Diabetic foot ulceration:
an avoidable complication

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Foreword

At present, more than 380 million people worldwide have diabetes. By 2035, this will rise to 592 million. Some 77% of these people live in low- and middle-income countries.[1] In emerging countries, in particular, health-care services are often difficult to access, and finding suitable therapies for diabetes can be challenging. Inadequate treatment of diabetes leads to complications such as diabetic foot syndrome (DFS). For an individual with diabetes, the lifetime risk of developing foot ulcers is at least 15%.[2] In western countries, DFS remains the most common reason for hospitalisation of patients with diabetes. In addition, DFS is the major cause of non-traumatic lower limb amputation. This supplement aims to improve understanding of DFS and to present essential aspects of prevention, diagnosis and treatment.

The major cause of foot wounds in people with diabetics is polyneuropathy (PNP), which leads to a loss of sensation. This frequently results in patients not seeking medical help — or if they do, it is often too late. Delays in therapy frequently lead to soft-tissue infection. Inflammation also often spreads over to the joints or the bone tissue, which can lead to sepsis or amputation. All patients with diabetes should therefore be routinely screened for PNP. They must also be informed about their risk and advised how to prevent injuries.

Peripheral arterial disease, which is common in people with diabetes, is also often responsible for non-healing wounds. Again, primary physicians should routinely screen patients with diabetes for this. Callus formation caused by deformities is another risk factor. Chiropodists and professional orthopaedic shoemakers should therefore be consulted in order to help prevent complications in patients with these risk factors.

Provision of therapy by multidisciplinary foot-care teams is the most promising approach to the treatment of DFS. For example, following the introduction of peripheral bypass surgery and establishment of a diabetic foot clinic in Copenhagen, the amputation rate reduced by approximately 75%.[3] In a multidisciplinary diabetic foot clinic, experienced physicians will cooperate with trained wound-care nurses, while orthopaedic shoemakers will be available to supply protective footwear.

Effective pressure-redistributing methods are of particular importance. As neuropathy prevents patients from using the offered devices consistently, the inclusion of padding within a wound dressing can help to provide offloading 24 hours a day. Similarly, total contact casts, which are also worn all day long, are useful. In serious cases, patients must be sent to a specialised hospital where septic surgery, advanced wound care, vascular surgery and, if needed, endovascular intervention are available.

Generally, structured treatment, whereby different levels of patient care (general practitioner, diabetic foot clinic and specialised hospital) are integrated and coordinated, is the best way to treat DFS successfully.

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Diabetes mellitus and the increased risk of foot injuries

With one person dying from diabetes-related preventable complications, including foot complications, every 7 seconds across the world, it is clear this is a major health challenge. Foot ulceration in diabetes remains the commonest reason for hospital admission in Western countries. From neuropathy to peripheral vascular disease, the challenges are significant and can result in premature death, but early diagnosis by aware health-care professionals, combined with supporting people in self-care, can help reduce the problems of diabetes to manageable proportions

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Diabetes is an increasingly common long-term condition worldwide, with the International Diabetes Federation (IDF) estimating that 387 million people are now diagnosed with it, a worldwide percentage of 8.3%.[1] This equates to 1 in 12 people across the world having diabetes and contributes to the 49 million deaths from diabetes that occurred in 2014. This is one person dying from diabetes-related preventable complications, including foot complications, every 7 seconds across the world.[1]

Ten million people are estimated to be at high risk of developing type 2 diabetes in England alone.[2] Furthermore, the IDF estimates that, of the world's population with diabetes, the condition is undiagnosed in about 46%, who more commonly live in low- and middle-income countries.[1] Being undiagnosed or having limited access to diabetes care places individuals at increased risk of the preventable vascular and neuropathic complications of diabetes, including foot disease, caused by neuropathy or ischaemia.[2-4.]

Prevention, early detection and referral to multidisciplinary teams for rapid access to treatment can save lives and reduce amputations worldwide. Diabetes is an expensive disease to manage, so education on and prevention of avoidable complications is the key to managing this. Person-centred care and information-giving are at the heart of this approach.[5,6]

Epidemiology of foot ulceration in diabetes

Foot ulceration in diabetes remains the commonest reason for hospital admission in Western countries.[7] Additionally, Tuttolomondo et al.[8] and Bakker et al.[9] identified that 20–40% of health-care costs relate to the treatment of preventable foot-care complications in diabetes. The lifetime risk for an individual with diabetes developing a foot ulcer is 25%, and up to 85% of all lower limb amputations in diabetes are preceded by foot ulceration.[7]

Foot problems occur in both type 1 (insulin-dependent autoimmune disease) and type 2 (metabolic disease).[3] Ulceration in diabetes is more common in men and more prevalent in people aged over 60 years. Foot ulcers occur more commonly in Caucasians than in those from Asian or African-Caribbean backgrounds[10] and increased rates of ulceration have been noted in populations who are socially deprived[11] or face inequalities in access to diabetes care and risk-factor reduction.[12] Foot ulceration can be the presenting feature of late diagnosis of type 2 diabetes, and so anyone presenting with foot ulceration should be screened for diabetes.[7]

Pathogenesis of foot ulceration

The World Health Organization recognised that diabetic foot syndrome (DFS) encompasses all complications relating to the

foot in someone with diabetes.[8] It defined DFS as 'ulceration of the foot, distally from the ankle (and including the ankle) associated with neuropathy, ischaemia and infection'. DFS encompasses a number of pathologies, including neuropathy, peripheral vascular disease, Charcot neuroarthropathy, foot ulceration and osteomyelitis.[13] People with DFS also often experience multiple diabetes-related complications, such as cardiovascular disease, nephropathy, retinopathy and cerebral vascular disease. Caring for such people is complex and requires a multidisciplinary approach to provide person-centred, effective diabetes care. This will be presented in more depth in the following articles in this supplement.

DFS often leads to preventable amputation, with data from the US identifying that more than 80,000 diabetes and foot ulcer-related amputations are undertaken per annum.[14] In the UK, approximately 120 leg, foot and toe amputations are carried out on people with diabetes each week, and it is estimated that 80% of these are preventable.[2,15] People with diabetes are nine times more likely to have a minor amputation and five times more likely to have a major amputation than those without diabetes.[16]

Amputations and foot ulcers have a huge impact on people's lives and increase their morbidity and mortality. Singh et al.[17] estimated that about 80% of people die within 5 years of having an amputation because of diabetes-related vascular or neuropathic disease.[15] Based on these statistics, the relative likelihood of death within 5 years following amputation is greater than that for colon, breast or prostate cancer. Sadly, diabetes does not carry the same emotive response or recognition within populations as does cancer, yet the risks can be so much greater.

Causes of foot ulceration

Diabetic foot ulceration has a multifactorial and complex pathogenesis, including peripheral neuropathy, foot deformity, trauma, abnormal foot pressures, abnormal joint mobility and peripheral arterial disease resulting in ischaemia. Boulton identified that infection is not a primary cause of foot ulceration, but a secondary event following injury/ulceration to the protective epidermis layer.[7]

Neuropathy

Prolonged hyperglycaemia can cause the destruction of neurons and intracellular metabolic changes that impair nerve function, leading to neuropathic changes.[3] Peripheral neuropathy results in insensitivity, especially to potential trauma or injury. The presence of callus in an individual with insensitive feet is highly predictive of foot ulceration.[7] Diabetic peripheral neuropathy significantly impairs nerve conductivity throughout the body, affecting autonomic, motor and sensory functions.[18] Neuropathy can initially affect the longest nerves, so can present in a glove and stocking distribution (Fig 1). It can also affect body systems such as cardiovascular, sexual functioning and digestion. This should be taken into account when assessing people with diabetes, as any such abnormality is an indicator of neuropathy.

Tuttolomondo et al. suggested that impaired sensation makes the foot increasingly vulnerable to damage caused by mechanical, thermal or pressure-related injury.[8] It is vital, therefore, to provide education and advice on foot wear in order to protect the foot and prevent ulceration. As sensory neuropathy increases, symptoms can range from numbness, to hyperalgesia and allodynia, which is commonly described as burning or cutting/piercing/stabbing pain that can be worse at night.[18] Between 16% and 26% of people with diabetes may experience painful peripheral neuropathy.[19]

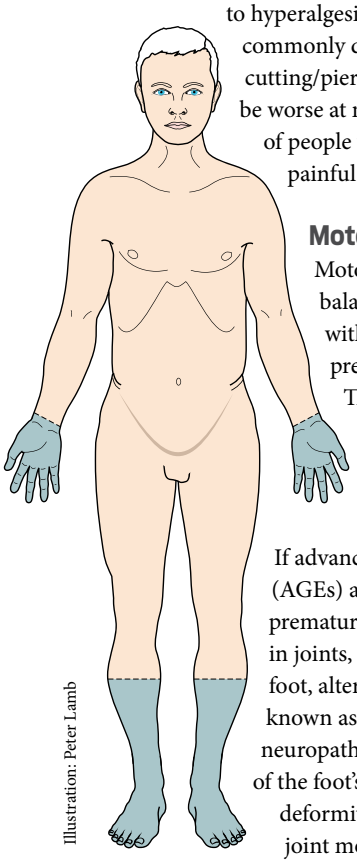

Illustration: Peter Lamb

Fig 1. The 'glove and stocking' presentation of sensory neuropathy[18]

Motor neuropathy

Motor neuropathy impairs the balance of biomechanical forces within the foot, causing altered pressure and muscle atrophy. This impairs proprioception and causes wasting of small muscles, which leads to altered loading under the foot when standing or walking. If advanced glycation end products (AGEs) are also present, cells will age prematurely, which can cause stiffness in joints, especially in the ankle and foot, altering pressure loading.[20] This is known as limited joint mobility. Motor neuropathy also impairs the integrity of the foot's anatomy, leading to foot deformities, clawed toes and impaired joint mobility, complicated by AGEs.[8] These processes compromise weight-loading on extremities in the foot, which can lead to ulceration and ischaemic necrosis of the tissues nearest to the callus.

Charcot neuroarthropathy

This is one of the more devastating complications of diabetes. It is a progressive, destructive condition that is characterised by acute fracture, dislocation and joint destruction in a person with neuropathy.[21] In its acute phase, it is often misdiagnosed and can lead to several deformities, resulting in ulceration and amputation.[7] Early diagnosis and management is, therefore, imperative to avoid rapid its rapid progression.[21]

Autonomic neuropathy

This presents as dry skin and subsequent textual changes. Skin dries and 'ages', causing fissures that can crack, opening the skin for bacterial attack. A preventive measure is to teach people with diabetes to inspect their feet daily, to wash and dry effectively, especially between the toes, and to moisturise their skin. This health message needs careful communication, as many men will deem being asked to moisturise their feet unacceptable, so other methods may need to be used to explain that this is a healthy approach to protecting their feet. Education should be delivered positively and pitched at a level that the individual understands; communication of information and language is as important as the information itself.[22]

Peripheral vascular disease

Peripheral vascular disease (PVD), which is also known as peripheral arterial disease, is also common in people with diabetes and is a major factor in ulceration.[23] It is a significant cause of morbidity and mortality, and affects lower limbs, reducing blood flow and causing intermittent claudication, pain on exertion, pain at rest in severe cases, critical limb ischaemia and gangrene.[24] About 10% of ulcers in people with diabetes are caused solely by PVD; in contrast, it has been suggested that 90% of ulcers are caused by neuropathy alone or present as a mixed aetiology (neuroischaemia).[7] PVD is accelerated by advanced cardiovascular risk factors such as smoking, hyperlipidaemia, hypertension and hyperglycaemia,[3] all of which are controllable with effective person-centred diabetes care.[25] People with PVD are at increased risk of cardiovascular mortality.[26]

The progress of intermittent claudication can be relatively stable, with 70–80% of people affected over a 5-year period remaining stable. However, 10–20% will experience some worsening of their condition and 5–10% will develop critical limb ischaemia.[24] Hirsh et al. suggested that, of this population, 5–10% will die from their PVD and a further 20% will experience a non-fatal cardiovascular event such as a stroke or myocardial infarction.[27]

When people present with foot ulceration and/or PVD, use of ankle brachial pressure index (ABPI) is standard as part of assessment. Care must be taken when interpreting results, due to the potential presence of arterial wall calcification and risk of false positives.[8]

Box 1: Individuals at greatest risk of diabetic foot syndrome are those with:

- Evidence of neuropathy
- Evidence of ischaemia
- Foot deformity
- Previous amputation
- Previous history of ulceration
- History of falls
- Sight impairment preventing self-detection of foot problems
- Other diabetes-related vascular or neuropathic complications present
- Poor social circumstances: living alone, poverty, isolation
- Older adults

Adapted from Boulton[7] and Phillips[25]

Identification and early intervention of the at-risk foot

Careful inspection and examination of the feet is an essential part of preventive diabetes care. Every person with diabetes, regardless of his or her age or the disease duration, should be assessed in this way. Practitioners should not rely on people presenting as symptomatic, as 50% of people presenting for the first time with insensitive feet have no previous history of neuropathy;[7] furthermore people with PVD may not present with obvious claudication.[7] Box 1 lists those who are at greatest risk of DFS.

Improving practitioner and person-centred knowledge

Education is the key to supporting consistency of care within diabetes.[25] Box 2 describes how to elevate care as required and refer all episodes of ulceration quickly for the best outcomes. Early referral of any foot wound in a person with diabetes is essential — timely referrals can save limbs and lives.

Prevention is ultimately based on the ability to identify the risk of ulceration during the diabetes review. Any identified risks must be shared with the individual and his or her family and/or carers, depending on their individual circumstances and where they reside.

The King's Fund states that person-centred care can enable people to be responsible for their own health and care.[6] Ongoing education about diabetes is the essence of good-quality diabetes care.[28] Two essential components of effective diabetes care involve recognition that:

- The individual with diabetes knows the most about his or her condition
- This individual is also the person who can also do the most to improve his or her diabetes control and thus reduce the risk of foot ulceration. However, they will require education and support to achieve this.

Conclusion

Informing people about the risks of diabetes and how their personal action can help prevent ulceration is a vital part of good person-centred diabetes care. Good communication

Box 2: Person-centred foot-care education

Everyone with diabetes should recognise the importance of the early warning signs of foot problems. These include:

- Swelling
- Redness
- Heat
- Pain
- Discharge

All people with diabetes and their family/carers should have emergency contact numbers for seeking help and advice if any of these early warning signs occur on one or both of their feet

Box 3: Summary of multifactorial pathogenesis of diabetic foot ulceration

Diabetic foot ulceration is caused by a multifactorial and complex pathogenesis. This can include peripheral neuropathies causing:

- Foot deformities
- Trauma-induced ulceration
- Abnormal foot pressures
- Abnormal joint mobility

In addition, peripheral arterial disease can result in ischaemia, placing the foot at risk of ulceration

skills, combined with the ability to consider physical and psychological health, social and cultural beliefs and the needs of the individual, will help enable self-management.[22]

This person-centred care approach requires a good relationship between the practitioner and the individual with diabetes. Health professionals have a unique role and relationship with people receiving diabetes care. Following foot-care pathways and expediting care as and when required can greatly improve the person's experience and ultimately reduce unnecessary amputations through better care processes and access to dedicated multidisciplinary diabetes foot-care teams as and when required.

