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Introduction

NEW APPROACH TO THE DIABETIC FOOT

Diabetes has reached epidemic proportions, and with it has come a growing number of complex diabetic foot problems. This book is written to help practitioners tackle these problems. It attempts to give enough simple practical information to enable practitioners to understand the natural history of the diabetic foot, rapidly diagnose its problems and confidently undertake appropriate interventions in a timely manner.

Three great pathologies come together in the diabetic foot: neuropathy, ischaemia and infection. Their combined impact results in a swift progression to tissue necrosis, which is the fundamental hallmark of the natural history of the diabetic foot. Progress towards necrosis can be so rapid and devastating that it has come to be regarded as a 'diabetic foot attack', similar to the heart and brain attacks of the coronary and cerebrovascular systems. A 'diabetic foot attack' can quickly reach the point of no return, with overwhelming necrosis. Thus, it is vital to diagnose it early and provide rapid and intensive treatment. Furthermore, it is important to achieve early recognition of the at-risk foot so as to institute prompt measures to prevent the onset of the 'diabetic foot attack'. Although there have been many advances in the management of the diabetic foot, it nevertheless remains a major global public health problem. All over the world, health-care systems have failed the diabetic foot patient and a major amputation occurs every 20 s. However, amputations are not inevitable.

In this book we describe a system of multidisciplinary care that has been shown to reduce the number of amputations. It is easily

Managing the Diabetic Foot, Third Edition. Michael E Edmonds and Alethea VM Foster.

reproducible and has been developed as a successful model of care throughout the world. This system is always being improved and refined, and this book describes the modern version of our diabetic foot management.

One important facet of this approach is the realization that neuropathy revolutionizes the practice of medicine and surgery. Classical symptoms and signs of disease are often absent because their expression depends on an intact peripheral nervous system. Thus, in traditional medical practice, a patient has a symptom, complains of this to their practitioner, who then makes a diagnosis. However, this approach may not work in the patient who has neuropathy. Instead, there must be a meticulous assessment of the patient to elicit subtle symptoms and signs that are clues to disease. Furthermore, an intact nervous system usually reflects symptomatically a 'picture' of what is going on inside the body, but in the presence of neuropathy this picture is absent and prompt use of imaging is required. Also, particular attention must be paid to inflammatory markers. Overall, it is important to practise what we call 'neuropathic medicine'. All practitioners looking after diabetic feet should understand this and adapt their practice of working with diabetic foot patients.

MODERN MANAGEMENT OF THE DIABETIC FOOT

This consists of four simple steps: assessment, classification, staging and intervention.

Assessment

A simple assessment of the diabetic foot is described to classify and stage the foot, looking for eight clinical features (Table 1.1). This assessment should take no longer than 5 min.

Table 1.1 **Eight clinical features in the assessment of the foot.**
Neuropathy
Ischaemia
Deformity
Callus
Swelling
Skin breakdown
Infection
Necrosis

Classification

The diabetic foot can be classified into two groups:

1. Neuropathic foot with palpable pulses
2. Ischaemic foot without pulses and a varying degree of neuropathy.

The neuropathic foot may be further divided into two clinical scenarios:

1. Foot with neuropathic ulceration
2. Charcot foot, which may be secondarily complicated by ulceration.

The ischaemic foot may be divided into four clinical scenarios:

1. Neuroischaemic foot characterized by both ischaemia and neuropathy and complicated by ulcer
2. Critically ischaemic foot
3. Acutely ischaemic foot
4. Renal ischaemic foot characterized by digital necrosis.

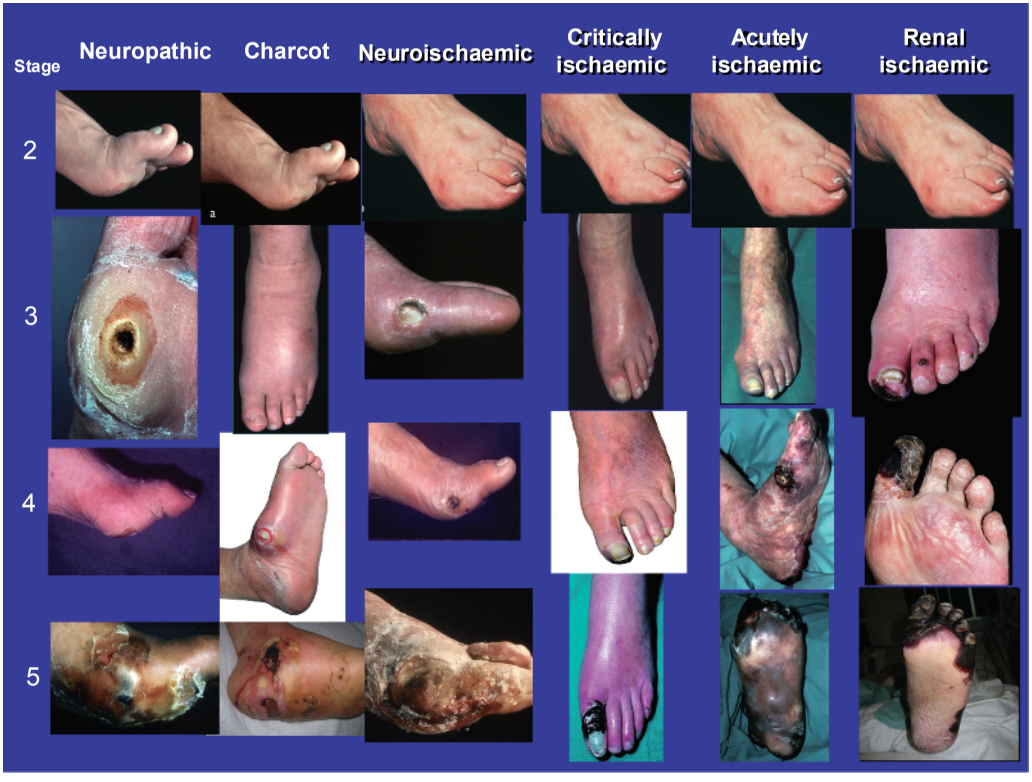


Fig. 1.1 The natural history and staging of the neuropathic and ischaemic foot.

