Chapter 13
The Role of an Orthopaedic Surgeon in the Management of Diabetic Foot Complications

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General Assessment of Diabetic Feet

An orthopaedic history is elicited from the patient. The following lines of enquiry should be pursued: swelling, pain, recent trauma, abnormal sensation, history and duration of foot ulcer, history and progression of deformity. The surgeon should ask about proximal joint problems—of the knee and hip, as often these can exacerbate foot deformities, or lead to gait problems, predisposing the patient to abnormal shear stresses in the plantar foot skin. The examination of the patient will involve a rudimentary assessment of the hip and knee looking for obvious deformity and contractures and stiffness of the joints.

Examination of the feet involves the following steps:

1. Footwear and insoles—custom or proprietary, wear pattern of the sole.
2. Hind foot alignment on standing—is the heel in anatomical valgus.
3. Gait pattern
4. Loss of medial arch
5. Integrity of plantar fascia
6. Tightness of the Achilles tendon and Gastrocnemius muscle.
7. Quality of the skin—healthy and pliable or dry cracked, presence and location of ulcers and their depth.
8. Ankle and hind foot deformity—are the deformities correctible?
9. Midfoot deformity and presence of exostoses

   Forefoot deformity and mobility: presence of claw, hammer and mallet toes

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10. Pulses, including doppler insonation and recording the signal quality—triphasic is normal; biphasic or monophasic indicates a degree of arthrosclerosis and loss of normal vessel wall elasticity. Recording the Ankle Brachial Pressure index which may also be falsely elevated in the diabetic patient
11. Sensation to light touch and protective sensation with the Semmes Weinstein 10 g monofilament
12. Temperature of the foot either by simple palpation, or with a handheld cutaneous thermometer

The standard X-ray imaging required is a weight bearing foot and ankle series. This is an AP, mortice and lateral view of the ankle and foot with an oblique and AP of the foot. Other specialist views can be useful such as an axial calcaneal view to localise a posterior tuberosity lesion.

More recently weight bearing computerised tomography has been used. This can identify any prominences in the loaded foot that might not be apparent on two dimensional radiographs such as plantar displaced or subluxed intermediate and lateral cuneiforms. Three-dimensional reconstruction views are helpful in allowing the surgeon to visualise a complex deformity as part of reconstructive planning process, to work out the shape and orientation of corrective osteotomies. There are certain centres that use 3D printers to create models of the deformed foot so the surgeon can plan and rehearse the orientation of the osteotomy required for corrective surgery [1, 2].

Ultrasound is useful in localising collections of pus in the foot or the tendon sheaths, directing surgical drainage.

MRI has a high sensitivity for confirming infection in the foot. A normal MRI virtually excludes infection. The diagnostic accuracy is 95%, with 95% sensitivity, and 80% specificity. It does not differentiate between infection and oedema, and therefore will not distinguish between an infective process or a Charcot neuroarthropathy. PET and SPECT scans have a role in identifying infections with similar diagnostic accuracy, sensitivity, and specificity. The wider availability of MRI scanners and the lack of exposure to ionising radiation leads to MRI being the investigation of choice [3, 4].

Other useful adjuncts include transcutaneous oximetry. This measures the partial pressure of oxygen in the skin at the site of the incision, usually in the foot. This investigation modality is usually employed by the vascular team and can be used as a predictor for ulcer and wound healing and likelihood for lower limb amputation [5, 6]. A partial pressure of greater than 30 mmHg in the subcutaneous tissue at the ankle indicates healing potential of wounds in the foot, with reported healing rates of 92% [7].

A panel of blood tests are useful: these include a full blood count, C reactive protein (CRP), bone profile, and glycosylated haemoglobin.

A leucocytosis and elevation of the CRP suggests an infection. An abnormal bone profile suggests bone involvement. The glycosylated haemoglobin is an indicator of metabolic control. Poor diabetic control is an unfavourable prognostic indicator of wound healing and wound complications following surgery [8–10].

Off-Loading Surgery for Diabetic Foot

Diabetic feet are vulnerable to ulceration, due to a combination of peripheral neuropathy, ischaemia, and deformity leading to loss of the normal load bearing structure of the foot. Patients have a distal symmetrical polyneuropathy resulting in a sensory loss in a stocking distribution with large fibre involvement causing tingling, paraesthesia, and eventually numbness. Vibration and touch pressure sensation are also impaired. Deterioration of the polyneuropathy gradually spreads to autonomic fibres resulting in loss of sudomotor function. There is small muscle atrophy of the intrinsic foot muscles; the imbalance between these and the long flexors leads to flexion deformities of the toes, and hyperextension at the metatarsophalangeal joints, causing claw and hammer toes in the forefoot.

The sensory neuropathy causes the loss of protective sensation resulting in the patient being unable to detect minor injury to the foot. The loss of sweating causes the foot to lose its natural pliability and the skin becomes cracked and fissured. The skin is vulnerable to minor trauma, which can lead to inoculation of the wound with microbes, resulting in an ulcer which if left undetected will deteriorate and become infected. Protective sensation can be assessed using the 10 g Semmes Weinstein monofilament [11, 12].

Subluxation of the metatarsophalangeal joints and the metatarsal head fat pad atrophies, leading to increased pedal pressure on the metatarsal heads, as demonstrated in pedobarographic studies. The skin is susceptible at the apex of the toe flexion deformity or at the subluxed metatarsal heads and is extremely vulnerable to injury and ulceration from direct contact with the floor or shoes (Fig. 13.1). Patients often develop calf tightness with gastrocnemius contractures which creates an equinus contracture at the ankle joint and resulting in increased forefoot pressures [13]. The ulceration may develop initially in the soft tissue; if the bone or joint becomes involved, osteomyelitis may ensue. The situation can deteriorate and potentially lead to a septic diabetic foot emergency requiring urgent surgical debridement.