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Want to avoid DFUs? A multidisciplinary team approach works best

As the majority of diabetic foot ulcers are avoidable, patients with diabetes should be routinely screened for risk factors. When such factors are present, holistic assessment followed by rapid referral to the appropriate member of the multidisciplinary team will ensure that preventive measures are implemented to avoid the occurrence of ulcers. Best results will be achieved if the patient is fully involved in this process

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The prevalence of diabetic foot ulcers (DFUs) in patients with diabetes mellitus is estimated as 3–10%.[1-3] DFUs are one of the most serious and disabling complications of diabetes, with 25% of patients with diabetes developing them in their lifetime, and 15% having to undergo hospitalisation and lower extremity amputations.[4,5] According to Tang et al., approximately 50–86% of lower extremity amputations result from DFUs.[3] These ulcers can significantly impair quality of life, causing patients pain and distress, low mood, decreased physical function, reduced social interaction and potential loss of income.[6,7]

Diabetic foot ulcers also have a major economic impact on the health-care system as their cost increases disproportionately to the severity of the condition. According to Co et al., diabetes accounts for approximately 12% of the world's health expenditure,[6] with Hick et al. stating that DFUs cost an estimated $1.5 billion annually.[8] The cost of caring for a diabetes patient with a DFU is approximately 5.4 times higher than that for a patient without a foot ulcer, due to more frequent visits to the emergency department and longer lengths of hospital stay.[9] It is vital, therefore, that the patient, carer and clinicians from all settings work together effectively to avoid the risk of such complications occurring. This article describes how this can be achieved.

Understanding risk factors for DFUs

It is essential to identify the major risk factors associated with lower limb ulcerations as these factors hinder and/or prevent wound healing. Physical, psychological and social risk factors can include peripheral vascular disease, age (>60 years), gender, duration of diabetes, basal metabolic rate (>25), obesity (BMI >30), oedema, inflammatory cytokines (as determined by laboratory tests such as full blood count, platelet count), susceptibility to infections, peripheral neuropathy and Charcot neuroarthropathy. Winkley et al. indicated that there is a threefold increased risk of recurrence in patients with one or more microvascular diseases, such as diabetic nephropathy, diabetic neuropathy and diabetic retinopathy.[10] Social risk factors include lack of access to care and a social support network. Non-adherence with prevention and treatment plans is another key risk factor. Patients with multiple risk factors have a higher incidence of DFU.[5,11,12]

Skin pathologies also play a vital role in predisposing patients with diabetes to foot ulceration. Fissures, toe web maceration and/or callus are significant risk factors for ulceration as they compromise skin integrity and allow entry of opportunistic pathogens.[13] Thick, hard skin on the sole of the foot is also prone to fissures and thus ulceration, especially in patients with neuropathic complications.

Most DFUs result from the accumulation of trauma, neuropathy and deformity caused by the pressure inflicted by incorrect footwear.[14] All footwear is associated with shear, but ill-fitting footwear is a particular cause of ulceration.[15] Footwear recommendations for patients with diabetes generally depend on their activity levels, the presence of

Box 1: Characteristics of footwear that will reduce the risk of ulceration[42]

- Good retaining medium
- Close-fitting in the medial and lateral quarters
- Adequate width and depth in the toe box
- Correct length (12mm space between the toe and shoe border)
- Correct width fitting
- Adequate heel seat
- Heel height no greater than 5cm
- Broad heel base in contact with the ground
- Upper part made from leather or another natural material
- Breathable material
- No foreign objects present

foot deformities and/or levels of plantar pressure. As a preventive measure, patients should be educated to check for foreign objects in their footwear before they put them on. Key aspects to consider when advising patients on footwear are listed in Box 1.

Alterations in biomechanics and gait

Studies have indicated that, due to the associated decrease in sensory or motor abilities, neuropathic changes may lead to functional gait variations.[16,17] Charcot neuroarthropathy in the lower extremity exacerbates foot deformities and/or limits joint mobility, which in turn results in abnormally increased foot pressure. Abnormal plantar pressure distribution, combined with limited joint mobility, is a common underlying cause of DFUs in individuals with peripheral neuropathy.[12,16,18]

Sensory-motor neuropathy is also a cause of biomechanical deformities of the foot, such as high-arched foot (cavus foot), clawed lesser toes, muscular wasting, hallux abductor valgus (bunions), hallux ridigus (structural hallux limitus and rigidus, although this can also be caused by other — functional— biomechanical factors) and fatty pad depletion.[12] Increased pressure over certain areas of the foot results in callosities.[11,19]

Charcot neuroarthropathy is one of the most destructive complications in diabetes due to its ability to induce subluxation, dislocation, deformity and ulceration within the lower limb.[19] The exact pathophysiology of Charcot neuroarthropathy is still unknown; however, it commonly develops in patients with long-standing diabetes complicated by neuropathy. Patients always have a good blood supply and are often unable to recall any injury. In some cases, they have a history of trivial injury. Patients often notice a change in their foot shape and/or crunching of bones when they walk. They experience rapid swelling, an elevated temperature within the foot, and sometimes discomfort or pain. This condition should not be left untreated as it will result in the foot forming the abnormal shape associated with Charcot neuroarthropathy.[19] As well as increasing plantar pressure, structural changes in the foot cause a longer stance time, resulting in a prolonged mechanical load on the tissues, leading to ulceration.[5,17]

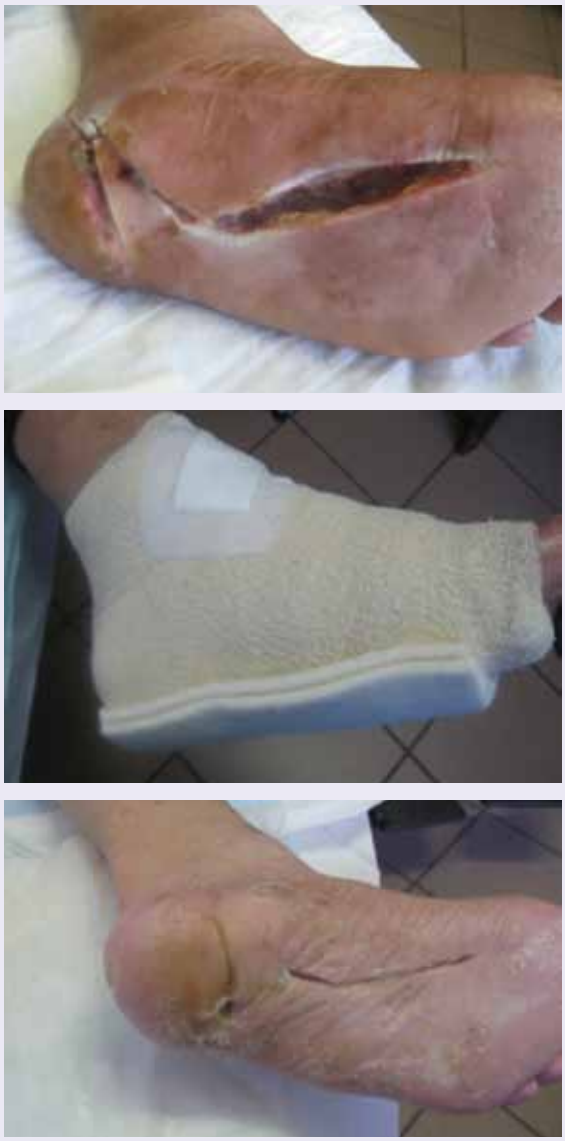

Fig 1. Example of the benefits of offloading: An infected diabetic foot ulcer at presentation (top).The patient had a 2-year history of ulceration in this foot, while his other foot had been amputated. Treatment included general podiatric care, wound dressings, offloading orthopaedic felt and a non-removable air cast boot (middle). After 9 weeks, there was a marked improvement in the condition of the wound (bottom)

Charcot neuroarthropathy can be managed successfully, and major deformity avoided, if multidisciplinary teamwork is used to enable early assessment, intervention and continued follow-up with a radiographer, orthotist, podiatrist and orthopaedic surgeon.

The role of the multidisciplinary team

DFUs are multifaceted and no individual or clinical specialist should be expected to address all aspects of management in isolation.[12] The multidisciplinary team (MDT) should ideally be composed of specialists such as a general practitioner with a specialist interest in diabetes, diabetes nurses, podiatrists, a tissue viability nurse, dietitian, a physiotherapist for rehabilitation, a psychologist and allied health-care staff,[20] although in some countries this will not be possible due to the scarcity of certain skills. For more comprehensive care, the MDT needs to be enhanced by a diabetologist, surgeon (vascular, plastic, orthopaedic), pain consultant, and infection and microbiology specialists.[12,20]

The main aim of the MDT is to enable patients to receive early or immediate access to relevant health-care professionals, education and, if required, interventions. A system needs to be in place that facilitates efficient and easy patient referrals between MDT members, effectively resulting in fast-tracking. This will also allow the MDT to flag other conditions that may have been overlooked. Studies have highlighted that a comprehensive and dynamic treatment pathway, that is coordinated by the MDT, is the most effective approach for DFUs.[12]

Foot self-care is an important factor in preventing DFUs, making it vital that patients are alert for the early signs of ulceration and aware of whom to contact for help.[2,12,21] The development of an ulcer is a major event and a sign of progressive disease. Patient education is essential to improve outcomes. The patient should receive information and support on lifestyle changes, exercise, optimal glycaemic control, how to spot early signs of complications, and why adherence will reduce the risks of ulceration and its life-threatening complications.[12] The patient is, of course, the key member of the MDT, and should be involved in all clinical decision-making about his or her care.

Assessment and management

MDT assessment and management aims to prevent the occurrence or deterioration of DFUs. This involves treating the underlying disease by improving glycaemic control, optimising skin integrity, promoting a healing environment through cleansing and, where necessary, the use of wound dressings, debriding devitalised tissue and callus, pressure offloading and arterial reconstruction.[22]

During assessment, a full medical and social history should be undertaken, and presenting comorbidities, previous management plans and any barriers to patient concordance should be documented.[23,24] Neuropathic status can be assessed using the modified neuropathic disability score[25] or, at the very least, a 10g monofilament, which measures whether or not a patient can detect pressure that can damage the tissues. A 128Hz tuning fork is used to detect whether the patient can detect gross vibration.

Vascular assessment is equally vital as misdiagnosis can result in morbidity as occlusion of blood flow can induce necrosis, potentially resulting in lower limb amputation. Assessment of the pedal pulses will help determine the integrity of the vascular system. If vascular insufficiency is suspected, the patient must be referred immediately to a physician or vascular surgeon.

The following assessment tools can be used to classify DFUs:

- Wagner[26]
- Texas[27,28]
- Pedis[29]
- Sinbad.[30]

Managing skin integrity

Patients often present with varying degrees of diabetic disease-related tissue injury resulting from pressure, friction and/or shear.[24] Wound characteristics vary from erythema to deep infected cavities involving underlying structures such as bone, tendon and fascia, with varying levels of malodour, exudate, slough and necrosis; pain can be absent or severe.[31] Regardless of the complexity of the wound, cleansing and maintenance of skin integrity through the use of dressings can help prevent tissue damage and promote healing.[32]

Cleansing

Reduced skin integrity is prevalent in patients with vascular insufficiency and diabetes.[31] The surrounding skin is cleansed in order to protect it from wound exudate and maintain its natural acid mantle. Wound exudate increases the natural pH of the skin. This causes the stratum corneum to swell, which alters lipid rigidity (fat cells harden) so that the skin becomes drier.[33] This, in turn, reduces the skin's protective barrier function, increasing the risk of infection, skin damage and ulceration.[24] Cleansing with a product that will maintain the acid mantel at a pH of 5.5 will optimise the natural antibacterial properties of the epidermis.

Wounds should be cleansed if dirt, foreign bodies or slough are present, but care must be taken to avoid damaging the wound bed with unnecessary or vigorous cleansing. Recommended practice[24,34,35] is summarised in Table 1).

Wound care

The TIME framework is a practical tool that can be used to facilitate wound bed preparation and promote healing. The framework can be summarised as:

Table 1. Recommended practice for wound cleansing[43,44]

Skin/tissue condition	Product use	Rationale
Granulating, clean wound bed and surrounding tissues	None	Maintain skin pH and protect the surrounding skin. Avoid disturbing of granulation tissue and peri-wound skin
Sloughy, malodorous, excess exudate, product residue	Irrigation products incorporating betaine (surfactants) and polyhexanide (PHMB)[45,46]	pH maintenance, removal of mild levels of exudate, slough and product residue
Critically colonised, moderate levels of slough, malodour, moderate levels of exudate, biofilm suspected	Irrigation products incorporating betaine (surfactants) and polyhexanide (PHMB)	Surfactants penetrate, clean and remove wound debris/biofilm. PHMB antimicrobial agent reduces bioburden[47]
Local infection, soft necrosis, strong malodour, high levels of exudate	Irrigation products incorporating betaine (surfactants) and polyhexanide (PHMB)	Surfactants penetrate, clean and remove wound debris/biofilm. PHMB antimicrobial agent reduces bioburden

Table 2. Simple guide for dressing selection for diabetic foot ulcers[40]

Stage of DFU	Clinical condition	Product choice	Rationale
1	Normal	Not applicable	Keep skin clean and dry and observe for cracks/friction marks on skin
2	High risk	Creams, emollients, debridement of callus	Prevent skin becoming hard and dry, which can lead to fissures
3	Ulcerated	Irrigation, iodine, silver, ionic silver alginate, PHMB, flat and cavity foam, silicone foam	Reduce bioburden, absorb excess exudate, protect peri-wound skin. Ensure dressing is adherent and its removal is atraumatic
4	Cellulitic	Sharp debridement, irrigation/gel, iodine, silver, ionic silver alginate, PHMB, flat and cavity foam, silicone foam	Reduce bioburden, remove non-viable tissue, absorb exudate and protect peri-wound skin. Ensure dressing is adherent and its removal is atraumatic
5	Necrotic	Intravenous antibiotics, sharp debridement, irrigation/gel, silver, ionic silver alginate, iodine, PHMB, flat and cavity foam, silicone foam	Reduce possible sepsis, bioburden, remove non-viable tissue, absorb exudate if cavity and protect peri-wound skin. Ensure the dressing is adherent and its removal is atraumatic
6	Major amputation	Following surgery: PHMB irrigation, foam, silicone foam	Products should be easy to lift for regular inspection, atraumatic, protect against infection, cushioning, and absorbent

- **T**issue management — remove non-viable or deficient tissue
- **I**nfecton or inflammation — aim to avoid or treat this
- **M**oisture balance — apply a dressing that will create a moist environment conducive to healing
- **E**dges — if the wound edges are not advancing or are undermining, then reassess the wound and consider new therapeutic strategies[36]

Tissue management can be achieved with cleansing and debridement, while local infection can be treated with antimicrobial dressings. More details are given in chapter 4. Most aspects of wound bed preparation involve the use of wound dressings, which should:[37,38]

- Promote a moist wound bed — exudate produced at the start of the wound healing process provides tissue cells with nutrients and biological elements required to promote healing. However, if a wound becomes too wet or dry, the cells from the wound edges are unable to migrate across the wound surface. In addition, the provision of nutrients to cells is diminished and the removal of waste products and dead cells ceases, which impairs healing
- Allow gaseous exchange — this enables oxygenation at the cellular level, promoting cell replication and migration across the wound surface and the removal of waste products
- Act as a barrier to bacterial invasion

Clinical scenario: the multidisciplinary team in action

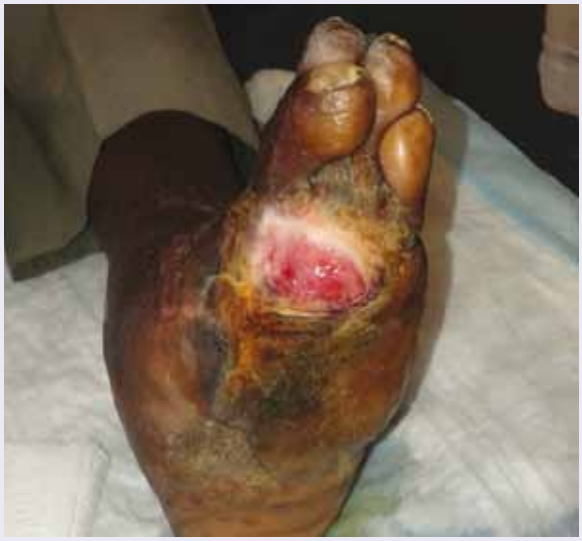

Fig 1. Wound at the start of treatment (week 1)

A 54-year-old man with type 2 diabetes mellitus presented with a 6-month old ulcer on the plantar aspect of his left foot. He was receiving treatment for hypertension and high cholesterol, and was a heavy smoker (general practitioner/ diabetologist/primary health-care nurse). He had previously undergone amputation of his hallux and first metatarsal ray (othopaedic/vascular/plastic surgeon).

