1

Neuropathic Case Studies

1.1 Introduction

In the past we have often described the neuropathic foot as a 'forgiving' foot, and there is little doubt that of the four main categories of patients – neuropaths, neuroischaemics, renals and Charcots – described in this book it is the neuropathic patients who do the best. It is, however, important never to underestimate the problems of the diabetic patient with neuropathy, which is a devastating deficit. In many of these patients, neuropathy affects other anatomical systems such as the cardiovascular, gastrointestinal and urogenital systems, and not just their feet and legs, and as a result they are incredibly frail and vulnerable, with greatly increased susceptibility to infections and other insults. When managing the neuropathic patient with foot problems, particular regard must be paid to all these susceptibilities and vulnerabilities. It is often said of these patients with neuropathic feet "Good pulses, not ischaemic, not in trouble" – but the neuropathic patient is actually very fragile and may rapidly develop severe problems and therefore can get into trouble very quickly.

In choosing sections for this chapter, we have included those that substantially affect the patient with diabetic neuropathic feet: highlighting first the role of infection, the "great destroyer", which is a real mask of Janus, putting on so many different and deceptive faces in the diabetic patient (a theme that is repeated in every chapter). Second comes the effect of neuropathy in conjunction with other co-morbidities that are present in the neuropathic patient. Third, we look at the effect of reconstruction of the deformed or unstable neuropathic foot. Fourth, we consider the significance of psychological factors, and finally, the importance of long term care.

The diabetic foot with neuropathy is very susceptible to traumatic damage, leading to a break in the skin, which then acts as a portal of entry of infection. Colleagues from Africa and India have frequently described devastating infections that rapidly destroy the neuropathic foot. Now that London is an international city we too now see patients with horrendous infections, including those who have travelled from Africa and India, and this type of rapidly destructive infection is described in this section. In temperate climes as well as tropical ones, there is increased susceptibility of the diabetic neuropathic foot to

Diabetic Foot Care: Case Studies in Clinical Management Alethea Foster and Michael Edmonds

infection, and we see very severe infections where the serum C-reactive protein (CRP) can be above 400 mg/l. Furthermore, when a neuropathic patient becomes systemically unwell then almost certainly the infection is very severe. We have also learnt that diabetic neuropathic patients are prone not only to foot infections but also to devastating infections elsewhere in the body.

Aggressive treatment of infection is important, starting with wide spectrum antibiotic therapy and then targeting therapy according to bacteria isolated. It is important to have a working knowledge of the principal bacteria and their local antibiotic sensitivities, including awareness of the prevalence of resistant organisms. However, in every patient, individual sensitivities of each organism isolated on culture should be sought to guide rational antibiotic therapy. There should be close co-operation between the microbiology laboratory and the diabetic foot service. Furthermore, antibiotic therapy should be accompanied by debridement of infective and necrotic tissue. When patients present with severe infection they need expert medical treatment including careful fluid replacement. Perioperative problems are common: in the early days of the Diabetic Foot Clinic we saw a patient who suffered respiratory arrest on codeine tablets and had to be temporarily ventilated.

We have also come to learn that diabetic neuropathic patients are frail vulnerable patients and we have also considered the impact that neuropathy and also co-morbidities can have on the diabetic patient. Peripheral neuropathy itself produces a major deficit of sensation in the lower limbs. It impairs proprioception and patients with neuropathy become very unsteady, and falls and associated traumatic lesions are common. Other complications, including postural hypotension and hypoglycaemic episodes without any warnings, make accidents even more likely. Neuropathy is a devastating deficit, and it is not just the feet and legs that are affected. The diabetic patient with a neuropathic ulcer will usually have evidence of nerve damage elsewhere. Autonomic neuropathy may affect the heart, gastrointestinal system and bladder. Damage to the nerve supply of the heart can lead to silent ischaemia and silent myocardial infarction. We have seen cases of sudden death in young, apparently robust neuropathic patients who had no peripheral vascular disease. There may be poor neurological control of ventilation leading to sleep apnoea and also susceptibility to pulmonary infections. In addition to neuropathy, co-morbidities may include poor vision through diabetic retinopathy and cataract.

Key principles to remember are that neuropathic patients may become destabilised by ulceration and sepsis and that neuropathic ulcers may present in various ways, often as medical emergencies with not only severe, rapidly progressing infections but also considerable metabolic upset.

Diabetic neuropathic patients who present with ulceration and infection often have deformity of the foot that has precipitated the initial ulceration. Such patients often need surgical debridement and in addition can benefit at the same time from surgical correction. If our early neuropathic patients seen in the 1980s and 90s developed severe deformity of the feet, the standard approach was to accommodate such deformity with appropriate footwear, but it is now possible to correct the deformity surgically as well as heal the ulcers.

In addition to the impact of other co-morbidities, psychological problems are also very important. Many diabetic foot patients, when they are first referred to us, are deeply

fearful, believing that they face inevitable amputation. The majority of patients rapidly gain confidence once they feel that they have found a safe haven where rapid and appropriate treatment will always be available. Patients build up relationships with the staff of the Diabetic Foot Clinic over many years, and even patients who do not always follow advice or do not always accept treatment are still cared for. However, some patients have concurrent psychological problems and remain deeply suspicious and often unwilling to accept care. Furthermore, there may be problems of self-delusion, when patients convince themselves that devastating foot infections are trivial.

We emphasize the need for long term management in a specialist Diabetic Foot Clinic for these patients.

Each diabetic neuropathic patient is unique, always beginning with the assumption that any person with diabetic neuropathy is a very vulnerable patient. It is important to have a thorough long term knowledge and understanding of the patient in order to apply effectively modern techniques for optimal diagnosis and treatment. Although we have always emphasized the need to make a clear distinction between the well perfused neuropathic foot and the neuroischaemic foot, it should never be forgotten that if a classical neuropathic patient lives long enough he is likely eventually to develop ischaemia. All the patients described in the neuroischaemic chapter will have started their diabetic foot lives as neuropathic patients. The diabetic foot is a moving target!

In this chapter we describe 36 cases of neuropathic foot disease. They fall into five main groups.

- Patients with differing presentations of infection and complications of infection.
- Patients with co-morbidities in addition to diabetes and neuropathy impacting on neuropathic foot presentations.
- Patients presenting with neuropathic foot deformity, ulceration and infection, when reconstruction to correct the deformity is carried out as well as surgical debridement for the infection.
- Patients with neuropathic feet in whom psychological factors have impacted on their management.
- Long term patients followed in the Diabetic Foot Clinic with neuropathic foot problems.

1.2 Differing presentations of infection and complications of infection

The first series of cases in this chapter are different presentations of infection because these are the most serious and dramatic problems in the diabetic neuropathic foot. Even a slightly raised body temperature can mark a severe infection with the capacity to deteriorate with alarming rapidity.

Case 1.1 A slightly raised body temperature can be a marker of severe infection, which can lead to collapse within hours

A 40 year old man with Type 2 diabetes for 6 years had proliferative retinopathy and peripheral and autonomic neuropathy. Vibration sensation was absent at both great toes. He sustained plantar blisters on both his feet after walking around the house barefooted on a nylon carpet after soaking in a long hot bath (Figure 1.1a). On the first occasion he was encouraged to rest and take time off work so that the blisters would heal quickly. He did not follow this advice and developed a large neuropathic ulcer on the left forefoot (Stage 3 foot), after which he agreed to rest as much as possible and the ulcer quickly began to heal (Figure 1.1b) and was completely healed in two months. He was reluctant to wear bespoke shoes but agreed to wear trainers. Two further episodes of blistering healed quickly.

However, five years later he developed another large blister on the right foot and attended the Foot Clinic. There was no obvious spreading infection but he had local cellulitis and a body temperature of 37.7 °C and it was decided to admit him (Stage 4 foot).

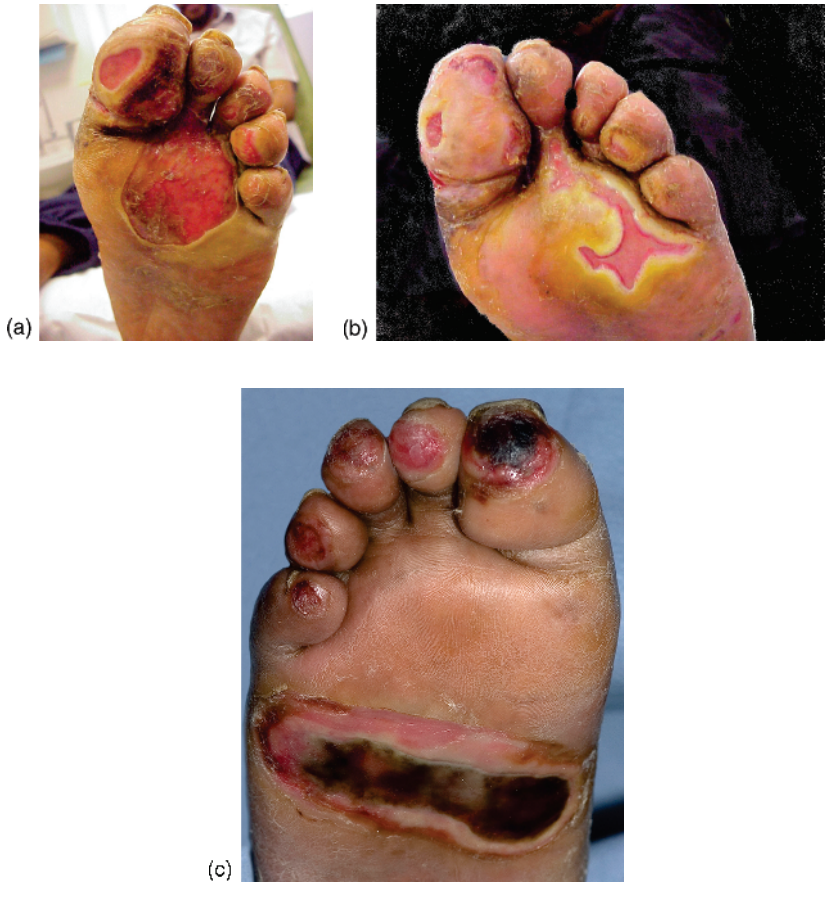


Figure 1.1 (a) Plantar blisters. (b) Healing foot. (c) The foot with necrosis.

He insisted on going home first because he wished to take his car home as it was parked in a hospital car park. He said that he would leave his car at home and ask his brother to bring him to the hospital. He was brought back by his brother later that afternoon. In two hours he had become extremely ill, with a body temperature of 40.2 °C. He was very drowsy and shivering and had a spreading cellulitis. Blood pressure was 90/60 mm Hg. He was admitted at once. He was immediately given quadruple IV antibiotics according to our usual regime of amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. He was started on IV insulin sliding scale. Blood cultures grew *Staphylococcus aureus* and Group G *Streptococcus* and his IV antibiotics were then rationalised to amoxicillin and flucloxacillin. He was also treated with clindamycin 450 mg qds IV. The CRP on admission was 166.5 mg/l, falling to 104.2 mg/l the next day, 41.8 mg/l after two days and 19.1 mg/l after four days. On admission the white blood count (WBC) was 12.77×10^9/l, with a neutrophilia of 10.66×10^9/l, falling after four days to a WBC of 4.64×10^9/l and neutrophils normal at 2.79×10^9/l. Glycated haemoglobin was 11.7%. The blister developed into an area of necrosis (Figure 1.1c) but this gradually improved. He was discharged after 10 days. After this episode he agreed to check his feet every day and come to the Diabetic Foot Clinic regularly and he has not relapsed.

Learning points

- Systemic symptoms and bacteraemia can develop rapidly from diabetic foot infections. It is very risky to allow any delays in admission, for example when patients wish to go home to collect possessions.
- A high fever, as in this case, is usually indicative of a very severe infection and is associated with a bacteraemia. Blood cultures should be carried out on all diabetic patients with foot infections and a fever, however mild this fever is.
- The clinician should beware of even mild fever as any fever is a sign of a serious infection. However, many diabetic patients do not develop fever even when they have a foot infection.
- Clindamycin was used because the patient had signs of toxicity with high fever and clindamycin is a powerful inhibitor of streptococcal toxin synthesis.
- When using clindamycin it is necessary to be aware of antibiotic-induced colitis, especially in elderly and postoperative patients.
- Serial measurements of falling serum CRP levels can confirm progress in resolving infection.
- *Staphylococcus aureus* is the commonest cause of infection in the diabetic foot and is often accompanied by a streptococcal infection of either Group B or Group G and rarely Group A. These organisms act in synergy and can lead to considerable tissue necrosis because hyaluronidase, produced by the *Streptococcus*, facilitates the spread of toxins produced by the *Staphylococcus*.

Case 1.2 Even if the pedal pulses are strong, necrosis develops when infection is severe

Another common presentation of infection in the neuropathic foot is of gangrene: the diabetic black toe. Previously, necrosis in a diabetic foot with palpable pedal pulses was deemed to be due to "small vessel disease", and the unfortunate patient often found himself on the receiving end of a major amputation. The diabetic black toe with bounding pulses a few centimetres away is now well known to be a complication of infection, where septic vasculitis damages the digital vessels, which become occluded by septic thrombus. As they are end arteries, the result is gangrene of the area supplied by the affected arteries, as in this case.

The patient, a 74 year old retired builder with Type 2 diabetes of five years duration, presented as an emergency at Casualty with infection and necrosis of the second and third toes of the left foot (Stage 5 foot) (Figure 1.2a), and was referred to the Diabetic Foot Clinic. He had strong pedal pulses but absent vibration sense at the big toes. His X-ray was normal despite the full-thickness necrosis. He was admitted and treated with quadruple IV antibiotics, amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. His CRP was 155.2 mg/l and his WBC was 16.85×10^9/l, with neutrophils of 14.41×10^9/l. He had a double ray amputation within 24 hours of admission (Figure 1.2b). He grew *Staphylococcus aureus* from the wound swab and *Proteus mirabilis* and Group G *Streptococcus* from tissue samples taken in theatre. His antibiotics were focussed to amoxicillin, flucloxacillin and ceftazidime.

His recovery from the ray amputation was complicated by his development of frank painful haematuria. He had a past history of renal stones and had had a bladder stone removed in an open operation. His haemoglobin dropped from 11.3 g/dl to 8.2 g/dl and he underwent a blood transfusion. CT scan and ultrasound revealed a 5 mm calculus at the lower pole of the left kidney. MSU showed a heavy mixed growth and the patient was treated with ciprofloxacin 500 mg bd. His haematuria resolved and he was reviewed by the

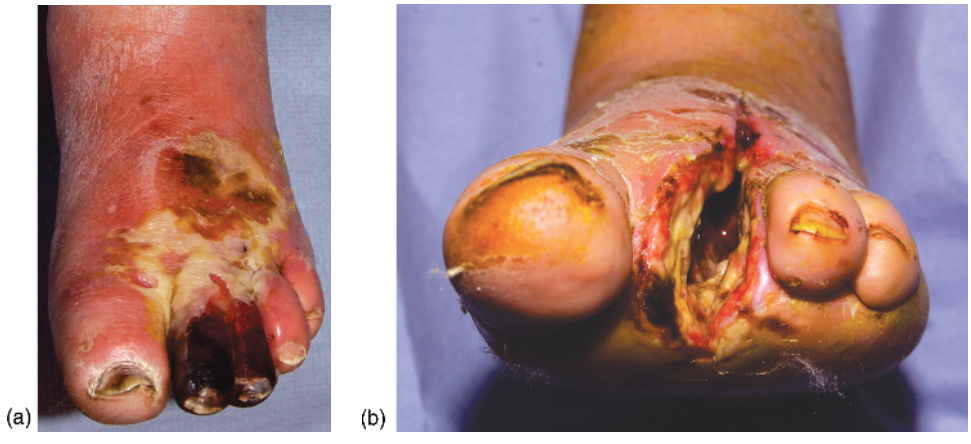


Figure 1.2 (a) Necrosis of the toes. (b) Double ray amputation.

urologists, who felt that no further treatment was necessary. His Type 2 diabetes was usually treated with oral hypoglycaemics but on admission, because of hyperglycaemia (20.5 mmol/l), he was treated with IV insulin sliding scale, which after two days was changed to a basal bolus insulin regime. Later on in the admission his foot wound grew *Enterococcus faecium* sensitive to vancomycin and teicoplanin. A PICC line was inserted into an antecubital vein, and he was discharged home on teicoplanin 400 mg daily IV and ciprofloxacin 500 mg bd orally. His foot healed after six weeks and the orthotists made him two pairs of shoes with cradled insoles. After healing of the foot, his diabetic therapy returned to metformin and glargine insulin at bedtime. He attended the Diabetic Foot Clinic regularly for nail care and removal of callus and his foot did not relapse.

Learning points

- Diabetic patients with Type 2 diabetes often have evidence of complications at the time of diagnosis because of glucose intolerance that may have been present for some years. Thus it is not surprising for this patient, who has a known history of diabetes for five years, and probably had glucose intolerance for some years before that, to have profound neuropathy.
- This patient had a typical polymicrobial infection of the diabetic foot, with *Staphylococcus aureus*, Group G *Streptococcus and Proteus mirabilis* isolated. The latter two organisms were isolated from operative tissue samples, thus illustrating the critical importance of sending operative tissue for culture. All three organisms in this polymicrobial culture were important and each needed appropriate antibiotic therapy.
- Many patients with Type 2 diabetes and foot infections will need insulin during the period of foot sepsis and subsequently during wound healing.
- Patients with Type 2 diabetes often have multi-system disease. Recovery from diabetic foot infections can be complicated by other illnesses, which need prompt attention or else they may compromise healing of the foot.
- Diabetic patients with urinary tract calculi are particularly prone to complications, especially infection.

Case 1.3 Neuropathic ulceration and infection lead to severe metabolic disturbance

When infection is not caught early and controlled well, then patients can become very ill. This poor lady went to see her general practitioner as soon as she detected an ulcer, but even so she developed severe foot problems.

The lady was 64 years of age and had had Type 1 diabetes for 25 years. She developed a small ulcer on the right first toe. It was her first foot ulcer. She visited her general practitioner, who prescribed flucloxacillin 500 mg qds. She had atrial fibrillation and was taking warfarin and digoxin. The next morning she felt unwell and was short of breath, and vomited on two occasions. Her family took her to Casualty. She had interdigital sepsis between the first and second toes with spreading cellulitis but good foot pulses. She had painless full thickness necrosis on the lateral aspect of the first toe with wet gangrene. She also had wet necrosis of the second toe, with infection extending up the tendon sheath, and wet gangrene of the medial aspect of the third toe. The plantar fat pads were necrotic in the base of the second and third toes (Stage 5 foot) (Figure 1.3a–d). She was in fast atrial fibrillation. Blood pressure was 60/30 mm Hg., CRP was 474 mg/l, WBC 23.82 $\times$ 10^9/l, with neutrophils 21.81 $\times$ 10^9/l, glucose 50.6 mmol/l, creatinine 152 µmol/l, sodium 129 mmol/l. She had evidence of ketoacidosis. The pH of arterial blood was 7.05 (normal values 7.35–7.45) and urine showed +++ ketones. Her INR was 7.22 (when anticoagulated we

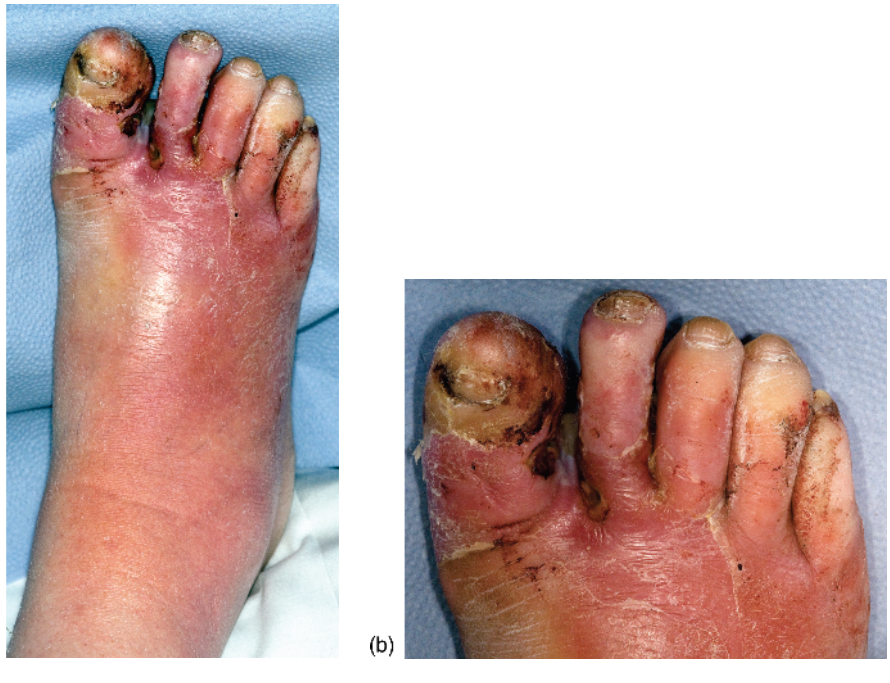


Figure 1.3 (a) Panoramic view of foot and lower leg. (b) Close-up of foot shows dorsal cellulitis.

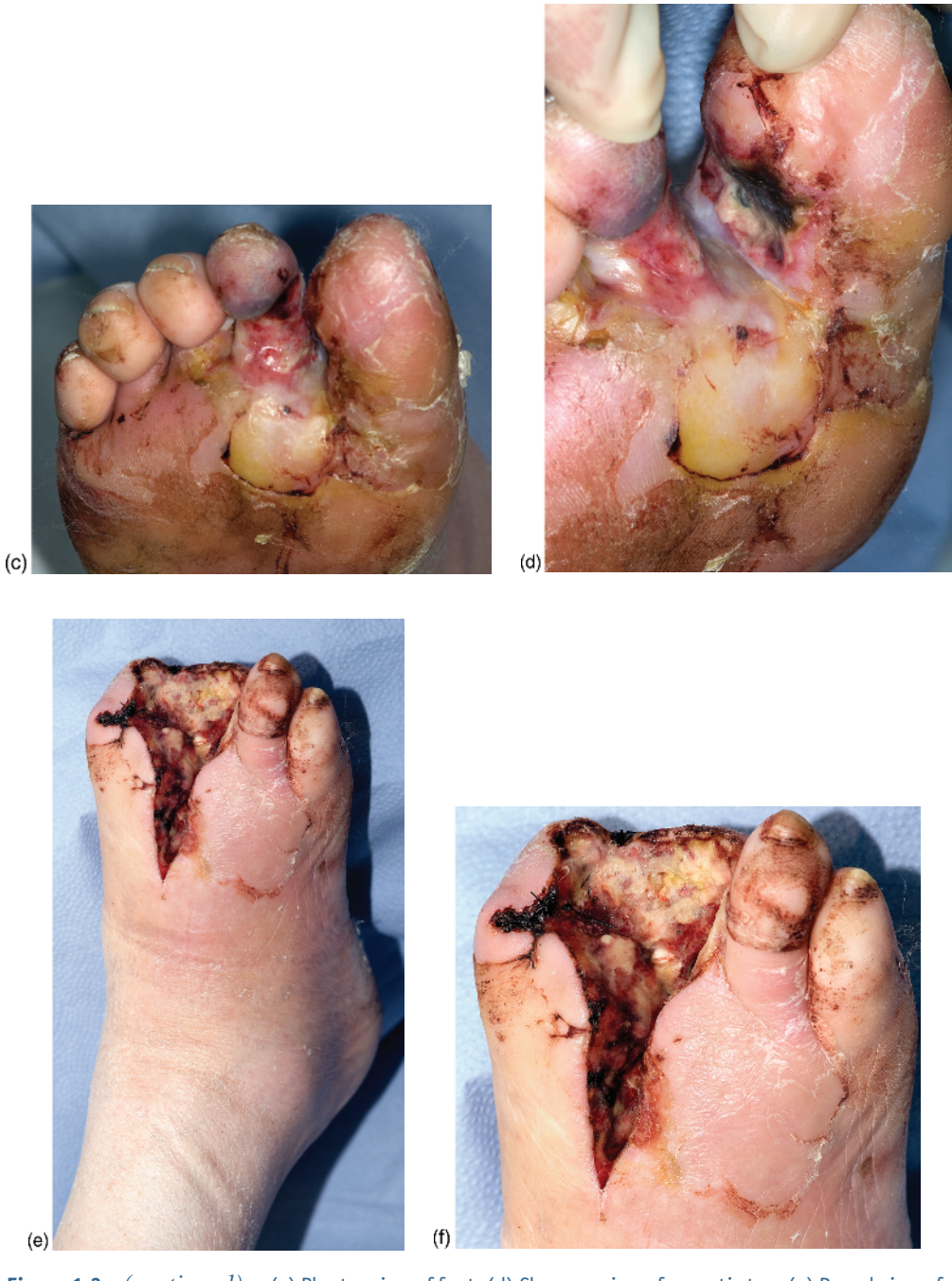


Figure 1.3 *(continued)* (c) Plantar view of foot. (d) Close-up view of necrotic toe. (e) Dorsal view of foot after surgery. (f) Close-up view of healing wound.

aim for INR around 3). A diagnosis was made of severe foot sepsis, diabetic ketoacidosis, cellulitis, fast atrial fibrillation and a deranged clotting mechanism secondary to her warfarin therapy and complicating sepsis.

