Chapter 3
Emergency Management of the Acute Diabetic Foot: Foot Attack

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Introduction

Diabetic foot ulceration (DFU) is the most common diabetic complication requiring hospitalisation. It results in significant morbidity and mortality and is the leading cause of non-traumatic lower limb amputations. Up to 25% of patients with diabetes will have a diabetic foot ulcer during their lifetime with 17% of individuals undergoing an amputation within 1 year of developing an diabetic foot ulcer [1]. Individuals with diabetes are at an increased risk of injury and subsequent ulceration due to the synergistic effects of peripheral neuropathy and impaired tissue perfusion; neuroischaemic ulceration.

Diabetic peripheral neuropathy is present in up to 50% of individuals with diabetes and effects the somatic (sensorimotor) and autonomic nervous systems.

Peripheral sensorimotor neuropathy often follows an insidious onset: initially affecting the feet before progressing proximally in a symmetrical manner (stocking distribution). The sensory component predominates in the early phase with patients complaining of numbness and paraesthesia or dysesthesia. As an individual's protective nociceptor reflexes diminish, they become at an increased risk of unappreciated foot integumental injury and subsequent ulceration.

Charcot neuropathic osteoarthropathy is an extreme consequence of somatic dysfunction whereby the peripheral sensorimotor neuropathy has caused the loss of proprioception and nociceptor reflexes within the joints of the foot. This leads to chronic joint subluxation, instability, and bony destruction, Charcot foot. In its acute phase the associated inflammatory response may be mistaken for infection

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and contributes to further deterioration of normal foot architecture. Motor neuropathy may alter the normal biomechanics of the foot through the creation of an imbalance between the flexors and extensors muscle groups of the foot. The resultant clawing of the toes and exaggeration of the longitudinal plantar arch exacerbates abnormal pressure loading over the plantar metatarsal heads, toe pulps and interphalangeal joints increasing the risk of injury and ulcer formation. Damage to the autonomic nervous system may further compound the effects of sensorimotor neuropathy through the loss of sweating, rendering the skin more prone to fissures and infection. Concurrently reduced sympathetic tone increases microvascular arterio-venous shunting exacerbating ischaemia caused by macrovascular peripheral arterial disease; paradoxically the arterio-venous shunting may lead to pink, warm foot despite underlying tissue ischaemia.

Peripheral arterial disease (PAD) is common in patients with diabetes and the length of affliction and level of glycaemic control is proportional to the risk and severity of PAD. For every 1% increase in haemoglobin A1c (HbA1c) there is a 25% increase in the relative risk of PAD [2, 3]. Seldomly causing DFU in isolation, PAD works synergistically with neuropathy causing neuroischaemic ulceration and is implicated in the aetiology of 50% of diabetic foot ulcerations.

The pattern of PAD in diabetes is macrovascular and diffuse, characteristically affecting the crural vessels whilst sparing portions of the plantar arch [4]. Concomitant microvascular dysfunction potentiates the effects of macrovascular disease with microcirculatory arteriolar shunting and impaired capillary vasoreactivity exacerbating tissue ischaemia. The combination of macrovascular disease and microvascular dysfunction has considerable implications on treatment strategies with relatively innocuous PAD in the non-diabetic population having the potential to significantly impact on tissue healing in patients with diabetes. In turn the presence of PAD is a predictor of non-healing and amputation and is implicated as a contributing factor in 90% of major lower limb amputations in diabetics [5]. Thus, even when only mild in severity, the early recognition of PAD is vital for limb salvage.

Diabetic foot infection is a common and potentially limb threatening problem often being the cause for emergency or urgent presentation. Traditionally thought to be integral to the initial formation of DFU, infection is now recognised as occurring because of ulceration or other types of foot wounds e.g. paronychia. Polymicrobial in nature, aerobic gram-positive cocci and gram-negative bacilli are the commonest causative organisms.

Infection may be initially limited to the ulcer or local integumentum, however a superficial diabetic foot infection can quickly spread from the subcutaneous tissues along the deep fascia impacting upon tendons, muscles, and bone. The anatomy of the foot makes it particularly prone for spread of infection due to its separate but intercommunicating compartments; as infection spreads compartment pressures elevate from the resultant oedema exacerbating ischaemia and tissue necrosis. This rapidly progressive diabetic foot infection requires prompt recognition and treatment without which deterioration may occur over a matter of hours leading to a non-salvageable foot and often life-threatening systemic sepsis. In this chapter, we focus on the emergency management of a patient with an acute diabetic foot infection—the ‘diabetic foot attack’.