Staging

Each of these six clinical scenarios (two neuropathic and four ischaemic) have specific stages in their natural history, and these stages have been described in a simple staging system (Fig. 1.1). This system covers the whole spectrum of diabetic foot disease and describes six stages in the natural history of each of the six clinical scenarios and emphasizes the relentless progression to the end stage necrosis. The stages are briefly summarized in Table 1.2 and will be described in detail in subsequent chapters devoted to each stage.

Intervention

A simple management plan is described for each stage, outlining six aspects of patient treatment within a multidisciplinary framework (Table 1.3).

ASSESSING THE DIABETIC FOOT

The initial approach to the diabetic foot starts with a simple assessment to enable the practitioner to make a basic classification and staging.

Table 1.2 Staging the diabetic foot.

Stage	Clinical condition
1	Normal
2	High risk
3	Ulcerated
4	Infected
5	Necrotic
6	Unsalvageable

Table 1.3 Six aspects of patient treatment, within a multidisciplinary framework.

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

There is a specific search for eight factors, as shown in Table 1.1. Many of these features can be detected by an examination that includes:

- Simple inspection
- Palpation
- Sensory testing.

A complicated examination is not necessary. This chapter describes the search for these individual features and then presents an integrated examination. It is often helpful to detect abnormalities by comparing one foot with the other.

Shoe inspection should be included in the foot assessment and this is described in Chapter 2.

Neuropathy

Peripheral neuropathy is the most common complication of diabetes, affecting 50% of all diabetic patients. Although neuropathy may present with tingling and a feeling of numbness, it is asymptomatic in the majority of patients and neuropathy will only be detected by clinical examination. An important indication of neuropathy will be a patient who fails to complain of pain, even when significant foot lesions are present. Neuropathic patients are at increased risk of injury while walking because of impaired sensation and motor function. Neuropathy also damages proprioception and falls are common.

The presentation of peripheral neuropathy is determined by the impairment of sensory, motor and autonomic nerves. Simple inspection will usually reveal signs of motor and autonomic neuropathy of the feet, but sensory neuropathy must be detected by screening or by a simple sensory examination.

The diabetic patient with peripheral neuropathy affecting the feet and legs may also have systemic symptoms resulting from autonomic neuropathy of the heart, gut and bladder. We have seen cases of sudden death in young, apparently robust neuropathic patients. Also, there may be poor neurological control of ventilation, leading to sleep apnoea and

susceptibility to pulmonary infections. Other neuropathic complications include postural hypotension and hypoglycaemic episodes without any warnings.

The peripheral nervous system is an early warning system. It detects external insults to the body and internal faults within and is programmed to direct appropriate protective responses to maintain the homeostatic integrity of the body. Diabetic patients with neuropathy have impaired homeostatic balance with diminished response to changes in the external and internal environment, and as a result they are very vulnerable and prone to disease. The signs and symptoms of disease in the foot and also in the rest of the body may be minimal and in some cases absent, reflecting 'silent disease'. Damage to the nerve supply of the heart can lead to silent ischaemia and silent myocardial infarction. Nevertheless, the pathology proceeds rapidly. There is a limited window of opportunity, and the end stage of tissue death in the foot and elsewhere is quickly reached.

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 (Fig. 1.2). In severe cases, pressure points also develop over the apices and dorsal interphalangeal joints of associated claw toes. However, claw toe is a common deformity and may not always

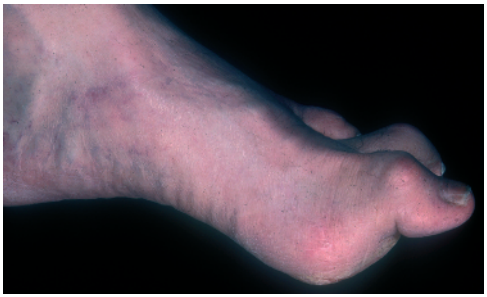


Fig. 1.2 Neuropathic foot showing motor neuropathy with high medial longitudinal arch, leading to prominent metatarsal heads and pressure points over the plantar forefoot.

be related to a motor neuropathy and atrophy of small muscles. It may be caused by wearing unsuitable shoes, trauma or may be congenital.

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. This is usually unilateral and will affect the patient's gait.

Autonomic neuropathy

The classical signs of peripheral autonomic neuropathy are:

- Dry skin which can lead to fissuring (Fig. 1.3)
- Distended veins over the dorsum of the foot and ankle (Fig. 1.4).

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.

Autonomic neuropathy can be tested using a Neuropad® to assess the moisture status of the foot. This is an adhesive pad containing a cobalt II salt. It is placed on the first metatarsal and in the presence of sweat

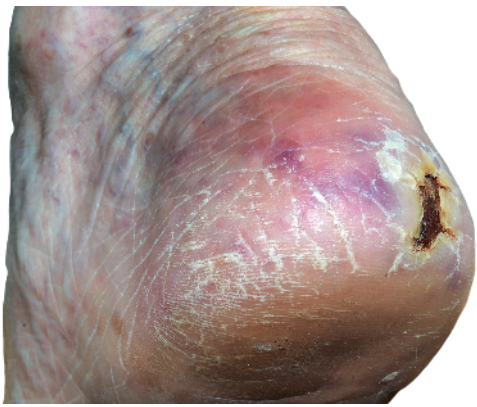


Fig. 1.3 Dry skin and fissuring of the heel with superficial necrosis in a neuroischaemic foot.