She needed resuscitation in the Intensive Care Unit and was given amiodarone to control atrial fibrillation, IV insulin sliding scale and vitamin K and fresh frozen plasma. She was treated with IV amoxicillin 1 g tds, flucloxacillin 1 g qds, metronidazole 500 mg tds and ceftazidime 1 g tds. She underwent amputation of the right first toe, right second toe, and right third toe at the metatarsophalangeal joint (Figure 1.3e, f). The tissue cultures grew *Staphylococcus aureus* and Group B *Streptococcus* and antibiotics were narrowed down to amoxicillin and flucloxacillin. After the initial medical stabilisation followed by surgical debridement, she then had vacuum assisted wound closure (VAC) therapy. Warfarin had been restarted and ten days later the patient had bleeding from the dorsal part of the wound. The leg was elevated and a Kaltostat dressing applied. Her blood pressure was 95/60 mm Hg. Her INR was 2.8. She was transfused two pints of blood. She had no further untoward events, and was discharged with regular follow-up in the Diabetic Foot Clinic and the foot subsequently healed.

Learning points

- Diabetic patients with severe co-morbidities can destabilise and become very unwell in the presence of ulceration and infection. This lady's foot did not look severely infected at first presentation to the general practitioner. Over the subsequent 24 hours she developed necrosis and became unwell and on presentation to the hospital she was critically ill.
- The foot infection destabilised the patient's anticoagulant therapy and she presented with an INR of 7.22. It also destabilised her diabetes and she presented with a life-threatening ketoacidosis.
- Patients with severe diabetic foot infections and associated metabolic instability should be resuscitated and stabilised as quickly as possible and then taken to the operating theatre for surgical debridement. However, such resuscitation should be quickly achieved, as the patient with necrosis and spreading infection needs urgent surgical debridement. It may be said that a patient is too ill to go to theatre but in the presence of spreading infection and necrosis it might also be said that the patient is too ill *not* to go to theatre.
- VAC therapy is very useful in accelerating the healing of post-operative wounds in neuropathic feet.
- During her recovery the episode of bleeding was possibly associated with her anticoagulant therapy but the INR was in therapeutic range. Bleeding could have been exacerbated by VAC therapy. However, anticoagulation is a caution not a contraindication during VAC therapy.
- There was synergy between *Staphylococcus aureus* and Group B *Streptococcus*, which is an important pathogen to which diabetic patients are particularly susceptible. Both bacteria produce toxins.

Case 1.4 Cutting the mushrooms

A recent report from India demonstrated that a high proportion of diabetic foot injuries are sustained within the home, and this case fits into this category.

A 60 year old lady had Type 2 diabetes diagnosed 9 years previously. She had peripheral neuropathy and hypertension, and was taking metformin 850 mg bd. She sustained an injury when a dirty knife fell on the foot and pierced the dorsum of the foot. The injury occurred when she was in the kitchen, and the knife was a kitchen knife with which she had been chopping mushrooms. She presented at the Diabetic Foot Clinic, previously unknown to us, one day later, complaining of pain and swelling, and she had difficulty in weight bearing. Her temperature was 36.6 °C and her blood glucose was 18.4 mmol/l. Her pedal pulses were palpable. There was a small laceration between the first and second rays on the dorsum (Figure 1.4a) surrounded by swelling (Figure 1.4b). There was bruising to the base of the second and third toes and tenderness on palpation (Stage 3 foot). X-ray was normal. Ultrasound showed induration of the subcutaneous fat at the dorsum of the first webspace and plantar to the big toe. There was no hypervascularity and no collection, and flexor and extensor tendons were intact. It was presumed that the knife wound had tracked through the web space to the plantar side. The patient was admitted and was treated with IV antibiotics, amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds because of the concern of possible deep infection. Her tetanus prophylaxis was not up to date so this was given. An ulcer swab from the laceration was negative. Her foot was reviewed twice a day, and for the first 24 hours she was put on IV insulin sliding scale. Diabetic control was subsequently obtained by changing the patient to a basal bolus regime of insulin. She made a full recovery, after which she went back to oral hypoglycaemic agents and was followed up in the Diabetic Foot Clinic.

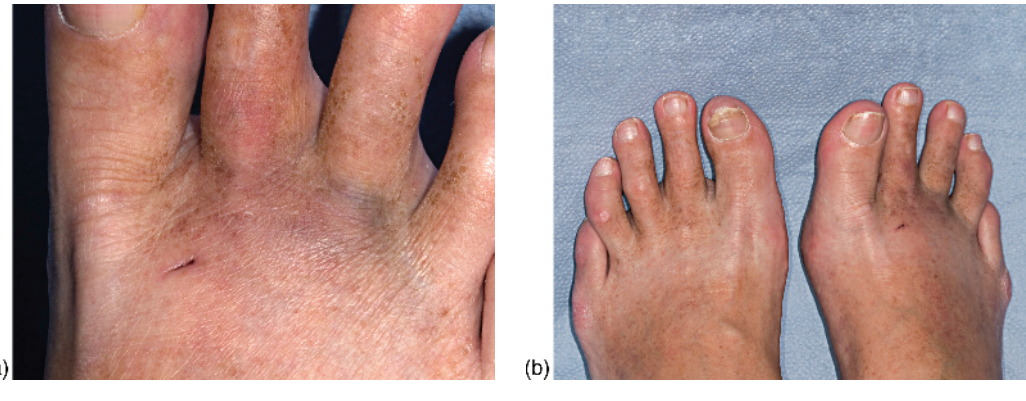


Figure 1.4 (a) Laceration from knife wound. (b) Swelling noted on lacerated foot compared with opposite foot.

Learning points

- Practitioners need to be careful about puncture wounds. Some of the worst cases of diabetic foot infections which we have seen began with a small and apparently innocuous wound.
- Practitioners should not be deceived by the seemingly trivial sign of a small puncture injury. Infection often is present at the base of the wound but then gradually takes hold.
- Speed of development of infection can be alarmingly rapid. Signs of infection after a penetrating injury only become apparent when the infective process has spread from the base of the puncture wound to the surrounding skin.
- Early presentation enabled this foot to be treated quickly and the risk of complicating infection to be averted.
- Pain is ominous in the neuropathic foot and should never be ignored.
- Puncture wounds often need admission to hospital as sepsis may be already apparent. Even when they are treated in the Diabetic Foot Clinic and not admitted, these patients should be reviewed every two to three days, despite the benign appearance of their foot.
- Many patients with severe infection will be apyrexial. Absence of fever – and even a normal white blood cell count – does not indicate that all is well and there is no infection.
- This lack of signs can make the patient even more difficult to manage, since a valuable sign of improvement is a decreasing temperature in a feverish patient.

Case 1.5 Infection leading to deformity

Although deformity usually leads to ulceration and then infection, occasionally infection can lead to tissue damage and then deformity.

This patient was 56 years old and had Type 2 diabetes for 5 years. She was obese and had a history of pulmonary emboli and was on warfarin therapy. She developed infection over the lateral aspect of the left foot with an ulcer probing to bone over the base of the fifth metatarsal (Figure 1.5a) (Stage 4 foot). She was admitted to hospital. She had a CRP of 54.6 mg/l and a WBC of 12.16×10^9/l with neutrophils of 9.24×10^9/l and was treated initially with amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. Deep wound swabs grew *Escherichia coli*, *Pseudomonas aeruginosa* and *Serratia marcescens*, all sensitive to piperacillin/tazobactam, to which she was changed, and her cellulitis resolved. However, she gradually developed an inversion deformity of the foot (Figure 1.5b) and was readmitted with recurrent sepsis. A swab grew *Enterobacter cloacae*, which was again treated with piperacillin/tazobactam. Her deformity has been accommodated in a total contact cast and her ulcer is healing. Surgery to correct the inversion deformity is under consideration, although the patient's past history of pulmonary emboli and obesity render her very high risk. She also has lymphoedema following her recurrent infection, and associated problems with healing an oedematous

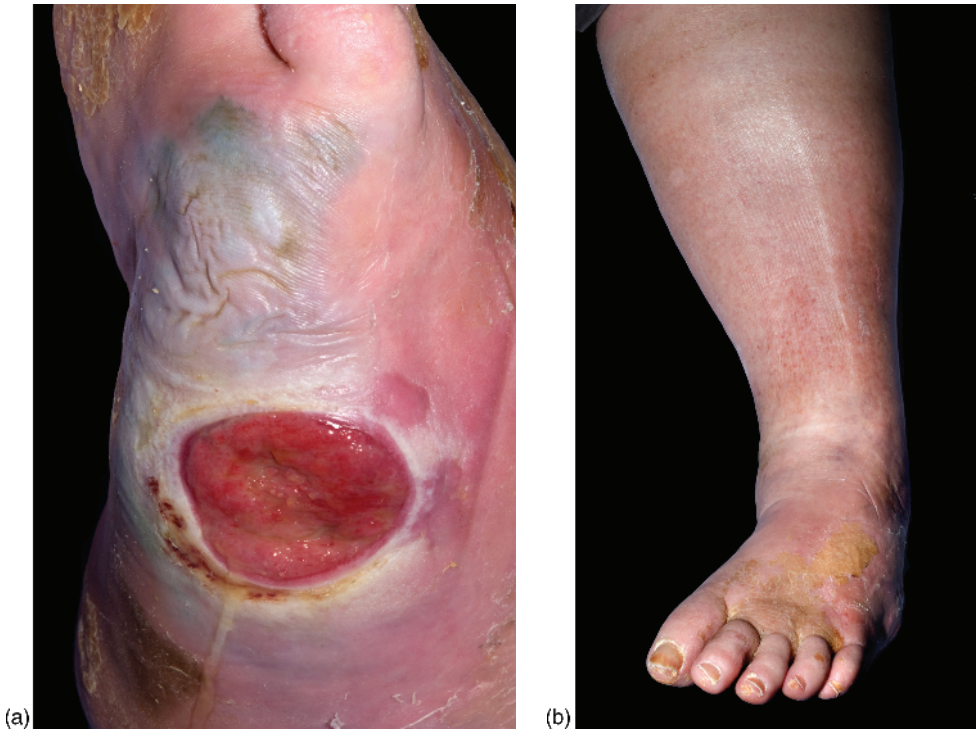


Figure 1.5 (a) Infected ulcer. (b) Inversion deformity.

limb are further reasons to avoid surgery. She has subsequently undergone successful reconstructive surgery of the foot. (Note added in proof)

Learning points

- Patients who have infections of the lateral foot often incur direct damage to the insertion of the peroneus brevis tendon to the lateral aspect of the base of the fifth metatarsal. Peroneus brevis is responsible for plantar flexion and eversion of the foot at the ankle. Damage to the insertion of the peroneus muscle leads to an inversion deformity.
- Ulceration and infection over the lateral aspect of the foot should be treated aggressively to prevent damage to the insertion of peroneus brevis.
- When the foot is debrided surgically for infection over the lateral aspect of the foot, care should be taken to preserve peroneus brevis function.
- Once this inversion deformity has developed the patient's foot should be accommodated in appropriate supportive footwear.
- This patient's initial foot cultures grew *Escherichia coli*, *Pseudomonas aeruginosa* and *Serratia marcescens*. These gram negative organisms are often regarded as insignificant in the diabetic foot. However, we believe that this is not correct. These bacteria can be definitely pathogenic in the diabetic foot, especially when they are in a pure growth or as part of a polymicrobial deep infection.
- Gram negative organisms should receive appropriate antibiotic therapy. Oral agents that are available to treat gram negatives are ciprofloxacin and trimethoprim. Parenteral agents include ceftazidime, aminoglycosides, meropenem, piperacillin/tazobactam, ticarcillin/clavulanate, tigecycline and ertapenem. It is crucial to obtain sensitivity patterns with gram negative organisms and not depend on empirical therapy alone.

Case 1.6 Neuropathic ulceration, sepsis, cardiac failure and more

This case was a lady with breast cancer, which was diagnosed during her admission for foot infection, and she also developed cardiac failure. Achieving healing of her foot was an urgent consideration so that she could commence chemotherapy for the cancer.

This 55 year old lady with Type 2 diabetes for 6 years presented with a necrotic ulcer of the right first toe, which was gangrenous at the base (Stage 5 foot). She also had a left-sided 10 cm labial abscess. She was admitted and treated with antibiotics comprising IV metronidazole 500 mg tds and vancomycin 1 g statim and then dosage as per serum levels and oral ciprofloxacin 500 mg bd. She was allergic to penicillin. A Group B streptococcus was grown from the labial abscess and the right toe necrotic ulcer. She underwent minor amputation of the first toe (Figure 1.6). However, post-operatively she had a pneumonia, which was treated with IV clarithromicin 500 mg bd. She was also put on insulin on a basal bolus regime. The patient also developed heart failure with pulmonary oedema, pulmonary hypertension and pleural effusions. An echocardiogram showed the left ventricle to be moderately dilated with normal left ventricular wall thickness, but left ventricular systolic function was reduced. She was treated with frusemide 80 mg orally, ramipril 10 mg orally, bisoprolol 1.25 mg orally and bendroflumethiazide 2.5 mg orally. She was also noted to have a lump in her breast with palpable axillary nodes. CT staging confirmed clinically locally advanced left breast cancer with involved axillary nodes. Every effort was made to control infection and heal the post-operative wound so that the patient could start chemotherapy.

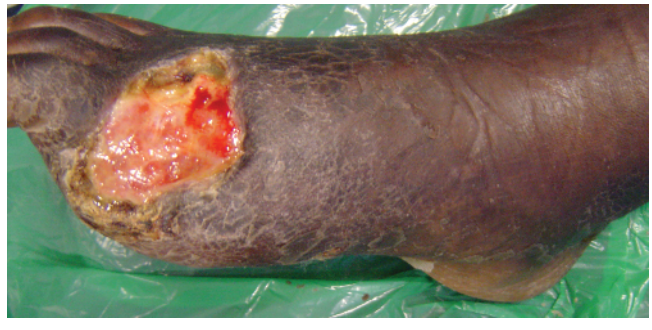


Figure 1.6 Postoperative healing wound.

Learning points

- This patient had Group B streptococcus from the labial abscess and the foot. This is increasingly recognised as an important pathogen in the diabetic patient.
- Diabetic patients with ulcers and infection may develop complications such as impaired cardiac function related to the infection. These need appropriate and accurate treatment to facilitate healing of the ulcer.
- This patient had significant complications, which impacted on the healing of the post-operative wound, particularly congestive cardiac failure. This needed urgent treatment to relieve the oedema and promote healing.
- It was also important to achieve healing of her post-operative wound as quickly as possible and control infection, so that the patient could start chemotherapy for her breast cancer.

Case 1.7 Infection in the neuropathic foot and Charcot foot

A useful technique for healing ulcers that have been surgically debrided following infection is vacuum assisted wound closure (VAC), which was used in the following case.

This was a 46 year old patient with Type 1 diabetes for 22 years who had peripheral neuropathy, autonomic neuropathy and proliferative retinopathy. He developed cellulitis over the dorsum of the right foot complicated by necrosis (Stage 5 foot). At his initial visit, when he presented with cellulitis, his foot swab grew Group B *Streptococcus* and he had a WBC of 14.87×10^9/l and a CRP of 157.6 mg/l, falling to 116.2 mg/l two days later. He was treated initially with IV amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds, and underwent surgical debridement of the necrotic area (Figure 1.7a). Post-operatively this was treated with VAC therapy (Figure 1.7b). He was discharged on oral amoxicillin 500 mg tds, flucloxacillin 500 mg qds and metronidazole 400 mg tds. He was readmitted electively for a split skin graft to the previously debrided wound (Figure 1.7c).

However, after discharge he developed swelling of the right foot and an area of necrosis on the lateral right foot at the distal end of the previous skin graft. He underwent further surgical debridement of the right foot and further VAC pump therapy. Four weeks later it was noted at a Foot Clinic appointment that his right foot was swollen. Discharge from the wound had not increased, and there was no obvious cellulitis or fever. His WBC was normal. An X-ray (Figure 1.7d) showed lucency in the navicular and an MRI showed bony oedema of the tarsal bones in keeping with Charcot foot and a fracture of the navicular (Figure 1.7e). He was treated with an Aircast and the Charcot foot became non-active six months later and the wound healed.

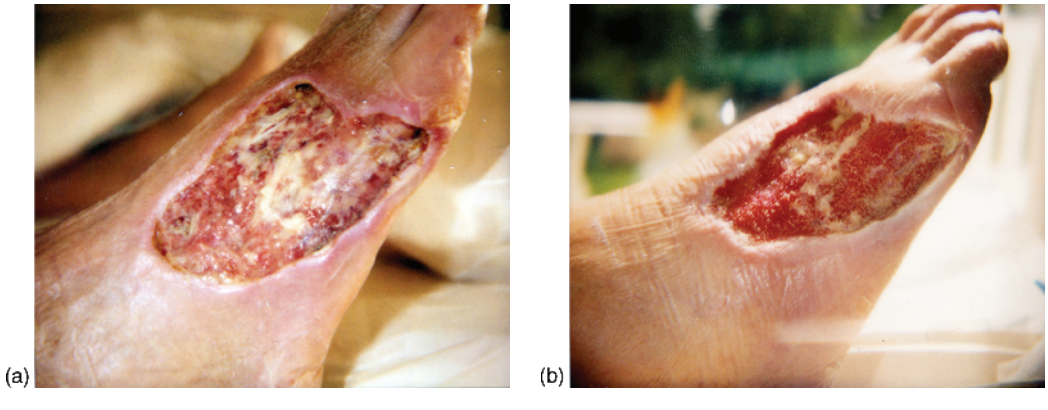


Figure 1.7 (a) Dorsal view of foot showing wound after debridement. (b) Dorsal wound after VAC therapy.

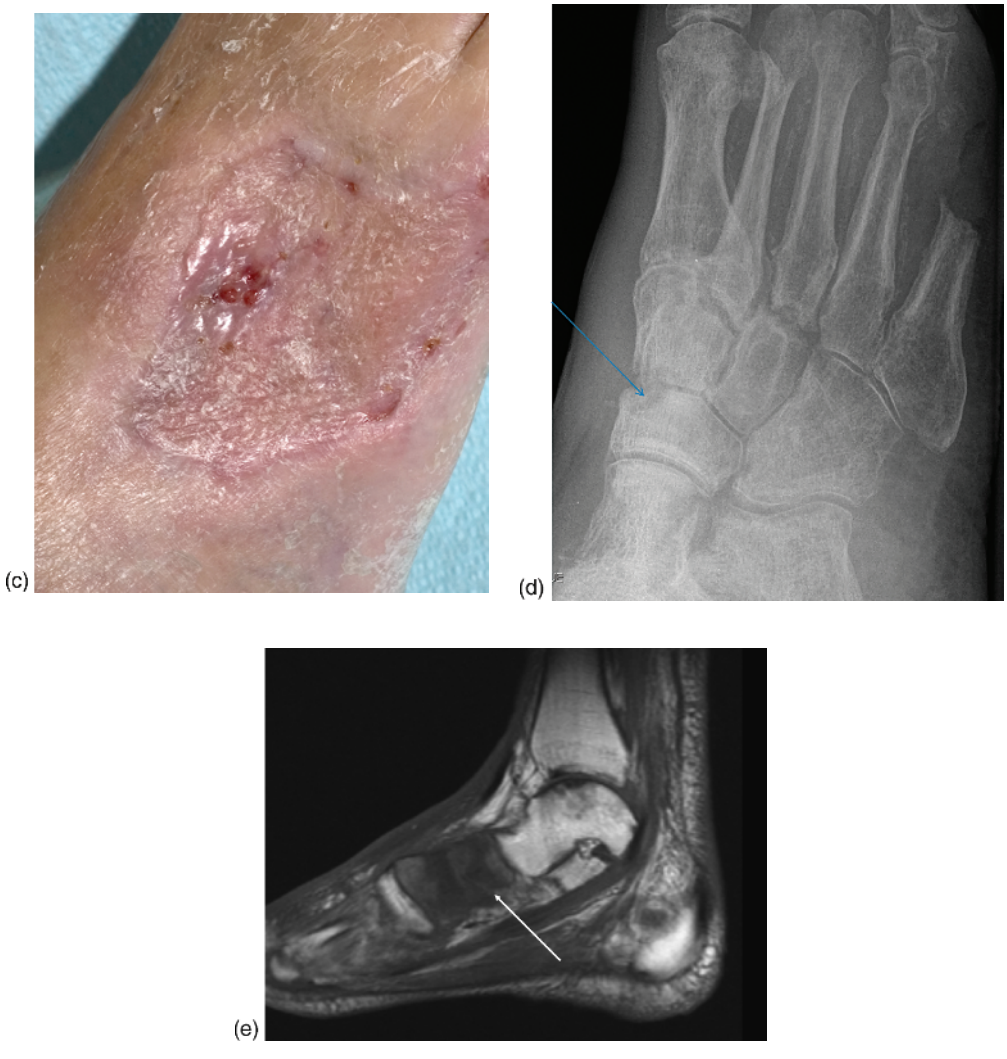


Figure 1.7 (*continued*) (c) Skin graft. (d) X-ray shows lucency of navicular (arrow). (e) MRI (T1, sequence) showing oedema of tarsal bones and fracture of navicular (arrow).

Learning points

- When the patient returned with a swelling of the right foot, it was difficult to be sure clinically whether this was a relapse of infection or the onset of an acute Charcot foot. There was no raised WBC or fever but these are unreliable indicators of infection. However, imaging with X-ray and MRI demonstrated typical Charcot changes.
- When imaging with X-ray and MRI does not distinguish between osteomyelitis and Charcot, it is best to treat for both conditions.

- The trauma of undergoing surgical debridement may have precipitated his Charcot foot.
- He was treated in an Aircast instead of a total contact cast in view of his history of developing necrosis, because the Aircast could be removed for daily wound inspection.
- We often use the VAC therapy on large neuropathic wounds. This can speed up the formation of a granulating bed suitable for application of a skin graft, and we also use the VAC to speed healing of skin grafts. However, we wait for 24 hours after surgery before applying the VAC, and apply it with caution to patients who are on anticoagulants.

Case 1.8 Group A streptococcal infection

Group A streptococcal infection is rare in the diabetic foot but can cause extensive tissue damage.

This 74 year old patient with Type 2 diabetes for 15 years was bitten in Ghana by an insect on the left anterior leg. The leg became swollen, painful and cellulitic, and then developed multiple bullae, which were haemorrhagic, leading to areas of necrosis on the skin (Stage 5 foot) (Figure 1.8a, b). He took a plane back to the UK and came straight to King's Casualty. He was admitted. His CRP was 386.7 mg/l and creatinine 146 µmol/l with eGFR of 44 ml/min and his WBC was 14.5×10^9/l with neutrophils at 12.46×10^9/l. He had a fever of 38.7 °C. Malarial screen was negative. A deep wound swab grew Group A *Streptococcus*.