Initial Management

Initial Assessment

Initial assessment begins with managing the individual according to the Resuscitation Council UK Guidelines with an A–E approach [6]. Blood glucose levels and the presence of ketones in the urine must be assessed urgently to diagnose metabolic derangements, diabetic ketoacidosis and hyperglycaemic hyperosmolar syndrome. Initial management and treatment should be done in accordance to guidelines ([7], also see Chap. 4). Other potential sources of sepsis should also be identified and treated accordingly.

History

A clear and focused history should be taken to determine onset, duration and extent of symptoms. Treatment prior to admission, including type and duration of antibiotic(s), should be recorded to reduce ineffective antibiotic prescribing. It is important to note that systemic symptoms (rigors, fevers and chills) are uncommon in patients with diabetic foot infection [8]. Preceding glycaemic control can provide an indicator of infection severity with hyperglycaemia a marker of severity of illness and predictor of poor outcome [8]. Cardiovascular co-morbidities such as hypertension, cerebrovascular accident, myocardial infarction and hypercholesterolaemia should be documented alongside other co-morbidities such as chronic kidney disease. History of previous surgery is useful to ascertain previous peri-operative complications and fitness for anaesthesia. Additionally, it is important to record the time of last oral intake, medications (particularly anticoagulants), allergies and a social history to determine timing and type of surgery if appropriate.

Examination

Basic observations should be recorded; respiratory rate, oxygen saturation, pulse, blood pressure and temperature to determine systemic response to infection. Fluid status can be determined by assessing skin turgor, mucus membranes, capillary refill time, peripheral temperature and pulse character.

To examine the foot all dressings must be removed including on the unaffected limb. Erythema, ulceration, pus, swelling and calluses should be noted and documented with particular attention paid between the toes and on the heel. The authors recommend taking photographs of the affected foot for accurate clinical documentation.

Changes in temperature, pain or tenderness, oedema and crepitus should be recorded. The finding of crepitus is significant as it indicates the presence of

gas-producing organisms (gas gangrene) in the soft tissues requiring urgent surgical debridement. 'Milking' of the foot along tendons may produce pus in the wound distally, suggesting proximal tracking of infection.

Osteomyelitis may underly diabetic foot ulceration and is frequently observed in ulcers that are chronic, extensive, overlying a bony prominence e.g., first or fifth metatarsophalangeal joints, or accompanied by a swollen "sausage" toe (Fig. 3.1). In these circumstances the clinician should undertake a 'probe to bone' test to establish bony involvement: a sterile metal probe is inserted into the ulcer with a positive test recorded upon encountering bone. A positive 'probe to bone' test is an accurate and inexpensive bedside test for osteomyelitis and widely used by the authors.

Pulse status throughout the leg and foot should be recorded alongside objective tests for peripheral arterial disease. An ankle brachial pressure index (ABPI) <0.9 indicates the presence of peripheral arterial disease however, a third of patients with diabetes produce an incompressible or falsely elevated ABPI due to calcification of the arterial wall. Toe pressures are more reliably used as they are rarely affected by atherosclerosis with a pressure <50 mmHg indicative of significantly impaired perfusion.

Validated scoring systems should be used to determine the severity of infection and need for revascularisation. The Infectious Diseases Society of America/International Working Group on the diabetic foot classification scheme is used to determine the presence and severity of a diabetic foot infection (Table 3.1). The Wound, Ischemia, and foot Infection (WIfI) is used to estimate the risk of major limb amputation and benefit of revascularisation in individuals with a threatened limb (Table 3.2).

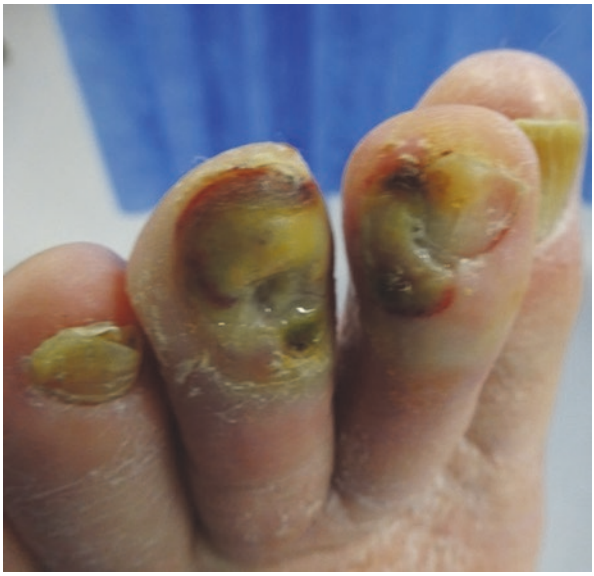

Fig. 3.1 "Sausage" toe indicative of underlying osteomyelitis

Table 3.1 The Infectious Diseases Society of America/International Working Group on the diabetic foot classifications of diabetic foot infection [8]

