Diabetic foot disease

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Abstract

Diabetic foot disease, or ulceration, is prevalent and is associated with high rates of lower limb amputation and mortality. Its underlying aetiology is complex and multifactorial. However, neuropathy and peripheral arterial disease represent two important precipitating risk factors. Regular, comprehensive foot examinations are important in the prevention of ulceration and cardiovascular complications as they provide an opportunity to assess risk, modify risk factors and deliver patient education. Charcot neuropathic osteoarthropathy is commonly misdiagnosed and should always be suspected in an individual with diabetes presenting with a hot and swollen foot. Diabetic foot ulcers are challenging to manage. The key to optimizing outcomes includes early diagnosis with referral for coordinated multidisciplinary care where prompt treatment of infection and peripheral arterial disease, as well as appropriate wound care and offloading can be initiated and monitored.

Keywords Amputation; bypass; debridement; diabetic foot; endovascular; peripheral arterial disease; revascularization; sepsis; wound management

Introduction

Diabetic foot disease, or ulceration (DFU), is a common and devastating complication of diabetes. It is associated with a poor quality-of-life due to the resulting reduction in mobility, frequent outpatient visits and prolonged hospital admissions. DFU is also associated with very high rates of lower limb amputation and mortality and is costly for health services to manage. It is estimated that up to a quarter of patients with diabetes have a lifetime risk of developing DFU. DFU is also associated with 5-year amputation and mortality rates of 10% and 40%, respectively. Unfortunately, even if healing is achieved, 40% of patients have a recurrence within 1 year. These sobering statistics highlight the need for attentive care and timely management to prevent diabetic foot complications. It is estimated that 85% of amputations are preceded by DFU and therefore can be prevented with improvements in ulcer care.

Aetiology

The aetiology of DFU is a complex and multifactorial process (Figure 1). The most important risk factors include diabetic peripheral neuropathy and peripheral arterial disease (PAD). With the high prevalence of PAD in Western populations,[1] there has been a shift from neuropathic to 'neuro-ischaemic' ulceration with concurrent neuropathy and PAD. Other important risk factors include patient-related factors (e.g. age and comorbidities such as renal disease and peripheral oedema), diabetes (e.g. duration of diabetes and glycaemic control), biomechanical abnormalities (e.g. foot deformities), infection and trauma (e.g. inappropriate footwear).

Somatic and autonomic diabetic neuropathy are progressive and can result in an insensate, dry (anhidrotic) and deformed foot which is at high risk of DFU.[2,3] Somatic neuropathy results in sensory loss, placing the patient at high risk of unperceived trauma. It may also affect motor nerves, leading to intrinsic foot muscle atrophy, limited ankle joint mobility and subsequent foot deformities (e.g. hallux valgus and claw toes) with elevated plantar pressure load. The metatarsal and heel areas are particularly vulnerable to this pathological foot load. Over time, callus (hyperkeratosis) can develop which can ultimately break down and result in ulceration. Autonomic dysfunction results in reduced sweating leaving the skin dry, fragile and predisposed to cracking and fissure formation. Autonomic neuropathy can also result in impaired microvascular regulation and foot perfusion.

Peripheral arterial disease is a chronic atherosclerotic condition that is estimated to be present in up to half of patients presenting with DFU. The abnormal metabolic state in diabetes results in accelerated atherosclerosis. In the context of DFU, PAD is associated with worse healing, higher amputation rates and higher mortality. Patients with PAD and DFU have an almost twofold increase in risk of foot infection and are ninety times more likely to undergo an amputation once infected compared to non-ischaemic, non-infected wounds.

Diabetic foot assessment

Clinical history

A detailed clinical history should be obtained from every patient presenting with DFU.[4] This should include questions regarding the patient's pre-existing conditions (e.g. renal failure, coronary heart disease and congestive cardiac failure), previous arterial intervention, previous amputation history, time since diagnosis of diabetes, glycaemic control, peripheral neuropathy, duration of ulceration, changes in the appearance of the ulcer and possible precipitating factors such as trauma or new footwear. Symptoms of systemic infection (e.g. general lethargy, fevers and rigors) should also be considered. Risk factors for DFU such as a history of neuropathy, symptoms of PAD (intermittent claudication or rest pain) or foot deformities should also be documented. However, it is important to note that the clinical presentation of PAD may be absent, subtle or atypical (e.g. leg fatigue or slow walking speed rather than pain) due to impaired sensory feedback and propensity to suffer from more diffuse or distal atherosclerotic disease.