Presentation

The ulcer, which was located on the plantar anteromedial aspect of the foot, was surrounded by hyperkeratotic skin. The wound bed was sloughy and malodorous, and producing extremely high levels of exudate. The foot was swollen. All pedal pulses were palpable; foot temperature was warm, but capillary refill was normal.

Management

The wound was swabbed and a sample was taken for culture (microbiologist). Broad-spectrum antibiotic therapy was immediately initiated (general practitioner). The hyperkeratotic skin was mechanically debrided (podiatrist), wound dressings were applied (diabetic wound nurse) and offloading techniques used (podiatrist/orthopaedic surgeon/orthotist). The patient was informed about his condition and the need to constantly control his glucose levels, to reduce/stop smoking and drinking alcohol, and to make lifestyle changes (dietitian/ diabetic nurse). He also received education on wound care. Normal type footwear was no longer appropriate, in particular due to the changes in the overall structure of the foot. Custom-made footwear was therefore recommended (podiatrist/ orthotist). The ulcer had almost healed at the final consultation at 12 weeks.

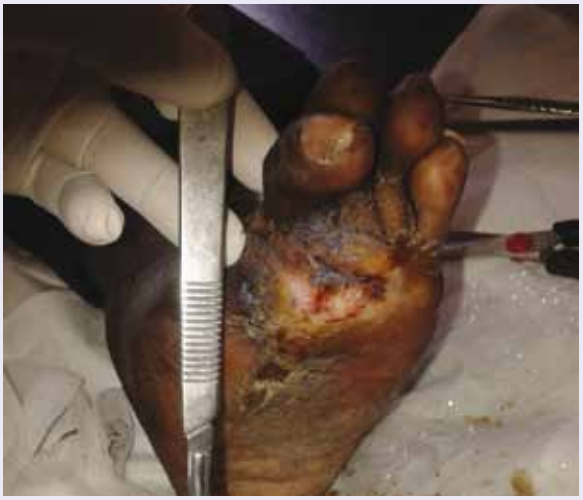

Fig 2. Wound at final consultation (week 12)

Bahle Ntelek and Mazizi Njokwenii

- Remove or absorb excess exudate — increased exudate encourages bacteria growth and can cause maceration
- Maintain wound bed temperature of 37°C — wound cells function at their best within an environment that has a normal core body temperature
- Be non-adherent to vulnerable tissues — to avoid damaging newly formed granulation and epithelial tissue and causing the patient pain at dressing removal
- Be compatible with other management elements of the foot-care regimen such as offloading devices.[39]

There is no conclusive evidence from large randomised trials that any one dressing is better or worse than another dressing.[40] However, with regards to the DFU, dressings must be easy to apply and quick to remove; they must not restrict mobility or offloading devices, or disintegrate under pressure; and they must be able to control exudate levels. A simplified guide to the use of wound dressings in the management of the DFU is given in Table 2. It is vital to include patients in dressing selection as this will increase the likelihood of adherence with treatment.

Offloading

Although regular foot examinations and early intervention will help prevent DFUs when deformities are present, patients with neuropathic changes will still experience a high incidence of foot ulceration. Offloading mechanisms play a vital role in the management and prevention of diabetic foot complications by redistributing pressure away from the fragile tissue that bears the most weight around the foot, and reducing the friction and shear that can contribute to tissue breakdown. Pressure, friction and shear impair normal capillary perfusion by stretching the blood and lymph vessels, which in turn damages the tissue, with the potential risk of deep tissue injury.[41] Offloading the high-pressure areas will redistribute pressure evenly across the plantar aspects of the feet.[18]

Several methods of offloading can be used to manage DFUs (Table 3). Total contact casting (TCC) is the gold standard treatment. It is indicated for the early phase of Charcot fracture dislocations and patients with existing DFUs. Non-removable cast walkers can be used on patients who do not wish to wear a TCC or who require complete immobilisation of an area to induce healing.

Therapeutic footwear is suitable for at-risk patients or those with a previous history of ulceration. There are numerous types of such footwear modifications, such as open toe, closed toe, custom-made and rocker type soles. These commonly have different indications, which are often outcome dependent. These indications will often be identified by the podiatrist, physiotherapist, or orthopaedic surgeon. Due to their cosmetic appearance, patients commonly prefer these to TCCs. Other devices that may help prevent or treat DFUs are orthotics, simple inner soles, pressure-relieving socks, walking aids and wheelchairs. These will be recommended by a podiatrist and/or orthopaedic surgeon, following assessment.

Table 3. Characteristics of the different categories of offloading devices

Type	Description
Total contact casting (TCC)	· Considered the gold standard · Redistributes pressures evenly over entire plantar surface · Must be applied by a trained practitioner · Permits daily wound inspection · Can reduce healing time by 6 weeks
Non-removable cast walker (non-RCW)	· Commonly composed of plaster of Paris · Not easily removed · Difficult to manage wounds located beneath the device · May cause skin irritation · Prevents daily wound inspection
Therapeutic footwear	· May consist of custom-made modifications e.g. extra cushioning, broad toe box, open toe box · Light weight, stable and reusable · Can increase risk of falls in patients with poor proprioception · Poor/incorrect use may result in further deformity/ulceration
Other devices e.g. orthotics, inner soles, pressure-relieving socks, walking aids and wheelchairs	· May be used in combination with other devices e.g. TCC, non-RCW and therapeutic footwear · Orthotics and inner soles can be custom made to best assist the patient · Walking aids and wheelchairs may require patient to have good upper body strength · Wheelchairs may provide complete offloading of the lower extremity · Wheelchairs may require modified homes eg, space and ramps

Conclusion

Teamwork plays an essential role in the prevention and management of DFUs. It can ensure that risk factors such as foot deformity and high plantar pressure are identified early and are followed by swift referral for treatment and ongoing patient education and support. Just as important is the need to consider the patient as a whole, rather than just focusing on the ulcer. Regular communication needs to be maintained between the patient and MDT as the management of DFUs is multifaceted and time-sensitive, and ulcers may take a considerable time to heal. The use of treatment pathways will ensure that the patient sees the right person at the right time in the right place. Engaging patients in the identification and elimination of risk factors, and educating them about the benefits of treatment options, such as offloading and surgery, will increase the likelihood of concordance and good outcomes.

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Assessing infected ulcers: a step-by-step guide

While every attempt can be made to avoid the development of diabetic foot ulcers, it is inevitable that some patients will present with them. Once they develop, these ulcers are hard to heal, placing the patient at increased at risk of infection and, ultimately, amputation. It is vital, therefore, that health-care professionals are able to recognise the signs of increased bioburden and infection, so that prompt treatment can be given

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Declaration of interest: Paul Chadwick and Joanne McCardle have presented at educational events sponsored by B Braun

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Fig 1. The monofilament test will help determine if peripheral neuropathy, a risk factor for infection, is present

A large proportion of patients with diabetic foot ulcers (DFUs) will develop an infection at some point. This is often due to the chronic, non-healing nature of these wounds: the longer a wound is present, the more likely it is that infection will develop and cause deterioration. In addition, non-healing wounds are far more likely to have a polymicrobial infection. These infections can spread rapidly and, if care is not taken to manage the infection, the wounds can become deep, and osteomyelitis (bone infection) and serious soft-tissue infection may develop.[1] These infected wounds can regress and surrounding tissues can be destroyed within as little as 24 hours to the extent that a limb cannot be salvaged.[2]

Early assessment, identification and prompt management of infection are essential to prevent limb loss in patients with diabetes. In addition, infection in the feet can spread elsewhere through the blood, leading to systemic infection and potentially life-threatening complications.[3]

Assessment of infection

When a DFU is being managed, the risk of infection must be considered at all times. However, the presence of microbes does not mean that a wound is clinically infected. All wounds are contaminated with a variety of microorganisms.[4,5] Normally, these microbes are harmless skin flora that are naturally found on the surface of the skin. Healthy and intact skin provides a physical barrier against these microbes.

Caution must be exercised, therefore, when a DFU develops. Any break in the integrity of the skin, even a simple skin fissure, should be considered as a possible portal of entry for bacteria, and managed in the same way as an ulcer to prevent deterioration. A break in the skin or wound could potentially allow microbes into the wound, which could cause infection. In other words, any

wound, no matter how small or shallow, may act as an open door through which infection may enter as an uninvited guest.

Once microbes have entered a wound, their presence can cause a series of different types of host reactions that can ultimately result in infection. According to the World Union of Wound Healing Societies (WUWHS), the presence of microbes in a wound can be classified into four categories, based on these host reactions:[5]

- **Contamination** — occurs when the microbial burden does not increase or cause clinical problems
- **Colonisation** — occurs when microbes multiply, but wound tissues are not damaged: the wound is progressing through the normal healing trajectory and has none of the clinical signs of infection
- **Critical colonisation** — occurs when microbes multiply and the wound moves from benign colonisation to an infected state with impaired healing but without tissue invasion or a host immunological response.[6] The classic signs of infection are not present, but subtle manifestations, such as wound stasis, friable or discoloured granulation tissue, are present. Currently, there is no consensus on how to define or identify critical colonisation.[7]
- **Infection (spreading or systemic)** — occurs when bacteria multiply, healing is disrupted and deep tissues are damaged. Bacteria might produce local problems, such as necrotic tissue, or cause systemic illness (sepsis).

The concept of wound biofilm has been proposed as an alternative to critical colonisation. This is discussed in more detail in on pages 28–29.

> “Rapid assessment will reduce morbidity, hospital admissions and limb loss”

How to diagnose infection in the diabetic foot

Bacterial infection in the diabetic foot is mainly diagnosed through clinical observation and investigation, and is based predominantly on the typical cardinal signs and symptoms of inflammation.

If you know the cardinal signs to look out for, recognition of infection can be reasonably straightforward. However, the cardinal signs may be masked in patients with diabetes, so infection should not be immediately excluded without a holistic assessment. If a clinician feels they do not have the experience to evaluate whether or not a DFU is infected, then the patient should be reviewed by an appropriate health professional in the multidisciplinary team (MDT).

An assessment should be performed whenever infection is suspected. The Infectious Diseases Society of America (IDSA) guidelines[8] and the Scottish Infectious Diseases Society[9] both state that holistic assessment of infection should be carried out on the three levels[8] outlined below.

Level 1: the whole patient

The whole patient should be reviewed, not just the hole in the foot; in other words, the wound should never be reviewed in isolation. Clinicians should talk to the patient and take a careful history as there could be tell-tale signs in other parts of the body. Patients may present with systemic signs of infection such as:

- Raised temperature — a simple check can determine this
- High respiratory rate — check blood pressure
- Clinical abnormalities in blood test results, such as elevated inflammatory markers and white cell count. (If infection is suspected, a blood test should be arranged immediately)
- Changes in general health — the patient's general health, particularly diabetes management, should be reviewed as infection can sometimes cause erratic blood glucose levels that may be out of character for him or her
- General fatigue and flu-like symptoms.

Level 2: the affected limb

The underlying causes of ulceration must be identified. This extends beyond the foot, and so the affected limb should be assessed. Clinical examination can identify if there is:

- Peripheral neuropathy (Fig 1)
- Peripheral arterial disease
- Spreading infection eg, cellulitis, tracking redness up the affected limb, swelling or pain.

Level 3: the infected foot

The wound should be closely assessed. Clinical examination and good observational skills can identify many of the cardinal signs of inflammation:

- Redness (erythema)
- Warmth
- Swelling or induration (tumor),
- Tenderness and pain
- Purulent secretions.

Classifying infection in DFUs

Once the patient, the limb and the wound have been examined, the infection can be classified. Table 1 shows the continuum between non-infected wounds and severe infection. When infection is present, a decision needs to be made on whether or not to admit the patient to hospital.[8] Severe infection should trigger an immediate admission to hospital. Intravenous antibiotics can then be started immediately. Delay at this stage can threaten both limb and life. Even if wounds are only moderately infected, hospital admission may be required. Intravenous antibiotics can sometimes be administered at home or in an outpatient setting. Outpatient parenteral antimicrobial therapy (OPAT) can be provided only if a local service infrastructure for it is in place.

To swab or not to swab

Taking a wound swab for culture should be seen as an additional aid to management, rather than a diagnostic process. This is because diagnosis is based on clinical signs and symptoms. The key benefit of culture results is that they can be useful in deciding which type of antibiotic to use.

It is not recommended that a wound should be swabbed or cultured unless an infection is suspected. Most people have normal flora on their skin and wounds, which could be picked up on a routine swab. The test would then come back positive because microbiological techniques cannot distinguish between contamination or colonisation and infection. It would be confusing if a clinically non-infected wound produced a positive culture.

For mild to severe infection, deep tissue biopsy or curettage in specialist centres is recommended. For non-specialists in diabetic foot ulceration, the best method is to:

- Debride the wound, but only if this is within your competency
- Cleanse the wound using normal saline in order to remove any debris
- Take as deep a wound swab as possible from the base of the wound.

After the wound has been swabbed, the following should be clearly marked on the request form:

- Site of the ulceration
- Date and time of the swab
- Swelling or induration (tumor),
- Background comorbidities, eg, diabetes
- Current or planned antibiotic treatment
- A microbiological request eg, culture and sensitivity.

Osteomyelitis

A repeated infection that requires antibiotics is usually a sign that the infection has progressed deeper. Osteomyelitis (bone infection) occurs when the infection has penetrated deeper and into the bone. A number of clinical tests can be carried out to

Table 1. Characteristics of the four grades of infection[8]

Clinical criteria	Grade/severity
No signs of infection	Grade 1/uninfected
After eliminating other possible causes of inflammation, such as gout, assess whether two or more of the following signs are present: · Local warmth · Erythema located >0.5–2cm around the ulcer · Local tenderness/pain · Local swelling/induration · Purulent discharge The infection must be local, involving the skin and the subcutaneous tissue only	Grade 2/mild
Erythema located ≥2cm around the ulcer and one of the findings above, or: · Infection involving structures beneath the skin/subcutaneous tissues (eg, abscess, lymphangitis, osteomyelitis, septic arthritis and/or fasciitis) and no systemic inflammatory response (see grade 4)	Grade 3/moderate
Local infection (as described above) and signs of systemic inflammatory response syndrome (SIRS), characterised by the presence of at least two of the following: · Temperature: >38ºC or <36ºC · Heart rate: >90 beats/min · Respiratory rate: >20 breaths/minute or $PaCO_2$ <32mmHg · White blood cell count: >12,000 or <4000 cells/µL, or ≥10% are immature (band) forms	Grade 4/severe

Table 2. Potential indicators of osteomyelitis	
If any of these are present, osteomyelitis should always be considered	
Positive probe-to bone test (PTB)	This is the gentle insertion of a blunt-ended metal probe into the ulcer. If bone can be palpated, bone infection is likely; if bone is not probed, it is unlikely that osteomyelitis is present[11,12]
Swollen or sausage toe (Fig 2)	The affected digit takes on a chronic swollen and often red appearance, like a sausage.[13] This is a potential indicator of osteomyelitis
Non-healing wounds of more than 6 weeks' duration, where all other possible factors in non-healing, such as exposure to pressure and impaired blood flow, have been addressed[13] .	

determine if osteomyelitis might be present. These are outlined in Table 2, although it should be noted that this guidance is for information only, as these techniques should be carried out by the MDT. If you suspect that osteomyelitis is present, refer the patient immediately to a specialist team. None of these tests provides a definite diagnosis in isolation, but their presence adds to the likelihood that osteomyelitis is present.