Clinical classifications of infection	IDSA infection severity
No symptoms or signs of infection	1 (Uninfected)
Infection defined as ≥2 of: • Local swelling or induration • Erythema >0.5 cm^2 around wound • Local tenderness/pain • Local increased warmth • Purulent discharge Excludes other causes (e.g. trauma, gout, acute Charcot, fracture, thrombosis, venous stasis)	
Infection confined to skin and subcutaneous tissue with no systemic manifestations	2 (Mild)
Infection with erythema ≥2 cm^2 and/or involving structures deeper than skin and subcutaneous tissues and with no systemic manifestations	3 (Moderate)
Infection with systemic manifestations defined as ≥2 of: • Temperature >38 °C or <36 °C • Heart rate >90 beats/min • Respiratory rate >20 breaths/min or $PaCO_2$ <4.3 kPa • White blood cell count >12 × 10^9/L, or <4 × 10^9/L, or ≥10% immature (band) forms	4 (Severe)

Table 3.2 Society for vascular surgery lower extremity threatened limb classification: wound, ischemia, and foot infection system [9]

Component	Score	Description		
Wound	0	No ulcer (ischemic rest pain)		
	1	Small, shallow ulcer on distal leg or foot without gangrene		
	2	Deeper ulcer with exposed bone, joint or tendon ± gangrene changes limited to toes		
	3	Extensive deep ulcer, full thickness heel ulcer ± calcaneal involvement ± extensive gangrene		
Ischemia		ABPI	Ankle pressure (mmHg)	Toe pressure or $TcPO_2$
	0	≥0.8	>100	≥60
	1	0.60–0.79	70–100	40–59
	2	0.40–0.59	50–70	30–39
	3	<0.40	<50	<30
foot Infection	0	No symptoms/signs of infection		
	1	Local infection involving only skin and subcutaneous tissue		
	2	Local infection involving deeper than skin/subcutaneous tissue		
	3	Systemic inflammatory response syndrome		

Investigations

Initial blood tests are required to help determine severity of infection and initiate management plans. It is important to note that half of patients with a diabetic foot infection have a normal white cell count [8]. However, a C-reactive protein level or other inflammatory marker level can help guide initial management and act as an adjunct in the diagnosis of osteomyelitis. A full blood count is also useful in determining baseline haemoglobin level and platelet function with anticipated serum grouped and saved. In patients with a history of cardiovascular disease a haemoglobin >80 g/L should be targeted. A coagulation screen should be carried out for clotting function with an INR ≤1.4 being acceptable for an individual to undergo regional anaesthesia [10].

Measuring urea and kidney function is useful to assess for organ dysfunction due to sepsis and to help guide suitable antibiotic choice and doses. A venous blood gas allows a quick immediate assessment of lactate, pH and glucose level. A HbA1C should also be sent concurrently. Blood cultures are required in those with pyrexia to determine micro-organisms involved.

Samples for microbiology culture and sensitivity should be taken; however, this should ideally be done aseptically by curettage or biopsy from the ulcer to determine the true causative organism as wound swabs are often positive for contaminants. Bony fragments evident in the wound should be biopsied and sent for culture and histopathology analysis.

Patients with a diabetic foot infection require an AP and lateral foot X-ray view to assess for osteomyelitis. It is important for the clinician to note that X-ray evidence of osteomyelitis may not be evident during the first 4–6 weeks of infection or could be mimicked by a Charcot osteoarthropathy. X-rays should also be assessed for the presence of soft tissue gas; an indicator of severe foot infection (Fig. 3.2). Advanced imaging techniques such as MRI may be useful following initial measures to control the foot control. However, they have little benefit during the emergency setting where the combination of accurate clinical history and examination, blood tests and plain radiographs are more useful in directing emergency treatment of the diabetic foot attack.