Physical examination

Essential components of the physical examination include an assessment of the wound, and the presence of potentially pre-ulcerative lesions (e.g. cracks, fissures, fungal infection, deformed nails, macerated web spaces) and foot deformities, neuropathy and PAD.

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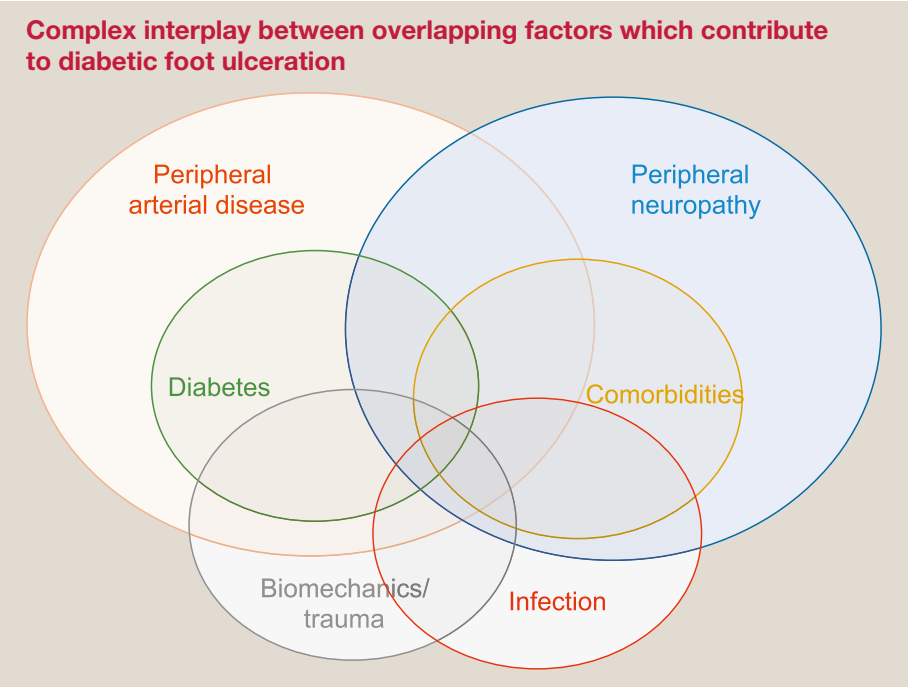

Figure 1 Complex interplay between overlapping factors which contribute to diabetic foot ulceration

Wound assessment should include a description of the number of ulcers, their location, size, depth, wound bed, margins, presence of deeper structures (e.g. tendon, bone, joint capsule) and any signs of infection. Ulcers have different characteristics depending on the underlying aetiology (Table 1). The depth of tissue can be evaluated using a sterile probe. Direct palpation of bone at the wound base raises concerns over the possible presence of osteomyelitis. Particular attention should be paid to detecting ulcers that may be hidden in the interdigital spaces or under callused skin.

Testing for neuropathy typically involves testing for sensation using a 10 g monofilament applied to ten testing sites on the foot (Figure 2). This may be used in combination with testing of vibration (using a 128 Hz tuning fork) and proprioception at the first metatarsophalangeal joint.

Vascular examination should include an assessment of peripheral pulses. However, it is important to recognize that the diagnosis of PAD can be challenging, and clinical examination and results of bedside tests can be confounded by the presence of calcified and incompressible vessels. Although there are a number of non-invasive PAD bedside tests available (e.g. ankle-brachial pressure index (ABPI), toe-brachial pressure index (TBPI), transcutaneous pressure of oxygen (TcPO2)), it has been demonstrated that visually displayed Doppler arterial waveforms at the ankle may be the best test for excluding PAD in patients with diabetes.[5] A haemodynamically significant arterial lesion

Characteristics of ulceration

Ulcer aetiology	Edge	Site	Surrounding skin
Neuropathic	Punched out	Pressure areas (vary with position_- Recumbent position - posterior and posterolateral heel. Weight-bearing position - plantar heel, metatarsal heads, digits and malleoli	Warm, well perfused
Arterial			Cool, associated with absent pulses and trophic changes (thin, shiny skin, lacking hair, poor quality nails)
Pressure	Undermined		Loss of subcutaneous fat
Venous	Sloped	Medial > lateral gaiter regions	Venous eczema, haemosiderin deposition, lipodermatosclerosis, oedema, visible veins, atrophy blanche
If do not conform to typical characteristics as above, consider mixed aetiologies, malignant and dermatological causes.			