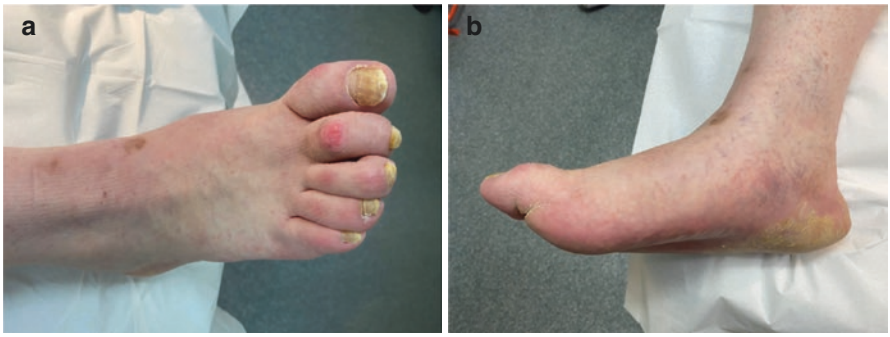

Fig. 13.1 (**a**) Hammer toe with ulcer on proximal interphalangeal joint; (**b**) Hammer toe of Hallux with associated swelling of digit, and trophic skin changes on heel

The aim of prophylactic offloading surgery is to reduce the risk of ulceration in this group of patients. The surgical armamentarium includes soft tissue releases and bony procedures. Gastrocnemius muscle can be released to reduce forefoot pressure on the metatarsal heads [14, 15]. This can be undertaken proximally at the level of the medial head of gastrocnemius, or in the midcalf aponeurosis with a Strayer's procedure [16, 17].

This procedure can be done under sedation with a local anaesthetic field block just distal to the popliteal crease, over the medial head of the gastrocnemius muscle belly. A midcalf release of the triceps surae aponeurosis can be undertaken if the proximal operation is inadequate. Lastly, if there is a significant contracture of the tendoachilles, the tendon can be lengthened with a Hoke triple hemisection [18, 19]. This results in slowing the Charcot process and reduces plantar pressures (Fig. 13.2). The percutaneous technique can be performed in an outpatient setting. Release of the Achilles tendon can cause weakness of the calf muscle and lead to loss of push off power during the gait cycle, as well as a calcaneus deformity of the posterior tuberosity of the heel and subsequent ulceration. It needs to be done with caution bearing those points in mind [20].

The imbalance between the long flexors of the foot and the intrinsic foot muscles contribute to an extension deformity at the metatarsophalangeal and interphalangeal

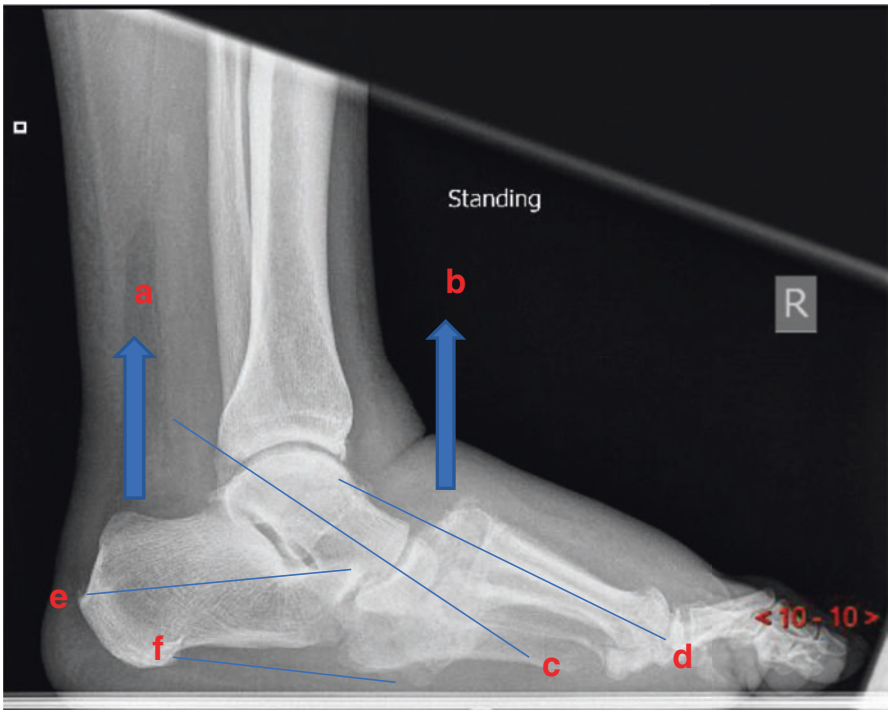

Fig. 13.2 Direction of pull of tendons. (**a**) Pull of tendoachilles; (**b**) pull of tibialis anterior; (**c**, **d**) loss of first metatarsal-talar alignment; (**e**) loss of calcaneal pitch leading to a rocker bottom deformity of a Charcot foot; (**f**) 'dropped' cuboid height

joints, resulting in claw, hammer, and mallet toes respectively. There is no direct physical evidence for this causal relationship, and it is thought that there are other factors involved with this pathological process. There is an increased plantar pressure on the affected skin and as a result the toes are vulnerable to ulceration on the dorsum of the proximal interphalangeal joint, at the pulp of the toe or under the metatarsal head where there is atrophy of the plantar fat pad [21, 22]. The dry, fissured skin is more vulnerable to ulceration and creates a portal of entry and inoculation of the deeper soft tissues, joint and eventually bone. The added complication of a microvascular angiopathy results in a poor environment for healing and the ulcer deteriorates.

This situation can be avoided with careful skin care and provision of shoes with a total contact insole, wide and high toe box. Patients often present to the orthopaedic surgeon with these deformities and a neuropathy. Release of the flexor digitorum longus tendons can be undertaken using a percutaneous needle tenotomy technique in outpatients. Studies have shown that this is a safe and effective treatment for diabetic patients with ulcer healing rates of 93% and no complications following treatment [23, 24].