Management

Emergency management of a patient with an acute diabetic foot infection also requires management of diabetes and other co-morbidities. Those awaiting emergency surgery with normoglycaemia (capillary blood glucose <10 mmol/L), no metabolic derangement and who will only miss one meal due to surgery should receive a reduced insulin dose. Those who will miss more than one meal or who have hyperglyacemia (blood glucose >10 mmol/L) without metabolic derangement require a variable rate intravenous insulin infusion (VRII). Where there are risk factors for hypoglycaemia (chronic kidney disease, acute kidney injury, low body weight, low total daily dose of insulin,

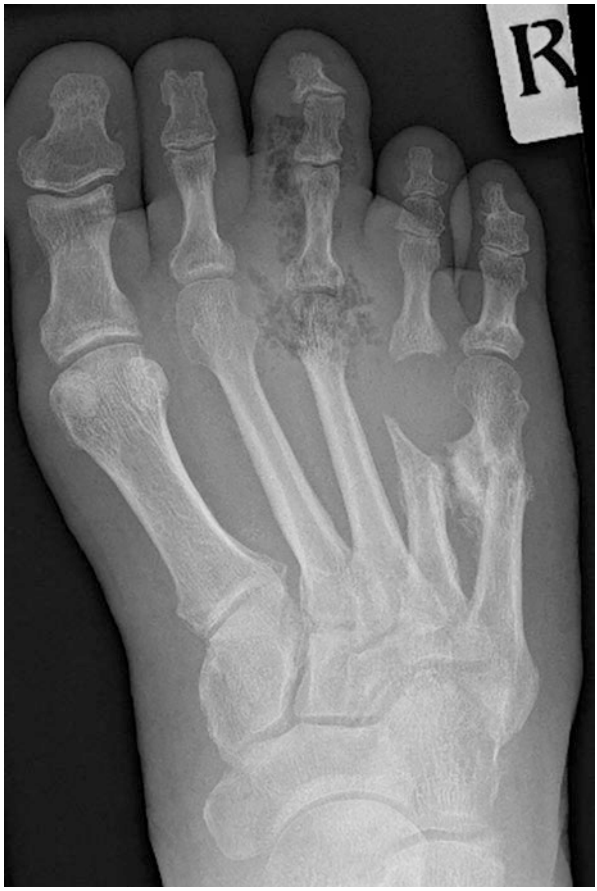

Fig. 3.2 Plain AP foot X-ray demonstrating locules of gas around the third toe. Previous partial resection of the fourth toe and through the distal interphalangeal joint of the second toe

insulin naïve), then a reduced VRIII should be used. Diabetic ketoacidosis and hyperosmolar hyperglycaemic states should be managed with a fixed rate intravenous insulin infusion according to local institute guidelines (see Chap. 4). Intraoperative blood glucose levels should be maintained between 6 and 12 mmol/L [11].

500 mL of crystalloid fluid should be used for immediate fluid resuscitation and broad-spectrum antibiotics given promptly. Antibiotics should be continued for 1–2 weeks and initially parenteral for severe infection. It is important to remember that diabetic foot infections are often polymicrobial in nature. The most common causative organisms are aerobic gram-positive cocci and gram-negative bacilli; therefore, antibiotics should be targeted accordingly. No antibiotic class or agent has been shown to be superior to others and so prescribing according to local policy, previous sensitivities and consideration of allergies should be undertaken.

Consideration is required for patients on anticoagulation and antiplatelet therapy who need surgery. Warfarin should be withheld for 5 days for patients undergoing surgery. If emergency surgery is required in 6–8 h then 5 mg of IV vitamin K for immediate reversal should be given. If emergency surgery is required sooner, then warfarin should be reversed with 25–50 μ/kg of four-factor prothrombin complex

concentrate. Direct oral anticoagulants should be withheld for 48 h prior to surgery. However, for those requiring emergency surgery, reversal agents exist. Idacrucizumab should be used to reverse dabigatran and andexanet for the reversal of apixaban, rivaroxaban or edoxaban. The management of anticoagulation in the emergency setting is complex and requires careful co-ordination and discussion with the anaesthetic and haematology teams. Regarding antiplatelet therapy, aspirin can be continued peri-operatively including those awaiting neuroaxial anaesthesia. Ideally Clopidogrel and Ticagrelor should be withheld for 5 days and Prasugrel for 7 days pre-operatively, however this may not always be possible in the emergency setting of a foot attack and rapid debridement should take priority with haematological input to reduce bleeding peri-operatively [12].

Surgery

Many diabetic foot infections remain above the subcutaneous fascia and can be managed with antibiotics alone. However, deep soft tissue involvement requires emergency surgical management. Purulent discharge, fullness in the plantar space, pain or tenderness in a previously insensate foot, infected and necrotic tissue, presence of an abscess or radiological evidence of gas are all indications for emergency surgical intervention. The aim of surgery for most patients is to facilitate the control of infection, and in turn limb salvage, through the drainage of compartmental pus and debridement of necrotic tissue. However, for the patient in fulminant diabetic foot sepsis the priority of surgery is preservation of life rather than limb salvage and on occasion a major limb amputation is required.