Table 1

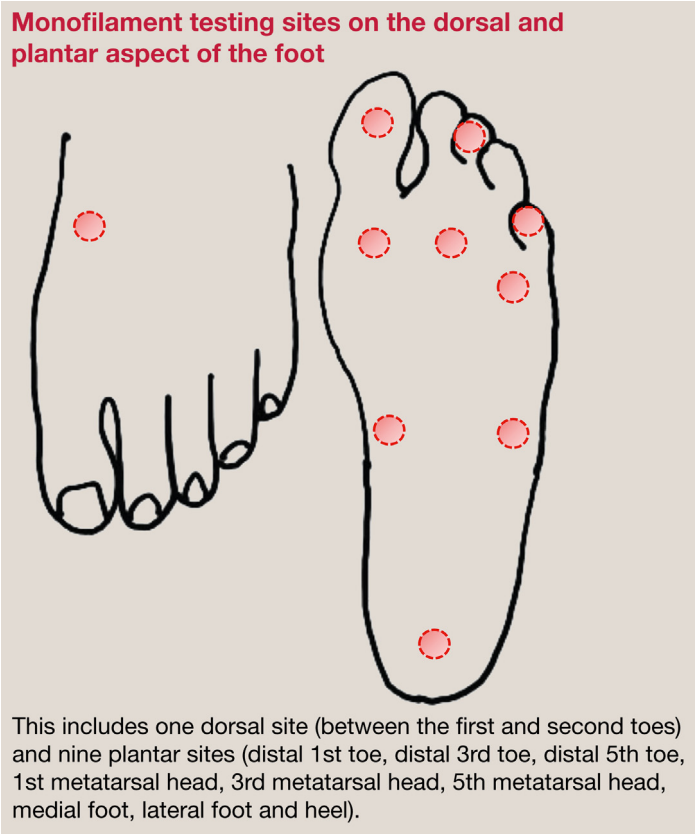

Figure 2

results in a change in the downstream waveform morphology. Regardless, ABPI and TBPI should be measured as they are likely to be important in risk stratification.

Diabetic foot ulcer clinical classification systems

Over recent years, a number of diabetic foot ulcer classification schemes have been developed and validated for risk stratification purposes and to enable better communication between health professionals. These include the Wagner, University of Texas, PEDIS, SINBAD and WIfI classification systems. The International Working Group on the Diabetic Foot (IWGDF) recommends the use of the SINBAD system for communication among health professionals and the WIfI system for assessment of perfusion and the likely benefit of revascularization.[6] These two important classification systems have been summarized in Table 2.

When examining a hot and swollen foot in an individual with diabetes, it is important to always consider a differential diagnosis of Charcot neuropathic osteoarthropathy ('Charcot foot').[7] In early stages this presents as a localized inflammatory condition of the bones, joints and soft tissue and can be easily misdiagnosed as cellulitis or gout. If a prompt diagnosis is made, further progressive bone destruction and deformity can be averted with offloading and immobilization. The classic deformity associated with later stages of this condition, is midfoot collapse ('rocker-bottom foot') which creates high midfoot plantar pressures and subsequent DFU (Figure 3).

Laboratory investigations

A full blood count, C-reactive protein, renal profile and glycated haemoglobin (HbA1c) can be informative. A raised neutrophil count and CRP may suggest the presence of infection. Similarly, procalcitonin level can be helpful in determining the presence and severity of a bacterial foot infection as well as monitoring the effectiveness of treatment over time. Anaemia can impact on the potential for wound healing and should be corrected. Baseline renal profile is necessary prior to any further contrast imaging if indicated. HbA1c is an important marker of overall diabetes control. However, in the context of anaemia an acceptable HbA1c may be falsely reassuring with regards to glycaemic control.

Radiological investigations

X-rays can be helpful in identifying bone deformities (e.g. fractures or subluxations), foreign bodies, osteomyelitis or subcutaneous gas (Figure 3c) in the soft tissue if an infection is suspected. Further imaging such as magnetic resonance imaging (MRI) may be necessary to evaluate the presence of osteomyelitis or a deeper collection, while ultrasound may also be helpful with regards to the latter. Computed tomography (CT) of the foot can be used to plan reconstructive foot surgery for example correction of deformity following Charcot foot.

