnormally high, the deformity is called pes cavus and leads to reduction of the area of the foot in contact with the ground during walking. Resulting abnormal distribution of pressure leads to excessive callus formation and ulceration under the metatarsal heads. This deformity is a sign of a motor neuropathy but may be idiopathic. It is often associated with clawing of lesser toes or a trigger first toe.

Fibrofatty padding depletion (FFPD) or displacement

A common complication is reduction of the thickness of the fibrofatty padding over the metatarsal heads (Fig. 1.8a,b).



(a)

(b)

Fig. 1.8 (a) Amputation specimen from 29-year-old non-diabetic patient showing thick fibrofatty padding. (b) Amputation specimen from 29-year-old diabetic patient with history of neuropathy and ulceration showing great reduction in fibrofatty padding.

Normal feet contain cushions of fibrofatty padding over the metatarsal heads which absorb plantar pressures. In diabetic neuropathy the fibrofatty padding may be pushed forward or depleted by previous ulceration, rendering the plantar metatarsophalangeal area a site of abnormally high pressures and prone to ulceration.

Hammer toes

A hammer toe is a flexible or rigid deformity characterized by buckling of the toe. The toe takes on the configuration of a swan's neck. In people with diabetic neuropathy, hammer toes are commonly caused by weakness of the small intrinsic muscles (interossei and lumbricals) of the foot, which can no longer stabilize the toes on the ground. Muscle imbalance results in the affected toes sitting slightly back and up on the metatarsal head.

This deformity results in increased pressure over the metatarsal head, over the prominent interphalangeal joint and at the tip of the toe.

Claw toes

Claw toes are similar to hammer toes, but with more buckling and greater deformity. There is fixed flexion deformity at the interphalangeal joint, associated with callus and ulceration of the apex and dorsal aspect of the interphalangeal joint. Although claw toes may be related to neuropathy, they are often unrelated, especially when the clawing is unilateral and associated with trauma or surgery of the forefoot. Claw toes may rarely result from acute rupture of the plantar fascia.

Hallux valgus

Hallux valgus is a deformity of the first metatarsophalangeal joint with lateral deviation of the hallux and a medial prominence on the margin of the foot. This site is particularly vulnerable in the neuroischaemic foot and frequently breaks down under pressure from a tight shoe.

Charcot foot

Bone and joint damage in the tarsometatarsal joints and mid-tarsal joints leads to two classical deformities: the rocker-bottom deformity, in which there is displacement and subluxation of the tarsus downwards, and the medial convexity, which results from displacement of the talonavicular joint or from tarsometatarsal dislocation. Both are often associated with a bony prominence which is very prone to ulceration and healing is notoriously difficult.

When the ankle and subtalar joints are involved, instability of the hindfoot can result. These patients with hindfoot Charcot's osteoarthropathy are far more difficult to manage than patients with Charcot joints in the forefoot or mid-foot.

Deformities related to previous trauma and surgery

Deformities of the hip and fractures of the tibia or fibula lead to shortening of the leg and abnormal gait, which predisposes to foot ulceration. Ray amputations remove the toe together with part of the metatarsal. They are usually very successful but disturb the biomechanics of the foot leading to high pressure under the adjacent metatarsal heads. After amputation of a toe, deformities are often seen in adjoining toes. We have seen three cases of development of a grossly inverted foot following 5th ray amputation where tendon attachments of the peroneal muscles to the base of the 5th metatarsal were destroyed (Fig. 1.9).

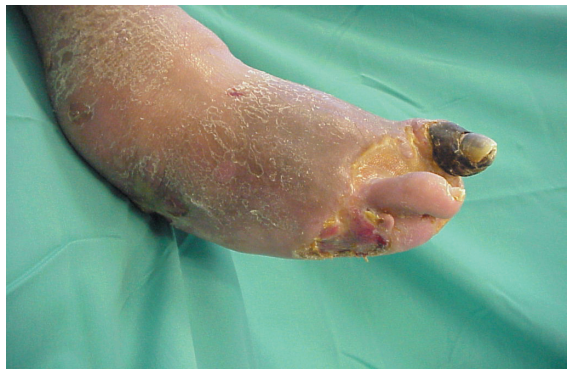


Fig. 1.9 Inversion deformity following surgical debridement.

Limited joint mobility (including hallux rigidus)

Limited joint mobility can affect the feet as well as the hands and involves the ankle, subtalar, mid-tarsal and toe joints. The range of movements in these joints will partially depend on the age of the patient but can be reduced by diabetes itself. In the diabetic foot, the two main joints to test are the ankle and the first metatarsophalangeal joint. For both of these joints the range of dorsiflexion and plantarflexion should be tested. Limited joint mobility of the first metatarsophalangeal joint results in excessive forces on the plantar surface of the first toe, which is unable to dorsiflex at the toe-off phase of the gait cycle, causing callus formation and ulceration on the underside of the hallus. It is common in barefooted and sandal-wearing populations. A plantar-flexed foot, with limitation of movement at the ankle joint commonly leads to plantar forefoot ulceration.

Colour

It is important to observe the colour of the foot including the toes. Colour changes may be localized or diffuse. Common colour changes are red, blue, white or black (Fig. 1.10).

Causes of the red foot

- Cellulitis
- Critical ischaemia, especially on dependency (dependent rubor)
- Charcot foot
- Gout
- Burn or scald.