Surgery

Surgery is aimed at targeting all pockets of infection and this requires a detailed understanding of the nine compartments of the foot (medial, lateral, four interosseous and 3 central; superficial, intermediate and deep).

The Loeffler–Ballard incision is the most widely described technique and commences proximally from behind the medial malleolar extending distally and laterally across the medial longitudinal arch, ending between heads of the first and second metatarsals [13]. This allows good access to the medial, central and 1–2 interosseous compartments. Modifications of this technique have been widely described and the authors use a modified technique (Fig. 3.3). The incision commences between the two metatarsal heads corresponding to the maximal distal extent of infection and progresses proximally towards the medial malleolus until healthy tissue is identified or all infection has been drained. In our experience, this rarely progresses to the level of the medial malleolus thereby reducing the

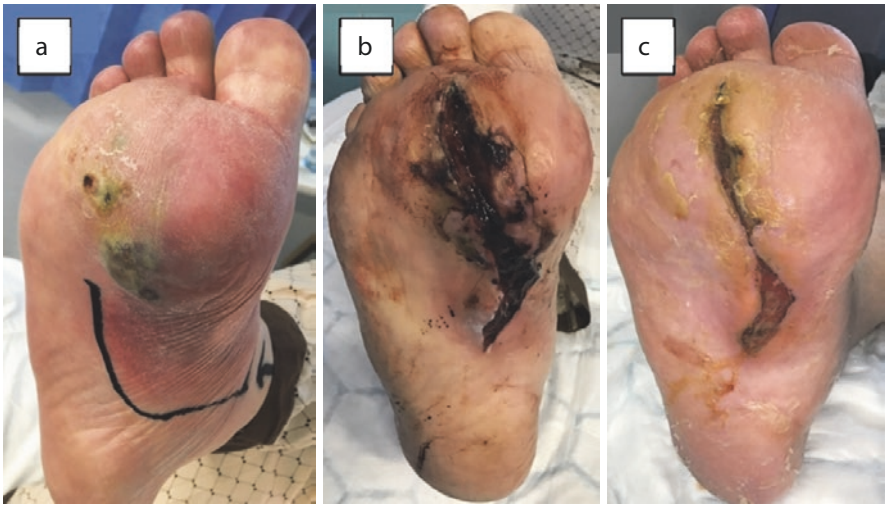

Fig. 3.3 (**a**) Diabetic foot infection; black mark demonstrating degree of erythema. (**b**) 5 days post modified plantar incision. (**c**) 4 weeks post modified plantar incision

morbidity of the surgery particularly in those patients with concomitant ischaemia. In cases of fulminant foot infection access to all nine compartments of the foot is vital. In these situations, the authors combine a standard Loeffler–Ballard incision with two longitudinal dorsal incisions commencing immediately proximal to the second and fourth webspace and extending the length of the adjacent metatarsal bone. When combined with a plantar incision, this facilitates access and lavage of all nine compartments.

Upon accessing the fascial spaces and drainage of pus, debridement of all non-viable tissue and bone should be undertaken regardless of size and extent. Exposed tendons should be resected to prevent future tracking of infection/pus. It is vitally important to document and send superficial, deep soft tissue, and bone samples for microbiological culture and sensitivity. This allows targeted peri-operative antibiotic therapy as pus samples alone are inadequate for this purpose.

Once the infection has been drained and all non-viable tissue excised, the incision(s) should be left open. Multiple surgical debridements are often required for the severely infected diabetic foot. The use of drains is at the operator's discretion, but the authors have found little benefit to their usage and believe them to be no substitute for aggressive debridement and planed 'relook' surgery.