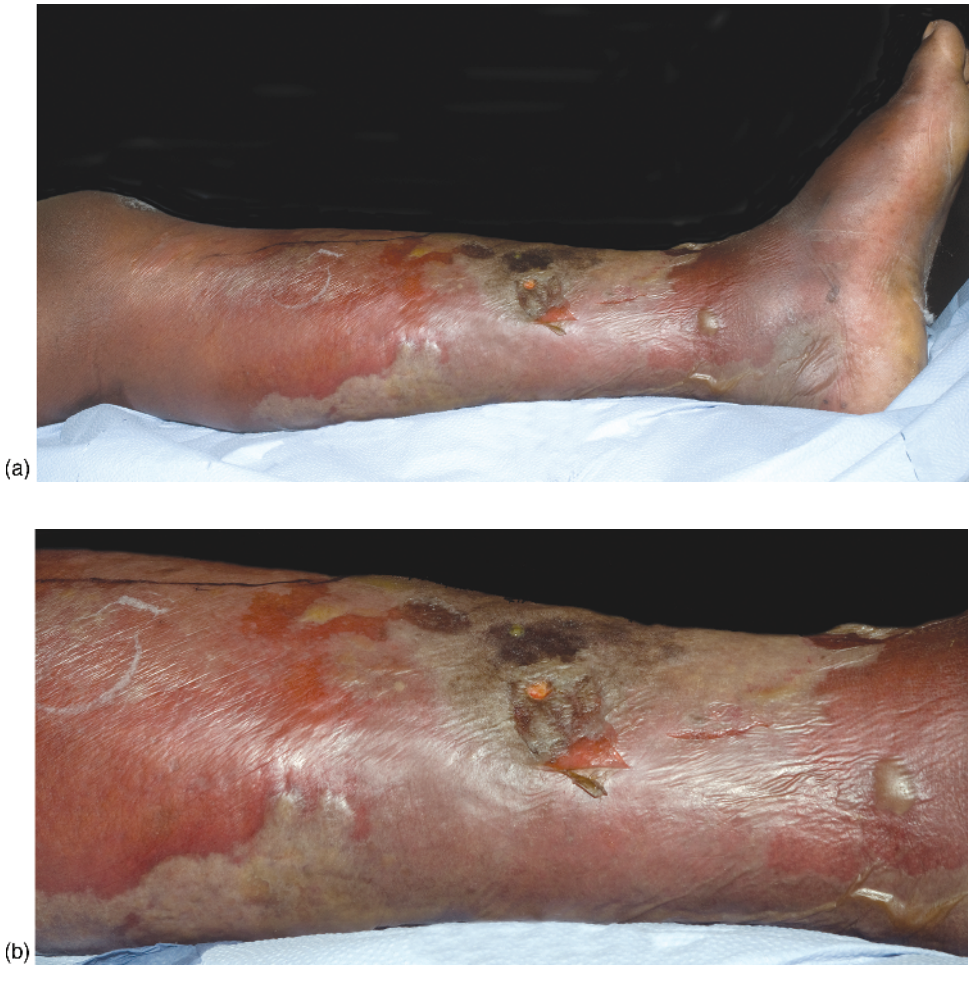


Figure 1.8 (a) View of the medial leg showing erythema, bullae and necrosis. (b) Close-up view of leg.

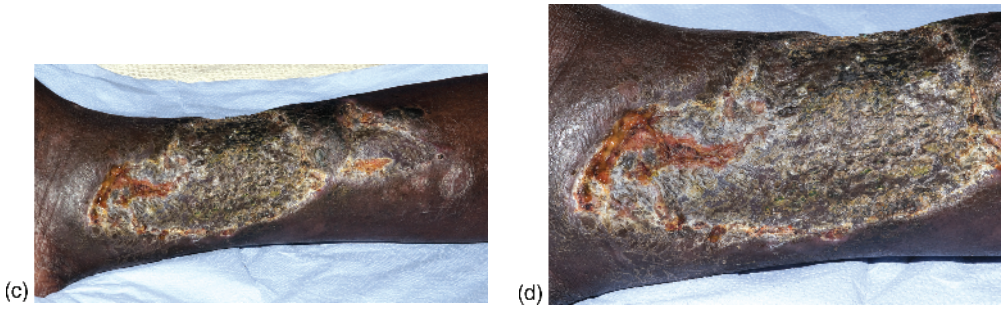


Figure 1.8 *(continued)* (c) Debrided lateral leg with skin graft applied. (d) Close-up of skin graft.

The patient was initially treated with IV piperacillin/tazobactam 4.5 g tds and vancomycin 1 g statim and then dosage as per serum levels and then high dose IV benzylpenicillin 2.4 g qds when microbiology results were available. He was given sliding scale IV insulin. Areas of further necrosis developed and he underwent operative debridement and fasciotomy. His CRP dropped to 297 mg/l but returned to 395 mg/l with a WBC of 19.82×10^9/l with 17.35×10^9/l neutrophils. He needed two further debridements to achieve complete eradication of Group A *Streptococcus*, and a skin graft to achieve healing (Figure 1.8c, d). Over the subsequent six weeks his CRP fell to within normal range and his creatinine fell to 77 µmol/l.

Learning points

- Group A *Streptococcus* can cause severe infection in diabetic patients as well as non-diabetic patients. It is recognised by the widespread cellulitis, bulla formation, which is often haemorrhagic, and extensive necrosis involving skin, muscle and fascia.
- Aggressive IV antibiotic therapy and prompt surgical debridement are crucial to the successful management of these infections.
- The antibiotic of choice is penicillin, to which Group A *Streptococcus* is particularly susceptible and has not been found to be resistant.
- This patient underwent fasciotomy because of the excessive swelling associated with deep infection. Subsequently he underwent surgical debridement on two occasions to remove all necrotic tissue.
- In the Afro-Caribbean leg it can be difficult to assess the extent of necrosis.

Case 1.9 A gas leak

Many neuropathic patients present late with extensive tissue destruction and polymicrobial infection.

A 45 year old chef with poorly controlled Type 2 diabetes of 9 years' duration, retinopathy, nephropathy and autonomic and sensory neuropathy walked barefoot at home and trod on a tin tack. When he saw blood on the floor he realised that there was a problem and pulled the tack out with a pair of pliers and applied a sticking plaster. Two days later the puncture wound appeared to have healed. Five days later he developed a large blister on the dorsum of his foot and applied papaya ointment, a traditional remedy for wounds in the West Indies. The foot did not improve and gradually became very swollen so he attended Casualty, ten days after the original injury. The Diabetic Foot Clinic team was called down to Casualty to see him. He had a fever of 39.0 °C with rigors. The dorsalis pedis pulse could not be palpated, but the posterior tibial pulse was bounding. The foot was swollen with blistering and dark blebs on the dorsum, and when palpated there was crepitus and pus oozed from deep within the foot (Stage 5 foot) (Figure 1.9a). When a scalpel was inserted into the dorsum of the foot in a bedside procedure there was an audible hiss of escaping gas. The foot smelled terrible and the odour literally made the eyes sting.

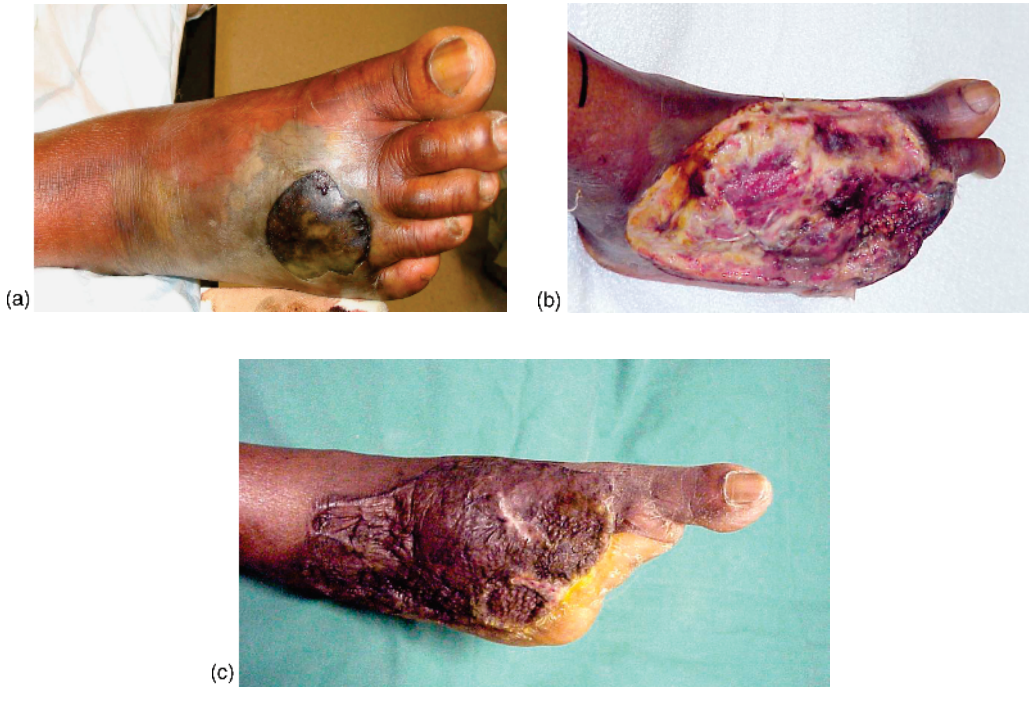


Figure 1.9 (a) Severe infection with bullae on dorsum of foot. (b) Post debridement. (c) Healed foot.

X-ray showed extensive gas within the soft tissue. Blood glucose was 27 mmol/l, CRP 279.6 mg/l and WBC 19.17 $\times$ 10^9/l with 16.30 $\times$ 10^9/l neutrophils.

Quadruple therapy with IV antibiotics of amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds was commenced. He underwent an extensive debridement and amputation of third, fourth and fifth toes (Figure 1.9b) on the same day, and all debrided material was sent to the laboratory for microscopy and culture. He grew *Citrobacter koseri* sensitive to gentamicin, ciprofloxacin and ceftazidime, mixed anaerobes sensitive to metronidazole and MRSA sensitive to vancomycin from the debridings. In view of the culture of MRSA, the amoxicillin and flucloxacillin were stopped and vancomycin 1 g statim IV and then dosage according to serum levels was substituted and ciprofloxacin 500 mg bd orally added. He underwent further surgical debridement including amputation of the second toe. The wound was inspected twice daily. There was no further collection of gas, and the CRP fell steadily and his temperature remained down. After three weeks, during which time the wound was irrigated with Milton twice daily, a healthy bed of granulation tissue had been achieved and a split skin graft was applied, and the wound healed in one month (Figure 1.9c). He was followed up in the Diabetic Foot Clinic. He was very reluctant to wear bespoke shoes ("cripple boots", as he called them) but agreed to wear a pair of boots with a design based on trainer-style sports shoes, and he was given two pairs. He also needed protective footwear for his kitchen work and this was manufactured according to a sample of protective footwear from work that he brought to the clinic.

Learning points

- Puncture wounds may appear to be small and insignificant but they can inject micro-organisms deep into the foot and cause devastating infections.
- Patients should be advised not to use traditional remedies but to seek help from an experienced Diabetic Foot Clinic as early as possible.
- *Citrobacter* is a genus of gram negative coliform bacteria in the Enterobacteriaceae family, which can be found in the gut. It can be responsible for soft tissue infections in diabetic feet.

Case 1.10 Trouble on holiday: severe infection in a neuropathic foot

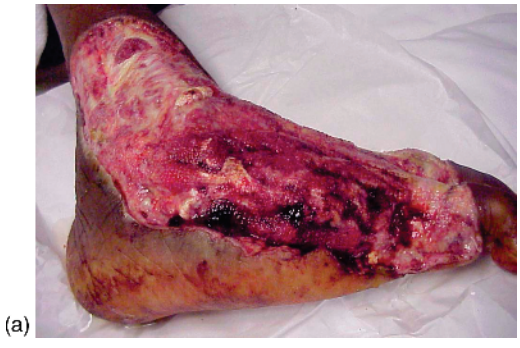
In Africa, extensive tissue loss is a feature of the diabetic foot. With the ease of modern travel, patients developing foot infections in Africa can readily present in other parts of the world.

A 62 year old African man, with Type 2 diabetes of 14 years' duration, presented to Casualty with a very swollen right foot with marked bulla formation and cellulitis (Stage 4 foot). He had noted swelling of the foot for a week and this had started whilst he was in Nigeria. His foot pulses were palpable. He had a pyrexia of 38.4 °C and a high WBC of 17.0×10^9/l, and his CRP was 200 mg/l. His blood glucose was 25.0 mmol/l. He was admitted to hospital, given IV amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds and also given IV insulin sliding scale. He was taken to the operating theatre the same day and underwent radical surgical debridement to remove all of the infected tissue. He had necrotising fasciitis. Three toes and their adjoining rays were amputated and the remaining first and second toes were denuded of proximal dorsal skin as was most of the dorsum of the foot and the dorsal wound extended above the ankle (Figure 1.10a). However, the plantar skin was intact (Figure 1.10b). A specimen of this tissue grew *Proteus mirabilis* and *Escherichia coli*.

He was initially treated with quadruple antibiotic therapy, which was then focused to amoxicillin and ceftazidime, and he made a complete recovery. He was left with a considerable loss of tissue cover on the dorsum. A VAC pump was used to stimulate formation of a bed of granulation tissue (Figure 1.10c) after which it was successfully skin grafted (Figure 1.10d). Bespoke footwear was provided to prevent overloading of the remaining plantar surface. This area had good fibro-fatty padding and no plantar scarring.

He had an admission two years later for multiple abscesses and severe infection of the right upper arm, right forearm and right chest. X-ray showed subcutaneous gas and soft tissue swelling. MRI showed a large collection in the right arm within the anterior and posterior subcutaneous fat and extending through the biceps muscle belly. The collection also extended to the anterior chest wall deep to the pectoralis muscle and to the right lateral chest wall deep to and within the serratus muscles and around towards the back and extending across the axilla. There were pockets of gas. Two blood culture bottles grew a Group B streptococcus. CRP was 429.0 mg/l with WBC 29.32×10^9/l, and neutrophils 26.60×10^9/l. He had surgical debridement. Six months later he was admitted with a right sided lateral plantar ulcer and cellulitis, after presenting late with advanced infection. His CRP was 236.7 mg/l, WBC 14.48×10^9/l, neutrophils 11.42×10^9/l. He grew *Enterobacter cloacae* and was treated with amikacin 7.5 mg/kg/bd initially and then as per serum levels and meripenem 1 g tds IV.

The following year, he was admitted with a further foot infection and CRP of 236 mg/l, and a foot swab grew *Staphylococcus aureus*, Group B *Streptococcus* and *Enterobacter cloacae*, and blood cultures also grew Group B *Streptococcus*. He was again treated with amikacin 7.5mg/kg/bd initially and then as per serum levels and meripenem 1 g tds IV. He occasionally attends the Foot Clinic but frequently misses appointments, and despite his propensity to develop severe infections his approach to his feet is one of *belle indifference*.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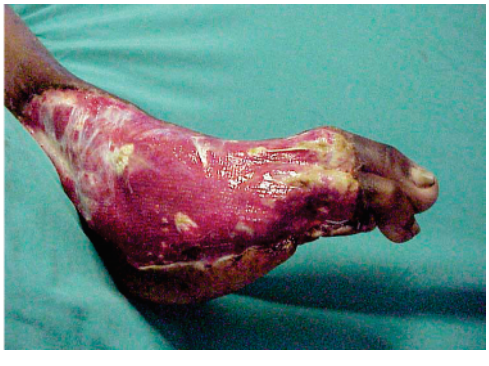
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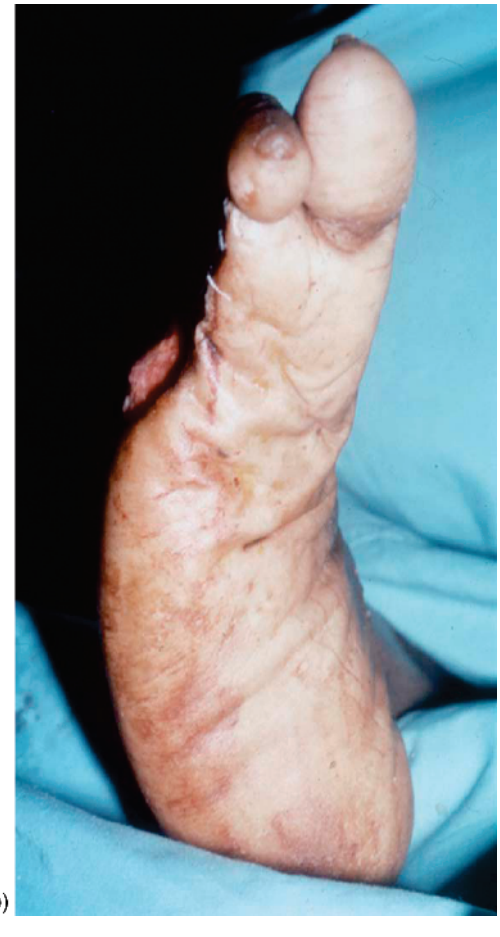
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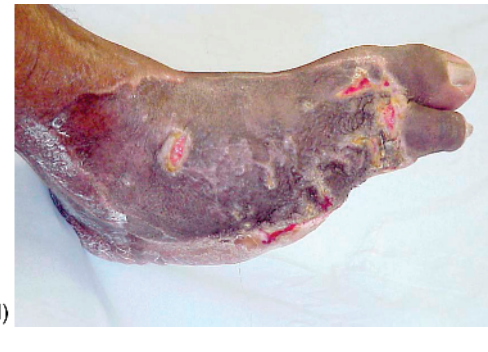

(d)

Figure 1.10 (a) Foot after surgical debridement. (b) Plantar skin. (c) Foot after treatment with VAC therapy. (d) Foot after successful skin graft.

Learning points

- Even a very small portal of entry, such as an insect bite, can lead to devastating infection, and patients from tropical countries may be particularly prone to develop these rapidly ascending infections.
- Necrotising fasciitis needs surgery without delay if the foot – and the patient's life – is to be saved.
- Wherever possible, the plantar tissues should be salvaged, as this will help to prevent further problems with neuropathic ulceration of the sole of the foot.
- The foot grew *Enterobacter cloacae*, a species of enterobacter that are enteric pathogens with limited sensitivities, being resistant to cephalosporins. Although laboratory testing may indicate sensitivity *in vitro* to the cephalosporins it is not advisable to treat with them, because enterobacter can induce chromosomal β-lactamases when challenged by a β-lactam antibiotic.
- Some surgeons believe that a transmetatarsal amputation should be performed if more than one ray needs to be removed, but it has always been King's policy to salvage as much as the foot as possible, since even if the foot is misshapen it can still function well with good foot care and footwear and orthotics.
- The VAC is a useful technique to obtain a good bed of granulation and to improve uptake of skin grafts.

Case 1.11 Denial leads to severe foot problems

Extensive tissue loss can also be a presentation of the neuropathic foot in the UK, especially when the patient has neglected his foot.

This 46 year old man had his Type 2 diabetes recently diagnosed when he had had a hospital admission with vomiting and fever. However, he refused any diabetic care or investigations. He was then admitted to hospital a year later with a large deep infection of the right foot extending over the lateral and dorsal aspects up to the ankle (Stage 5 foot). The second and fourth toes were already necrotic and the third toe had blue discoloration (Figure 1.11a–c). On admission there was a CRP of 187.5 mg/l. His creatinine was 51 μmol/l. WBC was 31.22×10^9/l, and neutrophils 27.67×10^9/l, indicating marked neutrophilia with a left shift in toxic granulation. X-ray of the right foot revealed osteopenia, but no injury was present. Glycated haemoglobin was 14.8%. Blood cultures were negative.

He was initially treated with quadruple IV antibiotics amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. Deep tissue swabs grew Group B *Streptococci* and *Staphylococcus aureus* and metronidazole and ceftazidime were stopped. He underwent extensive debridement of the foot, following which his CRP fell to 143.3 mg/l. Three days later a further surgical debridement was performed with amputation of the second, third and fourth toes. His wound was treated with VAC therapy (Figure 1.11d, e) and 18 days later he returned to theatre for further amputation of the first and fifth toes (Figure 1.11f) and his wound proceeded satisfactorily towards healing (Figure 1.11g).

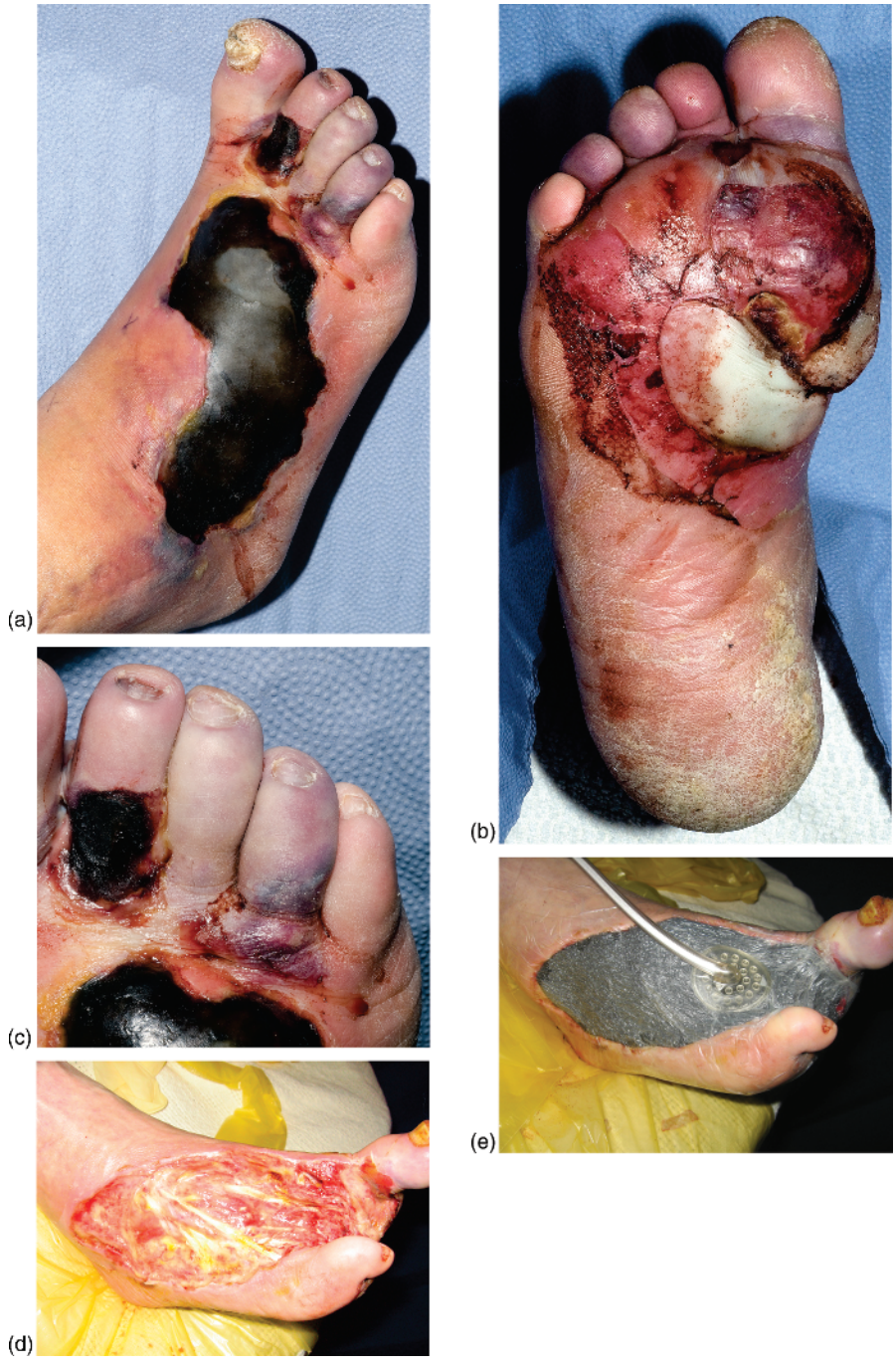


Figure 1.11 (a) Dorsum of foot with necrosis. (b) Plantar view showing extensive ulceration. (c) Blue discolouration of toes. (d, e) Post-operative wound before and after application of VAC therapy.