If PAD is suspected in a patient with active DFU then early arterial anatomical imaging is required to confirm the diagnosis and plan revascularization. First-line anatomical imaging is most commonly a non-invasive full lower limb arterial duplex

Description of the SINBAD and WIfI classification systems

SINBAD			WIfI				
Category	**Definition**	**Score**	**Category**	**Definition**			**Score**
Site	Forefoot	0	Wound	No ulcer (ischaemia rest pain)			0
	Midfoot/hindfoot	1		Small, shallow ulcer on distal leg or foot without gangrene			1
				Deeper ulcer with exposed bone, joint or tendon, ± gangrenous changes limited to toes			2
				Extensive deep ulcer, full thickness heel ulcer ± calcaneal involvement ± extensive gangrene			3
Ischaemia	Pedal blood flow intact, at least one pulse palpable	0	Ischaemia	ABPI	Ankle pressure	Toe pressure or TcPO2	
	Clinical evidence of reduce blood flow	1		≥0.80	>100	≥60	0
				0.60–0.79	70–100	40–59	2
				0.40–0.59	50–70	30–39	2
				<0.40	<50	<30	3
Neuropathy	Protective sensation intact	0	Foot infection	No symptoms/signs of infection			0
	Protective sensation lost	1		Infection defined by the presence of at least 2 of the following: • Local swelling or induration • Erythema (>0.5 to ≤2cm around the ulcer) • Local tenderness or pain • Local warmth • Purulent discharge			
Bacterial infection	None	0					
	Present	1					
Area	Ulcer ≤ 1cm	0					
	Ulcer > 1cm	1					
Depth	Ulcer confined to skin and subcutaneous tissue	0		Local infection (as defined above) involving only skin and subcutaneous tissue			1
	Ulcer reaching muscle, tendon or deeper	1		Local infection (as defined above) with erythema >2cm, or involving structures deeper than skin/subcutaneous tissue			2
				Local infection (as described above) with the signs of SIRS, as defined by 2 or more of the following: Temperature> 38°C or <36C° Heart rate >90 beats/min Respiratory rate >20 breaths/min or $PaCO_2$ <32 mmHg White blood cell count >12,000 or <4000 cu/mm or 10% immature (band) forms			3
Reference	Ince P, Abbas ZG, Lutale JK, Basit A, Ali SM, Chohan F, Morbach S, Möllenberg J, Game FL, Jeffcoate WJ. Use of the SINBAD classification system and score in comparing outcome of foot ulcer management on three continents. Diabetes Care. 2008 May;31(5):964-7.		Reference	Mills JL Sr, Conte MS, Armstrong DG, Pomposelli FB, Schanzer A, Sidawy AN, Andros G; Society for Vascular Surgery Lower Extremity Guidelines Committee. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: risk stratification based on wound, ischemia, and foot infection (WIfI). J Vasc Surg. 2014 Jan;59(1):220-34.e1-2.			

ABPI, ankle-brachial pressure index; $PaCO_2$, partial pressure of arterial carbon dioxide; SIRS, systemic inflammatory response syndrome; $TcPO_2$, transcutaneous pressure of oxygen.