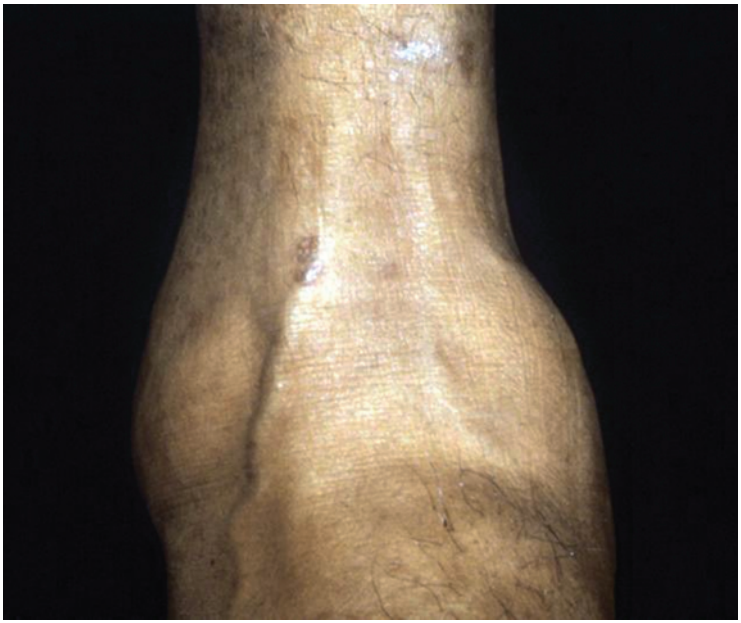


Fig. 1.4 Distended veins over the dorsum of the foot and ankle.

the pad will undergo a blue to pink colour change. Failure to change colour indicates autonomic neuropathy.

Sensory neuropathy

Sensory neuropathy can be simply detected by monofilaments (Fig. 1.5) or neurothesiometry (Fig. 1.6).

The advantage of the assessment with monofilaments or neurothesiometry is that it detects patients who have lost protective pain sensation and who are, therefore, susceptible to foot ulceration.

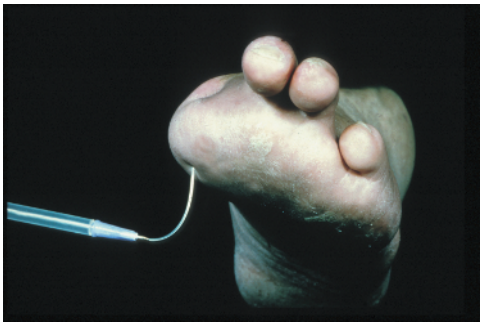


Fig. 1.5 Nylon monofilament buckles at a force of 10 g when applied perpendicularly to the foot. If the patient cannot feel this pressure then protective sensation has been lost.

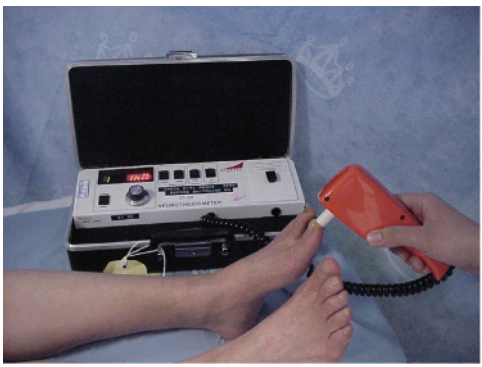


Fig. 1.6 A neurothesiometer is a device that delivers a vibratory stimulus which increases as the voltage is raised.

A simple technique for detecting neuropathy is to use a nylon monofilament, which, when applied perpendicular to the foot, buckles at a given force of 10 g. The filament should be applied at 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 a tested area, then significant neuropathy is present

and protective pain sensation is lost. After using a monofilament on 10 consecutive patients, there should be a recovery time of 24 h before further usage.

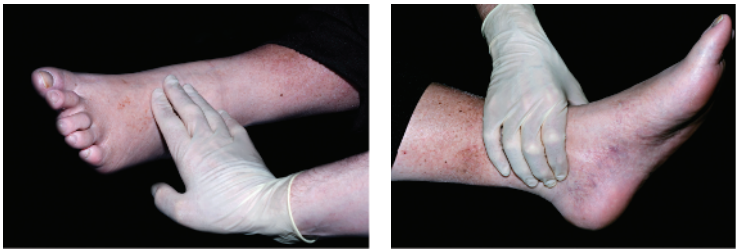
The degree of neuropathy can be further quantified by the neurothesiometer. When applied to the foot, this device delivers a vibratory stimulus, which increases as the voltage is raised. The vibration threshold increases with age, but, for practical purposes, any patient unable to feel a vibratory stimulus of 25 V is at risk of ulceration.

If monofilaments or a neurothesiometer are not available, then a simple clinical examination detecting sensation to light touch using a cotton wisp and to vibration using a 128 Hz tuning fork will suffice, comparing a proximal site with a distal site to confirm a symmetrical stocking-like distribution of the neuropathy.

Recently, the 'Touch the toes' test, also known as the Ipswich touch test, has been developed. This involves lightly touching the tips of the first and fifth toes on the right foot, then the first and fifth toes on the left foot and finally the third toes of the right and left foot. If the touch is not felt at two or more toes then the patient has significant neuropathy.

Also recently, Vibratip™, a simple, disposable, pocket-sized device that uses a button battery-driven vibrator motor to deliver a uniform source of vibration to the skin has been used to assess vibration in diabetic patients.

Touch and vibration are carried by large myelinated nerve fibres, but some 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 the neurothesiometer and a clinical assessment of light touch and vibration is normal. As yet, there is no inexpensive method of detecting and quantifying small-fibre neuropathy. However, a simple assessment of cold sensation can be made by placing a cold tuning fork on the patient's foot and leg.



(a) (b)

Fig. 1.7 Palpation of (a) the dorsalis pedis pulse on the dorsum of the foot and (b) the posterior tibial pulse at the ankle.

Ischaemia

Classical symptoms of ischaemia, namely claudication and rest pain, are often absent because of concomitant neuropathy. The most important manoeuvre to detect ischaemia is the palpation of foot pulses:

- The dorsalis pedis pulse is lateral to the extensor hallucis longus tendon on the dorsum of the foot (Fig. 1.7a)
- The posterior tibial pulse is below and behind the medial malleolus (Fig. 1.7b)
- If either of these foot pulses can be felt then it is highly unlikely that there is significant ischaemia.