In some cases, it may be appropriate to undergo a primary amputation to manage the diabetic foot infection. Limited toe amputations can be carried out for individuals with wounds limited to the middle or distal portion of the toe. Incisions ensuring complete drainage of infection and tension-free coverage should be chosen. Fish-mouth or transverse incisions are traditionally used for partial amputation of the toe and Racket incisions for complete amputation (Fig. 3.4). Ray

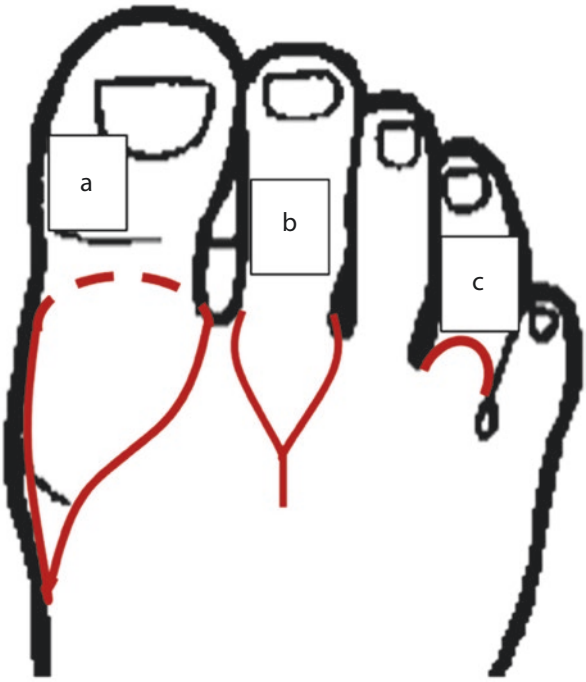

Fig. 3.4 (**a**) Racket incision. (**b**) Ray amputation. (**c**) Fish-mouth incision

amputations involve an amputation through the metatarsal head and may be necessary in severe diabetic foot infection where the entire digit is involved (Fig. 3.4). For the patient with a non-salvageable foot in fulminant diabetic foot sepsis (Fig. 3.5) it may be appropriate to undergo a guillotine amputation as a life-saving measure (Fig. 3.6). When the patients' condition is stabilised, a formal amputation can then be carried out.

Wounds should be dressed with a non-adherent dressing and padding and inspected within 48 h. If there are concerns regarding tissue viability or residual infection, then they should be inspected within 24 h. Patients should undergo strict bedrest for the first 24 h to allow for initial wound healing and prevent post-operative bleeding. Post-operative ward destination depends on the level of cardiovascular/organ support required and the authors have a low threshold for seeking high dependency level care. All patients should be encouraged to eat and drink as soon as able to reduce morbidity associated with prolonged fasting.

Revascularisation

All patients with suspicion of PAD should undergo formal imaging as described in Chap. 6. The need for revascularisation can be guided by intra-operative findings. Patients with known PAD but with good bleeding during initial surgery may be able

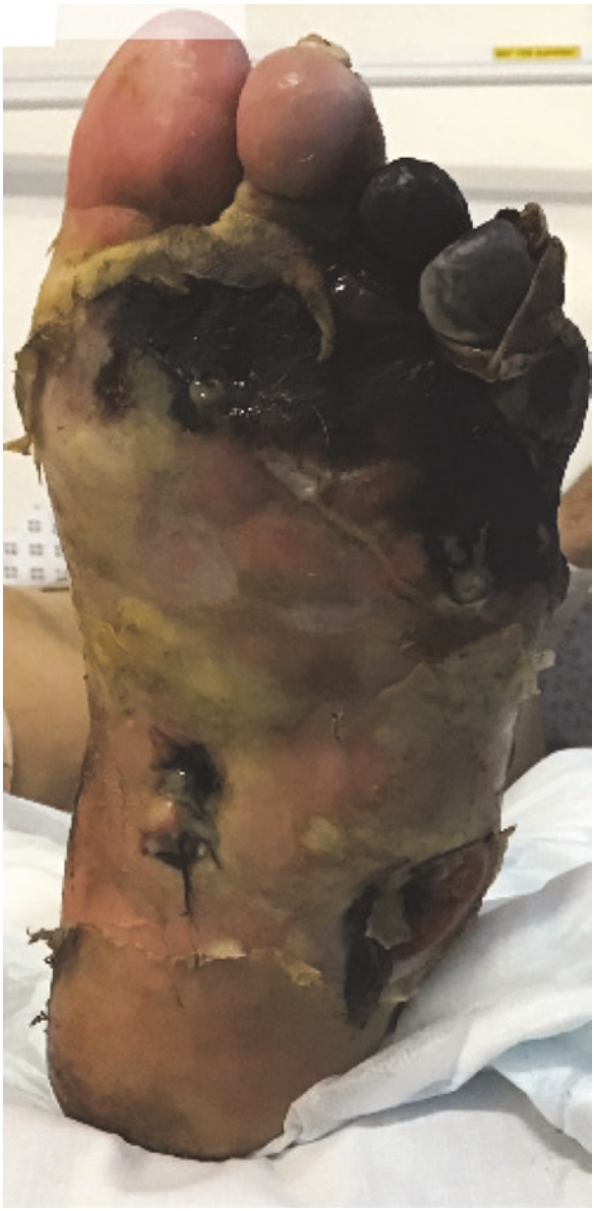

Fig. 3.5 Patient with a non-salvageable foot presenting with fulminant diabetic foot sepsis

to undergo a 'watch and wait' approach. However, if the wound fails to heal then prompt revascularisation should be arranged. Those with poor bleeding intra-operatively may be required to undergo emergency revascularisation once cardio-vascularly stable and source of infection removed. Revascularisation options available are discussed in detail in Chaps. 8 and 9.