Table 2

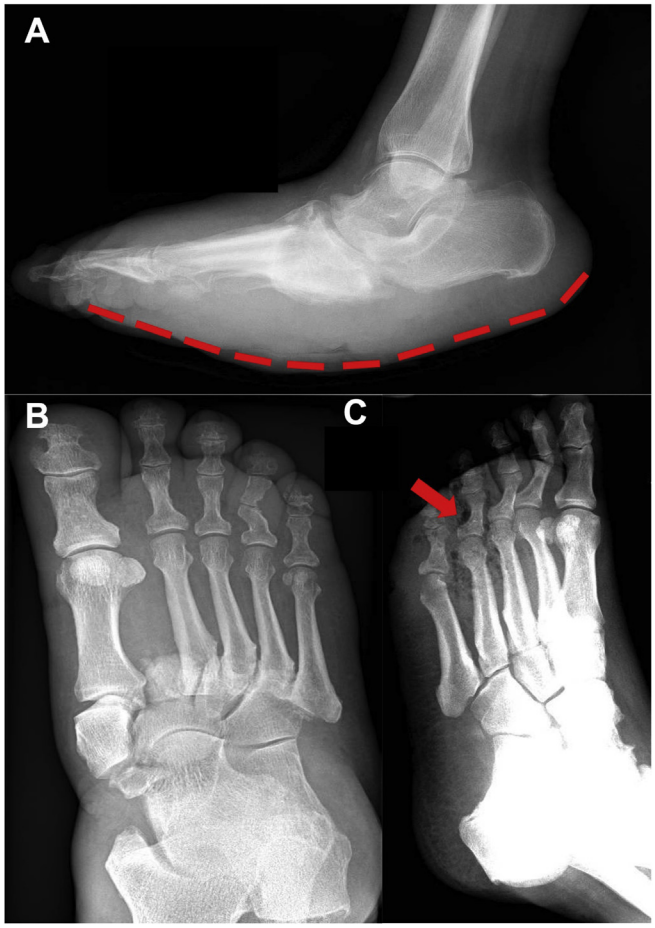

Figure 3 Radiographs of diabetic foot. **(a)** Lateral radiograph of a Charcot foot rocker-bottom deformity (dotted red line). Note that there is arch collapse and a rotated calcaneum. **(b)** Radiograph of Charcot foot with Lisfranc fracture dislocation. Note that there is a lateral displacement of the lesser metatarsals with respect to the 1st metatarsal and significant diastasis between the 1st and 2nd metatarsals. There is also a transverse fracture seen at the base of the right 2nd metatarsal. These findings are consistent with a Lisfranc fracture dislocation. **(c)** Subcutaneous gas seen on a radiograph in a patient with diabetic foot sepsis (red arrow).

ultrasound (DUS). Second-line imaging may include computed tomography angiography (CTA), magnetic resonance angiography (MRA) or digital subtraction angiography (DSA). In cases with evidence of chronic venous insufficiency a venous DUS should also be performed as treatment may be indicated in order to promote wound healing.

Microbiology

The IWGDF recommend the use of the IWGDF/IDSA (Infectious Disease Society of America) classification for the diagnosis of infection. This has been integrated into the WIfI classification system as described in Table 2 above. Clinical features of infection include erythema, warmth, swelling, purulent discharge and pain. Although pain may not be a common feature in patients with peripheral neuropathy, its presence may indicate the presence of a deeper collection warranting further imaging. In the absence of these features, or high index of suspicion for infection, wound cultures should not be taken as antimicrobial treatment will not be indicated.

Superficial swabs are typically not useful as they identify only colonizing microorganisms which may not be truly representative of the causative pathogen(s). Therefore, deeper specimens collected by curettage or biopsy are preferable where possible. Definitive diagnosis of osteomyelitis necessitates culture of bone specimens collected aseptically during surgery or percutaneously. Common infecting microorganisms include Staphylococci, Streptococci and anaerobic bacteria.

Diabetic foot management

Prevention of foot and cardiovascular complications of diabetes

The prevention of new and recurrent ulceration is a key priority.[6] The National Institute for Health and Care Excellence (NICE) recommends that all patients with diabetes should have at least one foot check annually.[8] These checks are an important opportunity to assess the risk of ulceration, modify abnormal risk factors (e.g. the provision of specialist footwear and orthoses, cardiovascular risk factor modification) and deliver patient education. Patients deemed to be at moderate or high risk for ulceration may be offered more frequent foot checks (3–4 times a year).

Cardiovascular risk factor modification is personalized to the individual but may include smoking cessation, glycaemic control, antiplatelet therapy and management of hypertension and dyslipidaemia.

Patients should be informed of their current risk of developing DFU, when and who to seek help from in the event of a foot emergency, the importance of daily foot inspections and general foot care advice. Foot care advice should include washing feet daily, followed by careful drying of feet (especially between the toes), moisturizing the feet (but not between the toes), avoiding walking barefoot, wearing socks with shoes, wearing appropriate shoes, checking inside shoes for foreign objects before wearing them, and cutting nails straight.

Management of diabetic foot sepsis

Local diabetic foot infection with signs of systemic inflammatory response (e.g. temperature of $>38^{\circ}C$ or $<36^{\circ}C$, tachycardia and increased respiratory rate) suggest a diagnosis of severe infection or diabetic foot sepsis. It is important to note that many patients with diabetes do not mount a typical physiological or biochemical response to severe systemic infection. Therefore, one should maintain a high index of suspicion as delays to treatment are associated with a high risk of morbidity and mortality.

Patients with confirmed or suspected sepsis should be treated according to the Sepsis Six pathway.[9] The administration of intravenous antibiotics and fluids should not be delayed. Metabolic disturbances including electrolyte imbalances, acidosis and hyperglycaemia should be promptly corrected. Additionally, emergency surgical debridement to control the source of sepsis is critical.

Management of diabetic foot wounds

Prevention and management of DFU requires well-coordinated multidisciplinary care across all healthcare settings. In the UK,

patients are managed in the community by 'Foot Protection Teams' comprised of healthcare professionals, often podiatrists, with specialist experience in diabetic foot assessment and management. More complex cases are often managed by 'multidisciplinary foot care teams'. Management should be holistic and view the ulcer as a sign of multi-organ disease. Well-organized interdisciplinary teams may include members from various related specialties, and can include diabetologist, vascular surgeon, foot and ankle orthopaedic surgeon, diabetes specialist podiatrist, diabetes specialist nurse, microbiologist, orthotist, radiologist, plastic surgeon, and tissue viability nurse.