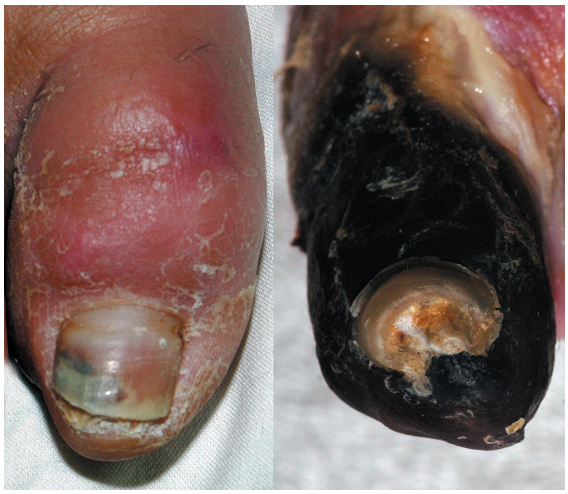


Fig. 1.10 Toes; red, and black. The red toe has cellulitis, and the black toe is gangrenous.

Causes of the red toe
- Cellulitis
- Osteomyelitis
- Ischaemia
- Gout
- Chilblains
- Dermatitis/eczema.

Causes of the blue foot
- Cardiac failure
- Chronic pulmonary disease
- Venous insufficency (often with brownish pigmentation—haemosiderosis).

Causes of the blue toe
- Severe infection
- Ischaemia.

The foot may have a pale white appearance in severe ischaemia, especially on elevation. In acute ischaemia, the foot is pale, often with purplish mottling. Emboli to the digital arteries can also result in a blue toe. In Raynaud's disease if there is acute vasospasm the toe becomes white.

Necrosis

Areas of necrosis and gangrene can be identified by the presence of black or brown devitalized tissue. Such tissue may be wet (usually related to infection) or dry.

Fig. 1.11 Henna has been applied to this hand. Note the black fingertips. This can be mistaken for gangrene.

Causes of the black toe
- Severe chronic ischaemia
- Acute ischaemia
- Emboli
- Bruise
- Blood blister
- Shoe dye
- Dirt
- Application of henna (Fig. 1.11)
- Tumour (melanoma).

Palpation

Palpation should take place to assess:
- Pulses
- Temperature of the foot
- Oedema
- Crepitus.

Pulses

The most important manoeuvre to detect ischaemia is the palpation of foot pulses, an examination which is often undervalued.
- The dorsalis pedis pulse is palpated, using the index, middle and ring fingertips together, lateral to the extensor hallucis longus tendon on the dorsum of the foot (Fig. 1.12)
- The posterior tibial pulse is palpated below and behind the medial malleolus (Fig. 1.13).

If either of these foot pulses can be felt then it is highly unlikely that there is significant ischaemia in the foot.

If both pulses are absent, the patient should undergo Doppler examination to measure the ankle brachial pressure index and to record the blood velocity profile or sonogram. This is described under Investigations, below.

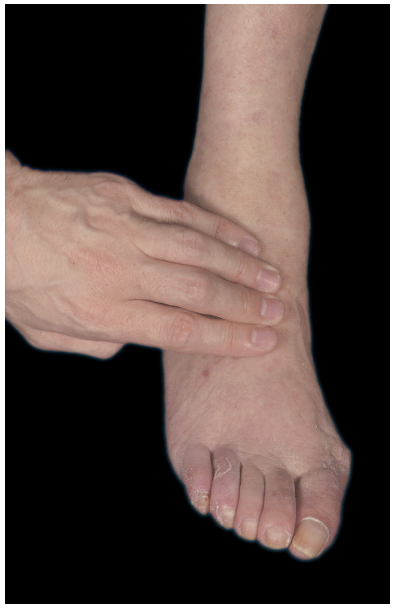


Fig. 1.12 Palpation of the dorsalis pedis pulse.

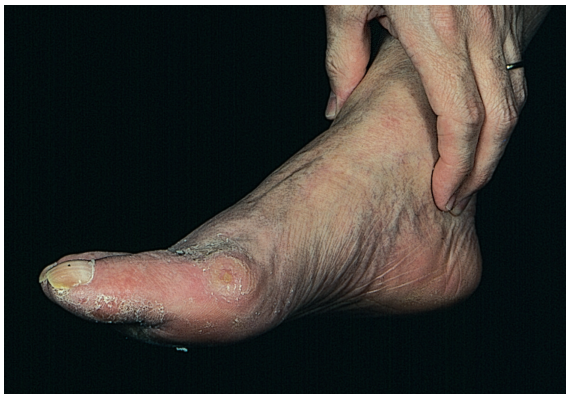


Fig. 1.13 Palpating the posterior tibial pulse.

Classically, in the neuropathic foot, the pulses are bounding, with a high volume.

Temperature of the foot

Skin temperature is compared between both feet with the back of the examining hand. Normally any one area on a foot will be within 2°C of the corresponding area on the other foot. Warm areas or hot spots outside this range indicate inflammation which may be due to infection, fracture, Charcot's osteoarthropathy or soft tissue trauma. An increase in unilateral pedal temperature, especially in the absence of ulceration, is best presumed to be Charcot's osteoarthropathy.

The temperature gradient is checked by using the back of the hands and gently moving them from the pretibial region of the leg distally over the dorsum of the foot to the toes while keeping in contact with the patient's skin. An asymmetric gradient may indicate either unilateral ischaemia on the colder side or unilateral inflammatory response such as Charcot's osteoarthropathy or infection on the warmer side.

In the neuroischaemic foot, coexisting autonomic neuropathy may keep the foot relatively warm, although an ice-cold foot is indicative of acute ischaemia.

Patients with neuropathy frequently complain of having cold feet when their feet are in fact warm.

Causes of the hot foot

- Soft tissue injury or fracture
- Cellulitis
- Charcot foot
- Gout
- Deep vein thrombosis.