Definitive diagnosis of osteomyelitis

It is recommended that a plain radiograph should be taken of all new foot infections. However, X-rays are not very sensitive in the early stages of infection and often lack specificity for osteomyelitis late in the course of infection[8,10] Osteomyelitis therefore should not be excluded on the basis of X-ray alone.[3]

If plain X-rays do not confirm osteomyelitis and there is a strong degree of clinical suspicion, then consider:

- Magnetic resonance imaging (MRI).[3] If this is unavailable or contraindicated — for example, if metalwork is present — the next level is:
- Radionuclide bone scan.

The definitive, gold standard diagnosis of osteomyelitis uses a combination of the findings of culture and histology from bone biopsy or bone debridement.[8] Caution is needed when obtaining a bone biopsy and where it is taken from: osteomyelitis could develop in a previously uninfected bone if an instrument is passed through an infected ulcer in order to obtain a bone sample. When diagnosis of osteomyelitis is inconclusive and MRI imaging and histology cannot be promptly arranged, diagnosis must be based on clinical features and any cultures/samples grown.

Challenges in assessing infection and detecting early increased bioburden

Up to 50% of patients with infected DFUs may present **without** the classical signs of redness, heat and swelling.[8] Therefore, infection should not be excluded in a patient who does not have the typical or cardinal signs. The absence of signs and symptoms of infection can be due to:

- A poor blood supply (peripheral arterial disease), which may mask the classic signs of infection, as a good blood flow is required to mount a good inflammatory response
- Sensory neuropathy may prevent the manifestation of pain, even when the wound can be probed to bone or is necrotic

In addition, the patient may be immunocompromised as a result of the diabetes, and so will not experience a normal inflammatory response.

This can be confusing for a practitioner, which reinforces the need for a holistic approach, as this may uncover other factors that will indicate a possible infection. In the absence of heat, redness, swelling and pain, more subtle signs of infection may be apparent in the wound site and surrounding tissue. These signs may include friable granulation tissue, wound undermining, malodour and increasing wound exudate.[2] These signs should never be ignored: they might represent early subdued infection or an increasing bioburden or critical colonisation,[5] which might tip the wound into an infected state.

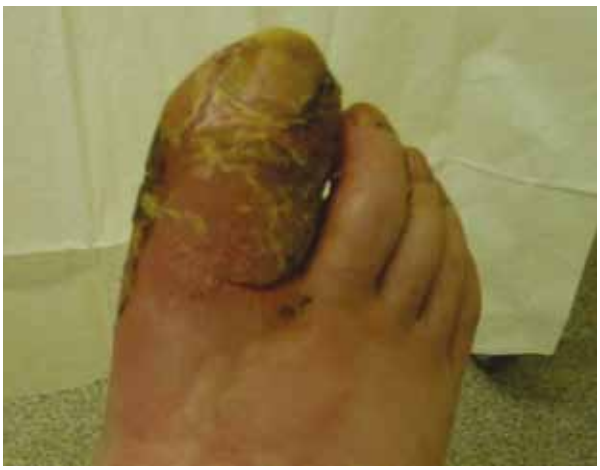

Fig 2. Swollen or sausage toe

Management of diabetic foot infection

The most important consideration for any patient displaying signs of infection, whether they range from mild to severe,[8] is referral to a specialist unit. Once the infection has been recognised and diagnosed, treatment will depend on the severity of the infection.

Rapid assessment and management will ultimately reduce morbidity, hospital admissions and limb loss in this vulnerable diabetes patient group.[8]

Management of infection should ideally be carried out by the MDT.[8] This allows a range of skilled health professionals to holistically manage not only the infection but also the associated complexities of diabetes foot ulceration.

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Clinical scenario: putting the IDSA classification into practice

Patient B, a 59-year-old male with long-standing type 2 diabetes, presents at a clinic with a new ulcer (Fig 1). On examination, there are signs that the patient has lost protective sensation (peripheral neuropathy), yet peripheral pulses are palpable with strong biphasic Doppler waveforms.

When examining the ulcer, you note there is a purulent discharge, the surrounding area is red, hot and swollen, and the probe-to-bone test is positive.

Using the IDSA classification system, what do you think the severity of the ulcer is?

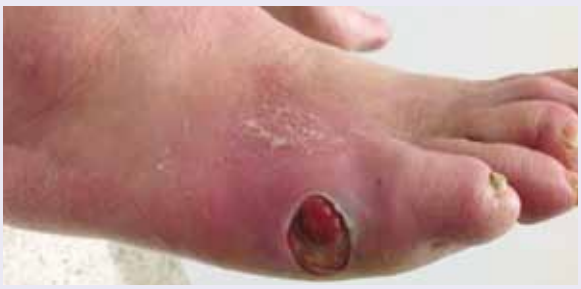

Fig 1. Ulcer at presentation

Answer

Determining whether infection is present is very much a clinical diagnosis. Using your senses is key to diagnosis. Looking at Fig 2, you note that the erythema present is located >2cm from the ulcer, the area is hot to touch and the fifth toe is swollen. When probing the ulcer, you are able to probe to bone. You also note purulent discharge. As standard, you record the clinical observations of this patient. His heart rate 88 bpm, his temperature is 37.4 and he reports feeling well in himself.

Fig 2. The signs of infection present

Using the above information, you refer to the IDSA classification chart. You note there are more than two classic signs of infection and, at this point, you suspect clinical infection is present. When determining the severity of infection, you tick on the IDSA chart that the erythema is >2cm and the probe-to-bone test is positive. This patient does not exhibit any symptoms of systemic inflammatory response syndrome (SIRS) and, as such, you diagnose this infection as IDSA grade moderate.

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Managing infection: a holistic approach

All wound infection presents risks for the patient, but the risks are multiplied in the presence of a comorbidity such as diabetes, when they can potentially be fatal. Where diabetic foot ulcer (DFU) infection is concerned, early recognition is crucial. Prompt treatment, comprising wound cleansing, debridement of devitalised tissue and use of antimicrobial dressings, can stop locally infected ulcers from deteriorating further

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Declaration of interest: none

Wound infection poses a significant risk for any individual. However, in patients with diabetes, it takes on an even greater significance. Serious wound infections can have devastating results and may lead to limb- or life-threatening sepsis, with the risk of hospitalisation and amputation being 56 and 155 times greater in patients with infected diabetic foot ulcers (DFUs).[1] As such, the management and prevention of wound infection is of central importance. Successful diagnosis and treatment of DFUs require a holistic approach involving five areas of management:

- Optimal diabetes control
- Effective local wound management
- Infection control
- Pressure-relief
- Restoring pulsatile blood flow.[2]

Despite awareness of the impact of infection on the management of DFUs, this is still a very real issue in clinical practice. A European study of individuals attending foot clinics with new areas of ulceration identified that 58% had clinical signs of infection,[3] while for an US one-centre study with a similar methodology the figure was 56%.[1] Infection need not have catastrophic results; if recognised early and managed correctly, negative healing outcomes can be avoided and the wound environment optimised to achieve wound healing.

Identification and classification of diabetic foot infection

The previous article in this supplement describes how to assess and classify infection in DFUs. However, not all patients will be aware that their foot is becoming infected. Diabetic patients with increasing bioburden and inflammation will experience an elevated metabolic demand, increasing the need for adequate oxygen and nutrients.[4] This can rapidly tip the equilibrium in those individuals already coping with the effects of poor vascularity because of macro- and microvascular insufficiency.[5] Combine this with sensory neuropathy, which can dramatically alter pain perception, and you will be faced with an individual who not only has inherent difficulty controlling his or her wound bioburden, but who might also have little perception that the infection is rapidly becoming problematic.

Factors that can dramatically increase the likelihood of infection in a DFU are:[6]

- A positive probe-to-bone test result
- Wound chronicity (a DFU present for >30 days)
- A history of recurrent DFUs
- A traumatic foot wound
- Peripheral arterial disease in the affected limb
- A previous lower extremity amputation
- Neuropathy (loss of protective sensation)
- Renal insufficiency
- History of walking barefoot.

How to identify pathogens in DFUs

DFUs with superficial infections that have not recently received antibiotic treatment are likely to be caused by a single Gram-positive bacteria such as *Staphylococcus* or *Streptococcus*. However, deep infections and those in areas of chronic ulceration are often polymicrobial[7,8] and might contain a biofilm.[9]

If wound infection is suspected, specimens should be sent for culture before starting empiric antibiotic therapy.[6] The process of taking a swab for culture is described in the

Clinical scenario: provision of good basic wound care following skin assessment

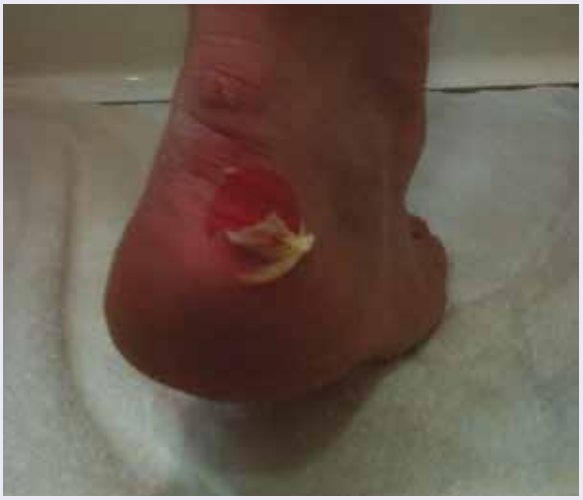

Fig 1. Wound on presentation

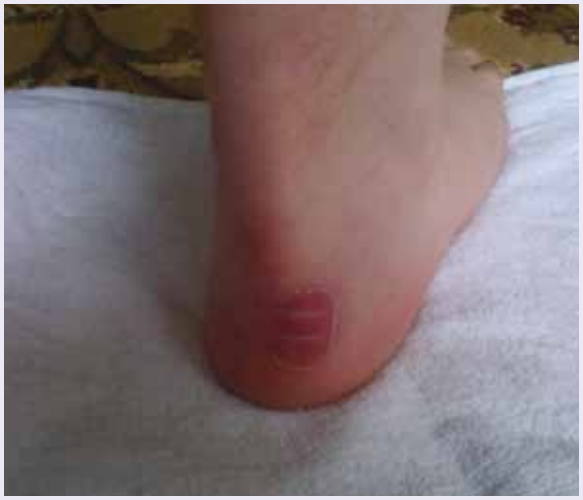

Fig 2. Wound on day 4

Mr A, who is 38 years old with stable type 2 diabetes, is attending a routine outpatient clinic department for a diabetic review and skin assessment. As a result of his obesity and chronic back pain, he is unable to inspect the skin on his foot and ankle. During the assessment, the podiatrist identifies a superficial ulcer on Mr A's right calcaneus. The ulcer is painful and, over the past 2 days, has been producing exudate that has soiled Mr A's footwear. Due to the swelling in his lower limb, the patient has difficulty putting on his shoes.

Question 1: What is the most likely cause of the ulcer?

a. **Neuropathy**
b. **Shear and friction**
c. **Excess moisture**

Answer b. The patient has sensed pain caused by friction and shearing forces as the shoe rubbed up and down his heel. This resulted in a ulcer.

Question 2: Based on Fig 1 above, is the wound bed:

a. **Sloughy?**
b. **Granulating tissue with epidermal de-roofing?**
c. **Necrotic?**

Answer b. The dermal layer is visible, and granular and epidermal layers are partially torn away.

Question 3: Again, looking at Fig 1, how would you cleanse the wound and surrounding skin?

a. **Cleanse with an antimicrobial solution containing PHMB**
b. **Cleanse with soap and water**
c. **Not cleanse in order to protect the granular wound base**

Answer a. The wound bed has been exposed to potential contaminants for 48 hours and so requires cleansing with an antimicrobial solution in order to prepare it for its first dressing application.

Question 4: What dressing requirements will meet this patient's wound-care needs?

a. **Cost effectiveness**
b. **Non-adherence to the wound bed; ability to absorb exudate; cushioned for comfort; atraumatic on application and removal; available to the clinician**
c. **You would use the same dressing as normal**

Answer b. A dressing must meet the patient's needs, Mr A requires a dressing that is comfortable, absorbent and pain-free during application and removal. In addition, his footwear must fit when the dressing is in place, so that it does not cause further damage to his vulnerable skin.

Question 5: On day 4 (Fig 2), after examining the wound and surrounding skin, would you:

a. **Cleanse it with an antimicrobial solution?**
b. **Cleanse it with soap and water?**
c. **Not cleanse in order to protect the granular wound base?**

Answer c. The wound bed is clean and free of debris, and so does not require cleansing. All that is required now is to re-apply the chosen dressing and arrange a follow-up assessment.

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Clinical scenario: avoiding deterioration

The diabetic foot must be regularly checked by a podiatrist or specialist nurse. The skin should be kept clean and shoes inspected regularly for any areas of pressure. The foot in the figure below has obviously not had this regular check. The skin is in a poor state and there is evidence of an emerging ulcer. Due to neuropathy, this patient might be unaware of the threat to his limb. He is an 85-year-old man with type 2 diabetes and has difficulty mobilising.

Question 1: What is the first consideration for treatment?

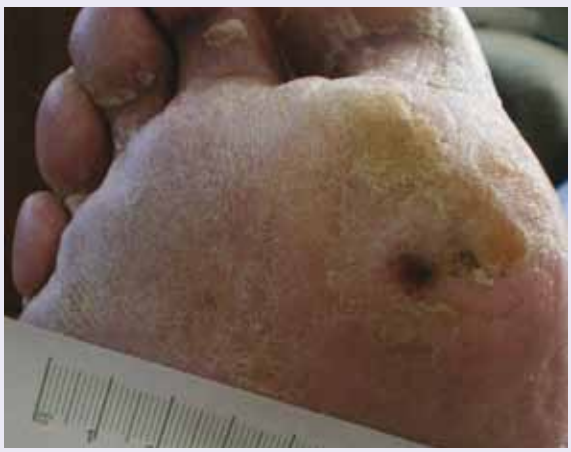
a) Check the shoes and replace with a softer, more accommodating pair
b) Refer to podiatry
c) Cleanse the skin with an antimicrobial solution containing PHMB
d) Offload the area
e) Apply a cream containing 25% urea
f) All of the above

Answer: f

Total contact casting is often used. If planned for this patient, it should be applied as soon as possible to prevent the damage from increasing.

The 25% urea cream is important to keep dry and cracked skin healthy. Higher concentrations of urea penetrate thicker skin better and will ensure it remains moisturised.

The diabetes specialist nurse or tissue viability nurse should always liaise with the podiatrist. Between them, they should ensure that the foot is kept healthy, thereby reducing the potential for further ulceration and, ultimately, amputation.



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previous article. Samples should be cultured for both aerobic and anaerobic organisms.[2,10]

Which antimicrobials to use

Topical antimicrobials

Management of infection should be based on the presenting signs of severity of the condition.[6] Superficial wounds with no systemic or local signs of infection require only local wound cleansing and simple dressings; the use of systemic antimicrobials is not indicated here.[6]

In recent years, topical antimicrobial agents have become the first line of treatment for managing bacterial burden in many disciplines, particularly tissue viability.[11,12] Their prophylactic use remains controversial, but can be justified in immunocompromised individuals or where there is a high risk of infection.[11-13] Current opinion suggests that the ideal antimicrobial is:[14]

- Associated with minimal systemic absorption
- Effective against likely contaminants and pathogens
- Fast-acting and close to the wound, with prolonged residual activity after a single dose
- Inexpensive
- Incapable of promoting bacterial resistance
- Non-carcinogenic and non-teratogenic (does not cause DNA damage that could result in carcinoma or foetal abnormality) to host cells
- Non-toxic
- Widely available.