Although most patients with DFU can be managed effectively in the outpatient setting, they do require intensive treatment. Depending on the individual patient's clinical and social situation, as well as local resources, patients may require admission for inpatient care. Specific factors likely to necessitate admission include severe infection, failure of outpatient management and the need for a surgical procedure.

Treatment of infection: Infection is present in up to half of DFUs and is strongly associated with an increased risk of hospitalization, amputation and mortality. A diabetic foot infection is defined by the presence of at least two of the classic signs of infection. Other secondary signs can include a malodourous wound and friable granulation tissue. Once infection is diagnosed, its severity can be classified using the IWGDF classification for defining the presence and severity of diabetic foot infections. Diabetic foot infection can progress rapidly, therefore, if infection is clinically suspected, broad-spectrum antibiotic therapy should be initiated immediately. The choice of therapy will depend on local microbiology guidelines for antibiotic therapy and, if available, recent culture results. In more complex cases, or where there is history of recent antibiotic therapy or history of antimicrobial resistance, then a microbiologist should be consulted. Response to treatment should be reviewed regularly and antibiotics should be changed according to sensitivities as soon as possible. The required duration of antibiotic therapy is typically 10–14 days for soft tissue infections, or 6–8 weeks for osteomyelitis (Figure 4). The recent OVIVA (Oral Versus Intravenous Antibiotics) trial demonstrated equivocal outcomes between oral and intravenous antibiotics for complex bone and joint infections.[10]

Treatment of infection also commonly necessitates some form of surgical treatment to control the infection and establish a healthy wound bed. This may range from bedside debridement to more extensive tissue and/or bone resection. Recent data suggests that uncomplicated osteomyelitis can be managed conservatively with a prolonged course (6–8 weeks) of oral antibiotics without the need for surgery.[11] However, this more conservative approach is less likely to be effective in the presence of PAD, end-stage renal disease, severe infection, resistant organisms and necrotizing soft tissue or bone infection.

Offloading: Ongoing repetitive injury caused by high pressure and shear forces prevents DFU from healing. Offloading is pivotal in the management of DFUs and refers to the use of devices or surgery to relieve this pressure and facilitate wound healing.[11] There are a variety of offloading strategies that can be utilized depending on ulcer location, local resources and patient choice.[12]

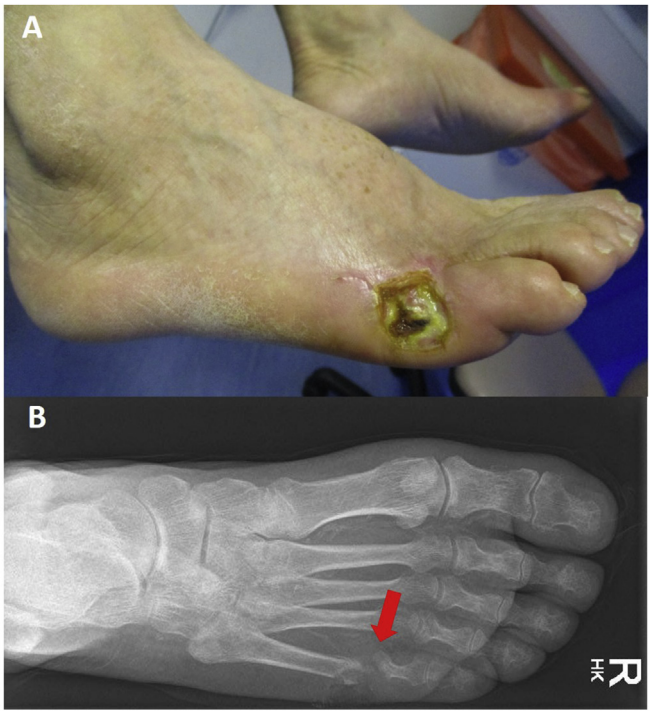

Figure 4 Diabetic foot ulceration. **(a)** Diabetic foot ulcer on the lateral aspect of the right foot. **(b)** Radiograph of the same ulcer demonstrating osteomyelitis of the 5th metatarsal head (red arrow). Note is also made of calcified pedal vessels.

These include therapeutic footwear and custom insoles, felted foam, padded dressings, toe spacers, removal cast boots and total contact casts. Total contact casts remain the gold standard for offloading. Total contact casts involve the application of a plaster cast to immobilize the foot and ankle, and transfers force transmission from focal points on the foot and distributes this over the wider surface area of the foot and calf.