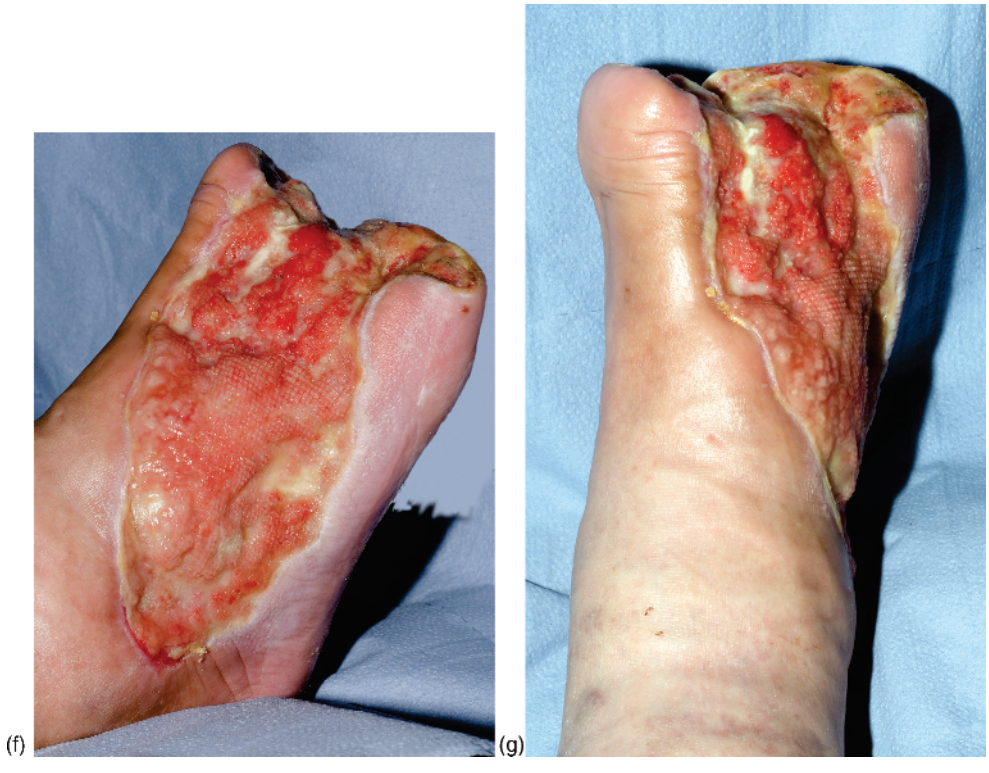


Figure 1.11 *(continued)* (f) Amputation of all the toes. (g) Healing dorsal wound.

Learning points

- This gentleman had extensive tissue necrosis and he was considered for major amputation. However, he had a very good peripheral circulation and extensive surgical debridement with IV antibiotic therapy enabled the foot to be preserved.
- This needed very close working between diabetic and orthopaedic services.
- Very frequent observation of the foot is essential to determine when further surgery is required.
- When a patient does not accept that his diabetes is a problem or attends for appointments, then he becomes very prone to problems.

Case 1.12 The neuropathic foot and dermatological problems

Some patients with diabetic foot infections develop dermatological problems, as in the following case of DRESS syndrome.

This 60 year old patient with Type 2 diabetes for 5 years was admitted with severe left foot sepsis and necrosis (Stage 5 foot) (Figure 1.12a). His CRP was 203.9 mg/l, blood glucose 17.5 mmol/l, glycated haemoglobin 11.4%, WBC 20.35×10^9/l, with neutrophils 17.61×10^9/l. He was initially given quadruple IV antibiotics amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. He underwent incision and drainage and debridement. Extensive necrotic tissue was noted in both plantar and dorsal aspects and incision of necrotic ulcer edges was performed with curettage and debridement. A large communicating cavity was found on the mid-plantar aspect of the foot up to the toes extending up into the dorsal aspect of the foot and a large amount of pus was drained, and debridement of putrefied, necrotised tissue was performed. The fourth toe was non-viable, with no obvious blood supply and extensive, infective necrotic tissue throughout it, and the third toe was also non-viable. The second toe was noted to have necrotic tissue surrounding it.

The tissue cultures grew *Citrobacter koseri*, *Streptococcus milleri* and mixed anaerobes. After surgery the white count fell to 8.77×10^9/l. The patient subsequently developed a widespread oedematous erythematous maculopapular rash on his scalp, chest, upper back and right forearm (Figure 1.12b). The patient also had an eosinophilia and abnormal liver function tests, but ultrasound of the liver was normal. He was treated as DRESS syndrome (drug related eosinophilia with systemic symptoms) and given prednisolone 60 mg daily.

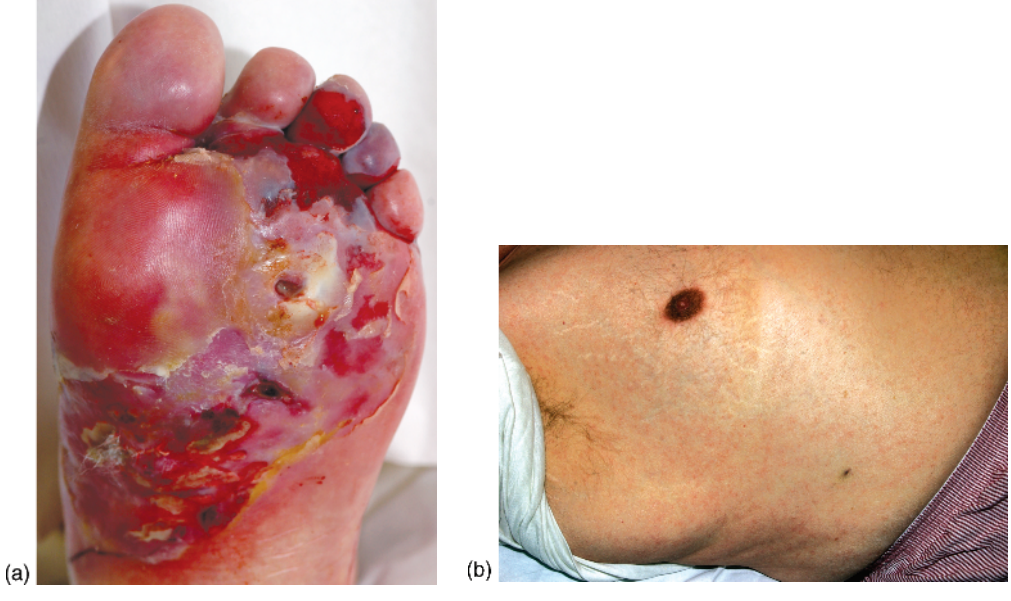


Figure 1.12 (a) Extensive cellulitis and necrosis. (b) Widespread rash.

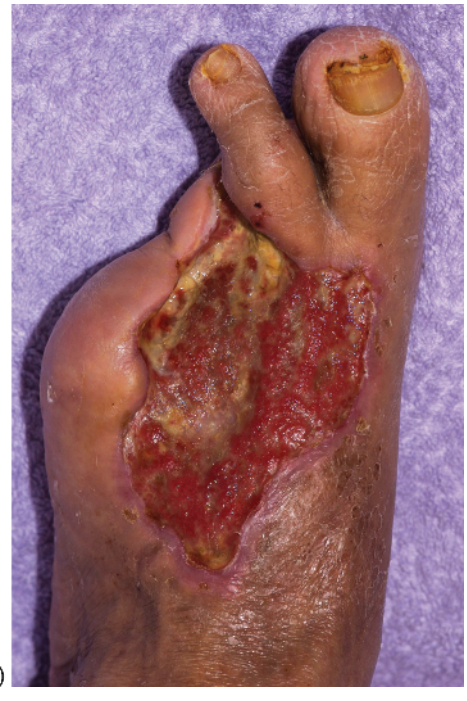
(c) 

Figure 1.12 *(continued)* (c) Healing foot.

Skin biopsy showed mild hyperkeratosis with minimal acanthosis, focal spongiosis and frequent apoptotic keratinocytes in the lower and, focally, in the upper layers of the epidermis. The superficial dermis contained a mild perivascular inflammatory cell infiltrate with scattered eosinophils. These features were in keeping with a drug reaction. The rash responded to prednisolone, which was gradually reduced in dosage and then stopped. The foot healed and he was followed up in the Diabetic Foot Clinic. Two months later, he had a relapse of his DRESS syndrome with an urticarial eruption over the left flank, which was again treated with a reducing dose of prednisolone. The foot continued to improve (Figure 1.12c).

Three months later, he had papules and nodules on the forearms and scalp as well as a demarcated linear papular urticated eruption on the right anterior cubital fossa. He was treated with Elocon (mometasone furoate) ointment to the inflamed areas of skin. After a further 3 months, the patient had a diffuse pruritic and erythematous rash on the chest and neck following treatment with metronidazole and was treated with chlorpheniramine. Finally, he developed thoracic shingles and was given acyclovir orally. He had residual post-herpetic neuralgia and was given dothiepin cream.

Learning points

- Diabetic patients with foot infections are exposed to many different antibiotics, which may provoke side effects including skin rashes.
- Dermatological drug reactions are self-limited diseases and therefore generally treatment is symptomatic. Prompt diagnosis and early withdrawal of all suspect drugs are the most important steps.
- Severe skin reactions include toxic epidermal necrolysis, Stevens-Johnson syndrome and DRESS syndrome, which stands for drug rash (or reaction) with eosinophilia and systemic symptoms.
- The symptoms of DRESS syndrome usually begin 1–8 weeks after exposure to the offending drug. Classic symptoms are rash, fever, lymphadenopathy and involvement of one or more internal organs. The rash is an erythematous skin eruption, often progressing to exfoliative dermatitis. It has a mortality rate of about 10%.
- DRESS syndrome responds to steroids, although it can relapse.
- This patient suffered a series of skin problems in which shared care with the dermatologists was crucial for accurate diagnosis and correct treatment.
- *Streptococcus milleri* is associated with pyogenic infection and abscess formation. Although often isolated in pure growth, it also found in polymicrobial infections.

Case 1.13 Diabetic patients with neuropathic feet may have a neuropathic bladder, which predisposes to urinary tract infections

The diabetic patient with neuropathy is prone to infection in other systems. Urinary tract infections are notable in diabetic patients with foot problems. These often emanate from a degree of neuropathy to the bladder: we have seen patients with distended bladders and concurrent foot problems, as in this case.

A 53 year old man with longstanding Type 1 diabetes for 48 years was admitted with ulceration and sepsis of the heel (Stage 4 foot). He was noted to have a distended abdomen and palpable bladder and urinary catheterisation drained 1500 ml of urine, which was cloudy and grew more than 100,000 organisms/ml of *Escherichia coli*, which was an ESBL (extended sensitivity β-lactamase*)* producer. The urinary tract infection responded to IV meropenem 1 g tds. A catheter was left in the bladder to drain completely for 12 hours. The patient then had urodynamic studies, which confirmed a neuropathic bladder, and this was treated by intermittent catheterization.

Learning points

- Patients presenting with neuropathic ulcer may have neuropathy affecting other systems of the body, and in particular the urinary tract.
- Symptoms are frequently masked by the neuropathy, so urinary retention should be actively sought since bladder distension is not painful in neuropathic patients.
- Bladder distension predisposes the patient to infection and a urinary culture should be obtained to detect the specific organisms so that appropriate antibiotic therapy can be prescribed.
- The *Escherichia coli* was an ESBL (extended sensitivity β-lactamase) producer. These organisms have developed resistance to extended-spectrum (third generation) cephalosporins (e.g. ceftazidime, cefotaxime and ceftriaxone) but not to carbapenems (e.g. meropenem or imipenem). ESBL enzymes are most commonly produced by two bacteria – *Escherichia coli* and *Klebsiella pneumoniae*. Another group of lactamases is AmpC β-lactamases, which are typically encoded on the chromosomes of many Gram negative bacteria including *Citrobacter*, *Serratia* and *Enterobacter* species, where expression is usually inducible. Thus, organisms considered susceptible by *in vitro* testing can become resistant during treatment with cephalosporins. These are gram negative organisms considered resistant to the cephalosporins and need treatment with the carbapenems.

Case 1.14 Prostatic obstruction

Although in a diabetic foot patient neuropathy may be the cause of a distended bladder, other aetiology such as prostatic enlargement should also be sought.

A 78 year old diabetic patient with Type 2 diabetes for 18 years was admitted with an infected ulcer on the right foot (Stage 4 foot) and had a surgical debridement by the orthopaedic surgeons. His serum creatinine quickly rose from 108 to 190 μmol/l, and then gradually fell to 106 μmol/l. The rise in creatinine was clearly associated with the episode of sepsis. At a routine measurement eight months later, the creatinine was normal, at 105 μmol/l, but three months after that it had risen to 264 μmol/l and the rising serum creatinine was initially assumed to be due to his diabetic nephropathy. After four more weeks it was 364 μmol/l. He was then admitted from the Diabetic Foot Clinic with a history of vomiting, fever and anorexia and a CRP of 177.3 mg/l. Although he had a small ulcer on the right foot there was no obvious focus of infection in the feet. Ultrasound of the kidneys and bladder showed both kidneys to be normal in size and shape. There was a bilateral hydronephrosis with an AP pelvic diameter of 2.7 cm on the left and 3.2 cm on the right. The bladder was of large volume and extended to the level of the umbilicus. There was a large residual volume after micturition of 1314 ml. Urine culture showed heavy mixed growth and he was treated with IV meropenem 500 mg bd. He had an enlarged prostate, which was clinically estimated to be 50 g. He did not want any active treatment, so a long term catheter was put in situ. His creatinine quickly fell to 130 μmol/l and did not rise again.

Learning points

- A raised serum creatinine in a diabetic foot patient may be related to diabetic nephropathy but there are other causes. Obstruction of the urinary tract should be investigated in all cases.
- The first rise of serum creatinine in this case history was related to infection in the foot. Although he had a foot ulcer at the time of the second rise in serum creatinine, it was not clinically infected, and indeed the second rise was related to the urinary tract infection and obstruction.
- Health care professionals working in Diabetic Foot Clinics have to be alive to the possibility of intercurrent illness in other systems in the body.

Case 1.15 Diabetic foot infection can masquerade as upper respiratory tract infection – always check the feet of diabetic patients

Although diabetic neuropathic patients with foot problems are prone to other infections including urinary infections, on certain occasions they do indeed have infection in their feet and a rise in inflammatory markers, which may mistakenly be attributed to infection elsewhere, including influenza.

This 40 year old patient with Type 2 diabetes for 6 years poorly controlled on metformin and glicazide first presented to Casualty complaining of flulike symptoms and cough. He was pyrexial and was a known asthmatic. He was treated with salbutamol nebulisers and sent home, with a diagnosis of a respiratory tract infection. His feet were not checked. The next day he came to the Diabetic Foot Clinic. He had infection of the left second toe with necrosis and cellulitis (Stage 5 foot) (Figure 1.15a). His foot pulses were palpable. He had a temperature of 38.6 °C. His CRP was 346 mg/l with a WBC of 9.62×10^9/l and neutrophils of 7.48×10^9/l (which is slightly raised).

He was admitted to hospital and initially treated with quadruple antibiotic therapy IV amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. Tissue grew *Staphylococcus aureus* and Group B *Streptococcus* and antibiotics were focussed down to amoxicillin and flucloxacillin. His foot X-ray was normal but that did not rule out osteomyelitis. His glycated haemoglobin on admission was 14%. He was given IV insulin sliding scale. He was treated with subcutaneous enoxaparin sodium as prophylaxis for deep vein thrombosis. In view of the necrosis he underwent amputation of the second toe (Figure 1.15b). Post-operatively he was treated with VAC therapy and made a good recovery, despite insisting on going home for Christmas earlier than the diabetic foot team thought was wise. He was switched to oral clarithromycin after he developed a maculopapular rash and the rash resolved. His foot healed.

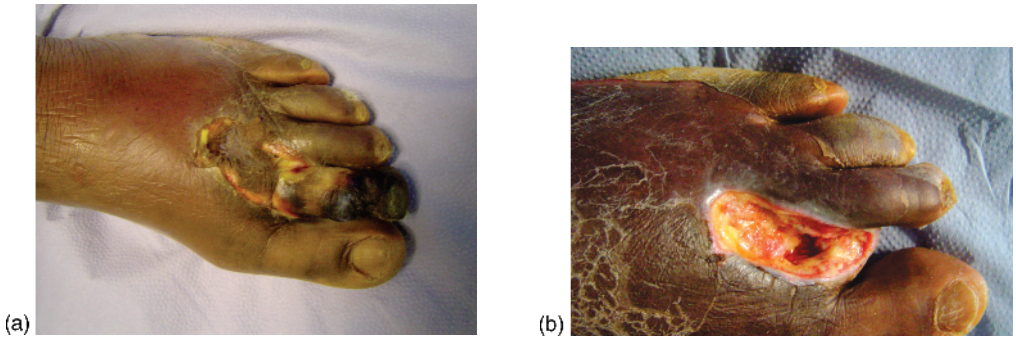


Figure 1.15 (a) Necrotic toe with spreading subcutaneous sloughing and cellulitis. (b) Amputation of second toe.

Learning points

- It should be mandatory to check the feet of any unwell diabetic patient.
- In this case, the patient had both a diabetic foot infection and a respiratory tract infection. When he presented on the second occasion with fever the respiratory tract infection diagnosed on the previous day could easily have been thought to be responsible for this, and it was only by checking the feet that his further foot infection came to light.
- All diabetic foot patients who undergo surgery and post-operative bed rest should receive venous thromboembolism prophylaxis.
- Osteomyelitis may not show up on X-ray for two weeks.

1.3 Co-Morbidities in addition to diabetes and neuropathy

Diabetic patients with neuropathy may have other co-morbidities, including microvascular and macrovascular complications. These patients may also have non-diabetic co-morbidities as well, which may further predispose them to ulceration and infection. These factors, in addition to peripheral neuropathy, may make them very vulnerable, as illustrated in the next series of patients, who demonstrated existing co-morbidities as they presented with their neuropathic feet. All these factors need active management to achieve resolution of infection and healing of ulceration.

Case 1.16 Diabetes, neuropathy and pancreatitis

This was a 51 year old man with Type 1 diabetes and an ongoing history of pancreatitis. He was admitted in diabetic ketoacidosis precipitated by acute pancreatitis and was treated in the Intensive Care Unit. He had clear evidence of neuropathy, with loss of sensation to mid-calf and absent vibration perception at the ankles bilaterally. He made a good recovery from his pancreatitis and was discharged, but eight weeks later he had a recurrence of his acute pancreatitis. During this admission a fissure on his left heel became infected, leading to cellulitis and osteomyelitis (Figure 1.16a). He was given IV piperacillin/tazobactam 4.5 g tds. Cultures from his heel grew *Staphylococcus aureus* and anaerobic *Streptococcus*. He underwent surgical debridement of the heel (Figure 1.16b) and two days after the operation he commenced VAC therapy. Seven days postoperatively he developed shortness of breath with right sided chest pain, and became hypoxic. On examination he had evidence of bilateral pleural effusions. Chest X-ray showed right sided basal consolidation. He underwent pulmonary angiography, which showed a filling defect in the right lower lobe subsegmental to the pulmonary artery, indicating a pulmonary embolus. His ECG did not show ischaemic changes and serum troponin I was less than 0.05 μg/l (lower limit of detection 0.05 μg/l), ruling out a myocardial infarction. His echocardiogram showed normal left ventricular systolic function. He was treated with therapeutic enoxaparin sodium. His heel improved on VAC therapy (Figure 1.16c), although he suddenly developed pain in his heel resulting from a pathological fracture of the calcaneum. This was treated with a non-weight-bearing cast in which a window was cut to observe the heel wound. Both fracture and wound eventually healed.

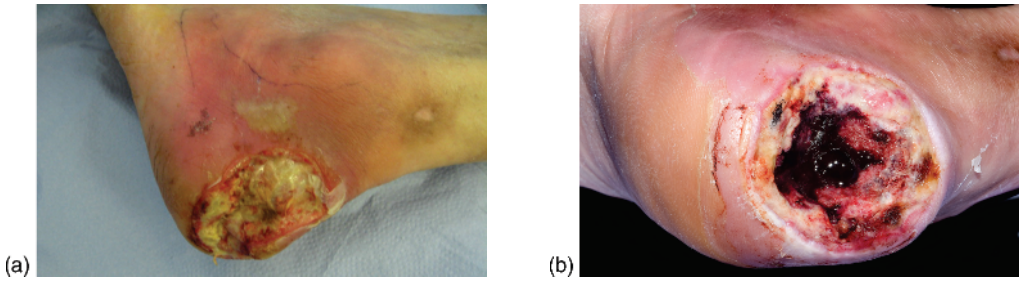


Figure 1.16 (a) Cellulitis and necrosis of the heel. (b) Post debridement.

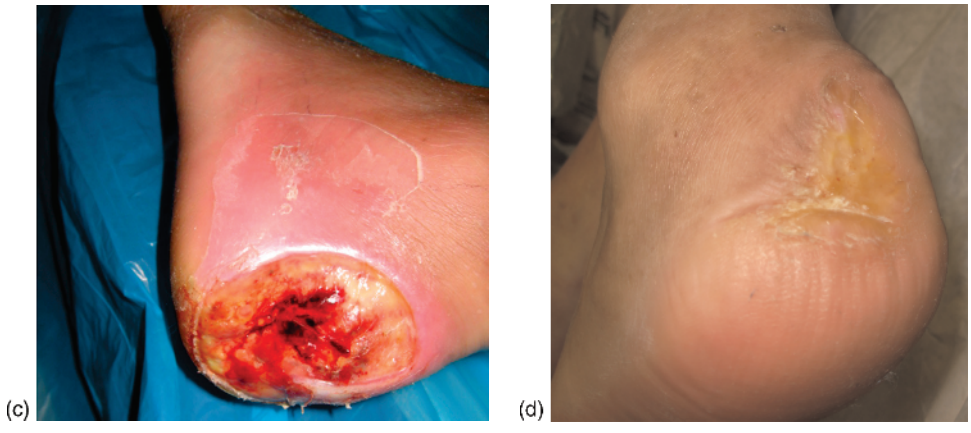


Figure 1.16 (*continued*) (c) Improvement on VAC therapy. (d) Healed wound.

Learning points

- Heel fissures are common problems on the diabetic foot and can act as portals of entry for infection. They should be treated early with debridement and emollients, to prevent ulceration.
- The calcaneal fracture was a pathological fracture related to osteomyelitis and underlying osteoporosis associated with his pancreatitis.
- Diabetic patients may not exhibit classical chest pain during episodes of ischaemia of the heart and the serum troponin is a measurement of cardiac tissue damage, which can be used in these circumstances to confirm or rule out a myocardial infarction.
- A serum troponin rise occurs in two situations. In ischaemic heart disease there is a rise in troponin because of the cardiac muscle damage from infarction secondary to thrombosis. Serum troponin also rises in septicaemia.
- It should be standard practice now for patients admitted to the hospital who are immobile to receive prophylactic anticoagulant therapy, as this patient did. Such therapy comprises a low molecular weight heparin such as enoxaparin sodium, which can be given subcutaneously once daily as long as the renal function is not significantly impaired, when heparin itself should be administered. Despite prophylactic anticoagulation in this case, the patient still developed a pulmonary embolus, which may have been related to his surgical debridement.

Case 1.17 Rheumatoid arthritis, neuropathy and an infected foot

Patients with diabetes and rheumatoid arthritis are very susceptible to ulceration and infection.

This 74 year old lady with Type 2 diabetes of 18 years' duration was referred to the Diabetic Foot Clinic by her general practitioner as an emergency. She lived with her 78 year old husband, who was in good health and drove her to the hospital in his own car. She had poorly controlled diabetes, despite taking maximum doses of oral hypoglycaemic agents, and a 40 years' history of rheumatoid arthritis, with severe deformity of feet and hands. This started when she was pregnant with her second child and had caused severe pain over the years, affecting her hands, wrists and elbows so that she could no longer play the piano, which caused her great sadness as she had been a talented amateur pianist. She also had problems with her ankles, knees and hips and she had undergone hip and knee replacements. She was taking methotrexate and prednisolone.

She had recently purchased a pair of "wide-fitting" shoes from a mail-order catalogue and had worn them around the house and on a visit to her daughter. "My wardrobe is full of almost-new shoes that I bought and can't wear because they make my feet hurt", she said. She had a VPT of 35 V and an ankle brachial pressure index of 1.00. She had severe hallux valgus of both feet. On the medial border of the right foot, directly over the prominent deformed forefoot there was a small ulcer with associated redness, warmth and swelling (Stage 4 foot). The base of the ulcer was yellow, soft and sloughy (Figure 1.17). She said that the ulcer itself was not painful but she had "deep pain" in both the feet whenever she walked. The ulcer did not probe to bone and an X-ray of the foot revealed severe erosions of the first metatarsophalangeal joint associated with rheumatoid arthritis, but no signs of infection in the bone. A diagnosis of soft tissue infection was made. She was admitted for IV antibiotics clarithromycin 500 mg bd and metronidazole 500 mg tds and oral ciprofloxacin 500 mg bd (she was allergic to penicillin), and the cellulitis resolved in five days and the foot healed after three weeks.