A small, hand-held Doppler can be used to confirm the presence of pulses and to assess the vascular supply. Used together with a sphygmomanometer, the brachial systolic pressure and ankle systolic pressure can be measured, and the ankle brachial pressure index (ABPI), which is the ratio of ankle systolic pressure to brachial systolic pressure, can be calculated. In normal subjects, the ABPI is usually >1, but in the presence of ischaemia it is <1. Thus, absence of pulses and an ABPI of <1 confirms ischaemia. Conversely, the presence of pulses and an ABPI 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 ABPI 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 (see Chapter 4).

Despite the difficulties of interpreting ankle systolic pressures in the presence of medial calcification, an ABPI of 0.5 or less indicates severe ischaemia whether the patient is calcified or not.

Deformity

It is important to recognize deformity in the diabetic foot. Deformity often leads to bony prominences, which are associated with high mechanical pressures on the overlying skin. This results in ulceration, particularly in the absence of protective pain sensation and when shoes are unsuitable. Ideally, the deformity should be recognized early and accommodated in properly fitting shoes before ulceration occurs.

Common deformities include:

- Claw toes
- Pes cavus
- Hallux rigidus
- Hallux valgus
- Hammer toe
- Mallet toe

- Fibro-fatty padding depletion
- Charcot foot
- Deformities related to previous trauma and surgery
- Nail deformities.

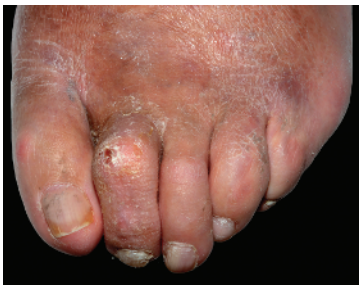


Fig. 1.8 Clawed second toe with early skin breakdown.

Claw toes

Claw toes (Fig. 1.8) have fixed flexion deformities at the interphalangeal joints and are associated with callus and ulceration of the apices and dorsal aspects of the interphalangeal joints. Although claw toes may be associated with neuropathy, they are often unrelated, especially when the clawing is unilateral and associated with trauma or surgery of the forefoot. Claw toes may result from acute rupture of the plantar fascia.

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 the abnormal distribution of pressure leads to high mechanical pressure over the metatarsal heads (Fig. 1.2).

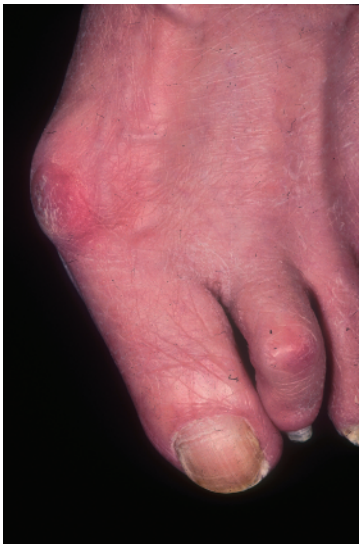


Fig. 1.9 Hallux valgus with erythema at first metatarso-phalangeal joint from tight shoe.

Hallux rigidus

This leads to limited joint mobility of the first metatarso-phalangeal joint with loss of dorsiflexion and results in excessive pressure on the plantar surface of the first toe, causing callus formation.

Hallux valgus

Hallux valgus is a deformity of the first metatarso-phalangeal 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 (Fig. 1.9).

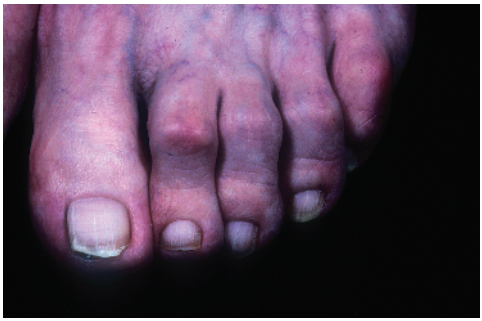


Fig. 1.10 Hammer toes (second, third and fourth toes) with hyperflexion of the proximal interphalangeal joints and hyperextension of the distal interphalangeal joints.

Hammer toe

Hammer toe is a flexion deformity of the proximal interphalangeal joint of a lesser toe with hyperextension of the distal interphalangeal joint (Fig. 1.10). The toe is at risk of dorsal ulceration.

Mallet toe

This deformity is a flexion contracture at the distal interphalangeal joint.

Fibro-fatty padding depletion

The plantar soft-tissue overlying the metatarsal heads is frequently pushed forward or destroyed by previous ulceration or infection. Also, motor neuropathy leads to anterior fat pad displacement and increased focal plantar pressures.

Charcot foot

Bone and joint damage in the metatarso-tarsal region is the commonest site of involvement and leads to two classical deformities:

- Rocker bottom deformity (Fig. 1.11), in which there is displacement and subluxation of the tarsus downwards
- Medial convexity, which results from dislocation of the talo-navicular or tarso-metatarsal joint.

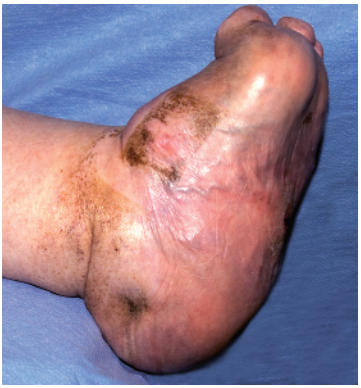


Fig. 1.11 Charcot foot showing rocker bottom deformity.

Both are often associated with a bony prominence which is very prone to ulceration. If these deformities are not diagnosed early and accommodated in properly fitting footwear, ulceration at vulnerable pressure points often develops (Fig. 1.12).

Deformities related to previous trauma and surgery

Deformities of the hip and fractures of the tibia or fibula lead to leg-shortening and hence 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. Removal of the fifth ray may lead to varus deformity of the foot if the fifth metatarsal base and its muscle attachments are not preserved. After amputation of a toe, deformities are often seen in adjoining toes (Fig. 1.13).

Nail deformities

It is important to inspect the nails closely as these may become the site of ulceration. Thickened nails are common in the population at large and may lead to ulceration.