Causes of the cold foot

- Chronic ischaemia
- Acute ischaemia
- Cardiac failure.

Patients on a very cold day may present with cold feet which merely reflect a severe vasoconstriction in response to a cold environment. It is sometimes difficult to differentiate this from the ice-cold ischaemic foot; however, the latter will remain cold in the clinic whereas the former will gradually warm up.

Oedema

Oedema already suspected on inspection can be confirmed by gentle digital pressure applied for a few seconds.

Crepitus

Very occasionally palpation may reveal gas in tissues as a fine crackling sensation.

Neurological assessment

Simple inspection will usually reveal signs of motor and autonomic neuropathy but sensory neuropathy must be detected by a sensory screening test or a simple sensory examination.

Motor neuropathy

The classical sign of a motor neuropathy is a high medial longitudinal arch, leading to prominent metatarsal heads and pressure points over the plantar forefoot.

Complicated assessment of motor power in the foot or leg is not usually necessary, but it is advisable to test dorsiflexion of the foot to detect a foot drop secondary to a common peroneal nerve palsy, which is usually unilateral and will affect the patient's gait. If pain is present in one leg only, a more detailed neurological examination is indicated to rule out compressive lesions of nerve roots supplying the lower limb—see under Painful neuropathy in Chapter 3.

Autonomic neuropathy

Signs of an autonomic neuropathy include a dry skin with fissuring and distended veins over the dorsum of the foot.

The dry skin is secondary to decreased sweating. The sweating loss normally occurs in a stocking distribution, which can extend up to the knee. The distended veins are secondary to arteriovenous shunting associated with autonomic neuropathy (Fig. 1.14).

Sensory neuropathy

An important indication of neuropathy will be a patient who has no pain even when significant foot lesions are present. Painless ulceration is definite evidence of a peripheral neuropathy. It is important to detect patients who have sufficient neuropathy to render them susceptible to foot ulceration. This can be carried out using a monofilament which, when applied perpendicular to the foot, buckles at a given force of 10 g. Ability to feel that level of pressure provides protective sensation against foot ulceration. It is helpful first to demonstrate the technique on the patient's forearm.

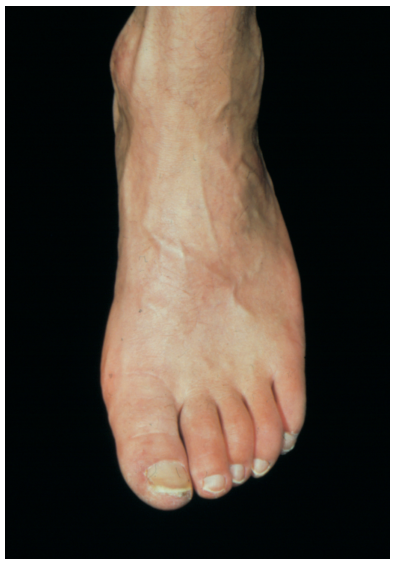


Fig. 1.14 Distended veins secondary to autonomic neuropathy.

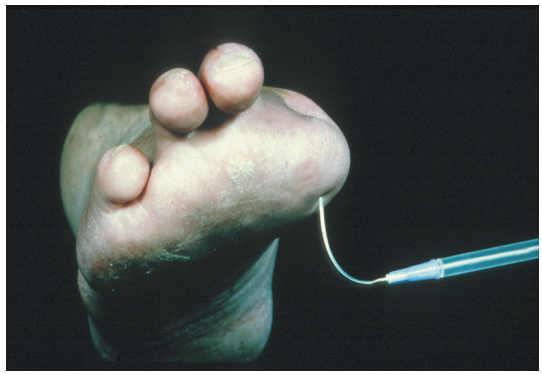


Fig. 1.15 A monofilament is applied perpendicular to the foot and pressed until it buckles at a given force of 10 g.

The number of sites tested varies according to different protocols. Sites examined include the plantar aspects of the first toe, the first, third and fifth metatarsal heads, the plantar surface of the heel and the dorsum of the foot. The filament should not be applied at any site until callus has been removed. If the patient cannot feel the filament at any of the tested areas, then protective pain sensation is lost, indicating susceptibility to foot ulceration (Fig. 1.15). The 10-g monofilament may become overstrained and inaccurate after use on numerous occasions and should be replaced regularly. A study has assessed differences in the performance of commercially available 10-g monofilaments. Monofilaments were tested using a calibrated load cell. Each monofilament was subjected to 10 mechanical bucklings of 10 mm while the load cell detected the maximal buckling force. Longevity testing was performed on a subset of the monofilaments by subjecting them to continuous compression until the buckling force was less than 9 g. Longevity and recovery testing suggest that each monofilament would survive usage on 10 patients before needing a recovery time of 24 hours before further usage.

If filaments are not available, then a simple clinical examination detecting sensation to light touch using a cotton wisp and vibration using a 128 Hz tuning fork will suffice. A recent study showed that the tuning fork is a valid and reliable test in screening for polyneuropathy and was superior to the monofilament. When using the tuning fork, it is best to compare a proximal site with a distal site to confirm a symmetrical stocking-like distribution of the neuropathy. The use of 'pin-prick' to detect sensory loss should be avoided.

Other useful but simple and practical tests for detecting neuropathy, if health-care practitioners have no access to formal equipment, include the Achilles tendon pinch, and the application of vertical pressure onto the nail plate. In practice, any patient who walks on a foot with ulceration or heavy plantar callus without concern has significant neuropathy.

Footwear assessment

It is important to examine both shoes and socks.