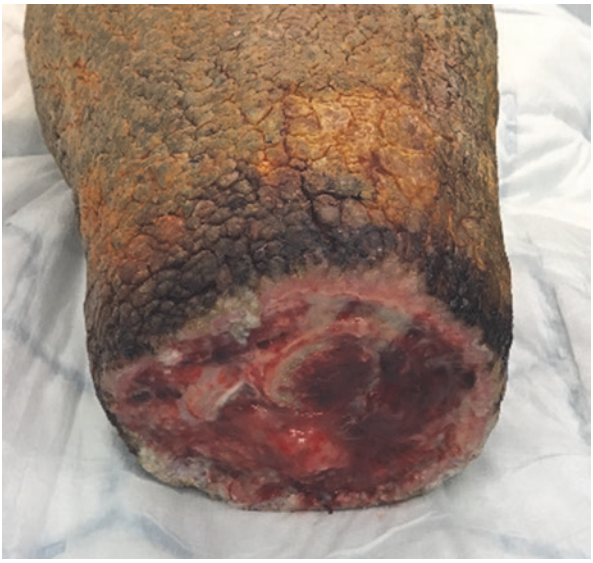

Fig. 3.6 One week post below the knee Guillotine amputation of limb for fulminant diabetic foot sepsis. This was revised to a through knee amputation at a later date

Ongoing Care

Antibiotics should be targeted to microbiology culture results and continued for 1–2 weeks for severe infection. Patients with diagnosed osteomyelitis and residual bone should be managed with a prolonged course of antibiotics. However, if no clinical improvement in infection within the first 2–4 weeks then further surgical resection or an alternative antibiotic regimen may be required. Treatment with antibiotics should ideally not exceed longer than 6 weeks [8].

Wound healing relies on optimisation of circulation and a multidisciplinary approach. Wounds should primarily be managed by vascular nurses and podiatrists to improve outcome. Regular wound inspection is imperative to assess for healing and prevent infection. Careful dressing management is required to control excess exudation and maintain a moist environment. Promotion of wound healing has been suggested by negative pressure wound therapy and systemic hyperbaric oxygen therapy however, there remains insufficient evidence to determine their benefit. The authors routinely utilise a topical negative pressure dressing with or without a lavage function for plantar wounds as we feel it facilitates exudate control and wound healing.

Appropriate footwear is necessary to offload the foot to reduce, redistribute and remove detrimental forces, preventing further ulcers. Custom-made footwear can be used to accommodate deformity and relieve pressure. Non-removable knee-high offloading devices can be used for plantar, midfoot or forefoot ulcers and the use of other offloading devices depends on the position of the ulcer [14]. All of these rely on the expertise of podiatrists and orthotic services.

Poor glycaemia control increases risk of infection and so all individuals should have their diabetic control optimised and referred to diabetic specialists as appropriate.

Upon discharge, all patients should be followed up in the community to ensure sufficient wound healing and to prevent further diabetic foot infections. The continued management of a patient with a diabetic foot infection requires a multidisciplinary approach to prevent further morbidity and mortality.

Summary

Patients presenting with an acute diabetic foot infection should be managed as a surgical emergency as early assessment and intervention is imperative to prevent morbidity and mortality. Diabetic foot infections remain the commonest diabetic complication requiring hospitalisation. Delays in management result in a higher risk of major limb amputation and potential for severe organ dysfunction due to sepsis. Therefore, the management of the patient presenting with a diabetic foot infection is complex and should be carried out in a multidisciplinary setting within a dedicated vascular unit. More should be done to prevent ulcer development with the use of community services such as podiatry and diabetic foot clinics. These services help identify the at-risk foot as well as regularly inspect and examine for ulcers. Additionally, the use of podiatry and orthotic services will ensure the routine wearing of appropriate footwear and prevention of ulcers. Lastly, educating the patient, family and healthcare professionals helps to recognise pre-ulcerative signs leading to the reduction in the associated morbidity.