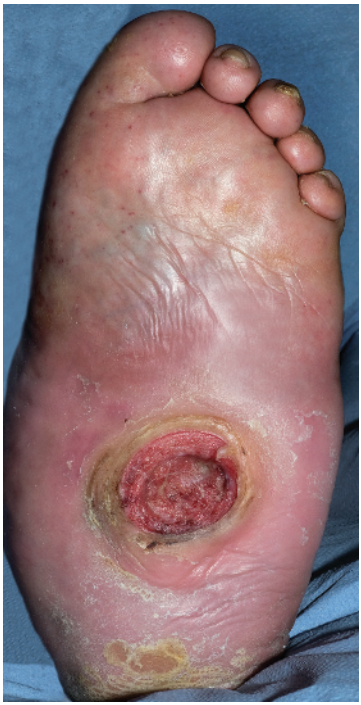


Fig. 1.12 Ulceration over bony prominence on the plantar surface of a rocker bottom deformity.

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 or trauma, the nail penetrates the flesh (see Fig. 2.5a and b).

Callus

This is a thickened area of epidermis which develops at sites of pressure, shear and friction. It should not be allowed to become excessive, as callus is a common forerunner of ulceration in the presence of neuropathy.

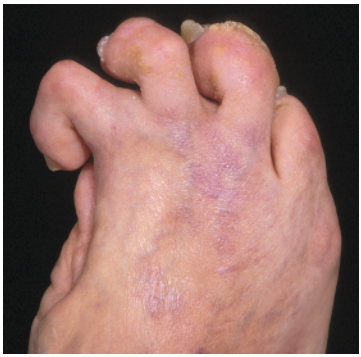


Fig. 1.13 Deformity of toe adjacent to amputated first toe.

Swelling

Swelling of the tissues of the foot is a major factor predisposing to ulceration, and often exacerbates a tight fit inside poorly fitting shoes. It also impedes healing of established ulcers. Swelling may be bilateral or unilateral.

Bilateral swelling

This is usually secondary to:

- Cardiac failure
- Hypoalbuminaemia
- Renal failure
- Venous insufficiency (sometimes unilateral)
- Inferior vena caval obstruction
- Lymphoedema (Fig. 1.14)
- Diabetic neuropathy, when it is related to increased arterial blood flow and arteriovenous shunting and is known as neuropathic oedema.

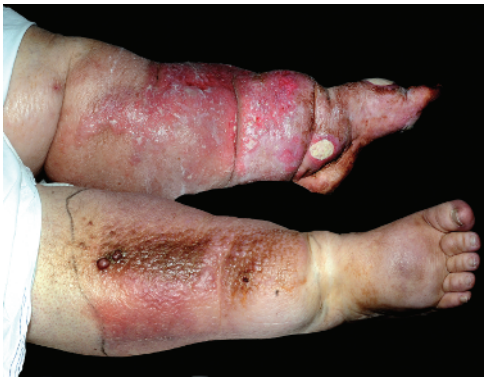


Fig. 1.14 Bilateral lymphoedema with cellulitis of both legs.

Unilateral swelling

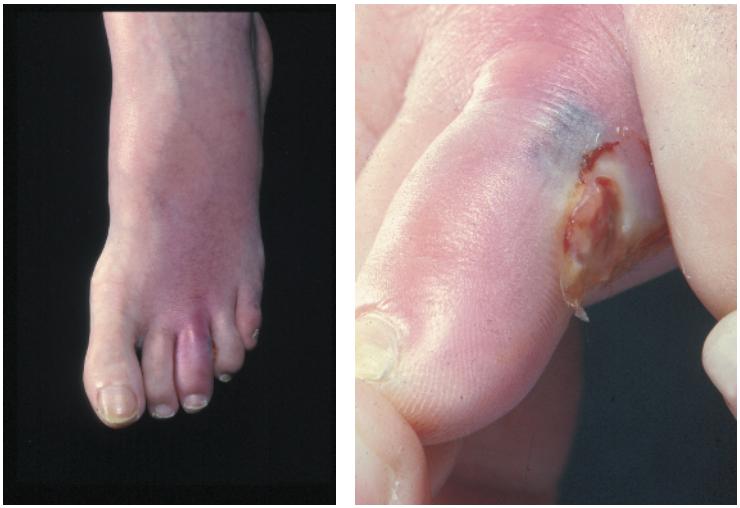
This is usually associated with local pathology in the foot or leg.

Causes are:

- Infection, when it is usually associated with erythema and a break in the skin (Fig. 1.15a and b)
- Charcot foot (a unilateral hot, red, swollen foot is often the first sign, and the swelling can extend to the knee)
- Gout, which may also present as a hot, red, swollen foot
- Trauma, sprain or fracture
- Deep vein thrombosis
- Venous insufficiency
- Lymphoedema, caused by lymphatic obstruction secondary to malignancy
- Venous obstruction by a pelvic mass, malignancy or ovarian cyst
- Localized collection of blood or pus which may present as a fluctuant swelling.

Skin breakdown

An active search should be made for breaks in the skin over the entire surface of the foot and ankle, not forgetting the areas between the toes



(a) (b)

Fig. 1.15 (a) Erythema and swelling of the third toe spreading up the foot. (b) Interdigital ulcer as a source of sepsis with cellulitis and blue discolouration adjacent to ulcer.

and at the back of the heel. Toes should be gently held apart for inspection (Fig. 1.15b). If jerked apart, this can split the skin. The classical sign of tissue breakdown is the foot ulcer. However, fissures (Fig. 1.3) and bullae/blisters (see Fig. 4.5) also represent breakdown of the skin.

Some lesions will be obvious; others will make their presence known by their complications, such as:

- Discharge or exudate
- Colour changes under callus or nail plate
- Pain or discomfort
- Swelling
- Warmth
- Erythema.

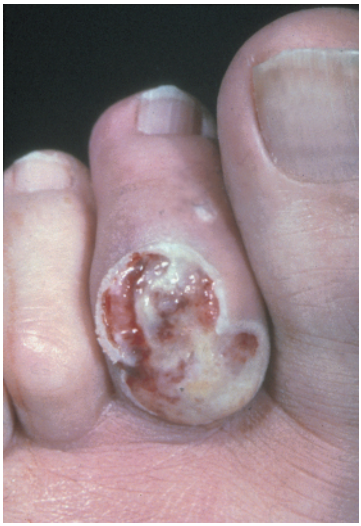


Fig. 1.16 Ulceration, cellulitis and purulent discharge from toe.