Dressing materials containing antimicrobial components should be capable of bactericidal activity against planktonic bacteria and, when used in combination with regular debridement, wound cleansing and good wound management, wound biofilm. In addition, the antimicrobial component must remain active for the duration of the dressing wear time, which can be 24 hours or more.[15]

The main compounds contained within antimicrobial wound dressings are silver, iodine, chlorhexidine, polyhexamethylene biguanide (PHMB) and honey. The rapid rise in the availability and variety of antimicrobial wound dressings is testament to the growing significance placed on the control of clinical bioburden. Even though there is limited evidence from randomised controlled trials (RCTs) on their efficacy, robust bodies such as the World Union of Wound Healing Societies (WUWHS)[16] and the European Wound Management Association (EWMA)[17] support the use of topical antimicrobials, including wound dressings, in the management of local wound infection.

Topical antimicrobials should be used only on high-risk patients with critically colonised wounds, on wounds with localised and/or spreading infection,[7] or as an adjunct therapy

to systemic antibiotics. Benefits include their ability to reduce the bacterial load, penetrate tissue where the vascular supply is compromised, and the minimal risk of microbial resistance and systemic complications.[16,18] By promoting a bactericidal and/or bacteriostatic environment, the balance of bioburden within the wound can be restored, enabling the body's natural defences to overcome the insult. This can help avoid the development of minor localised infection, with its potential to become systemic and require antibiotics.

> "Successful treatment of infected diabetic foot ulcers requires a holistic approach"

Safe practice

As stated above, topical antibiotics should be avoided in contaminated or colonised wounds, which, in line with the principles of wound bed preparation, should be thoroughly cleansed and debrided as necessary. It is recommended that the condition of the wound should be regularly assessed (at every dressing change and then formally after 2 weeks) in order to ensure that the antimicrobial dressing is used only as indicated. If there has been a reduction in wound size, exudate and pain, and the bioburden appears to be under control, then switch to a non-antimicrobial dressing. If the wound was initially critically colonised and has failed to progress or has deteriorated, consider referring for specialist advice. If the antimicrobial dressing has been used in combination with antibiotics and there has been no improvement after 14 days, re-assess the wound and re-evaluate the holistic management of the patient. Consider whether interventions such as debridement will kick start the inflammatory stage, ensure that the antibiotic regimen is being adhered to and reflects the laboratory culture results, that the wound management is evidence based, and the patient is adherent with therapy including the use of offloading devices. Finally, antimicrobial dressings should be discontinued if the patient has a sensitive reaction to them.[15]

Systemic antimicrobials

These are indicated where signs of localised, advancing or systemic infection are present. The route and specific type of systemic antimicrobial used will depend on the severity of the clinical signs, the structures involved, the immunocompetence of the individual and the local guidelines on antibiotic administration in place.[6]

Results of microbiological culture will determine how antibiotic therapy should be adjusted to target the specific pathogens found within the wound. However, the results will take time to obtain. As it is essential that antibiotic administration is not delayed, broad-spectrum antibiotics are likely to be required until definitive culture results are available. In severe, non-responsive or spreading infections, or where serious osteomyelitis is suspected, hospitalisation and intravenous (IV) antibiotic therapy may be indicated, especially if there are systemic signs of infection.[6]

There is no evidence to support the superiority of any specific antibiotic regimen in the management of DFU infections.[6,19] Most health-care communities recommend an antibiotic pathway that is judged effective against the bacteria likely to colonise wounds. This is specific to the microbiological flora present in the local area and the known susceptibility of these bacteria to specific antibiotics. It is also determined by the wound characteristics and the need to minimise the development of antibiotic resistance.[20] In the absence of such an antimicrobial formulary, information on commonly effective oral agents for the empiric treatment of mild to moderate diabetic foot infection can be found in Lipsky et al.[6]

Treatment of infections

Basic wound care

When treating wound infections, the clinician should first seek to reduce the potential for further wound contamination; sterile dressing products and instruments should be used, while effective hand washing is vital. Gloves should be worn to avoid cross-infection.

Wound cleansing is necessary to remove debris such as pus and slough, and to enable thorough assessment of the wound bed. More detail on wound cleansing is given in the second article in this supplement. After cleansing, the wound should not be left exposed as this will greatly increase the risk of cross-infection to other sites or individuals. This is especially significant when there are ulcers on both feet. Use of wound dressings that act as a barrier to bacterial ingress will help avoid this.

Dressings should be selected that meet the needs of both the patient and the wound characteristics. If wound bioburden is an issue, an antimicrobial dressing can be of

benefit until the balance of bioburden is tipped in favour of the patient's natural defences. At this point, it should be possible to safely change to a more inert dressing material. Again, more information about wound dressing requirements can be found in the second article in this supplement.

Mild infection

When wounds with clinical signs of mild infection are being treated with antibiotic therapy, they can be covered with a conventional wound dressings. However, as stated above, the WUWHS[16] and the EWMA[17] recommend the use of topical antimicrobial dressings in the management of mild, localised infections. Such dressings may be able to prevent the development of more serious sepsis and avoid use of systemic antibiotics. Wounds treated in this manner need to be closely monitored and, if deterioration occurs, appropriate antibiotics should be commenced immediately. Alternatively, antimicrobial dressings can be used in conjunction with oral antibiotics from the start. This regimen is indicated when the patients' immune response is believed to be suboptimal or increased antimicrobial penetration is considered necessary.

Oral antibiotic therapy should cover activity against Gram-positive *Staphylococci* and *Streptococci*. If a single agent fails to address the infection, a second antibiotic is added. Empiric therapy against meticillin-resistant *Staphylococcus aureus* (MRSA) should be considered if the patient has a previous history of carriage/infection, if there is a high incidence of MRSA infection in the population or if the infection is resistant to treatment.[6,21]

Moderate infection

Moderate infections involving deep tissue are commonly polymicrobial and usually require a combination of antibiotics to cover both Gram-positive bacteria (e.g. *Staphylococci*, including MRSA) and Gram-negative bacilli. Anaerobic bacteria may also need to be managed. *Pseudomonas aeruginosa* is frequently cultured in chronic and/or heavily exuding wounds and in hot climates. These bacteria do not always inhibit wound healing, however; if they become problematic, a targeted therapy approach is needed.[2,22] Antimicrobial dressings can be used to provide broad-spectrum activity against aerobic and anaerobic bacteria, spores, viruses and fungi.

Severe infection

This poses a significant risk to the limb and the individual as, without effective treatment, local tissues can rapidly face massive destruction, the release of cellular and bacterial toxins and the induction of unstable catabolic processes. As such, these infections require intense monitoring and aggressive management. Invariably, the patient will need to be admitted to hospital, even if only until infection control is established and blood glucose and general metabolic equilibrium is achieved.[6]

Wukich et al.[24] identified that the median hospital stay for individuals with severe diabetic foot infections was 60% longer than that of patients with moderate infections. Severe and some moderate infections normally require at least local wound debridement to reduce the necrotic and bacterial burden. However, this may be extensive, depending on the amount of damage already present and the viability of the remaining tissue. Wukich et al.[24] found that, in their study population, 55% of patients with severe diabetic foot infections required some type of amputation, compared with 42% in the moderately infected group. However, major amputation is not always required.

Severe infections require the prompt administration of a broad-spectrum antimicrobial regimen, usually intravenously for the first few days of treatment. This can then be changed to a more targeted approach when the results of microscopy and sensitivity testing are available. Similarly, it may be possible to switch to oral medication when the patient's systemic symptoms have improved.[6]

Continued IV administration may be considered where response to treatment is poor or where tissue penetration of antimicrobials may be difficult to achieve, such as in osteomyelitis or in the presence of peripheral arterial disease. This may be continued in the community setting if the patient is systemically well enough and these facilities are available locally. The duration of antimicrobial therapy required depends on the severity of the infection, the presence or absence of osteomyelitis and the clinical response to treatment.[6] Information on IV agents for empiric treatment of moderate to severe diabetic foot infections can be found in Lipsky et al.[6]

Debridement

Debridement is the removal of non-viable tissue and debris from the wound. This includes necrotic tissue from within the wound, non-viable or dysfunctional wound and/or peri-wound tissue such as callus, tissue with heavy bioburden, biofilm and debris, and may also include senescent tissue from the wound bed. (Senescent tissue is pale anaemic tissue that fails to progress through the normal healing process.) The role of debridement is to prepare the wound bed for tissue repair by reducing or removing non-viable tissues (often referred to as necrotic burden). This tissue can act as a culture medium for bacterial growth: it inhibits phagocytosis, prolongs the inflammatory response and delays wound healing. By removing dysfunctional tissue such as peri-wound callus, excessive hyperkeratotic skin squames and fibrotic anaemic granulation, it is possible to optimise the potential for healing and restore the natural acute healing response.

Similarly, by draining purulent exudate and removing heavily contaminated tissue and debris, it reduces the bacterial load (often referred to as bacterial bioburden), restoring the balance in favour of the host's immune response.[25,26]

Debridement can be an extensive surgical procedure performed in an operating theatre or a more limited treatment-room procedure. It can be a one-off procedure or it may be required on an ongoing basis to manage the wound and peri-wound tissues.[27]

Not all wounds are suitable for debridement. It is generally contraindicated in dry ischaemic ulcers, or where there is a high risk of exposure and damage of deep structures. It may also be deferred if there is no potential for wound healing.[28] It is essential, therefore, to thoroughly assess the individual, his/her wound and to have a clear rationale and plan for treatment.

There are a number of methods of debridement to choose from. Main determinants include: the patient's general condition, the specific wound characteristics, the clinician's skills and availability of resources, patient choice, the speed at which debridement needs to be undertaken, and the cost of the method used.

Types of debridement

No one method of debridement has been found to be effective in achieving complete healing in all wounds.[29] However, the gold standard method in the management of DFUs is regular, local, sharp debridement.[7] This entails use of surgical blades, scissors or curette to rapidly remove necrotic tissues and debris from the wound bed. This must be undertaken by a suitable trained and experienced health-care professional, usually in a clinical environment.[28] Contraindications are ischaemic ulcers or a high risk of bleeding — for instance, if the patient is taking anticoagulant therapy.

Peri-wound callus and excess skin scales from the surrounding area can normally be safely debrided by an appropriately trained health-care professional, such as a podiatrist. Dense skin is usually removed with a sterile surgical blade; however, diffuse areas of hyperkeratosis can also be controlled by the judicious use of a monofilament polyester/polyacrylic debridement pad.[30] More radical wound debridement, which may include resection of infected bone, is usually carried out under anaesthetic by surgeons in an operating theatre and is referred to as surgical debridement.

However, there are a number of other wound debridement techniques that may be employed:

- Autolytic debridement: usually done at the outpatient setting using hydrogel or a hydrocolloid to remove dry necrotic tissues from the wound bed. This may be considered the least traumatic route to debridement; however, it relies heavily on adequate tissue perfusion and leucocyte activity. It also takes the longest amount of time

Clinical scenario: preventing infection

Mr A presents at your clinic with a new ulcer in his medial arch caused by a new pair of shoes rubbing his foot. He only noticed the ulcer when he saw blood on his socks and cannot recall how long it has been present. This patient clearly has loss of protective sensation and you know he has a background of peripheral vascular disease.

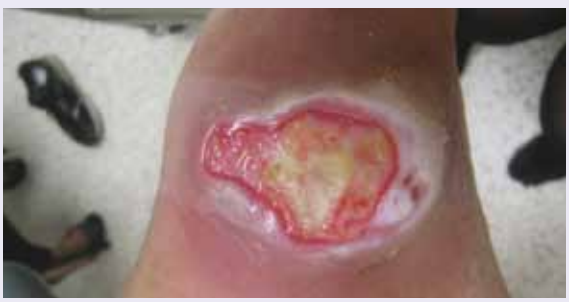
Question 1: What is the most likely ulcer aetiology?
a. Ischaemic b. Neuropathy c. Neuroischaemia
Answer: c

Question 2: Looking at the figure below, how would you describe the wound bed?
a. Clean and granulating
b. It has lots of fibrin slough with some granulation
c. Necrotic
Answer: b

You suspect the ulcer looks infected and immediately refer Mr A to his GP for oral antibiotics. As the wound has an ischaemic component, you do not debride the wound bed.

Question 3: Which of the following dressings options do you think is most suitable?
a. Hydrogel b. Antimicrobial c. Hydrocolloid
d. Alginate e. Foam
f. A product that has a combination of these
Answer: There is no right or wrong answer, only the clinician's perspective.

This demonstrates the vast array of products you could use. Here, an antimicrobial product that is in a gel or paste form, such as an ionic silver alginate dressing, could be useful. The antimicrobial component would reduce bioburden, while the gel/paste would promote autolytic debridement to help reduce fibrin slough. A secondary dressing is needed.



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to perform. It may therefore not be considered effective or appropriate for use in many individuals with DFUs

- Enzymatic debridement: agents such as papain, bromelain or bacterial collagenase are occasionally used to remove hard devitalised tissue from the wound bed
- Biologic debridement: specially bred medical larvae, when applied to the wound bed, can ingest devitalised tissue and bacteria. They are also believed to promote granulation tissue formation. Their use is limited, due to their unavailability in some countries and the high unit cost of treatment
- Mechanical debridement: this is one of the oldest methods of wound debridement. Gauze dampened with saline is applied to the wound bed and left to dry. On removal, it takes with it any exudate, debris and devitalised tissues present on the wound surface. This is usually painful and carries the risk of removing granulation tissue from the wound bed. Its use is therefore in decline globally, as more effective and atraumatic methods become widely available
- Hydro-surgical technique: this implies the use of a high-velocity jet of water to remove all necrotic tissue and suctioning all debris from the wound. It is becoming more popular, but is expensive and used mainly on hospital inpatients
- Ultrasonic-assisted debridement: a special device applies a low-frequency wave 25 kHz with irrigation fluids to remove devitalised tissues and bacteria from the wound bed. It is easy to use, requiring minimal training, and can be undertaken in an outpatient setting. The relative high cost of this technique may make it unavailable in many health-care environments.

Table 1. Potential advantages and disadvantages of initial primary surgical or primary medical treatment for diabetic foot osteomyelitis[38]

Surgical	Medical
Advantages	
Removes necrotic bone *	Avoids surgical procedure
Removes bacteria and biofilm*	Potentially avoids hospitalisation
Removes bony prominences*	Preserves more of the foot
Opportunity to stabilise the foot	May shorten duration of the hospital stay
Disadvantages	
May increase risk of reulceration	Increases risk of infection recurrence
Expensive	Risk of reulceration if foot deformity is not corrected
Risk of operative morbidity	Antibiotic-related toxicities
May destabilise the foot	Risk of developing antibiotic resistance
Risk of transfer ulcers	Risk of *Clostridium difficile* infection

Exceptions to each of these items may apply in individual cases or in specific health-care settings.
*May only be partial or temporary

When to refer to a surgical specialist

The management of DFUs is best undertaken by the multidisciplinary team.[31-37] Although most patients can undergo local wound debridement in an outpatient setting by an appropriately trained nurse or a podiatrist, there are instances where a more aggressive approach is indicated. This may include surgical intervention. The Infectious Diseases Society of America (IDSA) recommends that patients with severe infections and complications such as peripheral arterial disease or inadequate home-care facilities should be hospitalised, at least in the initial stages of infection management.[6] This may also include patients who are not improving on outpatient therapy. Clinically, this represents patients who require deep extensive debridement, have a tendency to bleed and/or are at significantly increased risk of osteomyelitis. Wherever possible, these patients should be managed and coordinated by clinicians who can mobilise adequate resources and expertise to ensure good clinical outcomes.