Examination of patient's footwear

- Is the shoe long enough?
- Is the toe box broad and deep enough?
- Are the heels low (below 5 cm)?
- Does the shoe fasten with a lace or strap to prevent friction? Slip-ons are unsuitable for everyday wear.
- Is the sole thick enough to provide protection from puncture wounds?
- Is the shoe lining worn, with rough areas that may prove irritating and warrant replacement?
- Are there foreign bodies within the shoes?
- Is there excessive wear under hallux suggesting a hallux rigidus?
- Is there wear across the whole of the tread suggesting pes cavus?
- Does the shoe avoid pressure points over the toes or margins of the feet?
- Does the heel cup fit snugly round the heel?
- What other types of shoes does the patient wear and when? Patients should be advised not to wear slippers around the house.

Examination of patient's socks

- Are the socks large enough?
- Are the seams too prominent?
- Is there a tight band at the top?
- Are the socks in good repair—no holes or lumpy darns?
- Are the socks made of absorbent material?
- Are the socks very thick, taking up too much space in the shoe?

General examination

As part of the diabetic foot assessment, and indeed the diabetic assessment, all patients should have a physical examination including the following systems:

- Cardiovascular
- Respiratory
- Abdomen
- Eyes:
  Visual acuity
  Fundi.

(A patient lacking necessary visual acuity to give himself a daily foot examination is a patient at risk, and his family or caregiver should help him.)

Investigations

Investigations include:

- Neurological
- Vascular
- Skin temperature
- Laboratory
- Radiological
- Foot pressures.

Neurological

The degree of neuropathy can be quantified by the use of the neurothesiometer (Fig. 1.16). When applied to the foot, it delivers a vibratory stimulus that increases as the voltage is raised. The vibration threshold increases with age, and, for practical purposes, any patient unable to feel a vibratory stimulus of ≥ 25 volts is at risk of ulceration.

A small number of patients have a small-fibre neuropathy with impaired pain and temperature perception but with intact touch and vibration. They are prone to ulceration and thermal traumas but test normally with filaments and neurothesiometer, and a clinical assessment of light touch and vibration is normal. As yet, there is no simple inexpensive method of detecting and quantifying small-fibre neuropathy. However, a simple temperature assessment of cold sensation can be made by placing a cold tuning fork on the patient's foot and leg.

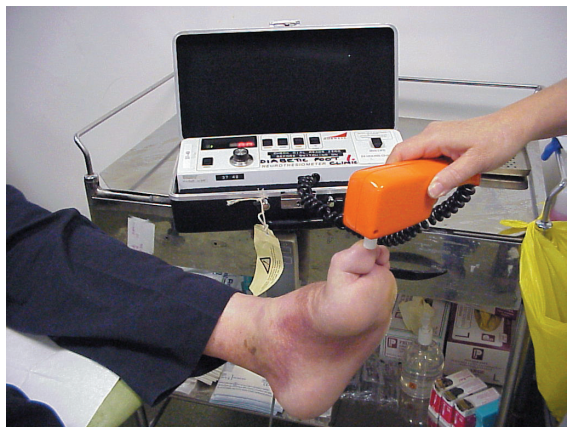


Fig. 1.16 The neurothesiometer.

Vascular

A small hand-held Doppler can be used to quantify the vascular status.

Used together with a sphygmomanometer, the brachial systolic pressure and ankle systolic pressure can be measured, and the pressure index, which is the ratio of ankle systolic pressure to brachial systolic pressure, can be calculated. In normal subjects, the pressure index is usually > 1, but in the presence of ischaemia is < 1. Thus, absent pulses and a pressure index of < 1 confirms ischaemia. Conversely, the presence of pulses and a pressure index of > 1 rules out ischaemia; this has important implications for management, namely that macrovascular disease is not an important factor and further vascular investigations are not necessary.

Many diabetic patients have medial arterial calcification, giving an artificially elevated systolic pressure, even in the presence of ischaemia. It is thus difficult to assess the diabetic foot when the pulses are not palpable, but the pressure index is > 1. There are two explanations:

- The examiner may have missed the pulses, particularly in an oedematous foot, and should go back to palpate the foot after the arteries have been located by Doppler ultrasound
- If the pulses remain impalpable, then ischaemia probably exists in the presence of medial wall calcification. It is then necessary to use other methods to assess flow in the arteries of the foot, such as examining the pattern of the Doppler arterial waveform or measuring transcutaneous oxygen tension or toe systolic pressures. Furthermore, absence of foot pulses would be an indication to investigate popliteal and femoral arteries.

Skin temperature

It is helpful to follow-up the clinical assessment of skin temperature with the use of a digital skin thermometer. An infrared thermometer is ideal and skin temperatures are compared between similar areas on each foot. This is particularly helpful in the management of the Charcot foot.

Laboratory

Laboratory investigations are determined by clinical findings, but the following investigations are useful as a baseline in most patients:

- Full blood count (to detect anaemia or polycythaemia), and white blood cell count (to reflect the presence of infection)
- Serum electrolytes, urea and creatinine (to assess baseline renal function)
- Serum bilirubin, alkaline phosphatase, gamma glutamyl transferase, aspartate transaminase (to assess baseline liver function)
- Blood glucose and HbA_{1c} (to assess diabetic control)
- Serum cholesterol and triglycerides (to assess arterial disease risk factors)
- C-reactive protein (as an acute inflammatory marker).