Foot surgery for offloading includes calcaneal tendon lengthening in individuals with metatarsal head ulceration and flexor tenotomy for clawed digits with apical and/or dorsal interphalangeal ulceration.

Local ulcer care: Debridement is the removal of non-viable or contaminated tissue which can serve as a reservoir for infection or impede normal tissue growth. Debridement is an essential component of managing acute and chronic diabetic foot wounds.[11,13] Serial debridement, performed as frequently as once or twice a week, is often required to achieve optimal wound conditions for healing. Methods of debridement can be broadly categorized as surgical or biological debridement and are often used in combination.

Minor surgical debridement can be performed at the bedside without the use of local anaesthesia, in the insensate neuropathic patient, if there is no risk of major bleeding. Minor surgical debridement involves the use of curette, knife or scissors to scrape or sharply dissect non-viable tissue and proteinaceous coagulum on the wound surface. Bleeding can be controlled with direct pressure.

Major surgical debridement, carried out in an operating theatre environment, is preferred in cases where more extensive debridement is required, for example in instances of deep

collections or soft tissue infections, acute presentations of diabetic foot sepsis, or when significant bleeding is anticipated. Local, regional (with or without sedation) or general anaesthesia is often necessary for such cases. For major debridement, previously discussed surgical tools of minor debridement can be used in combination with hydro-surgical debridement (e.g. using the Versajet system) and resection of bone with bone cutters, Rongeurs or oscillating saw as required.

In the presence of ischaemia and severe infection, such as diabetic foot sepsis, emergency debridement should be carried out without delay to prevent the rapid spread of infection. Following debridement urgent vascular imaging and revascularization should be performed if necessary. In cases of less severe infection, debridement may be delayed until urgent revascularization has been performed to facilitate wound healing and reduce the chance of further tissue loss due to ischaemia.

The general principle of surgical foot debridement is to leave only healthy tissue behind.[13] Demarcated healthy tissue can be identified fairly easily by a change in appearance and colour (e.g. fat may change colour from a shiny yellow to a dull grey); this may be challenging if undertaken as an emergency for sepsis control ahead of revascularization. Particular focus should be given to debridement of exposed tendons which can act as pathways for rapid spread of infection. If tissue is not clearly demarcated, incision can be started at the middle of the wound and extended until viable bleeding tissue is reached to identify a potentially healthy edge. Non-viable skin should also be debrided. To promote viability, tissue should be handled with care, with minimal use of diathermy and avoidance of undermining which could result in devascularization. Debridement should always be followed by generous irrigation to reduce bacterial load and debris.

Biological debridement techniques include autolytic dressings, enzymatic ointments, osmotic agents and larval therapy.[11] They work slowly but are useful for wounds with small areas of non-viable tissue, particularly those that are difficulty to debride surgically. Autolytic dressings (i.e. hydrogels, hydrocolloids, alginates and hydrofibres) promote release of endogenous proteolytic enzymes to dissolve sloughy or necrotic tissue. Enzymatic ointments (e.g. collagenase) contain proteolytic enzymes that selectively disrupt devitalized tissue and work best in moist or fibrotic wounds. Osmotic debridement (i.e. honey) works by drawing fluid from healthy tissue to facilitate endogenous autolytic debridement. Larval therapy (also known as maggot debridement therapy) involves placing live irradiated maggots (*Phaenicia sericata*) on the ulcer for 3–5 days, either loose or housed in a biobag. The maggots digest necrotic and non-viable tissue and secrete an antibacterial compound that reduces the wound bioburden while reducing inflammation and facilitating tissue remodelling.

Following debridement, primary, secondary or tertiary wound closure may be achieved depending on location and area of the wound. On occasion, interrupted absorbable sutures can be placed at the time of debridement to loosely approximate skin edges to facilitate future wound closure. Consideration can be given to the use of local antibiotics, for example Stimulan or Cerement preparations ahead of closure. Useful adjuncts to achieving wound closure include negative pressure wound therapy, split skin grafts, and decellularized cadaveric skin. Other surgical approaches include reconstructive techniques such as musculo-/fascio-cutaneous flaps.

Revascularization: In the context of DFU, the primary aim of revascularization is to restore blood flow and perfusion to improve the chance of wound healing.