Surgery versus antibiotics

Traditionally, surgical intervention has been a central pillar in the management of DFU infection, with radical soft-tissue or bone debridement being commonplace. However, this view is now changing. Although surgical procedures to optimise vascular perfusion are increasing in importance, radical debridement is now less common. Both surgical and medical approaches to the management of osteomyelitis have their advantages and disadvantages (Table 1).

In some cases, an aggressive surgical approach is clearly required — for instance, in the presence of virulent, life-threatening infection; conversely, some individuals will clearly benefit from a medical approach. However, debate continues as to whether surgery is more effective than antibiotic therapy alone in the management of uncomplicated infected wounds, as discussed by Lipsky in 2012.[6] Lipsky later stated that, due to a lack of robust research studies, there is no accepted standard in place.[38] However, research by Lázaro-Martinez et al.[39] appears to identify that a medical-only approach might be appropriate for some individuals. Their study, despite containing methodological

Clinical scenario: identification and management of infection in an unwell patient

Mr B presents at your clinic with a black toe that has been present for the past few days. The patient also reports that his leg has been hot and throbbing for at least 2 weeks. Today, he feels unwell, lethargic and is nauseous. Your first step is to define whether or not the patient has an infection and, if so, to grade its severity.

Question 1: In order to do this, you must take this patient's clinical observations. Which of the following observations would you most likely associate with the above clinical picture?

a. **Heart rate 82 bpm, blood pressure 140/75mmHg, temperature 37.4˚C**
b. **Heart rate 98 bpm, blood pressure 140/40, temperature 38.9˚C**

Answer: b

Question 2: Using the IDSA classification, how would you score this patient?

a. **Uninfected**
b. **Mild Infection**
c. **Moderate Infection**
d. **Severe Infection**

Answer: d

Question 3: What is your immediate management plan?

a. **Provide oral antimicrobials and send the patient home**
b. **Provide outpatient-based intravenous antimicrobials and send the patient home**
c. **Admit the patient to hospital**

Answer: c

Question 4: The patient is admitted to hospital. In order to ensure he receives optimal care, which of the following examinations is required as a minimum to draw a clear and concise clinical picture of the patient?

a. **Full blood count including inflammatory and metabolic markers (including HbA1c)**
b. **Plain X-ray**
c. **Blood cultures**
d. **Wound culture**
e. **Assessment of peripheral neuropathy**
f. **Assessment of peripheral vascular disease**
g. **Full and detailed patient history**
h. **All of the above**

Answer: h

A detailed medical history and clinical examination is the starting point. This should determine if the patient has peripheral neuropathy and/or peripheral vascular disease, and whether further vascular examination is required. Start by feeling for both foot pulses (dorsalis pedis and posterior tibial) and listening to the Doppler waveforms. Duplex ultrasound or CT angiography may be needed to aid pre-operative planning.

A plain X-ray will help identify any bone involvement and gas within the soft tissues. A full blood count will determine the systemic extent of any infection, including non-specific inflammatory markers, and any metabolic abnormalities. Blood cultures will help exclude bacteraemia.

While a patient with complex soft-tissue infection will initially be treated empirically, it is highly desirable to obtain sufficient cultures to guide treatment.

This scenario is based on a real patient presentation. The patient, who had an infected ulcer on the apex of the second toe, was admitted to hospital under the vascular surgery team. He often walked barefoot and this, plus the loss of protective sensation and trauma, resulted in ulceration. A break in the skin led to clinical infection. The patient had a history of poor glycaemic control, which can impair the immune response. This was compounded by the peripheral vascular disease.

X-ray revealed osteomyelitis in the toe, as well as gas within the soft tissues. Within 12 hours of admission, the patient underwent an amputation of the second toe with extensive debridement to remove all necrotic tissue.

Although the patient was treated empirically with intravenous antibiotics, subsequent tissue culture revealed MRSA and the antimicrobials were later directed towards this. The medical team also worked to stabilise the metabolic abnormalities of this patient, who required revascularisation through percutaneous angioplasty at a later date.

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Identification and management of biofilm in diabetic foot ulcers

Over 90% of chronic wounds contain bacteria and fungi in the biofilm phenotype and this frequently contributes to delayed healing.[1] Biofilms are complex microbial communities that are attached to the wound bed and embedded in a protective self-secreted matrix or extracellular polymeric substance (EPS), which enables them to survive environmental stresses including systemic and topical antimicrobial agents.[2] While the role of biofilm in chronic wounds, including diabetic foot ulcers (DFUs), remains poorly understood, it is recognised as a significant barrier to healing.[3]

Identification

Recognition and identification of biofilm is a complex specialist task, and the only definitive techniques available to detect it rely on advanced microscopy or specialist culture techniques.[4] As these diagnostic techniques are not easily available in most clinical settings, a more practical approach is required. It is increasingly suggested that a biofilm-dominated wound exhibits particular clinical signs and symptoms including:

- Significant exudate
- Poor-quality granulation tissue
- Tenderness
- Reactive hyperaemia around the wound
- Progressive necrosis across the wound bed
- Quick reformation of surface substance
- History of antibiotic failure or persistent recurrent infections despite the appropriate use of antimicrobials
- Infection lasting more than 30 days
- Negative culture results despite high clinical suspicion
- Response to corticosteroids and TNF-alpha inhibitors
- Recalcitrance despite appropriate wound management and host support.[4-6]

However, as with other types of infection, some of these signs may be suppressed in patients with DFUs, due to the abnormal inflammatory response.[5] Metcalf et al. propose a mechanism for diagnosis that focuses on both visual and indirect indicators (Fig 1, opposite).

Activity

Consider wounds that you have recently managed. How many of the indicators in the algorithm have you seen?

Management strategies

Given the high incidence of microbes in DFUs, it is highly likely that these ulcers will contain biofilm, and so an appropriate management strategy should be followed. Most publications on biofilm management focus on three key activities:

- Debridement
- Cleansing
- Appropriate wound management.[7]

Debridement and cleansing are designed to reduce the biofilm burden, while use of an appropriate dressing should prevent recontamination and suppress biofilm reformation.[2].

Mechanical debridement is key, both to fully visualise the wound bed and disrupt the biofilm. Wolcott et al.[8] identified that biofilm is considerably more susceptible to antimicrobial treatment for 24–48 hours post-debridement, and so serial debridement will help remove more mature biofilm, while a topical antimicrobial will address immature, less well-formed biofilm. Sharp debridement is the most significant mechanism in the prevention and control of biofilm .[4]

Cleansing with antimicrobial agents on their own has little impact on the biofilm as their ability to penetrate the EPS is minimal. However, once physical debridement has disrupted the biofilm, it is more susceptible to external agents. Use of cleansing agents that contain surfactants also encourages removal of surface debris and bacteria.

Appropriate wound management helps prevent the reformation/re-adhesion of the biofilm matrix. The wound should be dressed with an appropriate antimicrobial dressing, based on the presenting clinical symptoms. This provides some antimicrobial protection for the wound. At present, there is much debate about the effectiveness of individual antimicrobial agents in the management of biofilm. However,

flaws, suggests similar healing rates, healing times and short-term complications in patients with neuropathic forefoot ulcers complicated by osteomyelitis who were randomised to receive either antibiotics or conservative surgery.[39] They found that, at least in individuals without necrotising soft-tissue infections, peripheral arterial disease and extensive foot ulceration, use of antibiotic therapy alone effectively managed osteomyelitis. This may mean that, for some, extensive surgery, with its inherent anaesthetic risk and resultant altered foot biomechanics, can be avoided. However, until further studies can be undertaken to support and build on these findings, it is likely that a combined

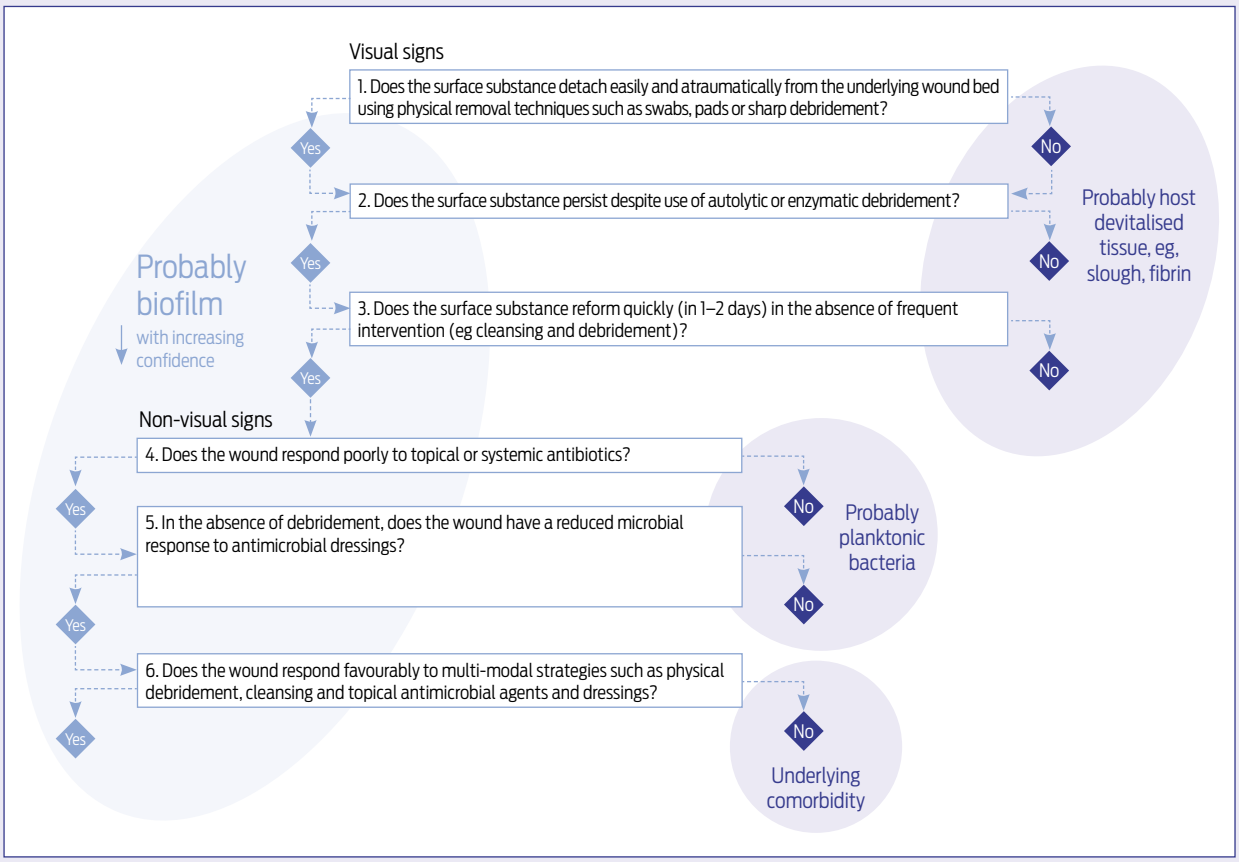

Fig 1. Proposed mechanism for the diagnosis of wound biofilm[5]

their effectiveness is underpinned by appropriate wound bed preparation.[7]

There is a growing interest in and increased clinical requirement to find a simple way of identifying biofilm in wound samples.[3] Until this is possible, it appears that it is becoming increasingly important to use clinical signs and symptoms as indicators of the presence of biofilms in DFUs, in order to deliver appropriate and timely care.

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surgical and medical intervention approach is needed in most cases.[6]

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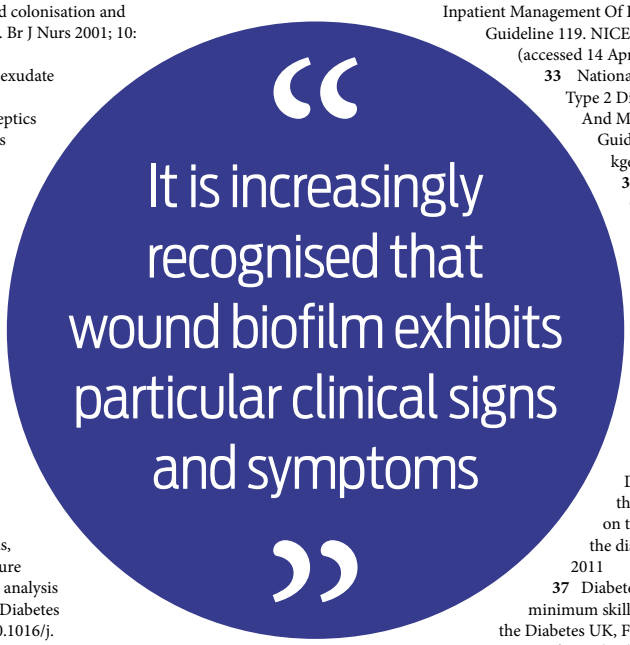

The deteriorating DFU: prioritising risk factors to avoid amputation

The risk of amputation in a deteriorating diabetic foot ulcer is high. This article identifies the three major risk factors associated with such an infection — tissue loss, ischaemia and infection — and explains how to identify which risk is most prominent, and what to do to reduce the risk of amputation. Examples are included of how this approach has led to successful patient outcomes

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Declaration of interest: none

Peripheral arterial disease and diabetes polyneuropathy are the precursors of lower extremity ulceration. They can predispose a limb to further tissue loss and place it at risk of infection. In the unfortunate event that this process ensues, the likelihood of amputation can increase. The treatment of diabetic foot ulceration requires close attention to the three factors that place a limb at risk of amputation: tissue loss, ischaemia and infection. These factors are like three intersecting rings that jockey for dominance over a patient's limb (Fig 1).[1] Clinicians must learn how to determine which factor is more dominant. This will help them ascertain which aspects of management should be given priority. Understanding the dynamic between these factors will shed light on what can be done to preserve or salvage a limb. This article provides clinical examples of each of these three rings of dominance. Case examples are also given to illustrate the application of this concept to clinical practice and implementation of the principles of limb salvage.