Key Points

- Initial assessment should begin with Resuscitation Council UK Guidelines with an A–E approach and address sepsis, diabetic ketoacidosis, and hyperglycaemic hyperosmolar syndrome as appropriate.
- Clinical assessment must include a medical history, clinical examination, blood tests and assessment of the arterial circulation.
- Validated scoring systems should be used to determine the severity of infection and need for revascularisation.
- Surgical debridement requires understanding of the anatomy and compartments of the foot and should be carried out by a surgeons experienced in management of diabetic foot complications.
- Successful wound healing requires a multidisciplinary approach, addressing antibiotic therapy, offloading and optimisation of the patients diabetes care.

References

1. Ndosi M, Wright-Hughes A, Brown S, Backhouse M, Lipsky BA, Bhogal M, et al. Prognosis of the infected diabetic foot ulcer: a 12-month prospective observational study. Diabet Med. 2018;35:78–88.
2. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati F, Powe N, et al. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med. 2004;141(6):421–31.
3. Nilsson SV, Nilsson JE, Frostberg N, Emilsson T. The Kristianstad survey. II. Studies in a representative adult diabetic population with special reference to comparison with an adequate control group. Acta Med Scand Suppl. 1967;469:1–42.
4. Jude EB, Oyibo SO, Chalmers N, Boulton AJ. Peripheral arterial disease in diabetic and non-diabetic patients. Diabetes Care. 2001;24(8):1433–7.
5. Heald CL, Fowkes FGR, Murray GD, Price JF, Collaboration ABI. Risk of mortality and cardiovascular disease associated with the ankle-brachial index: systematic review. Atherosclerosis. 2006;189(1):61–9.
6. Resuscitation Council UK. Underlying principles First steps Airway (A). 2015. https://www.resus.org.uk/library/2015-resuscitation-guidelines/abcde-approach.
7. Savage M, Dhatariya K, Kilvert A, Rayman G, Rees J, Courtney C, et al. Joint British Diabetes Societies guideline for the management of diabetic ketoacidosis. Diabet Med. 2011;28(5):508–15.
8. Lipsky B, Senneville E, Abbas Z, Aragon-Sanchez J, Diggle M, Embil JM, et al. Guidelines on the diagnosis and treatment of foot infection in persons with diabetes (IWGDF 2019 update). 2020. https://iwgdfguidelines.org/wp-content/uploads/2020/11/Lipsky_et_al-2020-IWGDF-infection-guideline.pdf.
9. Mills JL, Conte MS, Armstrong DG, Pomposelli FB, Schanzer A, Sidawy AN, et al. The society for vascular surgery lower extremity threatened limb classification system: risk stratification based on Wound, Ischemia, and foot Infection (WIfI). J Vasc Surg. 2014;59(1):220–234. e2. https://doi.org/10.1016/j.jvs.2013.08.003.
10. Horlocker TT, Vandermeuelen E, Kopp SL, Gogarten W, Leffert LR, Benzon HT. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine evidence-based guidelines. Reg Anesth Pain Med. 2018;43:263–309.
11. Dhatariya K, Dhesi J, Selwyn D, Dileep L, Agnes G, Mike G, Diabetes Guideline Working Group, et al. Guideline for perioperative care for people with diabetes mellitus undergoing elective and emergency surgery. London: Centre for Perioperative Care (CPOC); 2021.
12. British Society for Haematology. Peri-operative management of anticoagulation and antiplatelet therapy. Br J Haematol. 2017;175(4):602–13.
13. Loeffler RD, Ballard A. Plantar fascial spaces of the foot and a proposed surgical approach. Foot Ankle. 1980;1(1):11–4.
14. Bus SA, Armstrong DG, Gooday C, Jarl G, Caravaggi CF, Viswanathan V, et al. IWGDF guideline on offloading foot ulcers in persons with diabetes. New York: Wiley; 2019. p. 1–36.

Further Reading

Lipsky B, Senneville E, Abbas Z, Aragon-Sanchez J, Diggle M, Embil JM, IWGDF Editorial Board, et al. Guidelines on the diagnosis and treatment of foot infection in persons with diabetes (IWGDF 2019 update). Diabetes Metab Res Rev. 2020;36(Suppl 1):e3280.

Schaper NC, van Netten JJ, Apelqvist J, Bus SA, Hinchliffe RJ, Lipsky BA, IWGDF Editorial Board. Practical guidelines on the prevention and management of diabetic foot disease (IWGDF 2019 update). Diabetes Metab Res Rev. 2020;36(Suppl 1):e3266.