Radiological

These will be determined by the clinical presentation, and may not always be necessary. However, in most cases, an X-ray of the foot will be required to detect:

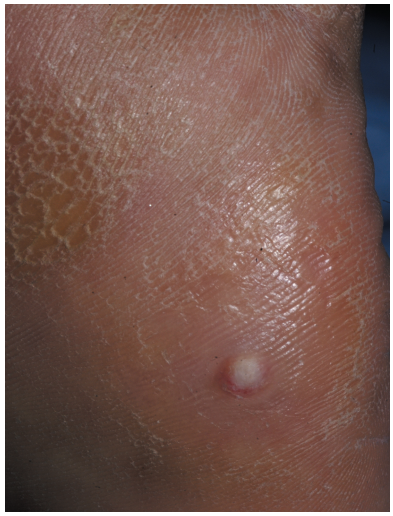
- Osteomyelitis
- Fracture/dislocation
- Charcot foot
- Gas in soft tissues
- Foreign body.

CASE STUDY
Foreign body

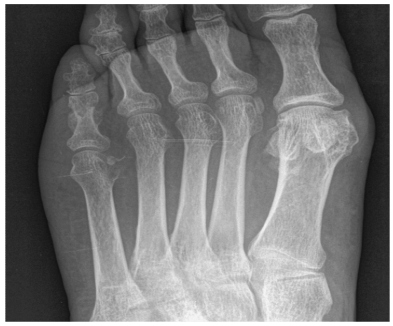
A 45-year-old woman with type 2 diabetes of 8 years' duration trod on a nail whilst decorating her house. She removed the nail and was seen in a casualty department and given tetanus prophylaxis. Subsequently she noticed a discomfort at the site of penetration of the nail, which was associated with a focal swelling of the soft tissues. This persisted for several months and she eventually presented at the diabetic foot clinic, where an X-ray showed a possible radio-opaque body adjacent to the head of the first metatarsal (Fig. 1.17a,b). An MRI showed increased signal from a possibly metallic object, and during the MRI she reported increased pain and burning in her foot which may have been related to movement of the metallic fragment within the magnetic field. An ultrasound showed a 4 × 5 mm hypoechoic area of granulomatous tissue just below the skin. There was a ring-down type artefact within it suggestive of focal dense material. The patient underwent surgery and a metallic fragment was removed.

Key points

- Persistent discomfort after a puncture injury may indicate a persisting foreign body
- It is important to X-ray the foot even though the foreign body has apparently been removed
- An ultrasound is a useful means of investigating foreign bodies.



(a)



(b)

Fig. 1.17 (a) Focal swelling of the soft tissues at site of penetration of nail. (b) A radio-opaque foreign body adjacent to 5th metatarsal head.

Foot pressures

These techniques measure the pressure distribution on the plantar surface of the foot. There are two main methods: 'out-of-shoe' and 'in-shoe'. The introduction of the optical pedobarograph considerably improved the accuracy of out-of-shoe pressure measurements. Developments in computer technology have led to microprocessor-like recording devices to quantify in-shoe foot pressures and these include the EMED system and the F-Scan system. These systems have the possibility of identifying patients at risk of plantar neuropathic ulceration and give a basis for the implementation of footwear adjustments or surgical intervention. We have found that they are also useful educational tools as described in Chapter 3.

CLASSIFICATION AND STAGING

After completing this basic assessment, it will now be possible to classify the diabetic foot and distinguish between the neuropathic foot and the neuroischaemic foot, as for practical purposes, the diabetic foot can be divided into these two distinct entities. Neuropathy is nearly always found in association with ischaemia, so the ischaemic foot is best called the neuroischaemic foot. In rare cases the foot may clinically be ischaemic without signs of neuropathy, but in practice, the diabetic ischaemic foot is treated in the same way as the neuroischaemic foot.

It is essential to classify the diabetic foot in this way by differentiating between the neuropathic and the neuroischaemic foot as their management will differ in many respects, and the particular vulnerability of the ischaemic foot renders recognition essential, ischaemia being a most important factor on the pathway to major amputation.

Usually there will be no doubt as to which category the foot should be placed in. However, if the examiner has any doubt as to the correct classification, then the foot should be regarded as neuroischaemic, because if a neuroischaemic foot is wrongly classified as neuropathic, with resulting failure to do further tests to confirm ischaemia and adapt the care plan accordingly, this may lead to preventable catastrophe and loss of the foot.

With the passage of time, nearly every neuropathic foot will become ischaemic. Thus it is very important to reassess diabetic feet annually so as to detect patients who drift from the neuropathic to the neuroischaemic foot.

Neuropathic foot

- The neuropathic foot is a warm, well-perfused foot with bounding pulses and distended dorsal veins due to arteriovenous shunting
- Sweating is diminished so skin and any callus tend to be hard and dry and prone to fissuring
- Toes are clawed and the arch of the foot may be raised
- Ulceration commonly develops on the sole of the foot, associated with neglected callus and high plantar pressures
- Despite the good circulation, necrosis can develop secondary to severe infection
- The neuropathic foot is also prone to bone and joint problems which we refer to as Charcot's osteoarthropathy.

Neuroischaemic foot

- The neuroischaemic foot is a cool, pulseless foot with poor perfusion and almost invariably also has neuropathy

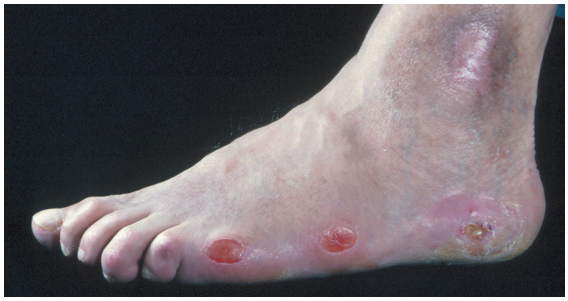


Fig. 1.18 Neuroischaemic foot with ulceration on the margins.