She was advised to use shoes that could accommodate the hallux valgus deformity, and agreed to this. Because of the severe deformity of the feet it was not possible to accommodate her in off-the-shelf, wide-fitting shoes, and bespoke boots were necessary. These had very deep and roomy toe boxes and deep cushioned insoles made of poron over a cradled insole to prevent her feet from rolling over into valgus and also supported her painful ankles. Heel counters were also fitted to increase her stability when walking, as she was very frail and unsteady and found it difficult to use crutches or sticks because they caused pain in her wrists and elbows. We asked the physiotherapists to review her and they visited her home and issued her with some helpful devices to make her life easier. She had no further foot problems but was seen regularly in the Diabetic Foot Clinic for nail care and regular review of the footwear. She was also followed in the Rheumatology Department. The King's Diabetic Foot Clinic ran a Friday morning satellite foot clinic for patients with rheumatoid arthritis, many of whom suffer from severe pain in the feet, and she attended this clinic, which was held concurrently with a rheumatology clinic, thus reducing the number of hospital appointments needed.

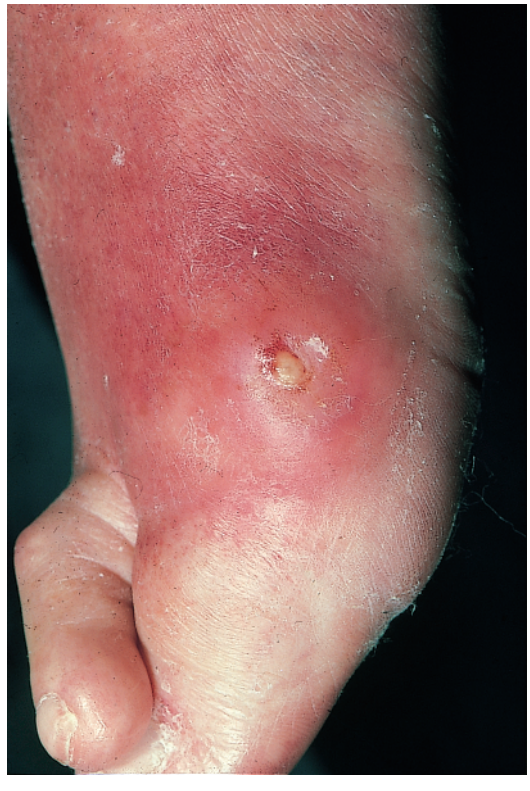


Figure 1.17 Sloughy ulcer.

Learning points

- Patients with rheumatoid arthritis may develop severe foot deformity. So long as there is no neuropathy they can often avoid ulceration because their feet are painful and they are careful to protect them, and if the feet are overloaded or the shoes are unsuitable the patient will be aware of the problem before it leads to ulceration. However, if they are also diabetic with peripheral neuropathy, as in the case of this lady, then foot ulceration is common. We have also seen cases of rheumatoid nodules of the feet that break down.
- Drugs used to treat rheumatoid arthritis predispose patients to infections, and rheumatoid patients who also have concurrent diabetes should be regarded as high risk patients.
- Diabetic foot patients should not purchase mail-order footwear without receiving advice from the Diabetic Foot Clinic, who should also check any shoes before the patient wears them.

1.4 Deformity, ulceration and infection treated by surgical debridement and reconstruction

Infections in the neuropathic foot often complicate ulcers that are associated with deformity. Long term outcomes improve if deformity can be corrected. In certain cases this correction can be carried out at the time of surgical debridement for an infection. We now describe four cases where reconstruction was successfully carried out at the time of their initial debridement. These patients were seen in the joint Orthopaedic/Diabetic Foot Clinic at King's, and we wish to pay special tribute to Mr. Venu Kavarthapu, Consultant Orthopaedic Surgeon, who carried out the reconstructions. All patients were followed up in the Diabetic Foot Clinic.

Case 1.18 Forefoot reconstruction can solve the problem of recurrent ulceration and sepsis associated with deformity

This was a 50 year old man with Type 2 diabetes for 10 years. Complications included maculopathy, peripheral neuropathy and hypertension. He had had previous amputations of the third and fifth toes. He developed a chronic ulcer under the second metatarsal head of the right foot, which was complicated by osteomyelitis. He was admitted as an emergency. He had a CRP of 70.9 mg/l and a WBC of 9.92×10^9/l with neutrophils 7.56×10^9/l (slightly raised). Although there was considerable cellulitis, no micro-organisms were grown. X-ray showed dislocation of the second metatarsophalangeal joint (Figure 1.18a). On MRI the second metatarsal showed abnormal signal on T1 and post-contrast extending through most of the metatarsal (Figure 1.18b). This was in keeping with osteomyelitis affecting almost all of the second metatarsal. The third metatarsal head showed abnormal signal on coronal section, again consistent with osteomyelitis (Figure 1.18c). All of these marrow changes appeared contiguous with extensive soft tissue change which extended up to the plantar ulcer.

The patient was treated with amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds IV antibiotics, underwent right second and third metatarsal exploration and had excision arthroplasty and internal fixation with a wire in the second toe (Figure 1.18d). He was discharged home on ceftriaxone 1 g IM daily and metronidazole 400 mg tds orally. The wire was removed after 4 weeks, the ulcer healed quickly and the foot did not relapse in the subsequent year.

Learning points

- Patients with persistent ulcer secondary to deformity should be assessed to determine whether the deformity can be corrected surgically.
- Patients presenting with infection secondary to ulceration can be treated with IV antibiotics, surgical debridement to remove infected tissue and formal reconstructive surgery to correct deformity.
- Forefoot reconstruction techniques can be very successful in correcting digital deformities.

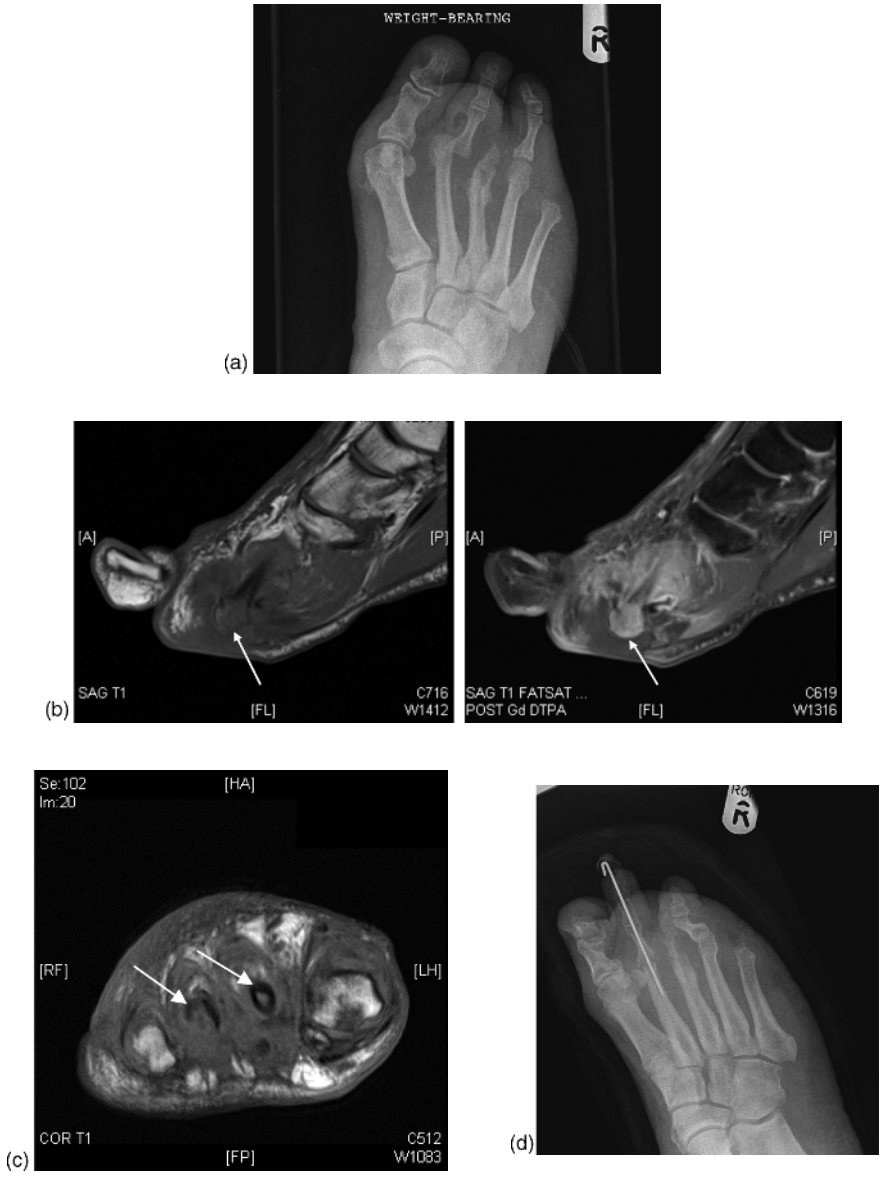


Figure 1.18 (a) X-ray shows dislocation of second metatarsophalangeal joint. (b) MRI sagittal sections of T1 and post gadolinium sequences showing oedema of second metatarsal head and shaft (arrow). (c) MRI coronal section showing oedema of second and third metatarsal heads (arrows). (d) X-ray following surgical procedure showing in situ wire in the second ray.

Case 1.19 The orthopaedic surgeon is a key member of the diabetic foot team

This 70 year old man had Type 1 diabetes for 40 years. His control was good with glycated haemoglobin of 7%. This patient first came to the Diabetic Foot Clinic 21 years previously presenting with ulceration of the left foot and also a left Charcot foot. His foot healed with podiatric debridement, antibiotics and an extended stay in hospital for bed rest. However, over the next 20 years he had many episodes of plantar ulceration. Despite receiving prolonged pressure relieving orthotics in the Diabetic Foot Clinic, the plantar ulcer did not heal. He was admitted as an emergency with infection of a plantar ulcer under the second metatarsal head. He had a fixed claw deformity, a stiff first metatarsophalangeal joint and a non-reducible chronically dislocated second metatarsophalangeal joint. The ulcer probed only to the soft tissues. X-ray showed, in the first and second metatarsophalangeal joint, loss of joint space, sclerosis and subchondral cysts. There was subluxation of the second metatarsophalangeal joint (Figure 1.19). He had a CRP of 38.2 mg/l and a WBC of 6.71×10^9/l, of which neutrophils were 4.91×10^9/l. He was treated with IV vancomycin 1 g statim and then dosage as per serum levels, metronidazole 500 mg tds and ceftazidime 1 g tds (he was allergic to penicillin). *Staphylococcus aureus* was grown from the foot ulcer swab.

He underwent surgical debridement and dorsal wedge shortening with Weil's osteotomy and proximal hemi-phalangectomy of the second toe of the left foot to correct the

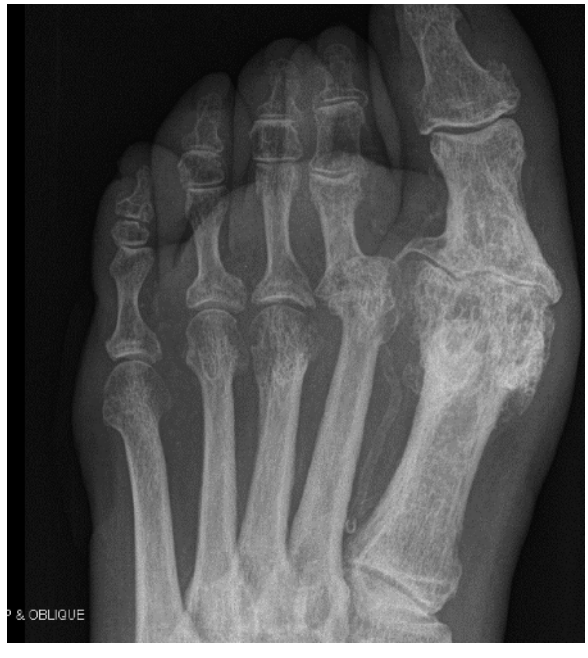


Figure 1.19 Foot X-ray showing subluxation of the second metatarsophalangeal joint and degenerative changes at the first metatarsophalangeal joint.

deformity that had predisposed him to recurrent ulceration. He was discharged on ceftriaxone 1 g IM daily and metronidazole 400 mg tds orally. His ulcer healed and his deformity was corrected.

Learning points

- Neuropathic patients with long term ulceration and recurrence should be assessed for any associated deformity that has been responsible for the ulceration.
- Where possible, these deformities should be corrected by the orthopaedic surgeon.
- When patients present with both infected ulceration and deformity it is now possible to perform surgical debridement and correction of deformity in the same operation. However, it is important that the infection has been investigated and the appropriate antibiotics administered intravenously to eradicate the infecting organisms.

Case 1.20 Very rapid deterioration of a patient with diabetic foot infection

This patient was 60 years old and had Type 2 diabetes for 24 years. He had had laser therapy for proliferative retinopathy. He attended with ulceration under the left first metatarsal head associated with mild cellulitis. His temperature at presentation to the Diabetic Foot Clinic was 37.2 °C. Over the subsequent 2 hours his cellulitis spread and he became very unwell. He vomited, and became faint and hypotensive. His WBC was 25.08×10^9/l with a marked neutrophilia and CRP was 127 mg/l. Serum creatinine was normal at 117 μmol/l. He was started on quadruple IV antibiotics amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds IV. His CRP was 178 mg/l on admission to the ward and next day rose to 264 mg/l. Blood cultures grew Group A *Streptococcus*, which was also isolated from his foot ulcer deep wound swab. He developed severe pain in his left groin and ultrasound showed multiple enlarged lymph nodes measuring up to 1.7 cm in short axis diameter. On the third day CRP had fallen slightly to 236 mg/l and white count was 18.57×10^9/l. Both parameters gradually fell over the following week and his cellulitis resolved. He was followed up in the Diabetic Foot Clinic, and the ulcer healed.

However, he had recurrent foot ulceration over the first metatarsophalangeal joint. Each episode was associated with cellulitis and he was very quickly treated with amoxicillin and flucloxacillin orally. He had one further admission with cellulitis and a CRP of 86mg/l and slightly raised white count of 11.45×10^9/l with 9.18×10^9/l neutrophils. He was again treated with quadruple IV antibiotics as above. A deep wound swab grew *Staphylococcus aureus* and his antibiotics were focussed to flucloxacillin. It had not been possible to prevent recurrent ulceration with conservative therapy. X-ray showed a severe hallux valgus deformity with previous amputation of the second toe (Figure 1.20a). He underwent surgical debridement and correction of his left forefoot deformity with metatarsal osteotomy and fusion of the first metatarsophalangeal joint. In the anaesthetic recovery room, he collapsed and vomited and was treated for a possible aspiration pneumonia with meropenem 1 g tds IV. A postoperative chest X-ray was normal and foot X-ray showed internal fixation of the first metatarsal and proximal phalanx of the great toe as well as of the second and third metatarsal heads (Figure 1.20b). He has had no further episodes of ulceration over the subsequent year.

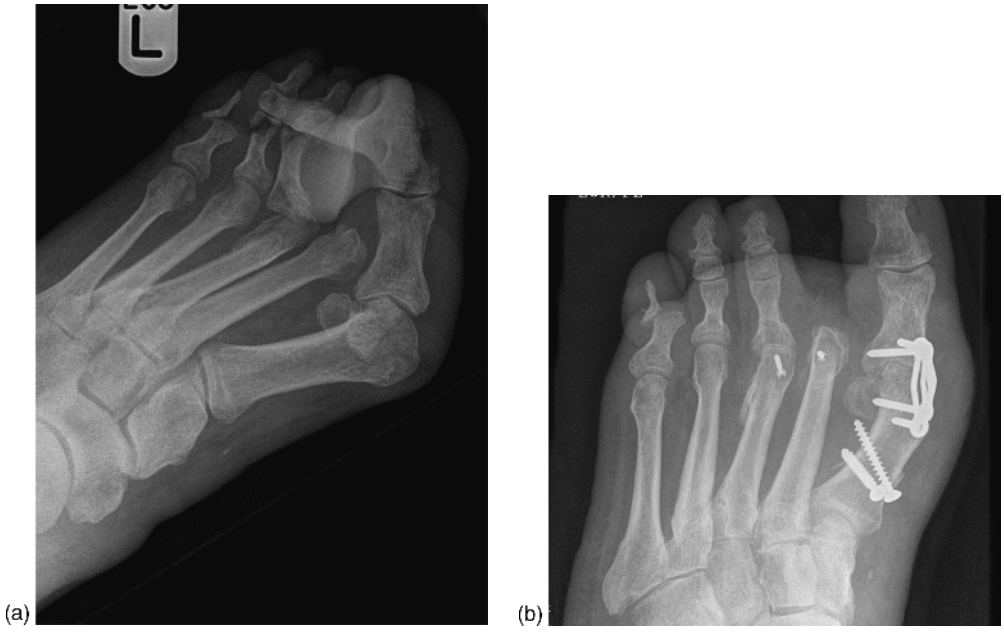


Figure 1.20 (a) Severe hallux valgus deformity with previous amputation of the second toe. (b) Internal fixation of the first metatarsal and proximal phalanx of the great toe as well as of the second and third metatarsal heads.

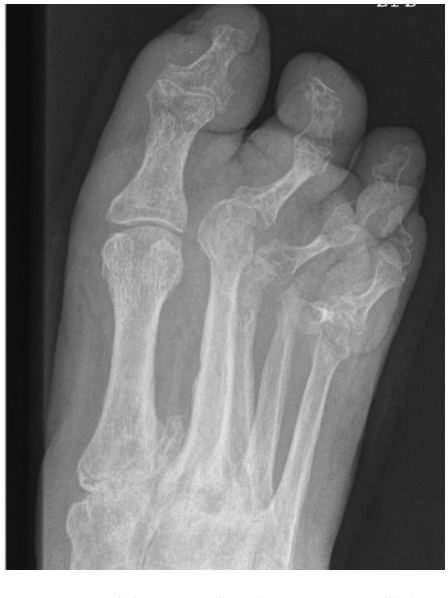
Learning points

- Diabetic patients with foot infection can become very unwell very quickly.
- Group A streptococcus can cause severe systemic symptoms.
- Pain in the groin from lymphadenopathy can be acute and can be indicative of a serious foot infection.
- The CRP usually reflects inflammatory events from the previous day.
- Recurrent foot ulceration associated with hallux valgus deformity should be addressed by correction of the underlying deformity.

Case 1.21 Indolent neuropathic ulceration

This patient was 48 years old with Type 1 diabetes for 33 years. She had a chronic non-healing ulcer on the plantar aspect of the right second metatarsophalangeal joint for 12 months despite cast therapy. She had a child and was very reluctant to undergo any treatment requiring hospital admission. However, she developed a foot infection with cellulitis and was admitted as an emergency for IV antibiotic therapy while her parents cared for her child. She was given antibiotics comprising IV vancomycin 1 g statim and then dosage as per serum levels, ceftazidime 1 g tds and metronidazole 500 mg tds as she was allergic to penicillin. She had a CRP of 73.7 mg/l and WBC of 10.52×10^9/l of which 8.32×10^9/l were neutrophils. Most previous wound swabs were negative, but one had shown MRSA. The cellulitis improved but her X-ray showed osteomyelitis of the second metatarsal head (Figure 1.21a). She underwent excision arthroplasty of the second metatarsophalangeal joint and Weil's osteotomy and proximal interphalangeal joint fusion of the fifth toe (Figure 1.21b, c). The alignment of the toes was satisfactory.

She was discharged with a CRP of 5.7 mg/l and a normal WBC, on teicoplanin 400 mg IM daily, and the ulcer healed after six weeks.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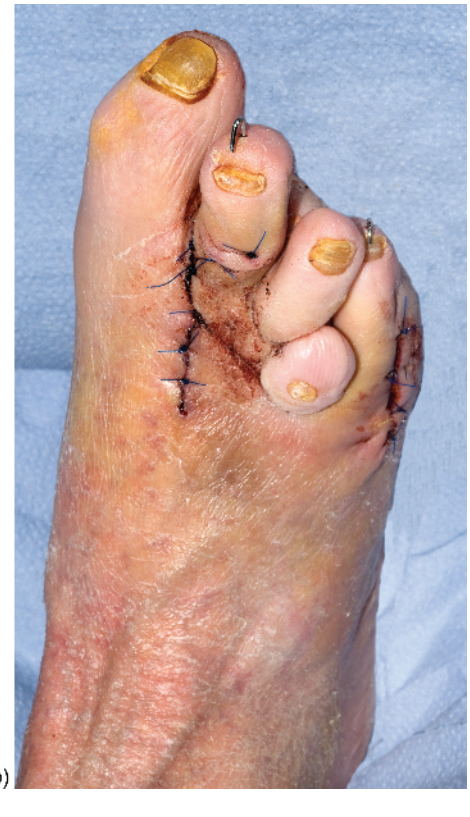
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Figure 1.21 (a) X-ray showing osteomyelitis of the second metatarsal head. (b) Dorsal view of the postoperative foot.

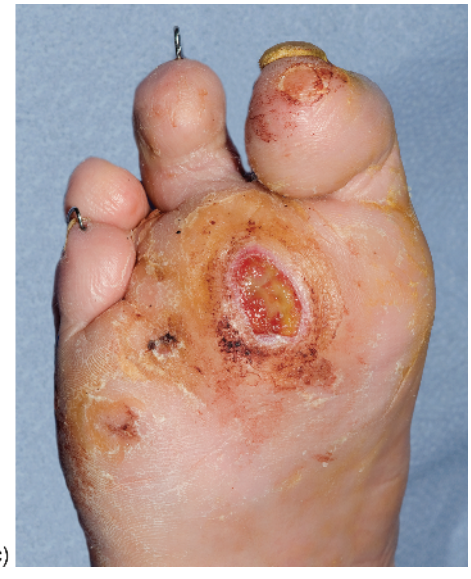
(c) 

Figure 1.21 *(continued)* (c) Plantar view of the postoperative foot.

Learning points

- Long standing Type 1 patients with chronic recurrent foot ulceration often develop infection. Most episodes can be managed as an outpatient within the Diabetic Foot Clinic.
- Recurrent episodes of ulceration and infection can lead to chronic osteomyelitis and destruction of underlying bone and joint, which is best treated by excision arthroplasty.
- This lady avoided hospital admission for many years, but when an episode of ulceration was associated with severe sepsis necessitating hospital admission and IV antibiotics she was then able to accept surgery.

1.5 Patients with neuropathic feet in whom psychological factors have impacted on their management

Concurrent psychological problems can lead to enormous barriers to care. The six patients described below cover a wide variety of mental health problems, personality disorders and inability to accept advice and treatment. Some patients have incorrect beliefs about how neuropathic ulcers develop and do not understand the association between neglected callus and subsequent ulceration. They need careful education. Some unfortunate patients actually cause their own ulcers, and even prevent them from healing. It is difficult to understand the motivation of these patients. Confrontation is not helpful: they will move away to another hospital where their psychological problems are not known. We have seen a small cluster of these patients: they are almost all young women. A previous history of eating disorders or brittle diabetes is often found, and several of our patients have been health care professionals.

Case 1.22 Neuropathic ulceration and psychiatric disease

This 65 year old man with Type 2 diabetes for 12 years and a history of paranoid schizophrenia attended the Diabetic Foot Clinic over many years. He developed ulceration and swelling over the left first toe (Figure 1.22a). On attending the Diabetic Foot Clinic for a routine appointment he was found to have deteriorated and now had osteomyelitis of his left first toe (Figure 1.22b), localised collection of pus, fever and CRP of 150.6 mg/l. He was advised that he needed hospital admission and first toe amputation but refused this. His foot swab grew *Pseudomonas* and he was treated with ciprofloxacin 500 mg bd as an outpatient. During this period he was attending his GP practice from Monday to Friday for daily dressing change and being attended by the district nurses at the weekends.

He returned after two weeks, during which time his foot deteriorated (Figure 1.22c). His temperature was 38.9 °C. He finally agreed to be admitted to hospital. On the ward, he was treated with piperacillin–tazobactam 4.5g tds. Blood cultures on admission demonstrated MRSA bacteraemia. Vancomycin IV 1 g statim and then dosage as per serum levels was started and the piperacillin–tazobactam was stopped. The patient was isolated according to the hospital's MRSA infection control policy. Previous specimens from the Diabetic Foot Clinic identified the patient to be MRSA negative. He continued to refuse amputation. The cellulitis settled after four weeks of IV antibiotic therapy He refused any surgical intervention to his left first toe. His CRP had fallen to 83.8 mg/l on discharge and he was followed up in the Diabetic Foot Clinic. He was given fusidic acid 500 mg tds and doxycycline 100 mg daily. Foot X-rays showed evidence of resolving osteomyelitis (Figure 1.22d) and the ulcer eventually healed (Figure 1.22e). The patient continues to attend the Diabetic Foot Clinic.