Patients with long standing neuropathic ulcers beneath the subluxed metatarsal heads can be treated with osteotomies to shorten the metatarsals. Historically open techniques with screw fixation have been used, but fell out of favour because of the high rate of complications with infection of the wound, and failure of fixation [25]. A minimally invasive surgical osteotomy using a burr minimises the soft tissue insult to the foot. The osteotomy is at the level of the neck and is not stabilised with an implant. This way the risks of wound and implant infection are mitigated, however the toes can end up ‘floating’ with the pulps of the digits not engaging the ground [26, 27]. Metatarsal osteotomies are more effective in leading to ulcer healing than standard non operative offloading treatments, with 96% of ulcers healing within 1 month of surgical off-loading compared to 68% healing after standard non-surgical offloading treatment [28].

The midfoot can develop a rocker bottom deformity with a plantar medial bony prominence caused by a loss of relationship between the navicular, medial cuneiforms and first metatarsal base (Figs. 13.3, 13.4, and 13.5). This creates a plantar

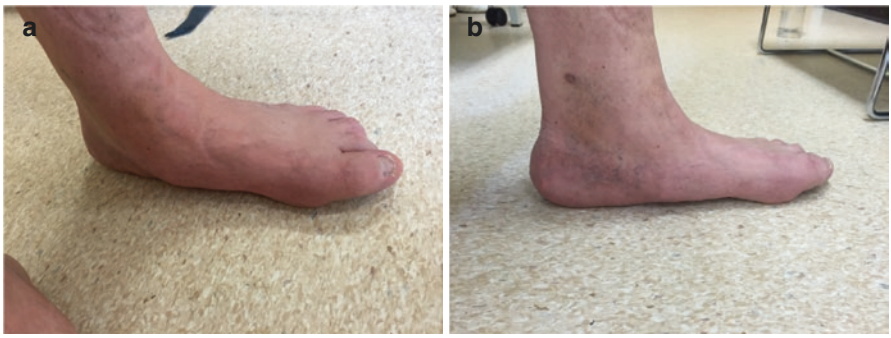

Fig. 13.3 (**a**) Medial rocker bottom Charcot foot deformity; (**b**) loss of medial arch

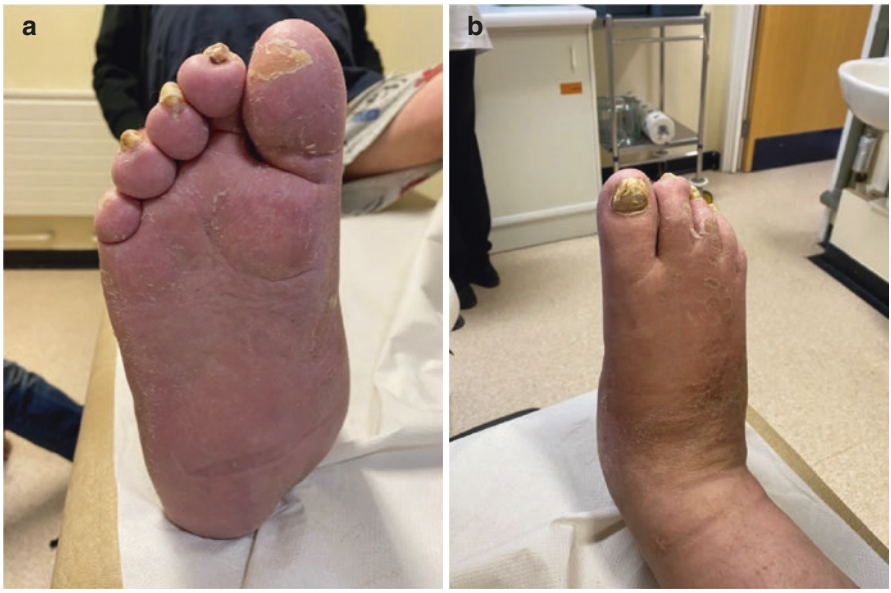

Fig. 13.4 Charcot foot with medial prominence and rocker bottom. (**a**) Trophic skin changes due to autonomic neuropathy; (**b**) dorsal foot swelling

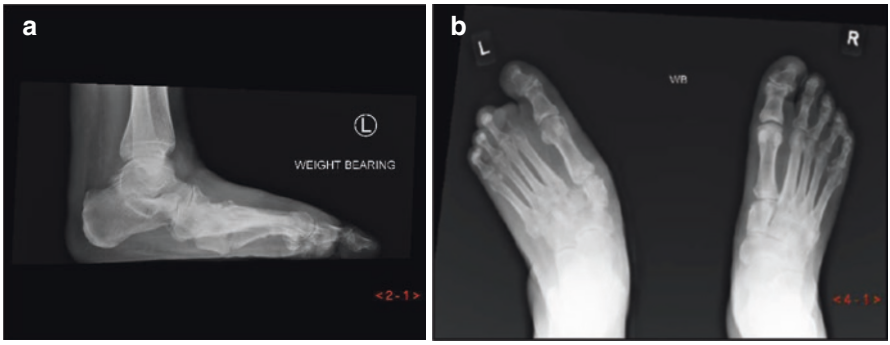

Fig. 13.5 Charcot foot XR with midfoot failure. (**a**) Loss of alignment between first ray and talus resulting in rocker bottom and abduction deformity; (**b**) failure of Lisfranc ligament complex leading to dissociation of second metatarsal from first ray with abduction of the forefoot

medial apex as the medial cuneiform is pushed out in the same direction. Pressure and friction on the skin leads to ulceration and eventually infection of the soft tissues and the underlying bone.