Tissue loss

Fig 2 shows a precarious plantar-based ulceration in a diabetic patient with polyneuropathy. There is no risk of ischaemia as non-invasive vascular studies have revealed adequate perfusion and vascular run-off to the distal extremity. Initially, treatment focuses on reducing the chronic, indolent nature of the wound thorough sharp debridement of devitalised tissue,[2] offloading[3] and the application of simple moisture-retentive dressings. Once such an ulcer is healed, it is paramount that the tissue is protected in order to reduce the risk of recurrence.[4,5] This can be

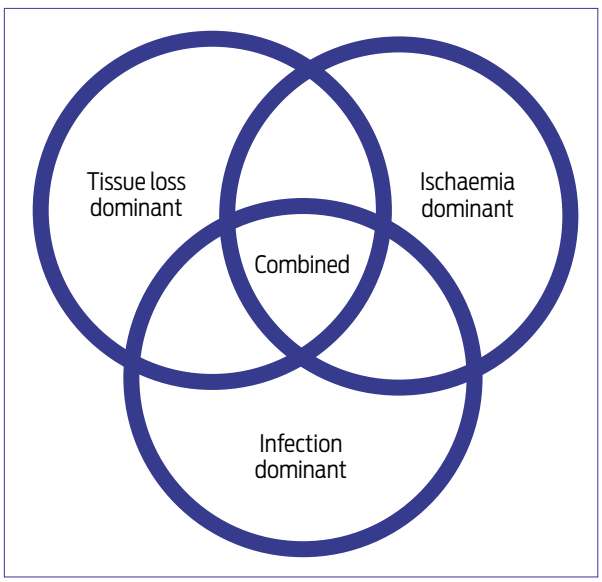

Fig 1. The three risk factors for amputation illustrated as three intersecting rings of dominance (adapted from Armstrong and Mills)[1]

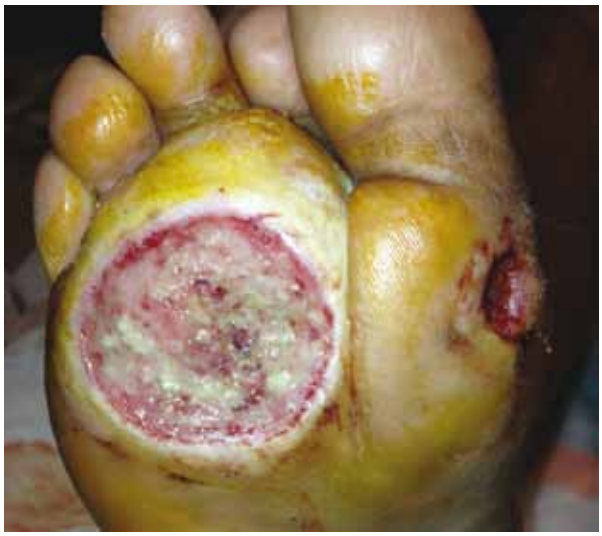

Fig 2. Tissue loss-dominant ulcerations with an aetiology secondary to diabetic polyneuropathy and digital deformity, which increases the forefoot pressure and causes thinning of plantar fat padding. This leaves the area susceptible to tissue breakdown

achieved with the use of custom inserts and depth inlay shoes. Often, abnormal biomechanics, such as a tight gastrosoleal muscle complex or clawed/hammer toe deformities, which contribute to the formation of these types of forefoot ulcerations can be addressed through surgical intervention.[6-9]

Ischaemia-dominant lesions

Fig 3 shows a dry gangrenous fourth digit with surrounding ischaemic-appearing tissue. The fifth digit has been amputated because of osteomyelitis and gangrene. The ischaemic area has progressed to encompass the fourth ray. There were no concerns about acute infection. The patient did not have palpable pedal pulses.

Treatment of this lesion involved a thorough vascular assessment with a lower extremity pulse and Doppler examination, as well as non-invasive vascular studies including ankle brachial pressure indices, plethysmography, digital pressures and arterial duplex imaging. In more urgent cases, such as acute limb ischaemia, some vascular specialists will opt for more advanced vascular assessments

> “Assessment is easier when you understand the three factors that place a limb at risk of amputation”

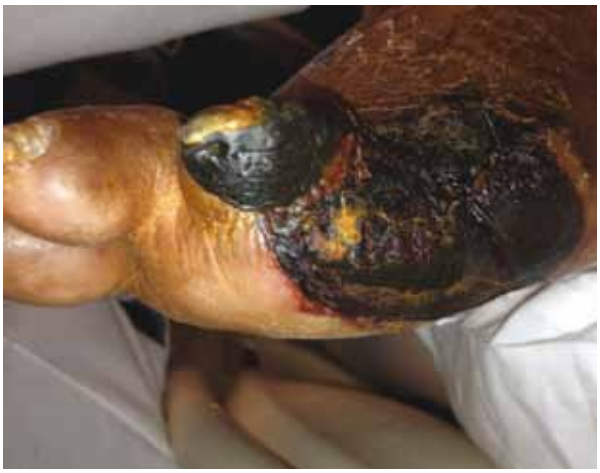

Fig 3. An ischaemia-dominant lesion with dry gangrenous tissue

with magnetic resonance angiography. The patient's overall medical status also needs to be evaluated and, depending on his or her condition, a strategy will be put in place either to monitor the foot or pursue vascular intervention.[10,11] If revascularisation is undertaken and successful, the tissue-loss ring may then become the more dominant factor. In these types of gangrenous conditions, limb salvage necessitates transmetatarsal amputation.

Infection-dominant lesions

Fig 4 depicts an acutely infected right foot. The patient has palpable pulses and the plantar aspect of the big toe is ulcerated. Thus, there is a component of tissue loss. However, in this case, given the acute signs of infection including increased redness, swelling, pain and purulent drainage, along with an elevated white blood cell count and markers of inflammation, the dominant ring is infection.[12] Plain radiography will need to be ordered if there is a strong suspicion of deep bone involvement and to evaluate for soft-tissue emphysema. Treatment involves a combination of surgical and medical interventions.[13,14] Once the infection is suppressed or eradicated, the dominant ring would then shift to become tissue loss. Treatment strategies would focus on wound closure and skin coverage. This may be accomplished with negative pressure wound therapy (NPWT) and split-thickness skin grafting.[15-17]

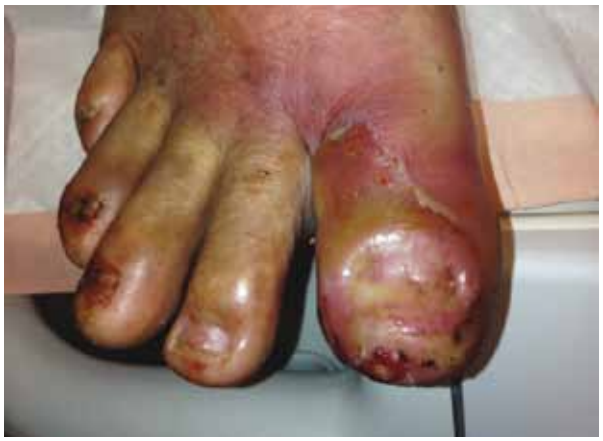

Fig 4. An infection-dominated foot with cellulitis and a deep-tissue abscess

Case studies

Here, real-life clinical scenarios are used to illustrate the three rings or factors that place a limb at risk of amputation.

Case study 1

A 68-year-old male with a past medical history of diabetes mellitus and peripheral neuropathy attended the emergency room complaining of increased pain, redness and swelling in his right foot. He related a 3-day history of worsening symptoms. He also complained of fevers, chills, nausea and vomiting, which he attributed to his history of gastroparesis.

Physical examination of the right foot revealed a dorsal-lateral foot ulceration overlying the fifth metatarsal region, with a necrotic-appearing wound bed with surrounding erythema, calor (heat) and oedema extending towards the midfoot. The infection extended directly from an ulcerated callus underlying the fifth plantar metatarsal phalangeal joint. On examination, soft-tissue fluctuance and purulence were noted (Figs 5a and 5b). Plain radiography revealed cortical lysis of the proximal phalanx and fifth metatarsal head. Magnetic resonance imaging revealed findings consistent with septic arthritis of the fifth metatarsal phalangeal joint and associated osteomyelitis of the proximal phalanx and metatarsal head. Non-invasive vascular studies showed no peripheral arterial disease.

Question: What appears to be dominant? Tissue loss, ischaemia or infection?

Answer: This is an infection-dominated foot. There is certainly a component of soft-tissue loss, given the quarter-size lesion. The necrotic appearance is a result of soft-tissue death from the infectious process and not from ischaemia.

Question: How is this condition addressed?

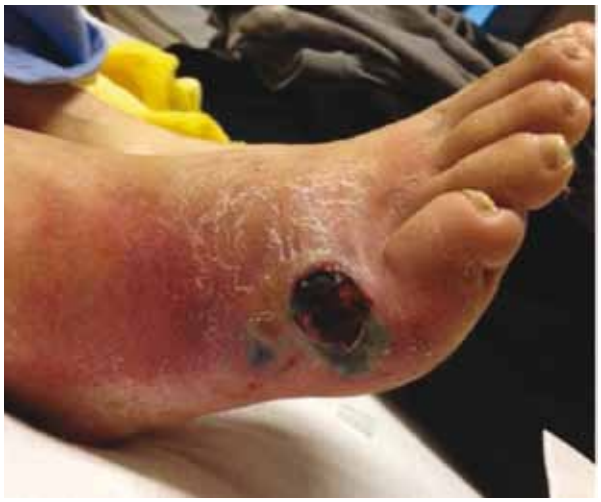

Fig 5a. A dorsal-lateral foot ulcer with necrosis in the wound bed and surrounding erythema, calor and oedema

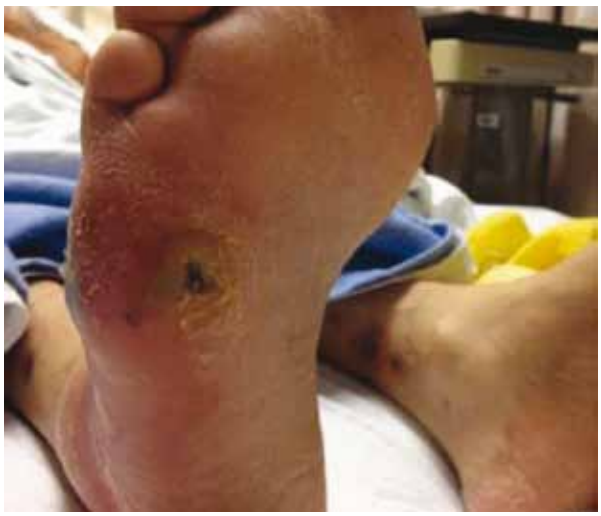

Fig 5b. The infection extended directly from an ulcerated callus underlying the fifth plantar metatarsal phalangeal joint

Answer: A combination of surgical and medical interventions is necessary for limb salvage. The patient underwent surgery to drain the infection and to debride and excise the devitalised bone and soft tissue. Deep tissue and bone specimens were collected for culture and histopathological analysis. Broad-spectrum antibiotic therapy was initiated. Eventually, the antibiotic therapy was narrowed down and targeted according to the final culture and pathology results. After a thorough debridement, lavage and administration of antibiosis, the infectious process was under suppression. There was also a resultant soft-tissue deficit from the debridement (Fig 6). The ring of tissue loss now became the more dominant factor.

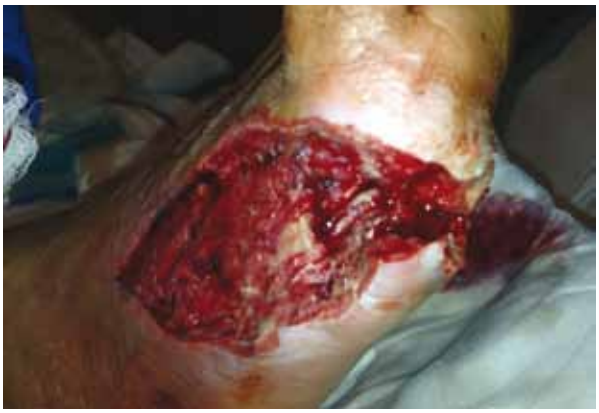

Fig 6. A large soft-tissue deficit after incision, drainage and debridement of all devitalised tissue

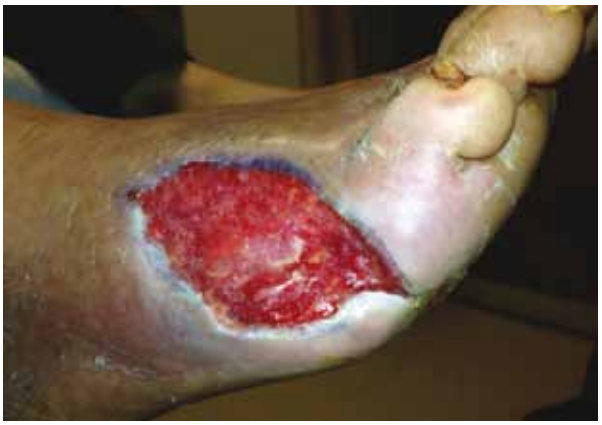

Fig 7. After 2 weeks of NPWT, the soft-tissue deficit was filled with healthy granulation tissue

The focus of treatment then shifted towards wound closure and skin coverage. NPWT was initiated, which accelerated granulation tissue formation (Fig 7). Two weeks later, a split-thickness skin graft was applied (Fig 8). This took well (Fig 9) and the patient was given prescriptive inserts and shoes for protection and prevention of ulceration.

Case study 2

A 67-year-old male with a past medical history of peripheral arterial disease, diabetes mellitus and peripheral neuropathy presented with rest pain, a gangrenous fourth digit and a chronic left lateral penetrating midfoot ulceration that appeared to be ischaemic in aetiology (Fig 10). His past surgical history included superficial femoral artery (SFA) angioplasty and a fifth toe resection. Despite intervention and local wound care, his wounds had not improved over 3 months. Non-invasive vascular studies were repeated and revealed an ankle brachial pressure index of 0. The toe pressure was also 0mmHg, with flat waveforms. Duplex ultrasound revealed an occluded SFA artery and stent, along

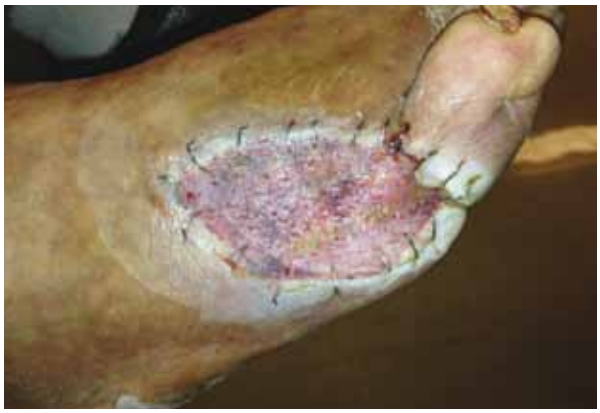

Fig 8. One week after application of a split-thickness skin graft

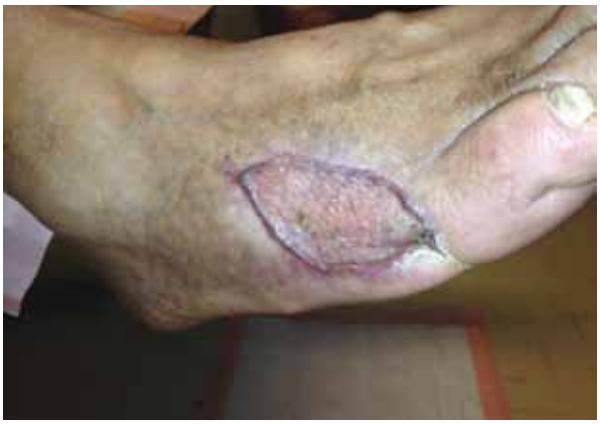

Fig 9. The skin graft was fully incorporated at 21 days postoperatively

Fig 10. Foot with a gangrenous fourth digit and chronic left lateral penetrating midfoot ulceration

with popliteal and trifurcation occlusion. These findings were consistent with critical limb ischaemia and recurrent arterial occlusive disease. There were no signs of acute infection.

Question: What appears to be dominant? Tissue loss, ischaemia or infection?

Answer: This is an ischaemia-dominant lesion. There is certainly a component of tissue loss given the ulceration and fourth digital gangrene. However, the degree and severity of ischaemia is predominant, which places this limb in the ring of ischaemia, as well as partly in that of tissue loss.

Question: How was this addressed?

Answer: The patient underwent successful vascular re-intervention. The presentation then changed to a primarily tissue-loss dominant ring. One week later, the patient underwent a transmetatarsal amputation to address the tissue loss (Fig 11). After successful healing (Fig 12), he was protected with a specially made insert, custom shoe and ankle-foot orthotic brace.