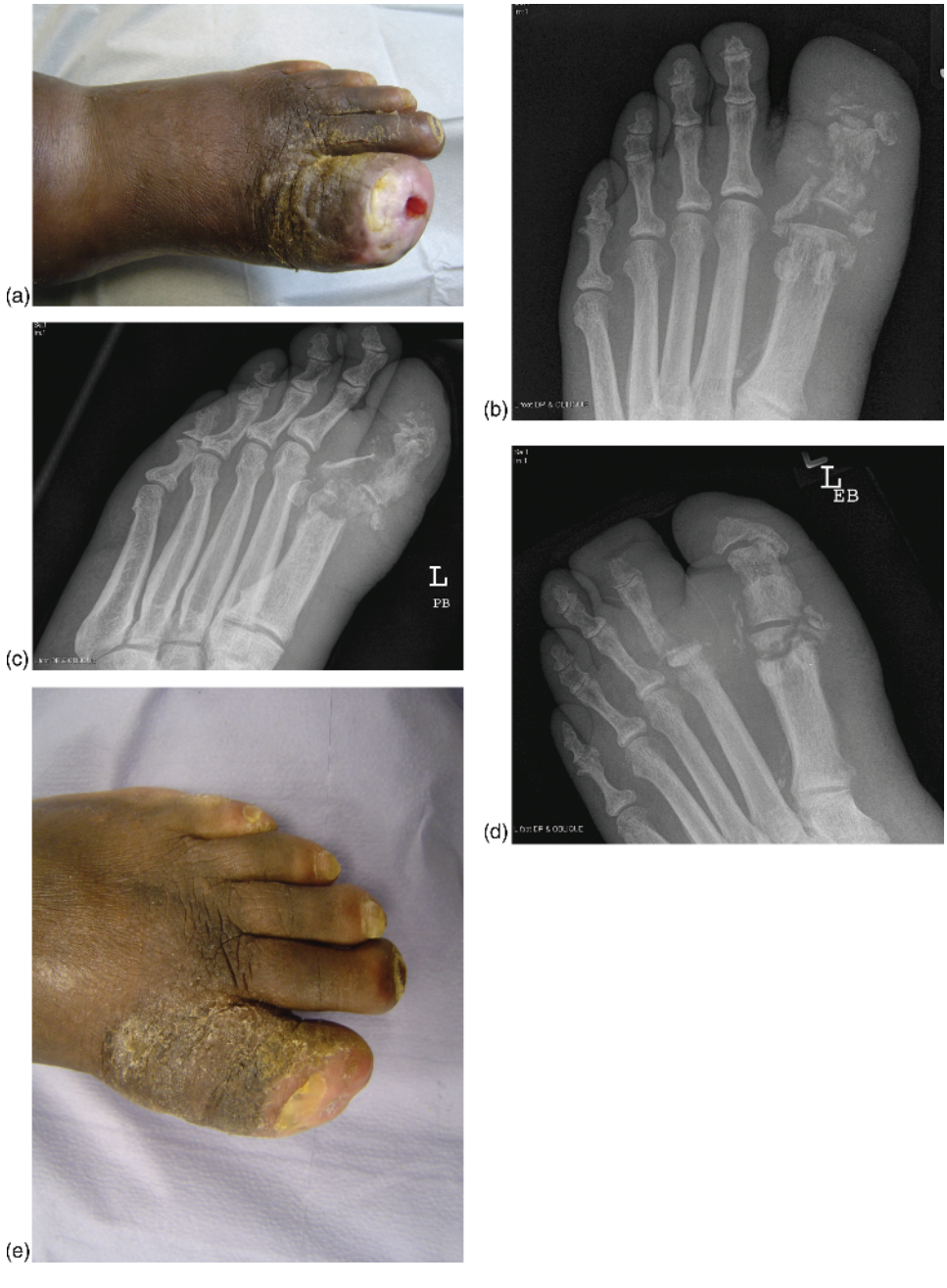


Figure 1.22 (a) Ulceration and swelling of left first toe. (b) X-ray shows osteomyelitis of proximal and distal phalanges of the left first toe. (c) X-ray shows worsening osteomyelitis. (d) X-ray showing reformation of proximal phalanx. (e) Healed ulcer.

Learning points

- Patients with mental health problems pose a great challenge and the Diabetic Foot Clinic is the ideal forum to follow them. It is their safe haven where they have continuity of care and often come to trust the personnel within it.
- It may not be possible to achieve all our goals and it may be necessary to compromise.
- This patient's ulcer eventually healed despite severe underlying bone destruction. Osteomyelitis can resolve on conservative treatment.
- It is always important to culture diabetic foot lesions and to take blood cultures so as to detect infecting organisms and bacteraemias including MRSA infections.
- Patients with MRSA infections should be isolated.

Case 1.23 In-growing toenail

A 59 year old man with Type 2 diabetes of 8 years' duration was referred to the Diabetic Foot Clinic by his general practitioner, when he visited him complaining of pain and throbbing in the right hallux, which was keeping him awake at night. His VPT was 15 V and pedal pulses were palpable. He lived alone following the death of his wife. The diabetes was well controlled with a glycated haemoglobin of 6%. He had never received foot care and had always cut his own nails. On examination the right foot was swollen, the medial nail sulcus of the right hallux was red and puffy (Figure 1.23) and pus was discharging from the medial sulcus. A diagnosis of onychocryptosis (in-growing toenail) was made. The medial sulcus was probed by the podiatrist and a splinter of nail that had penetrated the flesh was found and removed and the edge of the nail plate was smoothed with a Black's file so that it did not press upon the swollen sulcus. A dry dressing was applied. A swab was sent for culture, and oral amoxicillin 500 mg tds and flucloxacillin 500 mg qds were prescribed. Two days later the results of culture were received and checked against the patient's records to ensure that the antibiotics were appropriate. *Staphylococcus aureus* was grown, and the amoxicillin was discontinued. The foot healed in two weeks.

At his follow-up appointment, the patient was advised to cut his nails straight across and to ensure that he never tried to cut out the corner of the nail or left a splinter of nail behind

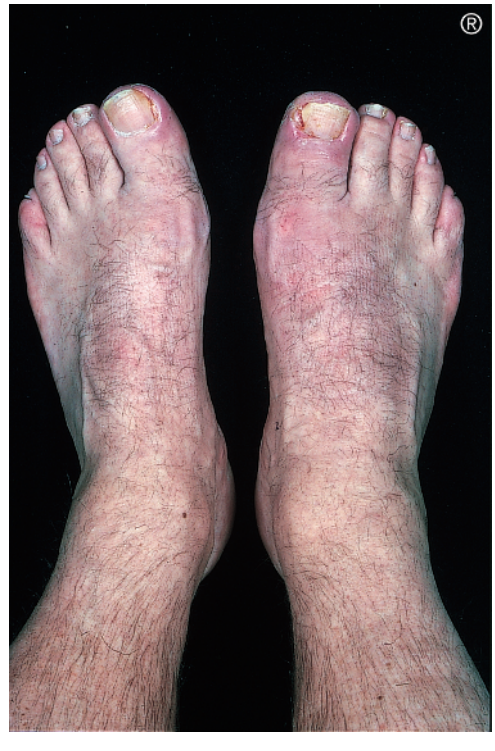


Figure 1.23 Swollen right foot with inflamed nail sulcus.

that might be forced into the flesh as the nail grew forward. He was advised to return to the Foot Clinic if any future problems arose and did not come back for ten years.

Unfortunately, his diabetes control had deteriorated, and he had developed complications. Initially the community podiatry service saw him and cut his nails and reduced callosities on the plantar surface of both feet. He developed a plantar ulcer on his left first metatarsal head; he was referred back to the Diabetic Foot Clinic by his community podiatrist, who was doing a training rotation working one day a week for three months at King's. She saw him at King's with the multidisciplinary team to ensure continuity of care. The foot was X-rayed: there were no signs of osteomyelitis. A swab was sent to the laboratory for culture: it grew *Staphylococcus aureus* and *Streptococcus* Group G and amoxicillin 500 mg tds and flucloxacillin 500 mg qds were again prescribed. The ulcer was debrided of callus. The orthotist made casts of both feet with a view to manufacturing bespoke shoes with cradled insoles. The ulcer healed in nine weeks and the patient continued to attend the Diabetic Foot Clinic every month and to see the Community podiatrist between these appointments. He lived alone, and gradually became unkempt in appearance and began to arrive late for appointments and sometimes to miss them. He also became very reluctant to wait to be seen if it was not possible for him to come into the clinic immediately. He arrived at the Foot Clinic one day wearing his bedroom slippers, saying that he had lost his bespoke shoes, but on the next visit he had found them again. On another occasion he wore unmatched shoes from two differently coloured pairs. He had no relatives or close friends to help him and was socially isolated. The general practitioner talked to him about his home situation: the patient was very reluctant to give up his home and move into sheltered accommodation, or to pay for a home help. He developed chronic neuropathic ulcers and the district nurses called daily to clean and dress his feet, but often he was not at home when they called. Close contact was maintained between the district nurses, the Foot Clinic, the community podiatry service and the GP practice, so that regular checks could be made on the patient's condition. He did not bathe regularly, and often did not take his medication regularly. Over the last two years of his life he had seven hospital admissions when his feet became infected. Use of a total contact cast to heal the ulcers was considered but it was felt that the risk of him not attending for review and developing devastating infection within the cast was too great for it to be risked. Eventually he failed to attend an appointment at the Foot Clinic, who notified the district nurses. They called at the house and could not gain entrance but spoke to a neighbour who was concerned because she had not seen the patient for two days. The police were asked to help. When they entered the house they found that the patient was dead. There was a coroner's inquest: the post-mortem revealed that he had died of a myocardial infarction.

Learning points

- Patients without significant neuropathy or peripheral vascular disease who can see their feet clearly are usually capable of self-care.
- All people with diabetes should be taught the principles of good nail care.

- If onychocryptosis does not settle and becomes a chronic problem then partial nail avulsion with phenolysation of the nail bed is an effective treatment, but is contra-indicated in patients with peripheral vascular disease.
- Nail conditions can lead to severe problems in the complicated diabetic foot and patients should receive expert help within a short time.
- Sometimes the home situation is not ideal and patients are unable to look after their feet well. In these circumstances, health care professionals have to make the best of the situation and endeavour to prevent catastrophes by offering frequent appointments and regular review. Home visits from the district nursing team are very useful.

Case 1.24 A patient's belief that podiatry caused a diabetic foot ulcer

A 58 year old woman with Type 1 diabetes of 38 years' duration was warned by her general practitioner that she should wear "sensible shoes" when she visited him and showed him a large area of callus on the plantar surface of her foot (Figure 1.24). He also wrote a referral letter to the Diabetic Foot Clinic asking if they could provide footwear, and she was seen later that week. She had palpable foot pulses and a VPT of 40 V. When the podiatrist debrided away the plantar callus a large blister was revealed. The patient became very upset, saying that the podiatrist had cut her foot and "caused an ulcer" and it was only after she talked to the diabetologist and the nurse that she understood that the problem had been present beneath the callus and that callus removal had only exposed the ulcer and not caused it. Dressings were applied and the patient was asked to rest and to return to the Foot Clinic in seven days' time, when the blister had healed. Bespoke shoes with cradled insoles were provided, and she remained ulcer free and attended the Foot Clinic regularly for callus removal and nail care.

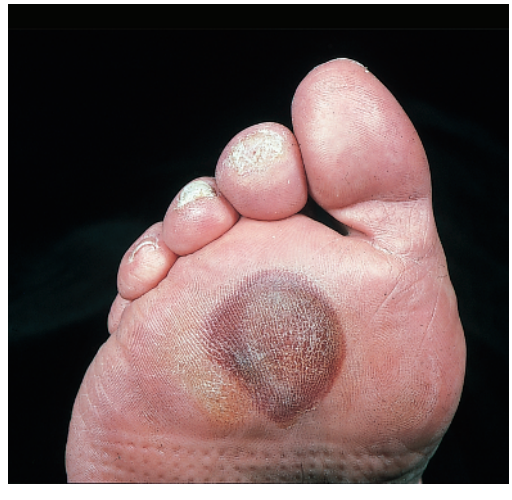


Figure 1.24 Callus with bulla.

Learning points

- Before callus or ulcers are debrided an explanation should be given to the patient as to the reasons for the debridement and the likelihood that an ulcer may be present beneath the callus.
- If patients become upset then a uniform response should be given by all the team members.

Case 1.25 Self-inflicted injuries

A 33 year old woman with poorly controlled Type 1 diabetes of 25 years' duration, proliferative retinopathy treated with laser, autonomic neuropathy and severe peripheral sensory neuropathy picked at her toenails instead of cutting them. She also had a habit of pulling pieces of loose skin off her heels, which were very dry. She was first seen in the Diabetic Foot Clinic when she first attended the Diabetic Department at King's at the age of 30 years. She was married with one small son. She had been referred to King's for "brittle diabetes", having had many episodes of ketosis requiring hospital admission and also complained of hypoglycaemia of sudden onset, of which she had no warning, which made it impossible for her to drive a car. When the condition of her toenails was noted she was offered treatment in the Diabetic Foot Clinic but refused to attend. She was referred to the Diabetic Foot Clinic again three years later, after attending the Diabetic Clinic for annual review. A random capillary glucose was unusually high, at 22 mmol/l and removal of her footwear revealed a hot swollen right foot with a blueish hallux. She said that two days before she had pulled off a loose piece of skin and the toe had bled but had not hurt. She was very reluctant to accept that admission was necessary, but telephoned her husband who came to the hospital and persuaded her to come in. Intravenous antibiotics were administered: however, the hallux gradually became black and necrotic and was amputated by the orthopaedic surgeon (Figure 1.25a). She was anxious to go home as soon as possible as she wanted to be with her family. Her small son became very upset every time his mother

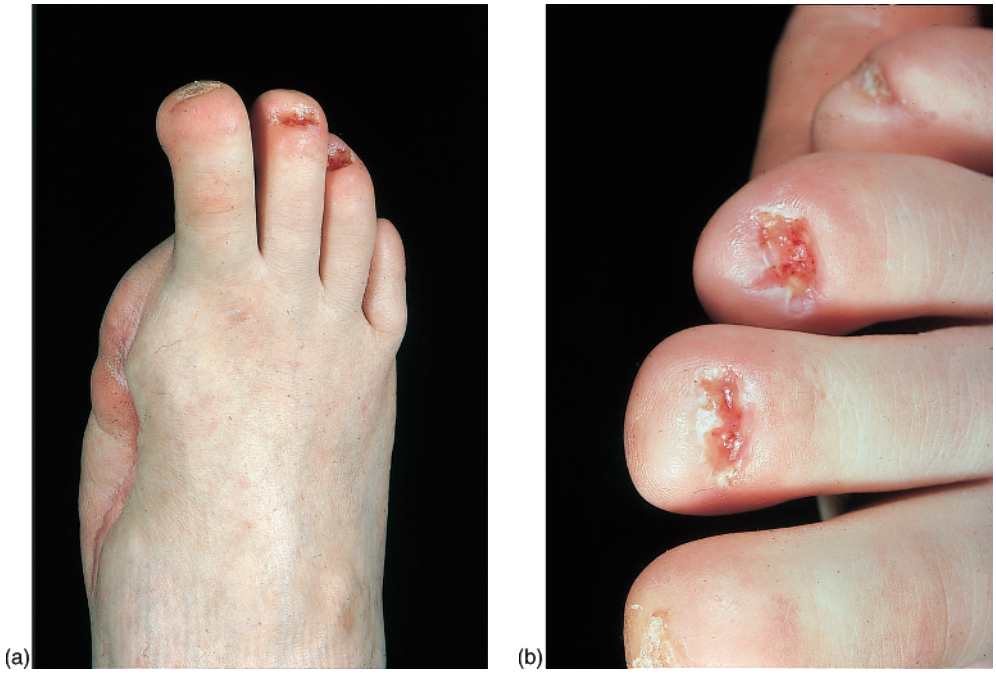


Figure 1.25 (a) View of foot with amputated toe. (b) Lesser toenails have been pulled off.

was admitted. She went home after two weeks in hospital. She was given an appointment the following week to come to the Foot Clinic, but failed to arrive. However, she attended the Diabetic Foot Clinic one month after that, walking in without an appointment. The amputation site was fully healed but nails had been pulled off all the lesser toes (Figure 1.25b).

Over the next 20 years she had numerous hospital admissions for infected neuropathic ulcers. Pulling pieces of skin off the heels eventually resulted in bilateral deep heel ulcers: these would heal in total contact casts but quickly relapse once the casts were removed, and it was clear that she was pulling pieces of tissue from the bases of the ulcers and the surrounding skin. She was seen by the Foot Clinic psychiatrist, but was unable to break the habit of pulling skin off her feet and remains with chronic neuropathic ulcers of both feet. She is aware that she is damaging her feet, but is unclear as to the motives behind this, and feels unable to stop. She has survived long term despite many ulcers and infections, and has no diabetic nephropathy and no cardiac problems.

Learning points

- Self-inflicted injuries can be a portal of entry for infection.
- Concurrent psychological problems make foot ulcers very difficult to treat.
- Self-inflicted ulcers are unrewarding to treat but care should never be withdrawn.
- Every diabetic foot clinic should have links with a psychiatrist as a member of the multidisciplinary team.

1.6 Long term patients followed in the Diabetic Foot Clinic with neuropathic foot problems

For many patients with neuropathic feet, long term attendance at a Diabetic Foot Clinic is the only way to avoid amputation.

Case 1.26 Long term management of a grateful patient who avoided losing his legs

The patient, a successful 40 year old banker, with Type 1 diabetes for 22 years, was referred to the Diabetic Foot Clinic after his local hospital proposed bilateral below knee amputations as the only solution to his indolent neuropathic ulcers. His feet had been ulcerated for over five years, and all his toes had been amputated during episodes of neuropathic ulceration complicated by infection. He had deep plantar ulcers on both forefeet five centimetres in diameter. However, X-ray did not reveal extensive osteomyelitis. The reason the local hospital gave for amputation was that they felt the ulcers would never heal and that unhealing ulcer is an indication for major amputation. This is a policy our Diabetic Foot Clinic has never adhered to. When told at his initial visit that his feet could be saved he wept with relief.

Initially he was treated with sharp debridement of his plantar neuropathic ulcers by the podiatrist (Figure 1.26a, b), and total contact casting. The ulcers became smaller and shallower, but after two months he developed infection of the left foot within the cast. There were no signs or symptoms until the cast was removed at a routine clinic appointment. There was oedema and crepitus in the soft tissues around the ankle, and X-ray revealed gas in the tissues. He was admitted the same day, and given IV amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. A tissue specimen taken in the Diabetic Foot Clinic grew anaerobic *Streptococcus*. His infection resolved over the next four days. In view of the problem under the cast, it was decided to discontinue total contact casting, and he was treated with regular podiatry to sharp debride the ulcers, felt padding to redistribute pressure from the ulcers, and orthopaedic boots with cradled insoles. He took prolonged courses of antibiotics. The ulcer healed (Figure 1.26c).

However, we were unable to achieve long term healing of the ulcers. The patient attended the Diabetic Foot Clinic for 17 years and his feet survived despite frequent episodes of ulceration and recurrent infections necessitating hospital admissions on nine occasions. By arrangement, most of these admissions were to his local hospital and were for acute sepsis, and on each occasion his CRP was very high, ranging between 188.3 and 371.6 mg/l. He never needed surgery and always responded quickly to treatment with IV antibiotics. On one occasion he had an ulcer on the lateral border of his left foot. He went to the bathroom in the middle of the night and knocked his foot on the bathroom door. The ulcer bled, and he and his wife were unable to staunch the flow. She took him to the local hospital casualty department, where it was found that he had fractured a small calcified

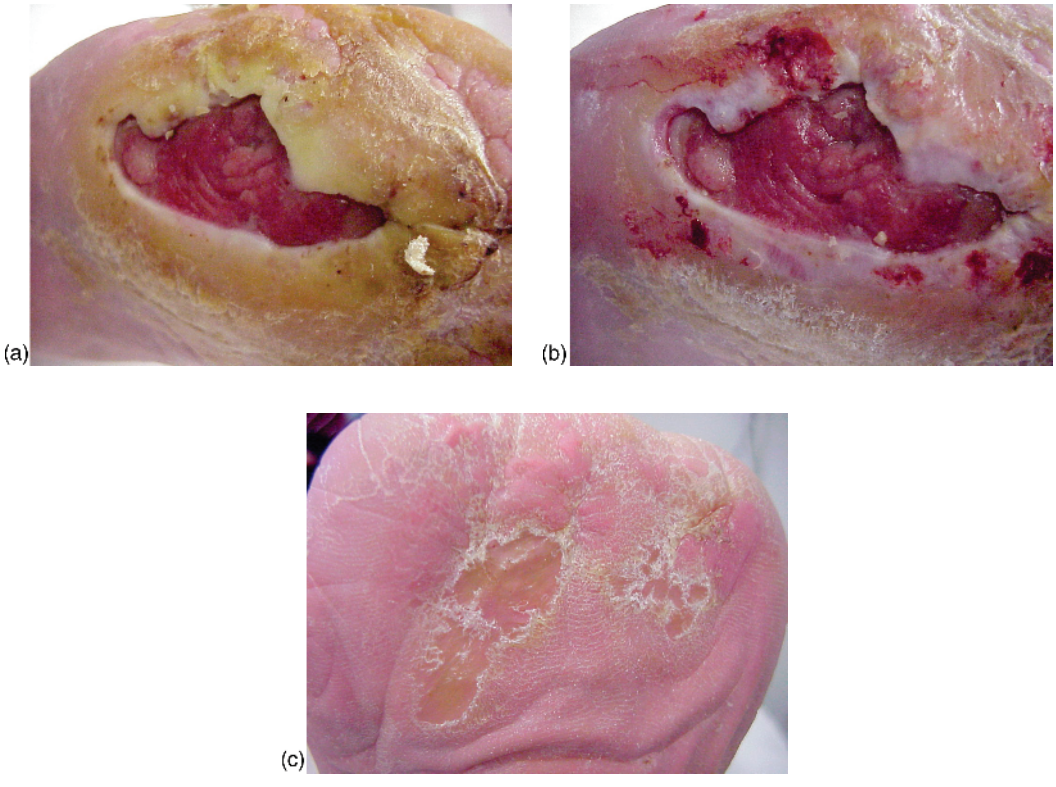


Figure 1.26 (a) Ulcer before debridement. (b) Ulcer after debridement. (c) Healed ulcer.

blood vessel within the wound bed. The leg was elevated and a compression bandage applied and the bleeding stopped. He was later referred to the dermatologists when he developed a cauliflower-like appearance of the skin on the dorsum of his foot. This was biopsied but no malignancy was found, and the dermatologists felt it was associated with long term lymphoedema of the foot.

Both his feet survived, despite recurrent episodes of ulceration and recurrent infections, for many years. He told us that he had weighed in the balance the benefits of having no ulcers against the benefits of leading an active life and doing the things he wanted to do, and he was convinced that whatever the future held he was not prepared to behave like an invalid and restrict his activities. We respected this decision. He eventually died of a stroke at home, having been unhealed for long periods, but always immensely grateful for the regular care that he received from the Diabetic Foot Clinic and the salvage of his feet.

Learning points

- Diabetic patients with neuropathy benefit from long term specialist follow-up care to treat their ulcers.

- It is impossible always to prevent recurrence of ulceration in many patients. The diabetic foot clinic needs to be available to receive and treat them.
- Patients with recurrent ulcers will get recurrent infections, which often need hospital treatment. However, neuropathic patients can often maintain mobility and remain free of major amputation. This patient had a long and successful career until the end of his life.
- A rare cause of haemorrhage in the neuropathic lower limb is a fractured calcified vein. This problem may lead to considerable bleeding. The leg should be elevated and pressure applied.

Case 1.27 Pain in the groin was this patient's warning sign of foot infection

Another lady with indolent neuropathic ulcers, whom we have followed over many years, was determined to live an active life and be independent.