The bony exostosis can be surgically removed to reduce pressure on the overlying skin, therefore preventing ulceration or resulting in healing of the ulcer (Figs. 13.6 and 13.7) [29–31]. Bony prominences on the plantar lateral aspect of the foot secondary to a 'dropped' cuboid, with disruption of the lateral plantar arch are more pernicious, and have a poorer prognosis. Simple exostectomy does not have

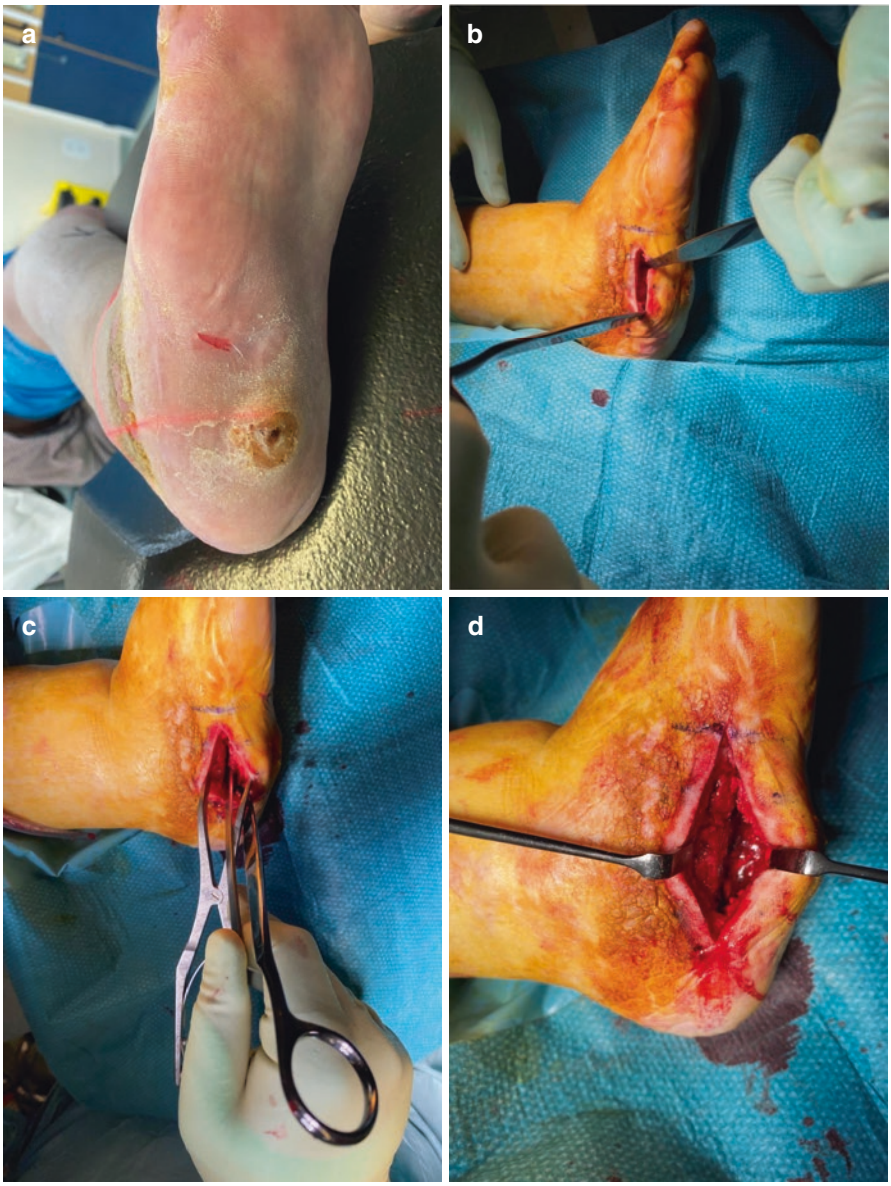

Fig. 13.6 Exostectomy. (**a**) Plantar ulcer; (**b**) lateral approach to exostosis; (**c**) exostectomy with osteotome; (**d**) completed exostectomy

the same effect and the foot deformity continues to deteriorate and the skin remains at high risk of re-ulceration. The foot needs reconstruction with restoration of the lateral and medial longitudinal arches with beams and plates. Studies have shown a negative association with loss of cuboid height and progression of the Charcot deformity [32, 33].

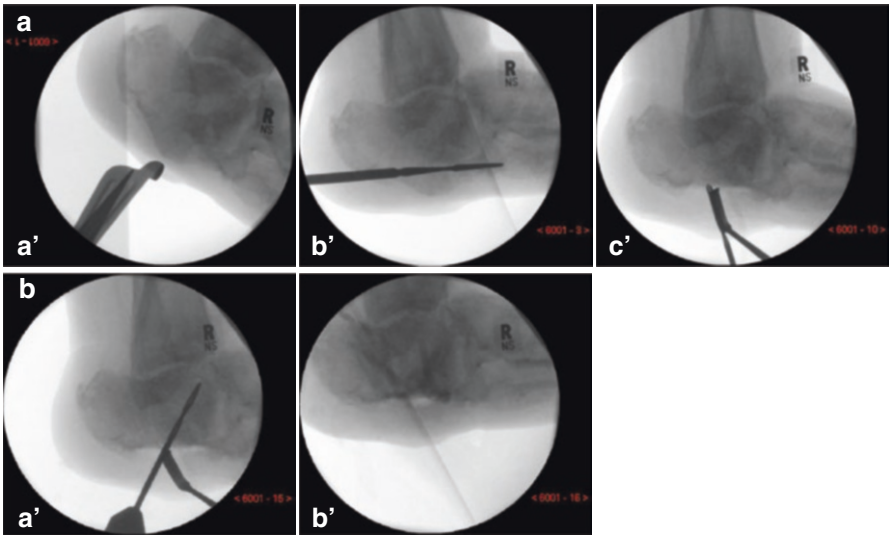

Fig. 13.7 (**a**) Intraoperative images of exostectomy: (**a′**) plantar exostosis; (**b′**) level of exostectomy; (**c′**) post exostectomy. (**b**) Calcaneal silo technique: (**a′**) drilling of calcaneum for SILO technique; (**b′**) injection of antibiotic bone void filler into calcaneal drill holes