- The colour of the severely ischaemic foot can be a deceptively healthy pink or red caused by dilatation of capillaries in an attempt to improve perfusion
- The neuroischaemic foot may be complicated by oedema, often secondary to cardiac failure or renal impairment
- Ischaemic ulcers are commonly seen around the edges of the foot, including the apices of the toes and the back of the heel, and are associated with trauma or wearing unsuitable shoes (Fig. 1.18)
- The neuroischaemic foot develops necrosis in the presence of infection or if tissue perfusion is critically diminished
- Even if neuropathy is present and plantar pressures are high, plantar ulceration is rare. This is probably because the foot does not develop heavy callus, which requires good blood flow.

After classification of the diabetic foot, it is necessary to make the appropriate staging in its natural history.

The natural history of the diabetic foot can be divided into six stages as shown:

- Stage 1: Normal foot
- Stage 2: High-risk foot
- Stage 3: Ulcerated foot
- Stage 4: Infected foot
- Stage 5: Necrotic foot
- Stage 6: Unsalvageable foot.

The simple staging system covers the entire spectrum of diabetic foot disease but it emphasizes the development of the foot ulcer in stage 3 as a pivotal event demanding urgent and aggressive management. However, each stage demands specific treatment.

Other classifications of the diabetic foot are essentially classifications of ulcers and do not cover the whole natural history of the diabetic foot: they may be useful for researchers who want to compare like with like, but are not a practical framework on which treatments can be based.

The simple staging system used in this book has been created to allow all practitioners, whether experienced in diabetic foot care or not, to make an initial assessment of the diabetic foot at whatever stage in the natural history it might be. The stage sets the place in the natural history and also determines treatment. The aim is to keep all diabetic feet at as low a stage as possible.

Stage 1

At this stage, the patient does not have the risk factors of neuropathy, ischaemia, deformity, callus and oedema. The patient is not vulnerable to foot ulcers. The patient's foot is free of diabetic complications but may be affected by other foot pathologies that occur in the general population. The foot is usually asymptomatic and any problems, including pain, are non-diabetic in nature.

Stage 2

The patient has developed one or more of the risk factors for foot ulceration including neuropathy, ischaemia, deformity, callus and oedema. The major risk factors are neuropathy and ischaemia and it is rare for the other three to cause problems when neuropathy and ischaemia are absent. When they are present, however, all these risk factors need addressing to reduce susceptibility to ulceration.

Patients without current active foot ulceration but with a history of previous ulceration should be regarded as at risk.

Within stage 2, there are specific conditions which are non-ulcerative but require treatment. These include:

- Severe chronic ischaemia
- Acute ischaemia.

There are also specific complications of neuropathy:

- Charcot's osteoarthropathy, including neuropathic fractures
- Painful neuropathy.

Stage 3

The foot has a skin breakdown. Although this is usually an ulcer, it is important not to underestimate apparently minor injuries such as blisters, skin fissures or grazes, all of which have a propensity to become ulcers if they are not treated correctly and fail to heal quickly. Ulceration is usually on the plantar surface in the neuropathic foot and on the margins in the neuroischaemic foot (Fig. 1.19).

Stage 4

The foot has developed infection, which can complicate both the neuropathic foot (Fig. 1.19) and the neuroischaemic foot (Fig. 1.19).

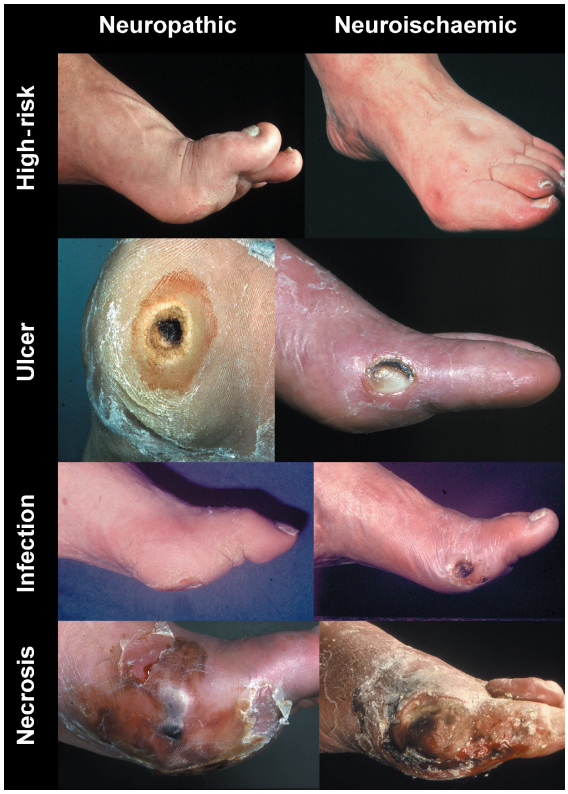


Fig. 1.19 Composite picture to show the natural history of the neuropathic and neuroischaemic foot as it passes from high-risk through ulceration, infection and necrosis.

Stage 5

Necrosis has supervened. In the neuropathic foot, infection is usually the cause. In the neuroischaemic foot, infection is still the most common reason for tissue destruction although ischaemia contributes (Fig. 1.19). In some cases ischaemia alone can lead to necrosis of a previously intact foot, with slow onset of dry necrosis and necrotic toes which appear shrivelled (Fig. 1.20). The diabetic foot in the patient with renal failure (the so-called renal foot) is very prone to develop necrosis, even in the absence of infection: for management see Chapter 6.

Stage 6

The foot cannot be saved and will need a major amputation.