The decision to perform revascularization can be challenging and involves consideration of the severity of disease (e.g. using the WIfI classification system), technical challenges (e.g. arterial lesion location and severity) and patient characteristics (e.g. comorbidities). In the UK, a nationwide NHS improvement programme (Getting It Right First Time; GIRFT) demonstrated unacceptable pathway delays to revascularization and significant variation in practice. Recognizing that time to revascularization is an important determinant of ulcer healing, the Vascular Society of Great Britain and Ireland (VSGBI) now recommend that outpatients with chronic limb threatening ischaemia (CLTI) should be revascularized within 2 weeks of referral, while inpatients should be revascularized within 5 days.

In many centres, diabetic patients with significant PAD will undergo digital subtraction angiography for planning of revascularization procedures due to higher resolution afforded by this modality, particularly when it comes to the smaller and commonly calcified tibial and pedal vessels. In patients with renal insufficiency, CO_2 can be used as a contrast agent to reduce the volume of iodinated contrast required for the investigation.

There are two main approaches to revascularization: open surgical or endovascular revascularization. These two approaches can also be used in combination in hybrid procedures. Over recent years, the choice of lower limb revascularization strategy has become contentious, with endovascular techniques increasingly prevalent. This choice should be personalized to the patient and relies on an assessment of a number of factors including the anatomical pattern of disease, metabolic demand of the wound, patient specific risk and preference. The Bypass versus Angioplasty in Severe Ischaemia of the Leg (BASIL) randomized control trial compared surgical bypass and endovascular intervention. Although perioperative mortality was higher in the bypass group, amputation-free and overall survival were similar in both groups at 1 year.[14] However, at 2 years follow-up bypass surgery was associated with a reduced risk of amputation and death.[15] Although only 42% of recruited patients were diabetic, the results do suggest that bypass remains a key treatment modality in patients who have suitable vein conduit and a life expectancy greater than 2 years. A number of ongoing multicentre randomized controlled trials aim to further increase our understanding of the best revascularization strategy. BASIL 2 aims to compare a vein bypass first strategy to an endovascular first strategy in the context of infra-popliteal disease; and BASIL 3 aims to investigate the best endovascular strategy for treating femoropopliteal disease, comparing drug coated balloons, drug eluting stents and plain balloon angioplasty.

Amputation: The decision making regarding when to amputate and at what level can be challenging. Although amputations are an unwanted and devastating outcome, they allow for removal of non-viable and infected tissue. An early and informed decision to proceed with major amputation supported by a multi-disciplinary team has the potential to reduce the time and patient deconditioning that can be associated with protracted efforts at limb salvage in the context of a limited chance of a functional foot. Amputation may actually result in an improvement in quality of

life, particularly in immobile patients suffering from recurrent non-healing wounds associated with lengthy hospital admissions, frequent hospital visits, regular dressing changes, and long courses of antibiotics required for treatment of resistant organisms.

Amputations can be classified into minor (below the level of the ankle) or major (above the level of the ankle). Major lower limb amputation is indicated in patients who have no further options for treatment. Below or through knee amputations have the advantage of greater mobility with prothesis while above knee amputation is a quicker operation to perform with higher rates of healing.

Minor amputations (e.g. partial/complete ray and transmetatarsal amputations) are important elements of diabetic foot management conducted as part of major debridement procedures. Patients with dry, well-demarcated digital gangrene of limited volume can be managed conservatively while awaiting autoamputation if there is no evidence of infection or pain.

In unstable patients with diabetic foot sepsis, a more appropriate strategy may be to perform a rapid guillotine amputation of the infected tissue and to return to theatre once stable for conversion to a formal amputation.

Conclusion

Diabetic foot disease, or ulceration, is a complex and devastating complication of diabetes. Preventing new or recurrent diabetic foot complications is a key priority, which can only be achieved through regular foot examinations and risk stratification with appropriate modification of risk factors and patient education. Foot complications can be challenging to manage. The important aspect of management include early diagnosis, well-coordinated multidisciplinary care, prompt treatment of infection and peripheral arterial disease, as well as optimal wound care and offloading. ◆

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Practice points

- Routine diabetic foot checks are important in the prevention of ulceration and cardiovascular complications as they provide an opportunity to assess risk, modify risk factors and deliver patient education
- A diagnosis of Charcot neuropathic osteoarthropathy should always be suspected in an individual with diabetes presenting with a hot and swollen foot
- In a patient with diabetic foot ulceration, the timely diagnosis of peripheral arterial disease and onward revascularization are important determinants of ulcer healing
- Patients with diabetic foot infection should be assessed for signs of sepsis and treated according to the Sepsis Six principles in addition to emergency surgical debridement for source control