Surgical Management of Bone Infection

A neglected ulcer diabetic foot ulcer will lead to soft tissue infection and eventually deeper structures, joint and bone, will become infected. Patients present with swollen digits, joints, redness, systemic malaise, cellulitis, septic arthritis, and osteomyelitis. The surgical strategies for treatment are radical debridement of the infected tissue back to a healthy margin, and systemic antibiotics. The rational for debridement is source control, and removal of biofilm. The bacteria accumulate in the biofilm and are immunologically privileged. They take refuge in a hyperglycaemic and poorly perfused environment, beyond the reach of white blood cells and the action of systemic antibiotics. These colonies are commonly polymicrobial. The surgical debridement disrupts the biofilm and disperses the bacterial colonies to their planktonic form, rendering them more susceptible to systemic antimicrobial therapy [34, 35]. Diabetic wounds demonstrate deregulated angiogenesis, and a suppressed inflammatory response. The hypoxic environment results in a poor wound healing prognosis [36].

Patients commonly present to the podiatrist or the foot protection team in hospital with an indolent non-healing ulcer, and a swollen digit or foot. There is erythema, and the foot is warm. The foot needs a full assessment including an X-ray and MRI. Occasionally if there is an occult collection of pus, an ultrasound scan of the foot and the tendon sheaths around the ankle can be useful. The blood tests are sometimes helpful with a raised white cell count and elevated inflammatory markers [35]. The patient's blood glucose is often deranged and those with the most severe infections or presenting with foot sepsis will have very unstable metabolic control.

Clinically the patient may demonstrate a general malaise, and sometimes demonstrate signs of confusion.

A deep bone biopsy should be acquired before commencing systemic antibiotics in accordance with local microbiology guidelines. The most commonest microbes are Staphylococcus aureus and epidermidis and Escherichia coli [37]. Streptococcus is also present in very severe skin infections [35, 38, 39].

The decision to operate is based on the presence of pus or wet gangrene, and the severity of the patient's clinical condition. If the patient is septic, urgent debridement is required for source control [40, 41]. The patient is taken to theatre for an emergency debridement and removal of the infected tissue back to a healthy bleeding margin [42]. Lavage of the wound is carried out with high volume saline, and aqueous chlorhexidine for a local bactericidal effect [43].

Specimens are taken from the infected tissue, and a marginal specimen is sent following debridement and washout. If a local debridement is performed, and there is concern that the margins have not been cleared of infection, it is sometimes necessary to preserve potentially infected bone to maintain the functional anatomy of the foot, then the bone can be treated with a topical local antibiotic bone void filler (BVF) [44, 45]. Following surgery the wound can be closed primarily if the skin is healthy, and a tension free closure can be achieved. If there is a defect application of negative pressure wound therapy (NPWT) dressing can be applied. If BVF has been deployed in the wound, the NPWT dressing is put on 24–48 h later. This allows the BVF to cure and reduces the risk of all the antibiotics eluting prematurely into the NPWT dressing.

Patients with deep bone infections without severe soft tissue involvement or in the absence of a septic presentation can be considered for a more planned approach. A bone specimen is required for targeted antibiotic treatment. If the focus of osteomyelitis is deep to an ulcer caused by a bone prominence, an exostectomy should be considered, and the underlying residual infected bone can be treated with antibiotic impregnated BVF. The BVF can be deployed using a silo technique which involves drilling the residual bone with holes, taking care to avoid penetration into neighbouring unaffected bone, and injecting the BVF into the holes (Fig. 13.8). A sucker can be placed into one of the adjacent drill holes and the suction pressure will draw

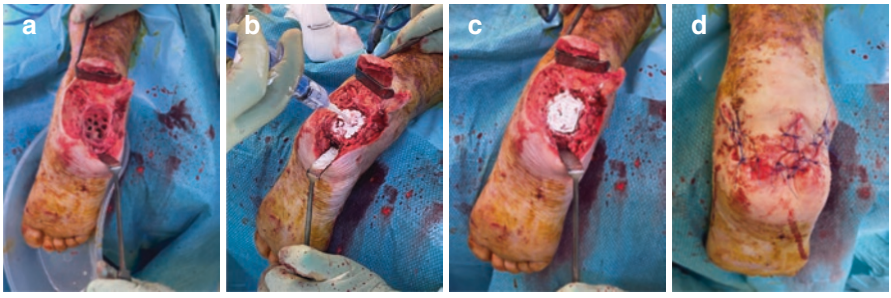

Fig. 13.8 Calcanectomy and silo technique. (**a**) Partial calcanectomy with drill holes; (**b**) filling drill holes with bone void filler; (**c**) filling of all holes; (**d**) closure of skin

the BVF, in its liquid phase, into the surrounding bone trabeculations, facilitating a deeper and wider penetration of the filler into the residual bone [45]. This technique can also be used for emergency debridements. It allows hindfoot osteomyelitis to be treated with local resection of infected tissue and avoiding radical resection of the bone to maintain the function of the lower limb.

Bone infections of the digits confined to the distal or intermediate phalanx can be treated with radical debridement of the toe at either the middle or proximal phalanx level to remove the infection completely (Figs. 13.9, 13.10, and 13.11). Antibiotic impregnated BVF is rarely required for these cases. Infection of the metatarsal heads are dealt with by removing the infected bone with a ray amputation and filling the residual stump with BVF. If the surrounding soft tissues are healthy the wound can be closed immediately, otherwise a larger defect can be treated with a NPWT dressing [46, 47].