This 40 year old lady with Type 2 diabetes for 5 years was referred to the Diabetic Foot Clinic 23 years ago with a deep infected ulcer – her first foot problem – and was admitted. She was a very house-proud housewife and always immaculately dressed. She had profound neuropathy, with a VPT of more than 50 V, and severe proliferative retinopathy requiring repeated laser therapy. At her first admission she needed a ray amputation for a deep plantar ulcer over the right fourth metatarsophalangeal joint with soft tissue infection and osteomyelitis. She stayed in hospital for three weeks and then attended the Foot Clinic regularly. She developed recurrent ulceration. After the right foot healed, she developed plantar ulceration on the left foot and needed amputation of the left first toe. During a subsequent episode of infection she lost her left fourth toe and had recurrent ulceration over several metatarsal heads. She then underwent resection of the remaining metatarsal heads on the left foot, which healed.

In total she had 20 infective episodes and needed six hospital admissions. She always insisted on removing her dressings at clinic visits herself, and putting them in the refuse bin without them being handled by staff. She also refused to use medical dressings on her feet, preferring to use round eye-makeup-removal pads, and brought a supply to clinic visits for dressing her feet. Her sight was not good enough for her to check her feet, and she was reluctant to ask her husband to do this, saying that she did not want him to see the ulcers in case it upset him. She never allowed him to accompany her to the Clinic but always drove herself there. The warning sign she relied on as a signal that she should come to the Foot Clinic in emergency was pain in the groin from lymphangitis. Despite her difficulties in checking her feet and detecting problems early, she has so far avoided major amputation, although she has frequent plantar ulcers (Figure 1.27) but leads an active life.

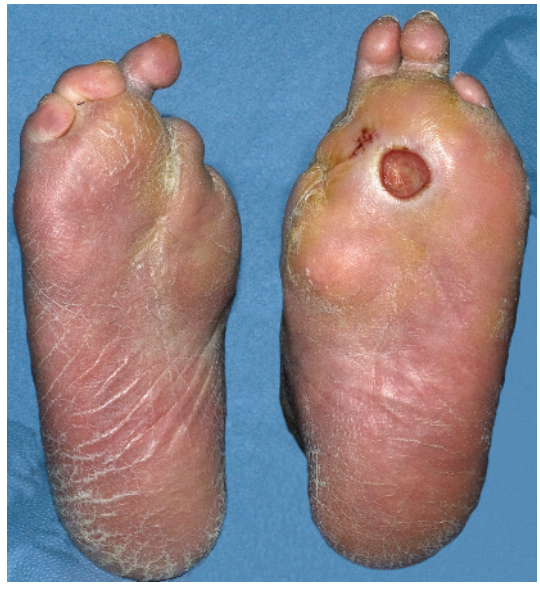


Figure 1.27 Healed minor amputations on both feet and plantar ulceration on the left foot.

Learning points

- Patients with neuropathy are vulnerable and need long term care.
- People with peripheral neuropathy are always susceptible to trauma and then infection, but can usually avoid major amputation if they seek specialist help in time.
- Partially sighted patients with proliferative retinopathy may not be able to see their feet and detect cellulitis early.
- Some patients have unusual ideas about wound care and dressings, which should be accommodated by staff wherever possible, as patients like to feel in control.

Case 1.28 "One damn thing after another"

A 37 year old man with Type 1 diabetes of 23 years' duration had a long history of plantar callus and neuropathic foot ulceration and was among the first patients treated at the King's Diabetic Foot Clinic. Initially he was issued with bespoke shoes with cradled insoles; his ulcers were regularly debrided and he did well until children at the school where he worked as a caretaker mocked him for wearing bespoke shoes and he decided to stop wearing them any more. Over the next three years he lost three lesser toes to septic vasculitis and infection, and this overloaded his plantar forefoot even more. He was very reluctant to take time off work and his in-patient admissions occurred during the school holidays, as during term time, except for routine monthly appointments outside school hours, he did not attend the Clinic. He developed a large deep ulcer with a diameter of more than three centimetres over his right first metatarsal head. This was only healed after a surgical procedure closed it through a racket incision (Figure 1.28a, b), but he then developed an ulcer over the fourth metatarsal head and said that he seemed to get "one damn thing after another".

The ulcer was healed after a period in a total contact cast during the long Summer vacation, but the foot broke down again within three months. The second toe had retracted and drifted laterally, and after a particularly busy time at work loading furniture his third metatarsal head area broke down (Figure 1.28c). He then agreed to wear a pair of bespoke shoes, so long as these were made to look "as normal as possible", and he was given a pair of blue and white trainer-style shoes with toe filler and cradled insole. His ulcers improved but he refused treatment with a total contact cast, saying that the foot would "just break down again" as soon as he came out of the cast. The head teacher at the school became very worried about him and suggested that he might consider taking early retirement. He became anxious about this, and accepted treatment with a living human skin equivalent, Dermagraft, and the ulcers healed within three months (Figure 1.28d).

He was encouraged by this and agreed to attend more frequently so that the callus on his feet did not have time to build up, and agreed with the Foot Clinic staff on a two week interval between treatments. Around this time, his wife began to accompany him to the Clinic. She asked questions about the pathogenesis of foot ulcers and the association between neglected callus and tissue breakdown, and she encouraged him to continue to attend the Foot Clinic more frequently. He had three years without ulceration. He then developed Charcot's osteoarthropathy in his left foot, and would not agree to treatment with a total contact cast until the foot had already developed a rocker bottom deformity. This was because it had been decided at the school that it would be dangerous for him to undertake his work while wearing a cast and he was still reluctant to take sick leave in case it was decided that he was unfit to work. Eventually a large plantar ulcer developed over the rocker bottom deformity (Figure 1.28e) and his wife persuaded him that the time had come to give up work, and he accepted early retirement on health grounds at the age of 54. The ulcer healed in a total contact cast; he continued to attend the clinic very regularly and to seek help at the first sign of any problem, and he did very well for the next seven years, with only brief episodes of skin breakdown, which were reported early and healed quickly. During this time he and his wife enjoyed several holidays abroad. She continued to work.

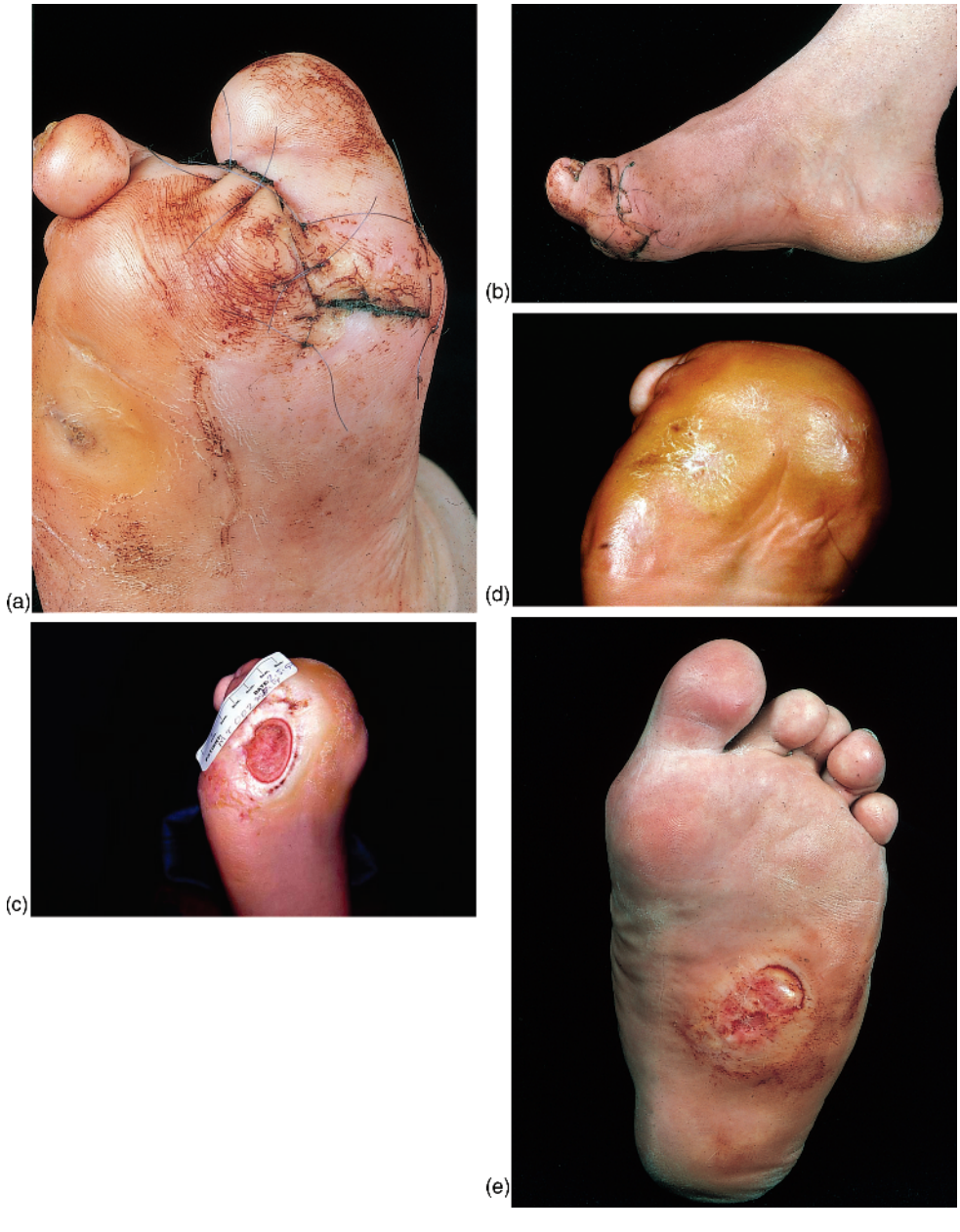


Figure 1.28 (a) Postoperative view of the right foot. (b) Lateral view. (c) Breakdown over the third metatarsal head. (d) Ulcer healed after application of Dermagraft. (e) Ulcer over rocker bottom deformity left foot.

When he was 61 years old, he developed unilateral oedema in his left leg, an abdominal mass was detected, he underwent laparotomy in another hospital and died on the operating table from uncontrollable bleeding.

Learning points

- This case study demonstrates again that, without effective offloading, plantar ulceration is very difficult to prevent and heal.
- Patients may be very sensitive about the appearance of their hospital footwear and every effort should be made to achieve a "normal" look.
- There are many different techniques that can be used to prevent and treat neuropathic ulceration, but the most important may be a change in the patient's lifestyle that enables him to rest the feet. Support from other family members is very useful and they should be encouraged to attend the foot clinic with the patient and to become involved in the treatment.

Case 1.29 Late onset neuropathic ulceration

Another of our original Foot Clinic patients had Type 1 diabetes and was a patient of Dr RD Lawrence.

This patient first presented at the Diabetic Foot Clinic at the age of 60 years with a deep long standing plantar ulcer complicated by osteomyelitis (Stage 4 foot). His Type 1 diabetes was diagnosed during World War II and he was a patient of Dr RD Lawrence at King's. Throughout his diabetic life, the patient's wife used a Lawrence metal template to measure out his portions of bread. This man worked on a building site. He had proliferative retinopathy treated with laser. He was admitted and underwent surgical debridement of the right foot. After three weeks of bed rest, the foot healed. However, he was doomed to almost continuous recurrences of ulceration. His left foot was less involved.

He had several admissions for infected neuropathic ulcers (Stage 4 feet). On one occasion, after his infection was under control, he was treated with Dermagraft and healed. However, the foot broke down again after four months. It was noted at the age of 83 years that his pedal pulses were no longer palpable and as he had further tissue loss he underwent angiography. There was diffuse disease of the superficial femoral artery but no significant narrowing. The anterior tibial artery and peroneal arteries were patent with no significant stenoses. He did not undergo angioplasty and the ulcer improved while he was in hospital.

He was becoming increasingly frail and during the last year of his life he had six admissions to hospital. He was first admitted with a deep necrotic heel ulcer, which grew MRSA, which was treated with teicoplanin 400 mg daily IM. Two months later, he had a recurrence of the heel ulcer, which became sloughy and probed to bone. MRSA was grown from the foot and he went home on teicoplanin 400 mg IM daily. After a further three months, he had an admission for diarrhoea and vomiting, thought to be caused by his antibiotics. The foot grew MRSA and *Enterobacter* species. He was treated with amikacin 7.5 mg/Kg/bd and teicoplanin 400 mg IV daily. Two months later, he developed dysphagia and weight loss. A barium swallow showed multiple tertiary contraction in the entire oesophagus. He also had ultrasound of the kidney and bladder, which showed significant residual volume after micturition, with 351.6 mls pre micturition and 222.5 mls post micturition. His serum creatinine was normal at 89 µmol/l. The heel ulcer was growing *Enterobacter* species and *Klebsiella aerogenes* and he was treated with IV amikacin 7.5 mg/kg bd and meripenem 1 g tds.

His penultimate admission was precipitated by a chest infection. His creatinine had risen to 144 µmol/l, CRP was 67.7 mg/l and a chest X-ray showed that he had volume loss in the right lower lobe with peribronchial thickening, indicating right lower lobe infection possibly secondary to aspiration. He treated with IV meropenem 1 g bd. By the time of discharge, his creatinine had fallen to within normal limits at 108 µmol/l and CRP was 6.2 mg/l.

However, he was back in hospital within a month, for his last admission. He was brought in after a severe hypoglycaemic episode during which he had aspirated. He developed pneumonia. X-ray showed right basal shadowing indicative of infection. His CRP rose to 96.0 mg/l and creatinine to 213 µmol/l. His WBC was 11.16×10^9/l, with neutrophils of 9.16×10^9/l. He was treated with meropenem 1 g bd but died on the ward of right basal

pneumonia, at the age of 84 years. He had lived with Type 1 diabetes for 55 years and members of the Diabetic Foot Clinic were invited to and attended his funeral.

Learning points

- Although this patient had had diabetes for most of his life he was afflicted by neuropathic ulcers for the last 24 years. The diagnosis of his original neuropathic ulcer with cellulitis was delayed and this led to considerable osteomyelitis. Although the infection was treated and resolved, the patient had recurrent episodes of ulceration and sepsis in that foot.
- Indolent neuropathic ulcers can respond to adjunctive treatment such as Dermagraft, which is an artificial dermis which is applied directly onto the ulcer.
- Despite modern diabetic foot care, neuropathic patients often suffer from recurrent foot ulceration. Such patients need ready access to a multi-disciplinary diabetic foot clinic throughout their life.
- We have noted a pattern of increasing fragility in diabetic neuropathic patients as they reach old age, with frequent admissions to hospital from the Diabetic Foot Clinic, heralding their eventual demise. The Diabetic Foot Team have an important role to support patients and carers through this difficult period. Cure may not be possible but relief of symptoms and making patients comfortable should be a priority.

Case 1.30 Long term follow-up needed, at home, at work and on holiday

Patients with neuropathic feet can never relax, and can never afford to forget about their feet. A long term patient, discovered this on holiday.

One of the first patients to be seen in the King's Diabetic Foot Clinic was a 50 year old Afro-Caribbean lady with Type 2 diabetes of 10 years' duration. At a routine annual appointment at the Diabetic Clinic, a research fellow investigating diabetic peripheral neuropathy asked her to remove her shoes so that he could measure her VPT. She had a neuropathic ulcer on the apex of her left hallux, of which she was unaware. The ulcer was surrounded by heavy callus and discharging pus (Stage 4 foot) (Figure 1.30a). The skin on both her feet was very dry with cracking and fissuring around the heels. The VPT was 35 V and the pedal pulses were full and bounding. Dorsal veins were distended. The ankle brachial pressure index was 1.82. The podiatrist debrided callus from around and over the ulcer, advised use of an emollient to the areas of dry skin around the heels and arranged to see the patient every two weeks for further debridement. Negative casts of both feet were taken and the patient subsequently provided with two pairs of shoes with cradled insoles. The patient returned two weeks later, having rested at home with feet elevated in the meantime, and the ulcer was much improved (Figure 1.30b). After a further two weeks the ulcer had healed (Figure 1.30c) and the patient was given her new shoes, and offered monthly appointments at the Diabetic Foot Clinic, which she accepted.

Four years later she went on holiday to the West Indies and walked barefoot on the beach. As soon as she returned to the UK she attended the Foot Clinic as an emergency with an infected left hallux and a healing neuropathic ulcer on the dorsum of the left fourth toe. In view of the associated warmth and oedema and unusually poor diabetic control she was admitted to hospital for bedrest and IV antibiotics. She was discharged after one week with complete resolution of warmth and swelling. The ulcers were fully healed in one month. She was followed for the rest of her life and had a further episode of ulceration aged 65 years when she dropped a dustpan onto her foot and developed an ulcer on the apex of her second toe, which healed in one week. This problem was detected early by the patient, who attended the Diabetic Foot Clinic as soon as she noticed it at her daily foot inspection. She worked as a domestic cleaner until the age of 65 years.

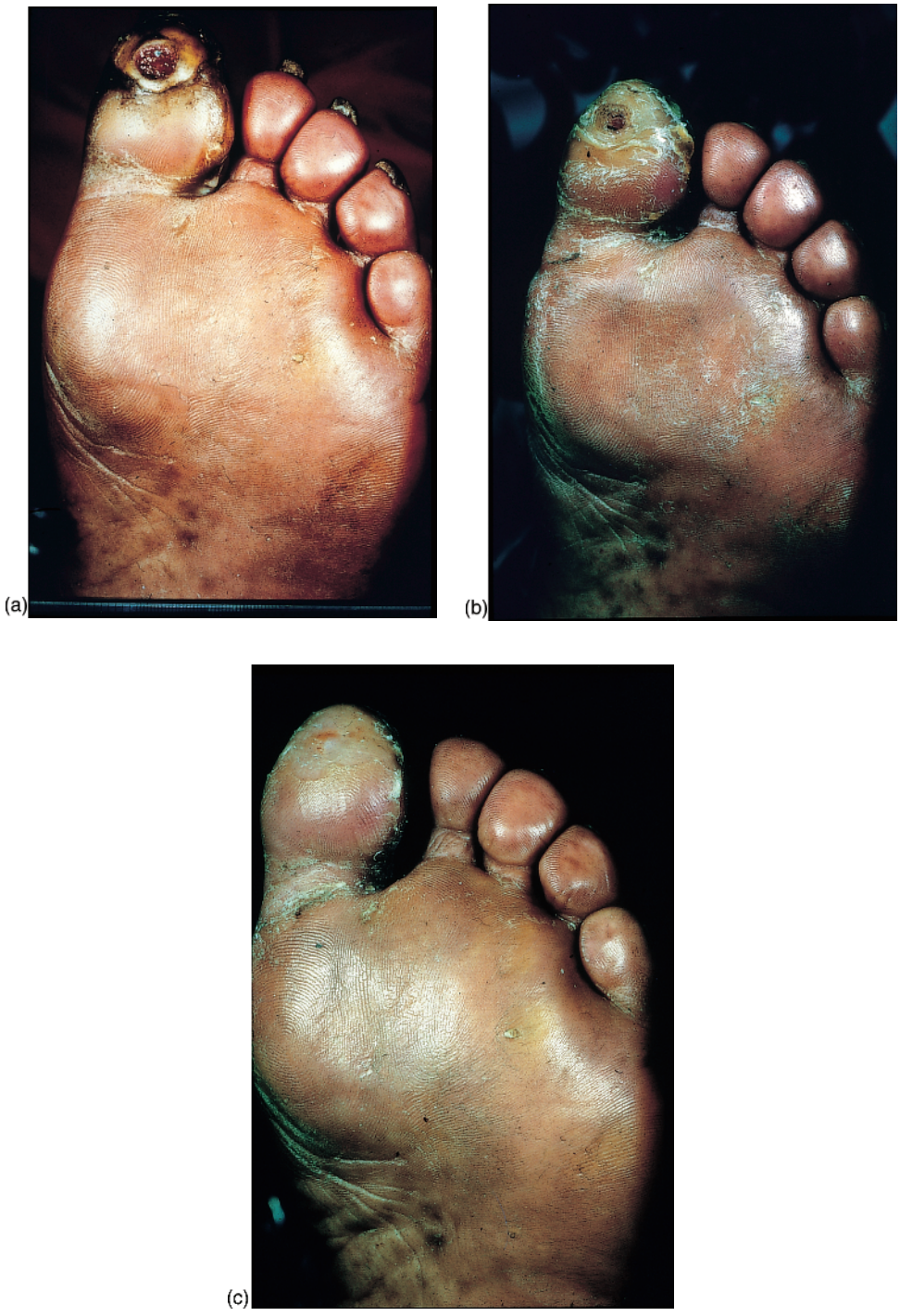


Figure 1.30 (a) Neuropathic ulcer on apex of toe. (b) Healing ulcer. (c) Healed ulcer.

Learning points

- Without inspection of the feet, neuropathic ulcers may not be detected because of lack of protective pain sensation, poor vision and unawareness of the feet.
- Neuropathic ulcers usually heal quickly with regular debridement of callus and rest and offloading and control of infection. Good shoes and insoles can help to prevent relapse.
- A change in environment often puts people with neuropathy or ischaemia at risk of foot problems.
- Patients who go on holiday are particularly vulnerable to trauma. One of the authors went on holiday with seven people, none of whom had diabetes and all of whom developed small foot problems, including blisters, deep fissures, traumatic injuries from wearing sandals, cuts from rocks in the sea and on the beach, and sea urchin spines in the feet. Because none of these people had neuropathy they were all aware of the problem, treated it, and healed without problems. Diabetic people on holiday often find it difficult to obtain specialist treatment locally even if they become aware of a problem.
- Long term outcomes can be very good in patients with a history of neuropathic ulceration so long as they have the services of a diabetic foot clinic and rapid access in emergency. When the neuropathy is profound then it is almost impossible to prevent occasional injuries to the feet, but, although ulcers cannot always be prevented, as in this case, they respond to treatment if caught early.

Case 1.31 Bathroom surgery

Self-treatment is best avoided if the feet are neuropathic. Bathroom surgery to remove callus led to ulceration in this lady.

A 54 year old lady with Type 2 diabetes for two years who worked as a domestic cleaner was referred to the Diabetic Foot Clinic by her general practitioner when she developed an ulcer on the sole of her foot (Stage 3 foot). The patient was aware that she had "a corn" and had been accustomed to shaving it with one of her husband's razors, but desisted when the foot "began to weep" and sought help. She had never received any foot care advice and had never attended the hospital Diabetic Clinic. She had a VPT of 40 V and bounding foot pulses. Her eyes had not been inspected for many years and she needed laser treatment for proliferative retinopathy. Her creatinine was 120 μmol/l and her glycated haemoglobin was 9%. She was referred to the Diabetic Clinic for her diabetic control to be improved.

Her right foot had a plaque of callus over the second metatarsal head with a small sinus connecting with an underlying ulcer (Figure 1.31a). The callus was removed to expose the true dimensions of the ulcer, and the patient was shown a digital photograph of the foot and was very disconcerted by the appearance of the ulcer. She was reassured that healing could be achieved, and was very cooperative with a proposed regime of bespoke shoes with cradled insoles, regular debridement of callus from around the ulcer and antibiotics to control infection. An X-ray showed no problem with the underlying metatarsal head. She was reluctant to give up work, but agreed to rest as much as possible. Her husband had taken early retirement from work and he was very supportive and agreed to undertake all of the domestic chores until her foot was better. She attended the Foot Clinic every week for debridement (Figure 1.31b). The foot healed in fourteen weeks (Figure 1.31c). She did not relapse and is alive and well 17 years later, still wearing bespoke shoes and attending the Foot Clinic regularly, and with good renal function and acceptable glycated haemoglobin levels.