Infection

When skin breakdown develops, it may act as a portal of entry for infection (see Chapter 5). A close inspection for signs of infection should be made. These signs include purulent discharge from the lesion and erythema, swelling and warmth of the toe or foot (Fig. 1.16).

In the presence of neuropathy, the signs of infection may be subtle and include increased friability of granulation tissue, wound odour, wound breakdown and delayed healing.

Necrosis

Finally, lesions of skin breakdown may progress to underlying necrosis (Fig. 1.17). This can be identified by the presence of black or brown devitalized tissue (see Chapter 6).

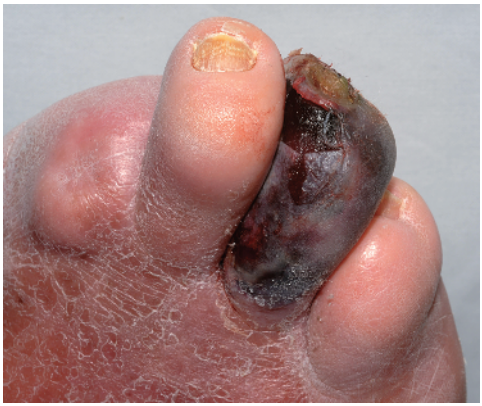


Fig. 1.17 Wet necrosis of fourth toe.

Integrated examination

In practice, the examination of the foot should be divided into three main parts: inspection, palpation and neurological assessment.

Inspection

The foot should be fully inspected, including dorsum, sole, back of the heel and interdigital areas with a full assessment of:

- Colour (as an indicator of ischaemia)
- Deformity
- Swelling
- Callus
- Skin breakdown
- Infection
- Necrosis.

Palpation

Pulses should be palpated and skin temperature compared between both feet with the back of the examining hand. The measurement of skin

temperature is particularly helpful in the management of the Charcot foot, when a digital skin thermometer is useful (see Chapter 4).

Neurological assessment

Peripheral neuropathy can be detected by using the monofilament, neurothesiometer, Vibratip™ or by performing a simple sensory examination or 'Touch the toe' test.

After completing this basic examination, it will now be possible to classify the diabetic foot and to make the appropriate staging in its natural history.

CLASSIFYING THE DIABETIC FOOT

For practical purposes, the diabetic foot can be divided into two main entities: the neuropathic foot with palpable pulses and the ischaemic foot without palpable pulses. It is essential to differentiate between the neuropathic and the ischaemic foot as their management will differ.

Infection is the most frequent complication of ulceration in both the neuropathic and ischaemic foot. It is important to diagnose it early and intervene rapidly. It is responsible for considerable tissue necrosis in the diabetic foot and is the main reason for major amputation.

The neuropathic foot

- This is a warm, well-perfused foot with bounding pulses due to arteriovenous shunting and distended dorsal veins
- Sweating is diminished, the skin may be dry and prone to fissuring, and any callus tends to be hard and dry
- Toes may be clawed and the foot arch raised
- Ulceration can develop 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 may have an abnormal response to minor traumatic injuries, and this can lead to bone and joint problems (the Charcot foot)
- When patients are followed for many years, the neuropathic foot may develop ischaemia and become a neuroischaemic foot.

The ischaemic foot

This is a cool, pulseless foot with reduced perfusion. It may also be complicated by swelling, often secondary to cardiac failure or renal failure. If it becomes infected, the ischaemic foot may feel deceptively warm.

The various subdivisions of the ischaemic foot will have characteristic appearances.

Neuroischaemic foot

The most frequent presentation is that of ulceration. Ischaemic ulcers are commonly seen on the margins of the foot, including the tips of the toes and the areas around the back of the heel (Fig. 1.18). They

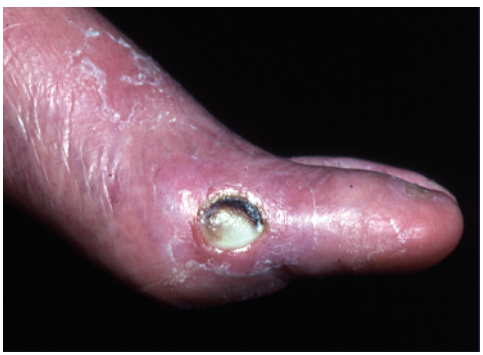


Fig. 1.18 Neuroischaemic foot ulcer.

are usually caused by minor trauma or by wearing unsuitable shoes. Even if neuropathy is present and plantar pressures are high, plantar ulceration is rare, probably because the foot does not develop heavy plantar callus, which requires good blood flow. Intermittent claudication and rest pain may be absent because of neuropathy and the distal distribution of the arterial disease to the leg.

Critically ischaemic foot

This presents as a pink, often painful foot with pallor on elevating the foot and rubor on dependency. The colour of the critically ischaemic foot can be a deceptively healthy pink or red, caused by dilatation of capillaries in an attempt to improve perfusion (Fig. 1.19).

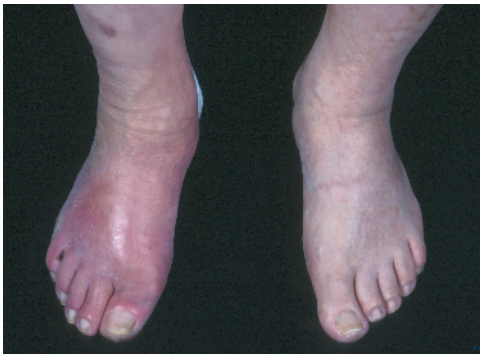


Fig. 1.19 Critically ischaemic right foot showing rubor on dependency.

Acutely ischaemic foot

This presents initially with sudden pallor and the foot becomes mottled (Fig. 1.20).

Renal ischaemic foot

A classical feature of this foot is the digital necrosis (Fig. 1.21).