Following surgery, the patient's inflammatory markers and glycaemic control are monitored. Patients who have undergone massive emergency debridement with large open soft tissue defects may require further washouts and debridement. If a

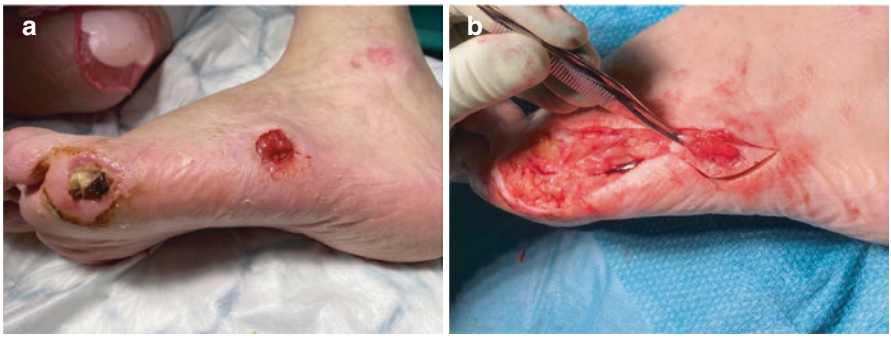

Fig. 13.9 Fifth metatarsal ulcers. (**a**) Fifth metatarsal head and base ulcers; (**b**) elliptical excisions of both ulcers

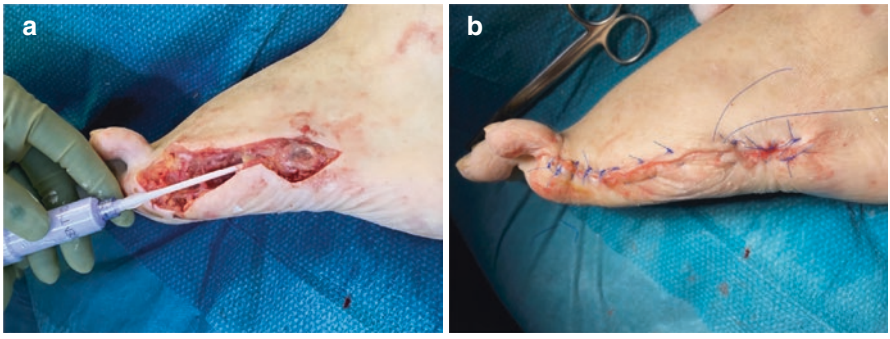

Fig. 13.10 Fifth ray amputation. (**a**) Fifth metatarsal ray amputation and injection of antibiotic impregnated bone void filler. (**b**) Direct skin closure

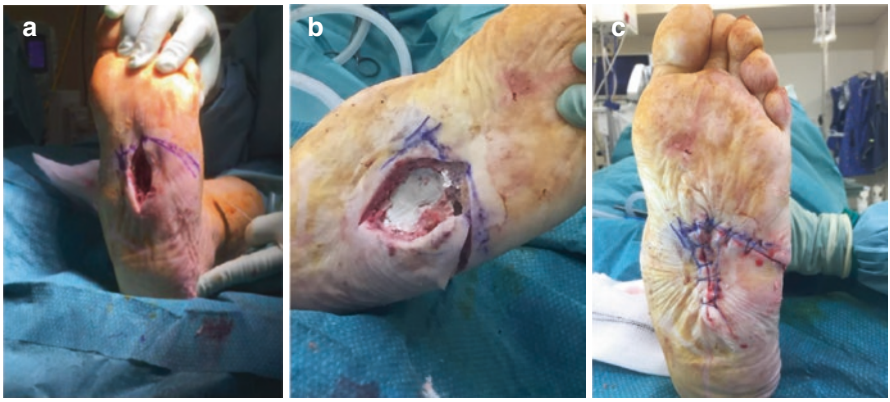

Fig. 13.11 Local flap. (**a**) Plantar central ulcer excised and local flap design; (**b**) exostectomy with bone void filler in residual bone; (**c**) closure of local flap

large soft tissue defect remains and cannot be closed by delayed secondary closure with NPWT, the patient can be referred to plastics for consideration of alternative means of wound coverage [48, 49]. Patients will require systemic antibiotics as per local microbiology guidelines with regards to duration and method of delivery—intravenously or oral [50].

Successful outcome following conservative treatment of diabetic foot infections is reported in some studies at around 63% [51]. The outcome following surgical treatment of diabetic foot infection varies between surgical reports with a long-term limb salvage rate between 70–90%; the recent use of antibiotic impregnated bone void filler has shown promising early results in some series [44, 52, 53]. The main criterion influencing the long-term outcome is the vascular status [54].

Charcot Foot Reconstruction

The structure of the foot can be described as a complex of a medial, and lateral arch connected by a transverse arch. The medial arch consists of the first metatarsal, medial cuneiform, navicular, talus, and calcaneum. The keystone of the medial arch structure is the talus, which is supported by a sling of soft tissue with the spring ligament, tibialis posterior and anterior tendons, and the peroneus longus tendon insertion at the base of the first metatarsal on its plantar aspect.

The lateral arch comprises of the fifth metatarsal, cuboid, and calcaneum, with soft tissue support from the calcaneocuboid ligaments and the shape of the joint. The peroneus longus forms a support beneath where it enters the plantar aspect within the cuboid groove.

The transverse arch is made up by the navicular cuneiform joint complex and metatarso-cuboid joints of the fourth and fifth rays, supported by the midfoot

ligaments, and the Lisfranc ligament locking the base of the second metatarsal shaft into the intermediate cuneiform as the key stone of this arch.

The plantar fascia takes origin from the base of the anterior calcaneal tuberosity into the plantar aponeurosis extending to the metatarsophalangeal joint level. This structure contributes to foot stability and maintenance of the arches by acting as a windlass mechanism.

The arrangement of the three arches creates a vault—an architectural construct which allows a load to supported by a dome ceiling, transferring the weight to each of the supporting pillars. In the foot this corresponds the weight of the tibia through the talus and into the base of the calcaneum and the five metatarsal heads, with the loaded dome of the talus being supported by the medial, lateral and transverse arches. Failure of the bones or supporting ligaments will lead to collapse of the arch and deformity of the foot.