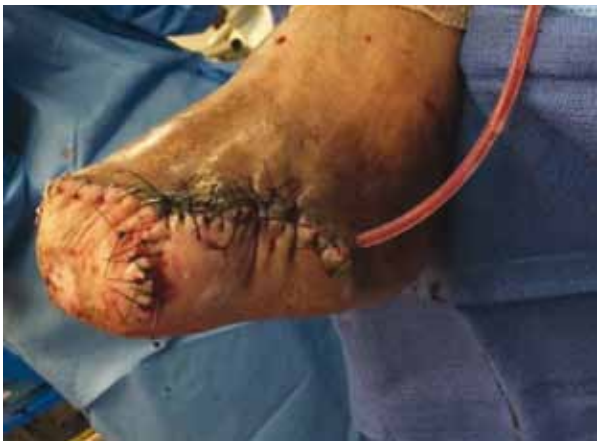

Fig 11. The patient underwent a transmetatarsal amputation one week after revascularisation

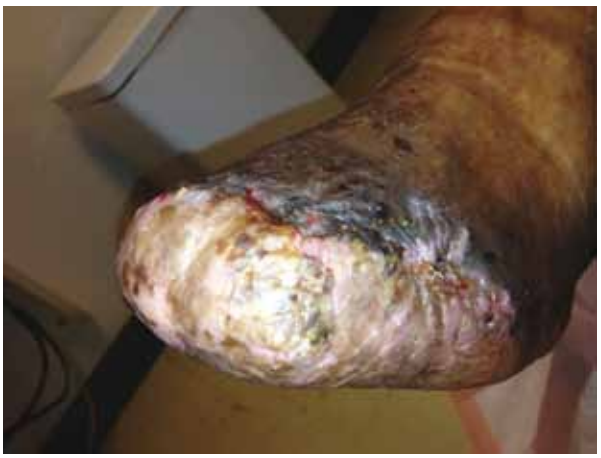

Fig 12. The patient showed significant progress at 3 weeks postoperatively and the wound healed uneventfully

Case study 3

A 40-year-old male with diabetes mellitus and peripheral neuropathy presented with chronic foot ulceration (Fig 13). Hand-held Doppler ultrasound revealed palpable and audible pulses. Wound evaluation, X-ray studies, culture and biopsy detected underlying osteomyelitis of the fifth metatarsal.

Question: What appears to be dominant? Tissue loss, ischaemia or infection?

Answer: This is a soft-tissue-dominant problem. There are no signs of acute infection. There is also no peripheral arterial disease. However, there is central wound necrosis with direct extension to bone. There is also associated bone erosion and infection. Thus, this limb also falls into the ring of infection. Additionally, the patient has a type of deformity that predisposes him to ulceration on the outside of his foot. Some tendons responsible for eversion of the foot were resected during

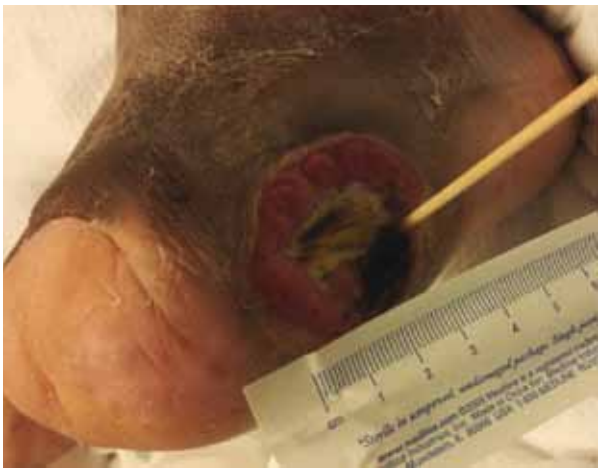

Fig 13. Chronic foot ulceration with underlying osteomyelitis

Fig 14. An inverted (adductovarus) and plantar flexed (equinus) foot deformity following a transmetatarsal amputation

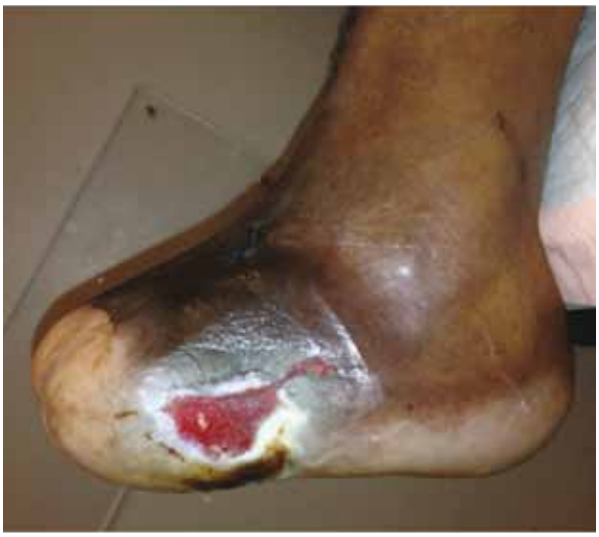

Fig 15. Following multiple wound debridements including resection of the osteomyelitis, NPWT was administered to encourage granulation tissue formation prior to split-thickness skin grafting

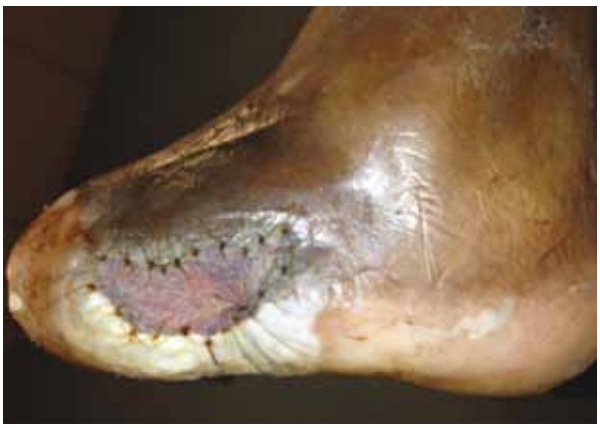

Fig 16a. One week after application of a split-thickness skin graft

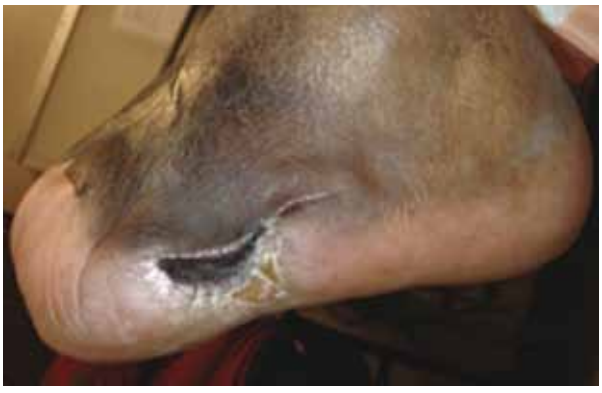

Fig 16b. The skin graft was fully incorporated at 4 weeks postoperatively

his previous transmetatarsal amputation, which led to an adductovarus and equinus foot deformity. This is where the foot turns inward towards the midline and points to the ground as the muscles responsible for inversion and plantar flexion overpower the foot (Fig 14).

Question: How do you address the infection and soft-tissue loss? Can anything be done to address the deformity?

Answer: Sharp debridement and resection of the osteomyelitis, which involved the fifth metatarsal residuum, were performed. Culture-driven antibiotic therapy was employed. Postoperative wound care comprised NPWT to encourage granulation tissue to fill the soft-tissue deficit (Fig 15). Two weeks later, a split-thickness skin graft was performed to obtain complete wound closure (Figs 16a and 16b). The deformity was corrected with muscle tendon-balancing procedures including percutaneous tendo Achilles lengthening and total tibialis anterior tendon transfer (Fig 17). Postoperatively, the foot and ankle were protected with a prescriptive insert, modified shoe and brace.

Wound (tissue loss), ischaemia and foot infection (WIfI)

A new and validated classification system has been proposed by the Society of Vascular Surgery (SVS). It focuses on the three factors that place a limb at risk of amputation: tissue loss, including gangrene; ischaemia; and infection (WIfI).[18,19] These are all key factors that need to be evaluated when assessing a lower extremity with ulceration. Each component of the WIfI has its own grading scheme, based on objective parameters. Each of the three conditions can be classified as none, mild, moderate or severe. This threatened-limb classification scheme can then be used to stratify the risk of amputation as well as to help determine whether or not a patient would benefit from revascularisation. The scoring system is intended to define the disease burden, and is analogous to the tumour, node, metastasis (TNM) system for cancer staging.

One other factor that needs careful evaluation is the abnormal biomechanics that occur in a neuropathic-stricken limb. In particular, in the diabetic foot an 'intrinsic minus' foot deformity occurs with the onset of motor neuropathy.[20,21] Hammer toe deformities and digital contractures form and develop, predisposing the patient to digital ulcerations over prominent bone and joint areas. Equinus contractures resulting from loss of flexibility due to gastrosoleal muscle complex may predispose a neuropathic limb to a host of problems including Charcot neuroarthropathy-related midfoot collapse and increased forefoot pressure and associated lesions.[22,23] Coupled with neuropathy, these biomechanical abnormalities predispose these limbs and feet

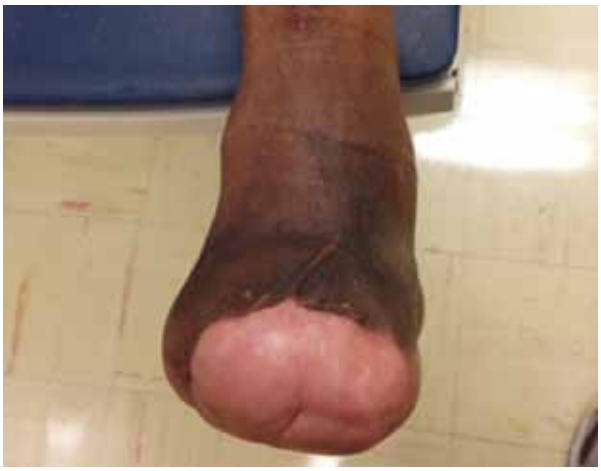

Fig 17. Postoperatively, the foot and ankle are in good alignment

to ulceration. These problematic lesions can be addressed through surgical intervention[6-9] or with total contact casting followed by protection in the form of prescriptive inserts and depth inlay shoes.[3,24,25]

Conclusion

The diagnosis and treatment of diabetic lower-extremity ulcerations can be very challenging. However, by understanding the three factors that place a limb at risk of amputation — tissue loss, ischaemia and infection — evaluation of lower-extremity wounds can become less complicated. By conceptualising the strategy of the three rings, it is possible to learn how to identify which one is most dominant. In turn, this will help determine which aspect to prioritise within a treatment plan. We hope this interactive guide will increase understanding of how to apply the principles of limb salvage.

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Wound-healing algorithm for dressing selection (adapted from Gray et al[1])

Implementation of the concepts of wound bed preparation, TIME and applied wound management (AWM) has resulted in a more systematic approach to wound care. The concept of applied wound management is based on three key physiological continuums: healing, infection and exudate (for wounds healing by secondary intention). When using this concept, clinicians can assess the status of the wound bed by observing the colour of the tissue present, which can range from black (necrotic) to pink (granulation). The algorithm presented here describes this colour continuum and explains the characteristics associated with each colour in the continuum and the associated treatment objectives. The concepts applies to all chronic wounds, including diabetic foot ulcers. It also explains how this knowledge can be used to aid dressing selection, with examples given from the B. Braun range.

Reference

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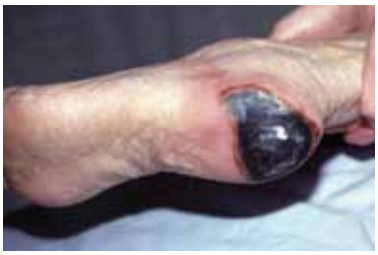
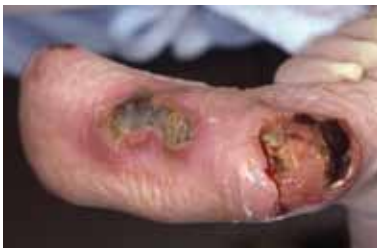
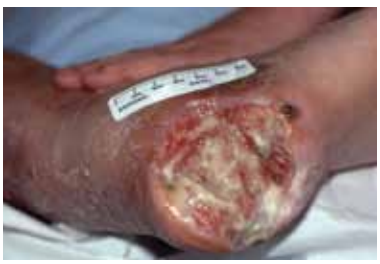
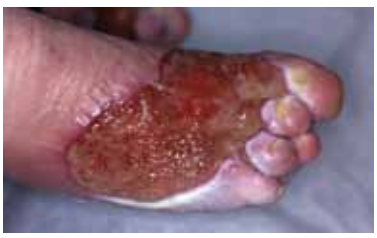
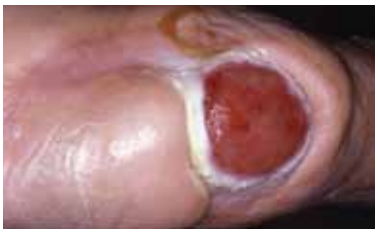
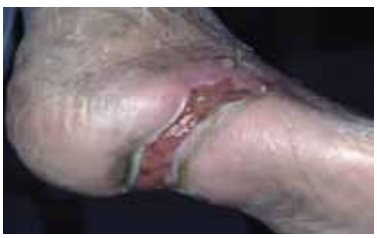
Tissue	Characteristics	Aim of treatment	
Black Necrosis	Ischaemic (dead) tissue is a breeding ground for bacteria. Can be more extensive than appears. Increased bioburden will be present	To rehydrate both eschar and the wound bed, remove devitalised tissue and prepare the wound bed for healing. Reduce bioburden.	
Green Infection	Bacterial invasion resulting in oedema, increased exudate, pungent odour, inflammation and pain. Discolours wound tissue. Results in tissue destruction and a stagnating wound. Increased bioburden will be present	To reduce the bacterial load in the wound and surrounding tissue, prevent or remove biofilm, and manage excess exudate.	
Yellow Slough	Mixture of dead white cells, dead bacteria, and rehydrated sloughy and fibrous tissue. Increased bioburden will be present	To remove slough and excess exudate, prevent or remove biofilm, rehydrate the wound bed, remove devitalised tissue, reduce bacterial burden and prepare wound bed for healing	

Suggested dressing regimen:
1. Cleanse with an antimicrobial irrigation solution (eg, Prontosan Irrigation Solution)
2. Apply an antimicrobial wound dressing (eg, Askina Calgitrol Paste)
3. Apply a foam dressing (eg, Askina Foam)

Medetec

Tissue	Characteristics	Aim of treatment	
Reduced yellow slough	Mixture of dead white cells, dead bacteria and rehydrated sloughy and fibrous tissue. Bioburden may be present	To remove slough and excess exudate, prevent or remove biofilm, rehydrate the wound bed, remove devitalised tissue, reduce bacterial burden and prepare the wound bed for healing	
Red granulation visible	Visible granulation tissue comprised of new capillary loops that will generate new tissue, resulting in wound contraction	To maintain a moist, warm and clean wound bed, prevent or remove biofilm, control exudate, protect vulnerable tissues and safeguard the wound bed from infection	

Suggested dressing regimen:
1. Cleanse with an antimicrobial irrigation solution (eg, Prontosan Irrigation Solution)
2. Use an antimicrobial gel (eg, Prontosan Wound Gel/Prontosan Wound Gel X)
3. Apply a foam dressing (eg, Askina Foam/Askina DreSil)

Tissue	Characteristics	Aim of treatment	
Red granulation	Visible granulation tissue comprised of new capillary loops that will generate new tissue and promote wound contraction	To maintain a moist, warm and clean wound bed, prevent or remove biofilm, control exudate, protect vulnerable tissues and safeguard the wound bed from infection	
Pink epithelialisation	Pink/mauve tissue that forms in the final stages of healing. The wound margins start to divide, rapidly forming the new epidermis	Maintain a moist, warm clean wound environment and protect the vulnerable epithelial tissue as it matures. Safeguard the wound bed from infection	
Healed	Pink to pale red skin that has completed the healing process. The tissue is mature with scar formation	Maintain skin integrity	

Suggested dressing regimen:
1. Cleanse with an antimicrobial irrigation solution (eg, Prontosan Wound Irrigation Solution)
2. Apply an absorbent/low-adherent dressing (eg, Askina Foam/Askina DreSil)
3. Apply skin-care products (eg, Trixo/Linovera

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