Reasons for major amputation:

- Extensive necrosis which has destroyed the foot
- Severe infection which puts the patient's life at risk
- Agonizing ischaemic pain which cannot be relieved

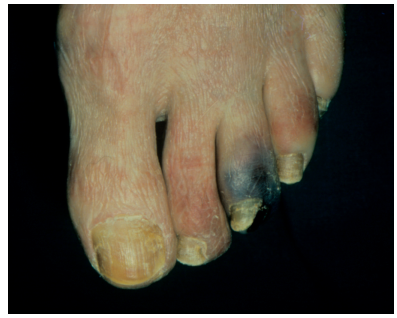


Fig. 1.20 Dry necrosis in a neuroischaemic foot.

- Unstable foot and ankle, usually secondary to Charcot's osteoarthropathy, which does not respond to external or internal fixation.

MULTIDISCIPLINARY MANAGEMENT

The aim in managing the diabetic foot is always to keep the patient at as low a stage as possible. At each stage of the diabetic foot it is necessary to take control of the foot to prevent further progression, and management will be considered under the headings shown in Table 1.3. The cornerstone of this approach to the diabetic foot is to encourage early presentation to allow early diagnosis and early intervention within the multidisciplinary diabetic foot clinic.

Multidisciplinary teams are essential for optimal management of the high-risk diabetic foot and there is no room for inter-professional rivalry. Over the years we have seen a healthy and welcome move towards the development of considerable mutual respect between team members from different specialties in many countries. We will describe the makeup and organization of the Diabetic Foot Clinic as it has evolved at King's College Hospital. While the situation we describe is not (of course) the only way of organizing patients with diabetic foot problems, it has proved to be successful in reducing amputations and improving outcomes for diabetic patients with foot problems.

Table 1.3 Multidisciplinary management

Mechanical control
Wound control
Microbiological control
Vascular control
Metabolic control
Educational control

No one person can take control of the diabetic foot. Successful management needs the expertise of a multidisciplinary team including the following:

- Podiatrist
- Physician
- Nurse
- Orthotist
- Surgeon
- Radiologist.

It is helpful if the multidisciplinary team works closely together, within the focus of a diabetic foot clinic, which ideally is situated in a hospital. It is extremely important that there is a geographical focus of diabetic foot care to serve the diabetic patients in a defined geographical area. This will be vital for patients who know where exactly to come for their routine and emergency appointments, and also vital for local health-care professionals who will know where to seek help and where to obtain advice and education. Busy diabetic foot clinics will need to be open and available to patients throughout the working week and suitable arrangements need to be made with their associated casualty departments to see patients out of office hours. This overall umbrella of care focused within a definite place is vital for the diabetic foot patient who often has multiple comorbidities and complications and needs urgent investigations and help.

The diabetic foot team should be based in such a diabetic foot clinic, and should meet regularly for joint consultations as well as for ward rounds and X-ray conferences and clinical sessions.

Some roles of team members may overlap, depending on local expertise and interest. At King's College Hospital the roles have evolved in the following ways as discussed below.

Roles of the members of the multidisciplinary diabetic foot team

Podiatrists

The podiatrists man the clinic's emergency service throughout the week, and undertake specialist wound care of ulcers, including debridement, and plaster casting for indolent ulcers and Charcot's osteoarthropathy. The podiatrists play a part in diagnosing problems, call in other members of the team, as appropriate, and also educate patients, their families and friends and other health-care professionals. They also provide routine preventive foot care.

One of the extended roles of the podiatrist in the UK is as a diabetic foot practitioner who specializes in looking after inpatients on the hospital wards. Podiatrists in the USA have become surgical specialists. Many podiatrists are now involved in clinical research. There is an urgent need for podiatry training in developing countries.

Physician

The physician plays a key role in the diagnosis of foot complications and is also crucial in the diagnosis and management of infection, working closely with the medical microbiologists. The physician also decides on the need for admission and organizes the admission, liaises with all members of the foot team and is responsible for the medical care of patients, including the management of diabetes and its complications.

Nurses

Nurses are also involved in ensuring optimal care of diabetes and its complications and in addition play an important role in the investigation of ischaemic patients, using Doppler sonography and transcutaneous oxygen and also in the assessment and management of the patient with neuropathy, including those with painful neuropathy. The health-care assistant prepares the dressing trolleys and also assists in dressing the ulcers.

Orthotists

Orthotists measure and take casts for the manufacture of insoles, shoes, orthotics and braces: they also deliver footwear education to patients and staff. The orthotists carry out joint consultations in the podiatry rooms and then measure and cast the patients in the orthotists' rooms, which are adjacent to the foot clinic.

Surgeons

Surgeons take part in joint consultations when the foot is infected to decide on the need for incision and drainage, surgical debridement and digital or ray amputation. Historically, in our Diabetic Foot Clinic, the orthopaedic surgeon works with the neuropathic patients, and one of their important roles is to assess the patient's suitability for surgical treatment of osteomyelitis. The vascular surgeon works with patients who have acute and chronic ischaemia and performs distal bypasses.

Radiologists

The interventional radiologists work in conjunction with the vascular surgeons to assess those patients suitable for revascularization of the ischaemic foot. The radiologists interpret X-rays and scans and perform angiograms and angioplasties.

Other members of the multidisciplinary team include microbiologists, physiotherapists, the rehabilitation physician and team, and the psychiatrists. The roles of all these people are very important but they do not usually work within the diabetic foot clinic.

The multidisciplinary team approach has reaped benefits both for patients and for staff in terms of expansion of traditional roles, increasing opportunities to learn from people in other disciplines, rapid access to patients and reducing numbers of hospital visits.