Charcot neuroarthropathy is a relatively rare condition which affects 1% of diabetics. It is painless in 80% of cases, and often preceded by minor trauma. It can affect the other side in 10% of cases. The pathophysiology of Charcot foot is created by a combination of a neurotrophic and neurotraumatic mechanisms. The autonomic neuropathy results in smooth muscle relaxation of the arterial wall leading to failure of local vasoregulation and arteriovenous shunting of the local microcirculation. With the hyperdynamic local blood flow, there is an increase in monocytes and osteoclasts, causing resorption of the bone. There is loss of bone density and osteopaenia.

This results in the bone being easier to fracture. The loss of proprioception and protective sensation results in unrecognised microtrauma, and fractures. There is loss of joint congruity with ligament injury, leading to subluxation and failure of the structural integrity of the three arches (Fig. 13.12) [55].

Diagnosis of the Charcot foot is a combination of clinical assessment and radiological investigations (Fig. 13.13). The skin quality, foot posture and joint deformity, signs of neurological and vascular deficiency are assessed. Callosities, loss of sudomotor response, loss of protective sensation, skin temperature, redness, paraesthesia, and dysaesthesia are noted. The redness in a Charcot foot diminishes in

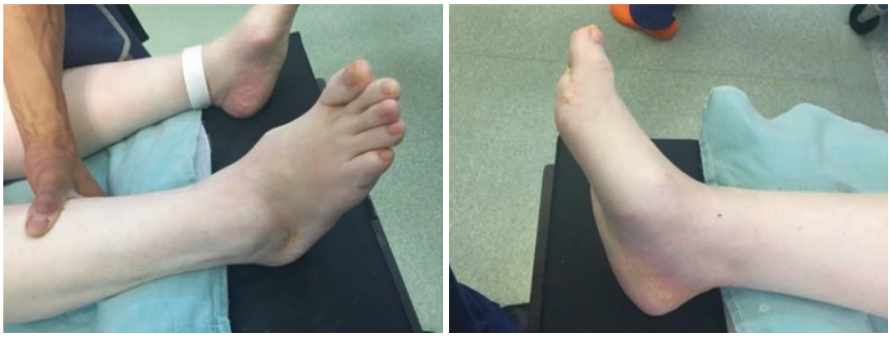

Fig. 13.12 Clinical photo of charcot foot

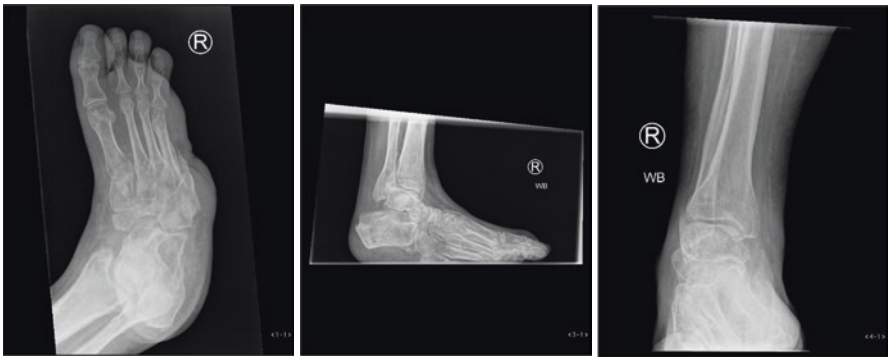

Fig. 13.13 Charcot foot X-ray

colour upon elevation suggesting a reactive hyperaemia. Vibration sense can also be diminished.

There are no serological criteria for Charcot foot. The inflammatory markers might be elevated if there is concurrent infection of a diabetic foot ulcer or underlying bone infection.

The modified Eichenholtz classification [56] for the staging of Charcot is as follows:

Stage 0: (Shibata)

Patients present with swelling erythema and warmth of the foot without any radiological changes. Management of the condition involves education, protected weight-bearing, and serial radiographic monitoring.

Stage 1: Inflammation

The acute Charcot process is a localised inflammation to a traumatic incident which precipitates the response in a vulnerable foot. This is caused by an imbalance between pro and anti-inflammatory cytokines. The bone responds with an acute phase release of pro-inflammatory cytokines, TNFα, and interleukin 1β, and interleukin 6, with a corresponding decrease in interleukin 4 and 10—anti-inflammatory cytokines. TNFα and interleukin 1β together initiates increased expression of receptor activator nuclear transcription factor κB (NF-κB) ligand (RANKL), activating monocytes and osteoclastic maturation, resulting in localised bone resorption. There is an intense and protracted inflammatory response of the bone as a result of the alteration of these cytokines, which results in bone destruction, and loss of structural integrity [55, 57]. Collapse of the medial arch at either the naviculocuneiform or talonavicular level creates an abduction deformity of the midfoot, and eventually the actions of tibialis anterior and tendoachilles causes a rocker bottom foot

deformity. As a result, a plantar medial bone prominence develops and becomes vulnerable to ulceration. (see Fig. 13.2).

The lateral arch integrity relies on the relationship between the cuboid and the calcaneum. As the calcaneocuboid ligament fails the cuboid drops, as the posterior tuberosity of the calcaneum is pulled proximally, and a prominence develops on the plantar lateral aspect of the foot.

Management of this stage involves protection in a total contact cast, which is changed initially weekly until the swelling settles and then every 2 weeks. The cast treatment can take between 2–4 months, or until the skin temperature settles to within 2° of the uninvolved foot, and there is evidence of radiographic resolution of bone fragmentation.

Stage 2: Coalescence

Absorption of bone debris, sclerosis, and bone consolidation and new bone formation occurs during this stage. This can be noted on the plain radiographs with increased bone density, and bony ankylosis of some of the larger bone fragments. Clinically the foot swelling and erythema decreases, and the temperature equalises to that of the unaffected foot. The foot is managed in a total contact plaster or a Charcot restraint orthotic walker (CROW).