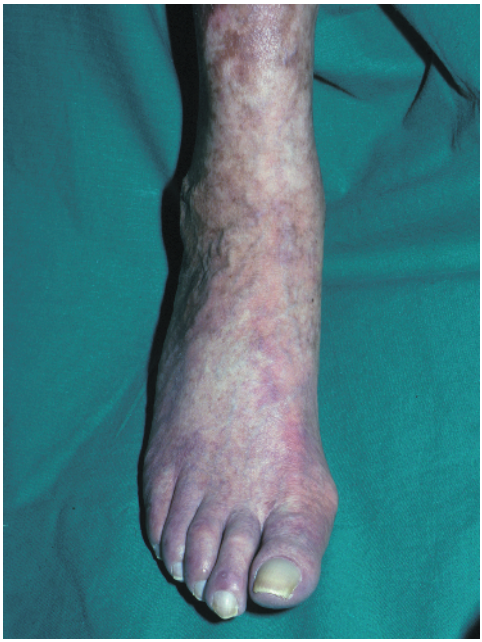


Fig. 1.20 Acutely ischaemic foot showing mottling of the skin.

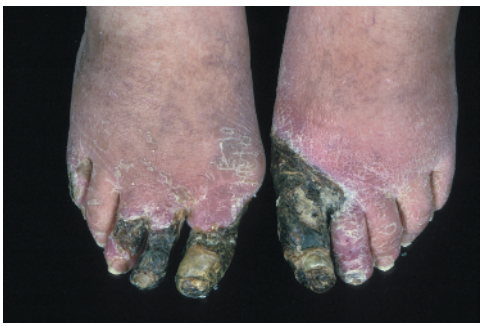


Fig. 1.21 Digital necrosis in renal ischaemic feet.

STAGING OF THE DIABETIC FOOT

The natural history of the diabetic foot can be divided into six stages, as shown in Table 1.2. The most frequently encountered types of diabetic foot are the neuropathic foot with ulceration and the neuroischaemic foot with ulceration. Their natural history is portrayed in Fig. 1.22. This represents pictorially the progression from Stage 2 to Stage 5 for each type of foot.

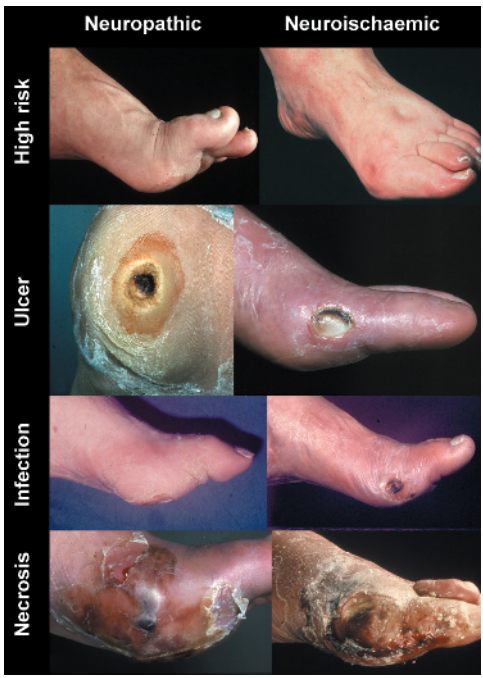


Fig. 1.22 Composite picture to show the natural history of the neuropathic and neuroischaemic foot as it passes from high risk through ulceration, infection and necrosis.

- *Stage 1* The foot is not at risk. The patient does not have the risk factors of neuropathy, ischaemia, deformity, callus or swelling. Thus, the patient is not vulnerable to foot ulcers.
- *Stage 2* The patient has developed one or more of the risk factors for ulceration and the foot may be divided into the neuropathic foot and the neuroischaemic foot.
- *Stage 3* The neuropathic and neuroischaemic foot has developed a skin breakdown. This is usually an ulcer, but because some minor injuries, such as blisters, splits or grazes, have a propensity to become ulcers, they are included in Stage 3. Ulceration is usually on the plantar surface in the neuropathic foot and usually on the margin in the ischaemic foot.
- *Stage 4* The ulcer has developed infection with the presence of cellulitis, which can complicate both the neuropathic and the neuroischaemic foot.
- *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.
- *Stage 6* The foot cannot be saved and will need a major amputation.

The staging system was developed primarily for the two main scenarios of the neuropathic and neuroischaemic foot, stressing the development of the ulcer as a pivotal stage in the natural history of the diabetic foot and the rapid progression through infection to necrosis (Fig. 1.22).

However, staging can be helpful for the four less common scenarios, namely the Charcot foot, the critically ischaemic foot, the acutely ischaemic foot and the renal ischaemic foot. The natural history of these feet, together with that of the neuropathic and neuroischaemic foot, has been described at the beginning of this chapter in Fig. 1.1.

The overwhelming feature of the natural history of all these scenarios of the diabetic foot is the rapid progression to necrosis, which clinically