It is not possible for the entire team to be working together throughout the week as most people have additional responsibilities outside of the diabetic foot clinic. However, it is useful if all members of the team are accessible in an emergency in addition to having a formal commitment to work within the diabetic foot clinic with other team members at certain times. The podiatrist should be based within the diabetic foot centre throughout the entire working week, if at all possible, to maintain an emergency diabetic foot service, where walk-in patients can be seen immediately. Other members of the team will combine to see special problems under the umbrella of the diabetic foot clinic. As well as the basic day-to-day service for patients with emergency and follow-up appointments, the King's College Hospital Diabetic Foot Clinic has evolved fresh approaches to deal efficiently with an ever-increasing workload. These approaches may include:

- Joint fast-track services
- Joint ward rounds and X-ray conferences
- Specialized clinics.

Fast-track services

Joint vascular fast-track services

A joint vascular clinic can be organized to offer rapid investigations and priority treatment and regular follow-up. Many angiograms and angioplasties can now be performed on a day-case basis.

Joint orthopaedic clinics

At these clinics, selected patients with problems that may be amenable to orthopaedic surgical intervention are selected and seen at a joint clinic. Patients suitable for surgery are admitted, operated on and followed long-term.

Inpatient care

Joint ward rounds

Patients are also admitted as emergencies from the diabetic foot clinic. They are usually admitted to the medical wards under the care of the diabetologist, but when there is an overt vascular problem they are admitted directly under the vascular surgeons. While they are inpatients they are cared for by the admitting team but there is a joint ward round weekly when patients are seen together, both on the medical and surgical wards. On these rounds, the ward staff and the diabetic foot practitioner are joined by a pharmacist and the diabetic foot clinic team. They see every inpatient and discuss their medication and other aspects of their care, with a view to avoiding any delays and achieving discharge as quickly as possible.

Joint X-ray conferences

These are held weekly and are attended by the vascular surgeon and his team, the interventional radiologist, the diabetic foot practitioner and the diabetic foot team. Individual cases are discussed and treatments planned.

Other associated foot clinics have arisen within the King's College Hospital Diabetic Foot Clinic to fulfil very specialized functions within diabetic foot care. These include those discussed below.

Joint renal/foot clinic

Ideally, a podiatrist from the diabetic foot clinic should attend the renal unit for one morning a week, assessing and treating diabetic patients on dialysis or with renal transplants, and working with the renal physician and renal nurses. The feet of every patient with diabetes should be inspected at each clinic visit and additional appointments arranged as necessary. With this programme, outcomes in renal patients with diabetes were as good as those seen in diabetic patients.

Painful neuropathy clinics

At King's College Hospital these are currently run by a diabetes foot specialist nurse and a physician. Patients with painful neuropathy need considerable support and reassurance. There is a large armamentarium of different treatments which are covered in Chapter 3.

Clinics to rehabilitate diabetic amputees

Diabetic patients who have undergone a major amputation are at very high risk of developing problems in their remaining foot. Ideally they will be seen regularly by the rehabilitation physician and the physiotherapists and orthotists, together with a podiatrist who provides regular foot care and rapid referral to the diabetic foot clinic if problems develop.

Defined pathways and timescales for treatment and follow-up

Patients in stages 1 and 2 can be seen in primary care. Patients in stages 3–5 are best seen in the multidisciplinary diabetic foot clinic, which takes early referrals from a primary care service. However, there should be very rapid referral pathways between the primary care service and the hospital.

- Stage 1—Annual review with basic foot education
- Stage 2—It is difficult to stratify the risk of ulceration within this group. Any patient with one or more of the following—neuropathy, ischaemia, deformity, callus, oedema—should be referred for education and podiatry, receiving 3-monthly or more frequent treatment.
  Patients with specific problems will need the following referrals:
  Severe chronic ischaemia to diabetic foot clinic or vascular clinic within 1 week.
  Acute ischaemia to diabetic foot clinic or vascular clinic same day.
  Any neuropathic fracture/acute Charcot's osteoarthropathy to diabetic foot clinic within 24 hours. Patients with history of Charcot's osteoarthropathy are always best managed by the hospital team as problems frequently develop in other joints. Renal patients should also be seen by the hospital team.
  Painful neuropathy to diabetic foot clinic within 2 weeks. This is a singularly disagreeable condition and patients should not feel abandoned
- Stage 3—To diabetic foot clinic within 1 week. Maximum treatment interval 2 weeks with provision for emergency access in case of deterioration
- Stage 4—To diabetic foot clinic same day (may need admission for intravenous antibiotics or outpatient treatment with oral or intramuscular antibiotics). Maximum treatment interval 1 week
- Stage 5—To diabetic foot clinic same day for admission: after discharge, maximum treatment interval 1 week until any remaining necrosis is dry and well demarcated, then every 2 weeks until fully healed
- Stage 6—To diabetic foot clinic same day for admission. Remaining foot should be inspected daily during perioperative and rehabilitation period. After discharge from hospital, should be followed up with maximum interval between treatments of 6 weeks.

PRACTICE POINTS

- The basic approach to the diabetic foot is assessment, classification, staging and multidisciplinary management using the simple staging system as a treatment-based system
- Diabetic feet should be classified into neuropathic and neuroischaemic feet to ensure that ischaemia is never missed
- The natural history of the diabetic foot falls into six stages: normal, high-risk, ulcerated, infected, necrotic and unsalvageable
- Multidisciplinary management consists of mechanical, wound, microbiological, vascular, metabolic and educational control
- The multidisciplinary foot care service should include podiatrist, physician, orthotist, nurse, surgeon and radiologist
- Joint clinics help to provide improved care to diabetic foot patients.

FURTHER READING

Practical assessment

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