Stage 3: Reconstruction/Remodelling

The foot now is the same temperature as the other foot, with an absence of swelling and redness. The joints are no longer collapsing, and the deformity is stable. The radiographic appearance is one of arthrosis or fibrous ankylosis. Fragments of bone appear rounded and smoothed off. For the plantigrade foot custom shoes with total contact insoles, a rigid shank and a rocker bottom sole are indicated. In a deformed foot with skin at risk of ulceration, exostectomy, or reconstruction with internal fixation may be necessary. Some patients complain of arthritic pain from their Charcot deformity—another indication for surgical reconstruction.

The intra and interobserver reliability of the Eichenholtz classification has not been validated, and it is difficult to identify the transition phases of the three stages. The lack of anatomical localisation makes the Eichenholtz classification difficult to apply in the treatment decisions for Charcot feet.

The goal of Charcot foot reconstruction is plantigrade with no bone prominences. At the end of the treatment the foot should fit into a custom shoe with a total contact insole. The surgical principles are rigid long segment reconstruction with beams and plates [58, 59].

External fixation can also be used to achieve long multisegment fixation [60]. Sammarco, in 2008, popularised long segment fixation, creating a "superconstruct"—in which the zone of collapse or injury is bypassed by anchoring the implant in healthy bone either side of the pathological joint segments [61–63].

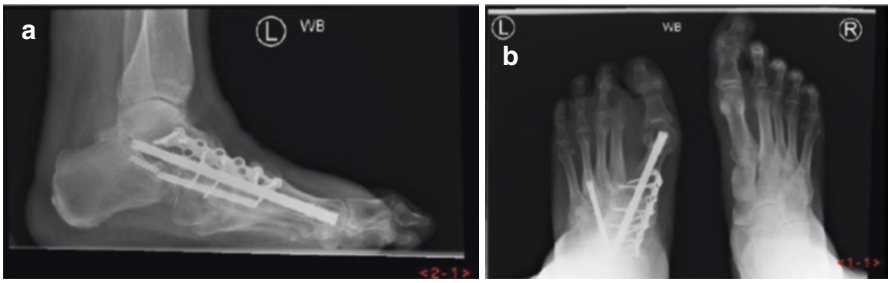

Fig. 13.14 Charcot reconstruction. (**a**) Reconstruction of medial arch by restoring alignment of first metatarsal and talus, and fusing with an intramedullary beam, neutralised with a medial plate. (**b**) Locking fourth tarsometatarsal joint to prevent collapse and failure of the lateral arch

Applying this principle, the medial ray is fixed from the head of the first metatarsal to the talus to reconstruct a midfoot Charcot rocker bottom deformity. The lateral arch is stabilised with fixation from the fourth or fifth metatarsal into the calcaneum. The intervening bones are fully prepared with removal of the articular cartilage, bone is resected with corrective osteotomies to restore the alignment of the arch, and to decompress the soft tissue. Incisions should be restricted to a single angiosome, to avoid disruption and mitigate the risk of wound breakdown (Fig. 13.14).

The implant design has gone through several iterations, with the current third generation of screws and plates giving the surgeon the best chance of creating a stable mechanical environment allowing the bones to fuse. The plate is applied to add further rigidity and neutralise rotational forces. The ankle and hindfoot joint can be fused using an intramedullary nail device augmented with a lateral or anterior plate for rigidity [63]. The literature reports an overall fusion rate of 86%, with a complication rate of 36%, and 95% of patients returning to weight bearing ambulation. The overall amputation rate is 5.5% [58].

In the infected Charcot deformity, reconstruction can still be a surgical option. The infection is initially treated, and the ulcer must be in remission before surgery is undertaken. Surgery is done in two stages. At the first stage the ulcer and infected bone are excised, and the residual bone treated with antibiotic impregnated bone void filler. The foot is temporarily stabilised with large diameter K wires, and the ankle placed in a plaster. The wound is dressed and monitored. Systemic antibiotics, as guided by local microbiology protocols, are given. The inflammatory markers and white blood cells are serially monitored for 2 months. Once these have normalised, the second stage definitive fixation can take place, utilising the principles of long segment rigid fixation, creating a superconstruct to bypass the zone of abnormal bone. The rate of fusion is 83% in the hindfoot and 60% in the midfoot for these two stage procedures as shown in recent series [64].

Healing of the bone takes approximately 3–4 months, and the patient is protected in a plaster for that time. Following removal of the plaster, the patient can be fitted with a custom-made shoe with a total contact insole and allowed to ambulate.

Key Points

- The role of the orthopaedic surgeon is to perform an orthopaedic foot and ankle examination and assessment of the patient presenting with diabetic foot complications.
- To undertake emergency debridement of foot infections. This depends on the organisation of services in the hospital. In some centres this responsibility falls to either the General or Vascular surgeons.
- Offloading surgery for diabetic feet to prevent ulceration of skin, or promote healing of ulcers, with a combination of soft tissue releases, and bony procedures
- Treatment of diabetic foot ulceration and bone infection. This should be done urgently to treat the septic foot and to preserve tissue, and ultimately function. "Time is tissue".
- Non-surgical management of Charcot foot is indicated for a deformity without bony prominences making the skin vulnerable to ulceration. The limb can be managed in a custom shoe with a total contact insole or a CROW boot.
- Surgical management of Charcot deformity:

  The bone exostoses predisposing vulnerable neuropathic skin to ulceration should be removed, and tendon releases performed to neutralise the forces driving the deformity.

  Surgical reconstruction to realign the medial and lateral rays, and restoration of the hindfoot alignment in relation to the tibiotalar axis. The aim is to create a rigid multisegment fusion.

  Fusion rates are reported to be between 60% and 80%, and overall amputation rate is 5.5%.

  Infected Charcot can be managed with two stage surgical reconstruction.

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