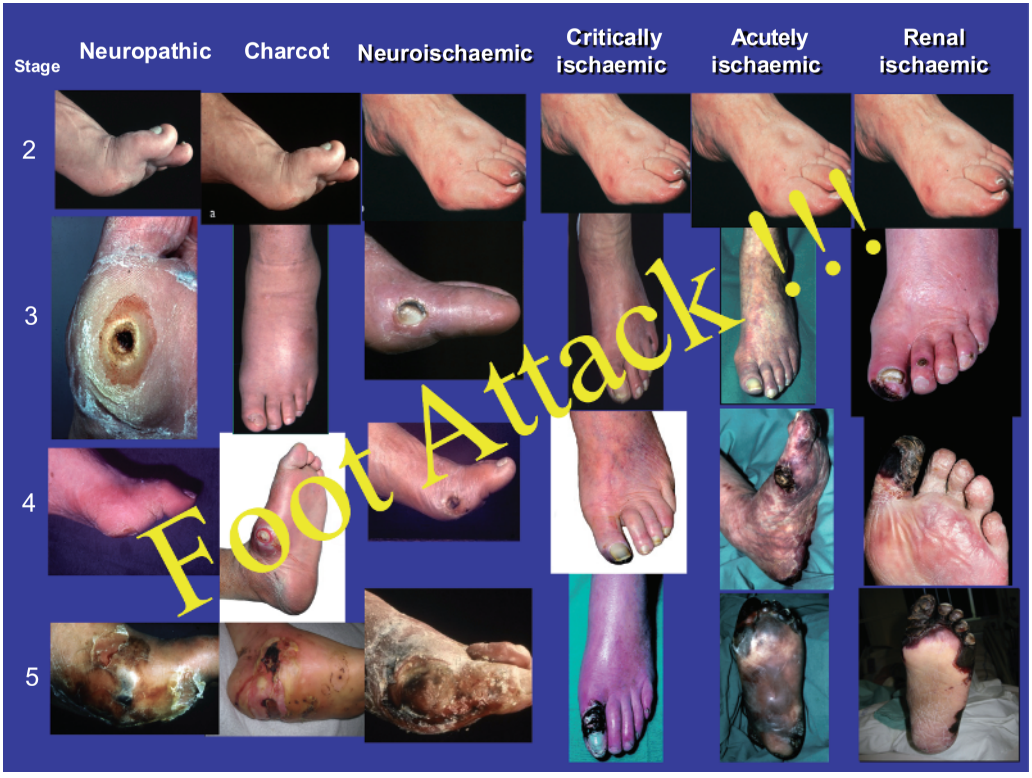


Fig. 1.23 Foot attack in the natural history of the neuropathic and ischaemic foot.

can be described as a 'diabetic foot attack' with similarities to the heart attack and brain attack (Fig. 1.23). Essentially, there are two drivers of the 'diabetic foot attack' which leads to necrosis: infection and ischaemia. Infection results in a septic vasculitis and is a prominent feature both of the neuropathic foot and the neuroischaemic foot. It can also complicate ulcers associated with the Charcot foot. In the remaining scenarios, the driver is ischaemia producing the clinical syndromes of critical ischaemia, acute ischaemia and the renal ischaemic foot.

INTERVENTION

At each stage of the diabetic foot it is necessary to intervene early and take control of the foot to prevent further progression. Management will be considered under the headings shown in Table 1.3. This book describes full details of practical management for each stage in the appropriate chapter with one colour-coded chapter for each of the six stages. The book contains sufficient, easily accessible information to enable the practitioner to make rapid, effective decisions which will prevent deterioration and progression to necrosis and amputation.

Multidisciplinary foot management

In the modern management of the diabetic foot, two teams have established importance: the multidisciplinary foot care team and the foot protection team.

The multidisciplinary foot care team

Successful management of the diabetic foot needs the expertise of a multidisciplinary foot care team which provides integrated care focused in a diabetic foot clinic. Members of the team comprise podiatrist, nurse, orthotist, microbiologist, physician, radiologist and surgeon, including vascular surgeon, orthopaedic surgeon and plastic surgeon. Some roles may overlap, depending on local expertise. The multidisciplinary foot care team should be available to assess outpatients with active foot

disease, not only in routine appointments but also as emergencies. Patients in Stages 3–5 are best seen within a multidisciplinary foot service. Day-to-day multidisciplinary treatment is carried out by podiatrist, nurse, orthotist and physician in the diabetic foot clinic. Further management is achieved by holding regular joint multidisciplinary clinics with vascular and orthopaedic surgeons in the diabetic foot clinic. Through these specialist clinics, it is possible to organise a 'fast-track' service in a 'one-stop' visit, comprising clinical assessment, same-day investigations and urgent treatment.

In the joint clinic with the vascular surgeon, the need for angiography can be rapidly agreed upon and either angioplasty or bypass surgery can then be promptly carried out. Ischaemic patients are followed up post-operatively to provide continuity of care for wounds and amputation sites. Follow up includes routine preventive foot care, regular updating of education to detect problems early, rapid and aggressive care of ulcers, regular arterial and graft surveillance and the provision of statins and aspirin for secondary prevention of arterial disease.

In the joint clinic with the orthopaedic surgeon, patients with Charcot osteoarthopathy and patients needing complex deformity corrections and limb reconstructions can also be seen. In the joint clinic with the plastic surgeon, patients with tissue destruction and non-healing wounds are seen and considered for skin grafts and free flaps.

The diabetic foot clinic should offer rapid access, early diagnosis and prompt help for patients with urgent foot problems, providing podiatric, nursing, surgical and medical treatment. The use of modern imaging is important in diagnosis, and the foot clinic can facilitate urgent visits to radiology, nuclear medicine, ultrasound departments and the vascular laboratory. The clinic can facilitate urgent measurement of inflammatory parameters, such as serum C-reactive protein (CRP). Each team member should be available quickly for emergency appointments, which can be run concurrently with routine clinics so that patients with new ulcers, pain or discolouration can be seen the same day. This includes patients

in whom overwhelming infection or ischaemia is 'attacking' the foot and leading to significant necrosis in a 'diabetic foot attack'. Such patients need rapid treatment and admission to hospital through the emergency service provided by the diabetic foot clinic.

When diabetic patients with severe foot problems have been admitted to hospital, they should be looked after by the multidisciplinary team. Some patients may be under the primary care of the vascular, orthopaedic or plastic surgeons and others under the care of the diabetologist. However, there should be joint ward rounds and multidisciplinary team meetings. This has been facilitated at King's College Hospital by the creation of the post of diabetic foot practitioner, who, as an experienced podiatrist, coordinates all aspects of inpatient care, including wound, microbiology, mechanical, vascular, metabolic and educational aspects, as shown in Table 1.3.

Overall, the diabetic foot clinic has taken the role of an 'operations' centre or a 'command' centre for the prevention of amputation in vulnerable diabetic neuropathic and ischaemic patients that are susceptible to trauma, ulceration, infection and necrosis.

The foot protection team

In contrast to patients with ulceration in Stages 3–5, patients in Stage 2 without ulcers, but at an increased risk of developing lower limb complications, should be treated by the foot protection team. Typically, members of this team include podiatrists, orthotists and other foot care specialists who are based in primary care. The important aspect of management is screening for patients at risk of ulceration and then providing follow-up care and education for those patients. However, there should be very rapid referral pathways between the foot protection team and the multidisciplinary foot care team if a patient develops ulceration.