The patient and her husband wanted to go to Majorca in the summer. When they first decided that they would like to do this they asked the Foot Clinic staff whether it would be wise and what special precautions they would need to take. (Since 1990 we have conducted a special "Holiday Footcare Programme" because of the high incidence of foot problems in people with diabetes who go on holiday. Many injuries are caused by the strange and unfamiliar holiday environment, extremes of climate, and burns and cuts from walking barefoot on beaches and in the sea. Moreover, some patients become so "light-hearted" when on holiday that their usual protective footcare regimes are relaxed and injuries are common.) She was warned to wear plastic sandals on the beach and in the sea, not to walk barefoot in the hotel room, to avoid sitting in hot sunshine, and not to discard her hospital shoes. She was advised to telephone the Diabetic Foot Clinic if any problems arose and to take a small first aid kit with her on holiday, containing antiseptic, dressings, bandages and tape. The holiday was very successful and the patient and her husband decided to return to Majorca the following winter for three months. She was then given the contact details of a podiatrist who practiced in Majorca, so that she could obtain regular preventive foot care, reduction of callosities and cutting of nails, and also

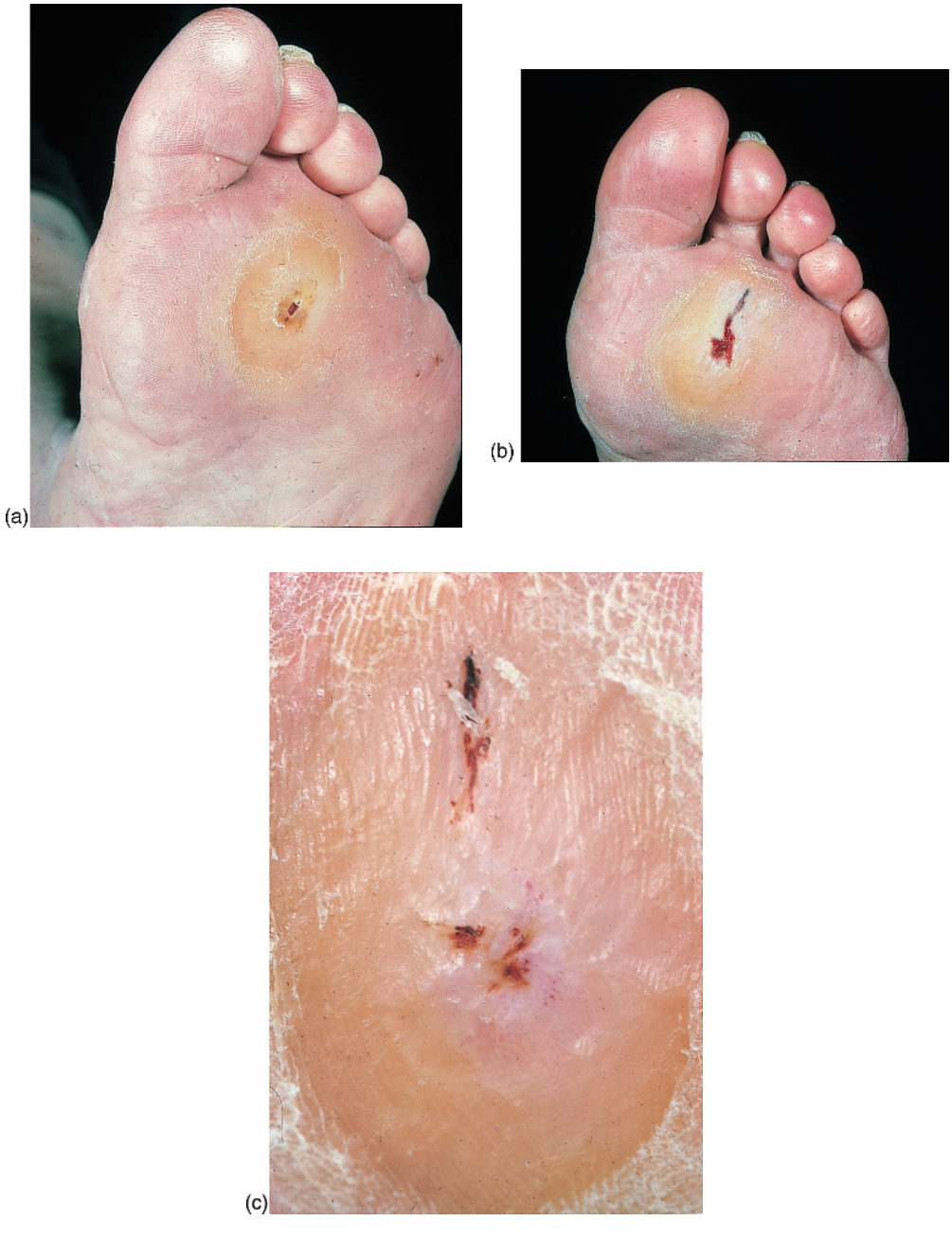


Figure 1.31 (a) Ulcer with callus. (b) The ulcer has been debrided. (c) Healed ulcer.

obtain local help if needed in emergency. The longer trip did not cause problems and now they always winter abroad.

Learning points

- In addition to looking at the feet, the Foot Clinic needs to ensure that good metabolic control is achieved.
- The support of the patient's husband helped her to continue working while achieving healing of the ulcer.
- Long term outcomes can be very good in patients who take their feet seriously and follow advice.
- Holidays can be hazardous for high risk diabetic patients. They should be carefully forewarned of possible dangers and told how to obtain help if problems arise.
- Many UK trained podiatrists work abroad and can see patients privately, if needed. The UK Society of Chiropodists and Podiatrists maintains a register of these practitioners.

Case 1.32 Contact dermatitis

A 76 year old widowed lady with Type 2 diabetes of 12 years' duration lived alone, but spent part of the year staying with her married daughter (who also had Type 2 diabetes) and son-in-law and their two children. She was very overweight with a body mass index of 32, and suffered from hypertension. Her diabetes was managed by the general practitioner and the practice nurse. She was determined to maintain her independence and never to become a burden to her family. She was a very cheerful and happy lady, who always chatted to staff and other patients about her grandchildren, and brought in home-made cakes as gifts. She believed that her diabetes was mild and "not really a problem. I just need to watch what I eat". Her diabetes was diet controlled.

She first came to the Diabetic Foot Clinic because while shopping one morning she knocked the front of her ankle on a supermarket trolley. She felt no pain at the time of the injury, but another shopper saw that the leg was bleeding and pointed it out to her, and a first aider at the shop cleaned the laceration and applied a dressing. When the patient returned home she applied further first aid. She filled a plastic bowl with warm water, mixed in half a bottle of Savlon antiseptic and soaked the foot and ankle for half an hour. The next morning the entire foot and lower leg were red and weeping. She applied gauze and a bandage and visited her general practitioner, who referred her to King's. Her VPT was 35 V and pedal pulses were palpable. The skin of her left foot and ankle was red, moist and weeping, with numerous small bullae (Stage 2 foot). She told us that she had tested the temperature of the water in the soaking bowl with her elbow and was sure that it was not hot enough to burn her foot. She was seen by the dermatologists, who felt that the problem was a contact dermatitis, due to an allergic reaction to the strong solution of Savlon (Figure 1.32).

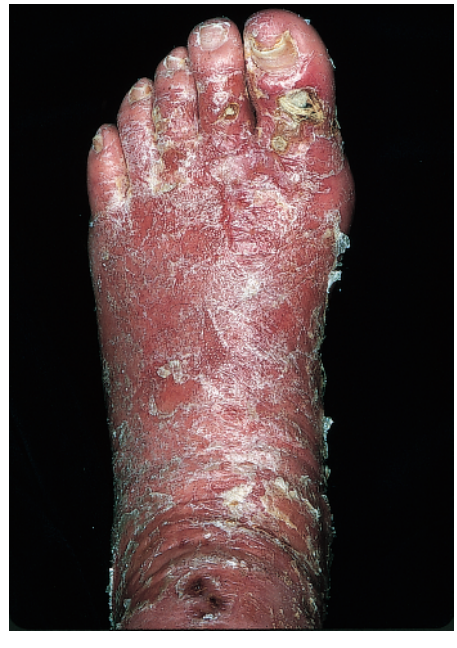


Figure 1.32 Contact dermatitis.

She was a advised to apply calamine lotion daily to the red areas. A district nurse called on her every day to check the foot and re-dress the laceration. Within a week the foot was dry and desquamating, and she made a good recovery.

In view of her neuropathy she was offered foot care and attended the Foot Clinic regularly. She then began to find the journey to and from the hospital difficult and did not want ambulance transport or a hospital car to take her, so treatment was sought from the community podiatry service and a podiatrist called at her home every three months. This was also arranged for when she was staying with her daughter.

Learning points

- All people with diabetes should be advised to seek professional help if they injure their feet.
- First aid for superficial injuries should be confined to cleaning, dressing and covering the wound with a dry dressing held in place by a loose bandage.
- The Diabetic Foot Clinic is not only a clinic but also a club, in which patients and staff form strong bonds of mutual comfort and support through good times and difficult times.
- Antiseptics should be carefully diluted in accordance with the instructions on the bottle.

Case 1.33 Colour change in the Afro-Caribbean foot

A 66 year old Afro-Caribbean woman with Type 2 diabetes of 22 years' duration was referred to the Diabetic Foot Clinic with swelling and discolouration of her left fourth toe (Stage 4 foot) (Figure 1.33). She had neuropathy with VPT of 29 V. Pedal pulses were palpable. She said that she had never received any help with her feet and cut her own toenails. She was awaiting operation for bilateral cataracts and her vision was poor. She was aware that she might have cut the flesh of the toe the previous week because she had seen a spot of blood on her white socks when she washed them, but could not see any problem with the foot until several days later, when she thought the toe was swollen and visited her general practitioner and was sent to the hospital. The medial sulcus of the toe was very swollen, with a splinter of nail penetrating the sulcus. Pus was drained and sent for culture, and she was given amoxicillin 500 mg tds and flucloxacillin 500 mg qds orally. *Staphylococcus aureus* was grown. The nail splinter was removed together with a small wedge removed from the side of the nail so that it would not press on the sulcus. The toe was cleansed with Hibitane in spirit and a dry dressing was applied. The district nurses were asked to visit her every day to check and redress the foot. At review after one week the toe was healed.

She was offered regular foot care, which she accepted, and attended the Foot Clinic for many years. Because her feet were not deformed and she had no callus or history of

Figure 1.33 Infected toe.

ulceration it was felt that bespoke shoes were not needed. However, she received careful footwear education and was advised to wear lace up shoes with low heels for everyday and for around the house, and agreed to do this. She lived with her husband and an unmarried daughter, both of whom also had diabetes and attended the Foot Clinic and were very supportive. Despite stalwart attempts to control her diabetes as well as possible, her glycated haemoglobin levels gradually rose, and despite high doses of oral hypoglycaemic agents it was not possible to achieve optimal control and she was started on insulin.

She developed a plantar wart on her right heel at the age of 73 years and needed reassurance that treatment was not necessary so long as the lesion was painless and not spreading, and after two years it resolved spontaneously. Her skin was dry and she was encouraged to use an emollient and liked cocoa butter, which her daughter also used. She died at the age of 83 years of a stroke.

Learning points

- Colour change in the Afro-Caribbean foot with infection may be subtle but careful inspection will usually detect it.
- Patients with neuropathy should not be expected to cut their own nails.
- A frequent problem is caused when the patient cuts the flesh of the toe instead of the nail, or does not cut the nail completely, leaving behind a splinter of nail that penetrates the sulcus as the nail grows forward.
- Light coloured socks can often reveal a foot problem because blood or exudate forms a discoloured patch.
- Patients with new foot discolouration should be seen as an emergency on the same day the problem is first noted.
- High risk patients benefit from long term follow-up in a specialist centre.

Case 1.34 Sudden death

When patients with neuropathy appear young and healthy, active and cheerful, it is easy to forget how serious their problems are. The following patient appeared fit and well, remained in full-time employment, and then died very suddenly, without warning and without prior complaint of any problem.

A 46 year old man with Type 1 diabetes of 30 years' duration was a very early patient of the Diabetic Foot Clinic. He was referred to King's by his practice nurse, who reported that he had a blue right second toe and a neuropathic ulcer over the right second metatarsal head. He was not a local patient, coming from rural Kent, and all his previous care had been at the general practitioner's clinic, with annual visits to the diabetic department of his nearest district general hospital. He was profoundly neuropathic, with a VPT of over 50 V. His pedal pulses were full and bounding, the dorsal veins on both feet were distended and the plantar skin was dry and flaking. The neuropathic ulcer was deep to bone and the forefoot was cellulitic (Stage 4 foot). He denied ever previously receiving any foot care, or education relating to the feet, and had been unconcerned about "the hole in my foot" because "it did not hurt so it could not be anything serious, could it?". He had proliferative diabetic retinopathy, which had received laser therapy in his local hospital, and his vision was poor. When the redness and swelling of the forefoot was pointed out to him he said that he could not really see any difference between the two feet and had only been aware of the ulcer because his sock was always wet.

A diagnosis of spreading infection in the neuropathic foot was made and it was thought that septic vasculitis had led to occlusion of the digital vessels of the blue toe with septic thrombus. (At that time many practitioners believed that diabetic patients suffered from "small vessel disease", leading to the spontaneous development of black toes in feet with bounding pedal pulses, and it was often thought that nothing could be done for the patient apart from major amputation, which was an alarmingly common practice even in patients who were purely neuropathic with no macrovascular disease of the leg arteries.) He was admitted the same day and started on IV amoxicillin 500 mg tds, flucloxacillin 500 mg qds and metronidazole 500 mg tds and underwent a ray amputation. Tissue culture grew *Staphylococcus aureus* and Group B *Streptococcus*. The orthopaedic surgeon applied strips of sterile Vaseline-impregnated gauze around the foot to hold it together as much as possible but was careful not to prevent free drainage. It was his practice to cut through the strips so that if there was severe post-operative swelling they would not form a constricting band around the foot. Histology of the digital artery demonstrated that the arterial wall was invaded by white cells and the arterial lumen occluded by septic thrombus, thus confirming the suspicions of septic vasculitis as a cause of digital necrosis in the neuropathic diabetic foot. The foot was irrigated with Milton (2% sodium hypochlorite solution) four times a day, with great care being taken to wash the Milton solution off the surrounding skin and to apply emollient cream. A healthy glistening bed of granulation tissue formed within a week.

The patient was discharged, with crutches and a wheelchair, after three weeks, and followed as an outpatient in the Foot Clinic, but during his time on the ward he was carefully educated. When his wife visited him on the ward she was asked to come down to

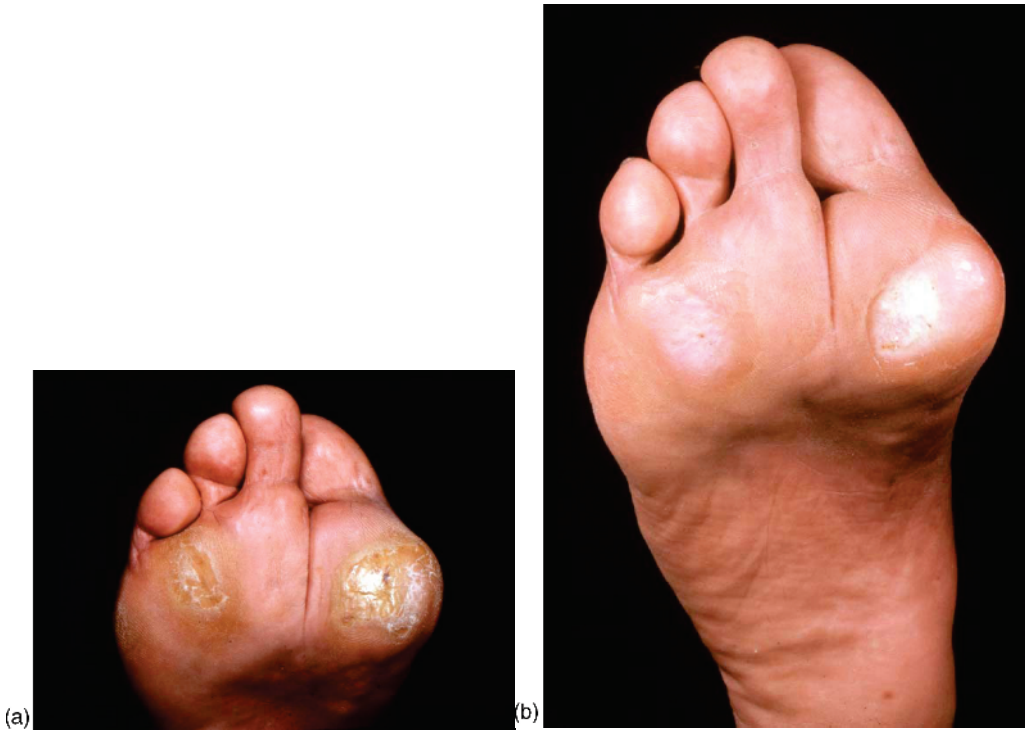


Figure 1.34 (a) Callus before debridement. (b) Callus removed by debridement.

the Foot Clinic with him, and we explained that due to his poor eyesight his feet were at risk and taught her how to perform a daily foot check, and she said that she would bring him to the clinic immediately if there was any change in the feet. She soon noted that he was developing heavy callus over the first and fourth metatarsal heads adjoining the ray amputation site (Stage 2 foot) and brought him to the Foot Clinic, where he underwent podiatric debridement of this callus (Figure 1.34a, b) to reduce the plantar pressures and prevent neuropathic ulceration. He was also reviewed by the orthotist at the next joint clinic and two pairs of bespoke shoes with cradled insoles were issued. His wife reported that he had found it very difficult to use the crutches to achieve complete off-loading because he frequently lost his balance and had to put his foot to the ground. She purchased a light-weight, folding wheel chair for him so that she was able to take him out and he did not become lonely, depressed and isolated. The foot healed quickly.

He had no further foot problems but died suddenly at work three years later. His wife came to visit the Diabetic Foot Clinic after his death and presented his wheel chair for the department's use

Learning points

- Infection of an ulceration over the metatarsal head can spread to the digital arteries of the adjoining toe, leading to septic vasculitis, occlusion of the arterial lumen by septic

thrombus, and a black toe. If this is caught early and treated with antibiotics the process can be reversed: if the patient presents late, as in this case, loss of the toe is inevitable.

- After a ray has been removed, overloading of the adjoining metatarsal area is common. Regular removal of callus and offloading with an insole can reduce plantar pressures and prevent ulceration.
- Sudden death sometimes occurs in young neuropathic patients with no warning and is usually due to "silent" cardiac ischaemia.

Case 1.35 A cardiac arrest in the Diabetic Foot Clinic

Among our patients we have encountered sudden deaths both at home and in the hospital – and a "near miss" within the hallowed portals of the Diabetic Foot Clinic.

A 76 year old lady with Type 2 diabetes of 12 years' duration and known coronary vessel disease treated by coronary angioplasty at age 74 developed a sore on the end of her left second toe after she injured the flesh when cutting her nails. She visited her general practitioner when the toe became pink, and he arranged for her to see the practice nurse every week for dressings. She attended every week for four months, after which the toe became swollen and red. He then referred her to the Diabetic Foot Clinic. She had neuropathy, with VPT of 37 V. The pedal pulses were palpable. The toe was clearly infected with red fusiform swelling typical of osteomyelitis (Stage 4 foot): the so-called sausage toe (Figure 1.35). A small ulcer on the dorsum of the toe probed to bone, and an X-ray showed lucencies and erosions typical of osteomyelitis. She was reviewed by the orthopaedic surgeons but was very anxious that the toe should not be amputated if there was the slightest possibility of saving it and a trial of conservative care was agreed. A swab was taken from the ulcer, which grew *Staphylococcus aureus*. She was prescribed oral antibiotics with good bony penetration, namely fusidic acid 500 mg tds and doxycycline 100 mg daily, and these were continued for eight weeks, following which the ulcer had healed and the bony changes had improved on X-ray.

She was followed in the Diabetic Foot Clinic for the next two years. She was then admitted to hospital suffering from congestive cardiac failure. She improved and was sent down from the ward for a routine Foot Clinic appointment to have her nails cut and filed. After this procedure she was sitting in her wheelchair in the waiting room when the receptionist, who always keeps a close eye on patients, saw her head fall forward suddenly. The patient had lost consciousness and a podiatrist was called, who could not palpate her pulse. The patient was laid on the floor and cardio-pulmonary

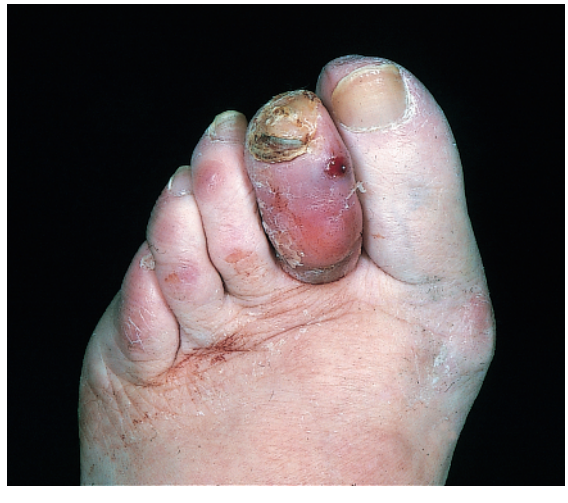


Figure 1.35 Sausage toe.

resuscitation (CPR) was commenced while the cardiac arrest team was bleeped. They arrived within two minutes and the patient was successfully resuscitated and moved to the Coronary Care Unit. She appeared to make a good recovery and was returned to the ward: however, two weeks later she had another cardiac arrest on the ward and could not be resuscitated.

Learning points

- Patients with breaks in the skin should be referred quickly for specialist care. Osteomyelitis can usually be prevented if infections are caught early and treated aggressively. This patient was kept under community care for too long.
- Osteomyelitis is the likely diagnosis in patients with "sausage toe", an ulcer which probes to bone, or when obvious bony changes are evident on X-ray.
- Osteomyelitis may respond to conservative care and amputation is not always necessary. Amputation of the toe is not always required in patients presenting with sausage toe. Management decisions are based on the clinical appearance, X-rays and the patient's wishes. Sometimes the response to antibiotics is very good and the ulcer heals, with complete resolution of the bone infection.
- When treating osteomyelitis conservatively, antibiotics with good bony penetration should be chosen.
- Diabetic foot patients are very frail. Many have multi-system disease. Our reception staff are instructed to keep a very close eye on patients in the waiting area. They get to know the patients well over the years, and are frequently the first to notice when a patient's behaviour is abnormal because of hypoglycaemia or when a patient is unusually pale or quiet.
- Patients should be closely observed at all times when they are at the Foot Clinic.
- All Diabetic Foot Clinics should have staff who are trained in cardio-pulmonary resuscitation (CPR) and a cardiac arrest trolley should be readily available. There should be a formal clinic policy relating to clinical emergencies so that when these arise all members of staff know what their roles are, i.e. who should call the cardiac arrest team and who should commence CPR.

Case 1.36 From neuropathic foot to ischaemic foot

We have included the last patient because he first presented as a pure neuropathic patient and subsequently developed peripheral vascular disease. The diabetic foot is a moving target.

This was a 30 year old patient with Type 1 diabetes for 16 years. He had bilateral proliferative retinopathy, bilateral cataracts and a previous retinal detachment, and his sight deteriorated so that he could not get a driving license. He was treated with bilateral photocoagulation. He attended the Diabetic Foot Clinic for many years with episodes of neuropathic ulceration and then attended in emergency, complaining that he had developed a small ulcer on the lateral border of his right fifth toe where he had never previously had an ulcer (Stage 3 foot). The ulcer had no surrounding callus and was in a "typically ischaemic" site, on the margin of the foot. His pedal pulses had become weak on the left and were impalpable on the right. He was admitted and underwent angiography. On the right there was a short segment occlusion at the level of the mid superficial femoral artery and a further short segment stenosis at the level of the right adductor canal; this narrowing and occlusion was angioplastied with a 5 mm balloon, and his ulcer healed in four weeks and did not recur.

Eighteen months later he complained of pain in his left calf when walking. He had angiography, which revealed disease of the left superficial femoral artery and a tight stenosis at the origin of the profunda artery. He underwent successful angioplasty and his claudication was relieved. Two years later he had a focal mid left superficial femoral artery stenosis of 70% and had another angioplasty. He continued to have episodes of ischaemic ulceration associated with infection for many years, but the ulcers quickly resolved. He died suddenly at home of a stroke ten years after his peripheral vascular disease was first diagnosed.

Learning points

- Diabetic patients with neuropathic feet can do very well, but only if they look after their feet well, seek help early, and receive multidisciplinary care when necessary.
- Neuropathic patients who are followed up may eventually become ischaemic. This process proceeds silently and may not manifest itself until the patient has developed ischaemic ulceration or gangrene.
- The burden of the neuropathic foot is great, both for patients and for health care professionals, but it becomes even greater when the neuropathic foot is complicated by ischaemia.
- Practitioners must be ever alert to the development of other diabetic complications apart from neuropathy.