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Review

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The diabetic foot

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Summary

Diabetes is reaching epidemic proportions and with it carries the risk of complications. Disease of the foot is among one of the most feared complications of diabetes. The ultimate endpoint of diabetic foot disease is amputation, which is associated with significant morbidity and mortality, besides having immense social, psychological and financial consequences. As the majority of amputations are preceded by foot ulceration, it is crucial to identify those at an increased risk. Diabetic foot ulcers may develop as a result of neuropathy, ischaemia or both and when infection complicates a foot ulcer, the combination can become limb and life threatening. Structural abnormalities such as calluses, bunions, hammer toes, claw toes, flat foot and rocker bottom foot need to be identified and managed.

Introduction

Diabetes is reaching epidemic proportions and with it carries the increased risk of complications. Disease of the foot is among one of the most feared complications of diabetes. The term 'Diabetic Foot' consists of a mix of pathologies including diabetic neuropathy, peripheral vascular disease, Charcot's neuroarthropathy, foot ulceration, osteomyelitis and the potentially preventable endpoint, limb amputation.[1] The lifetime risk of a person with diabetes developing foot ulceration is reported to be as high as 25%.[2] It is estimated that more than a million people with diabetes require limb amputation each year, suggesting that one major amputation is performed worldwide every 30 s.[3] Amputation is associated with significant morbidity and mortality, besides having immense social, psychological and financial consequences.[4,5] As the majority of limb amputations in patients with diabetes are preceded by foot ulceration, it is essential that strategies are directed towards preventing this.[1] Subjects with diabetic foot problems are also likely to harbour other associated complications of diabetes such as nephropathy, retinopathy, ischaemic heart disease and cerebrovascular disease. Hence, these subjects are more likely to benefit from a multidisciplinary approach with a view to addressing these challenging complications. Furthermore, there is evidence to suggest that the incidence of major amputation can be reduced by implementation of a multidisciplinary team approach.[6]

Epidemiology

Diabetic foot complications are more frequent in males and individuals aged over 60 years.[1] Reliable data on the accurate estimation of incidence and prevalence of diabetic foot problems are lacking. Based on recent studies, the annual population-based incidence for diabetic foot ulcers is 1–4%, with a prevalence of 4–10%. The lifetime risk is

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Table 1 Factors predisposing to diabetic foot complications

Neuropathy
Peripheral vascular disease
Trauma
Infection
Poor glycaemic control
Improper footwear
Others: old age, smoking, low socioeconomic status, psychological factors

estimated to be ~25%.[2] In the Northwest of England diabetes foot care study, a large cohort of diabetic subjects ($n = 9710$) in the community healthcare setting was followed up to determine the incidence of new foot ulcers. The study reported a 2.2% annual incidence rate of new diabetic foot ulcerations.[7] Interestingly, this study also reported a 33% lower risk of foot ulcers in South Asians with diabetes in the UK when compared with Europeans. This ethnic difference was accounted for by lower levels of peripheral arterial disease, neuropathy, insulin usage and foot deformities in South Asians.[8] The most feared and costly complication of diabetic foot disease is amputation, which occurs 10–30 times more often in diabetics than in the general population.[9,10] Diabetes accounts for up to 80% of non-traumatic amputations, with 85% of these being preceded by a foot ulcer.[10] Amputation carries with it a significantly elevated mortality at follow-up, ranging from 13% to 40% at 1 year to 39–80% at 5 years.[2]

Pathogenesis

Diabetic foot problems are caused by a number of factors such as neuropathy, peripheral vascular disease, trauma and infection. Table 1 lists the various contributory factors predisposing to diabetic foot complications. Diabetic foot complications are usually the result of an interplay of these varied causative factors, of which neuropathy is considered to be the most important.[11]

Diabetic neuropathy is present to some degree in >50% of patients ⩾60 years and increases the risk of foot ulceration by 7-fold.[11–13] Diabetic neuropathy can affect the sensory, motor and autonomic functions to varying degrees. The insidious nature of neuropathy may go unnoticed by the patient, thus emphasizing the importance of regular assessment of the diabetic foot. Motor neuropathy leads to muscle atrophy, foot deformity, altered foot biomechanics, and redistribution of foot pressures which eventually predispose the foot to ulcerate. Sensory neuropathy renders the foot '*deaf and blind*' to stimuli, which would normally elicit pain or discomfort. This predisposes the foot to repetitive trauma, which may go unnoticed until ulceration ensues. Autonomic neuropathy results in loss of sweating, with the resultant dry skin being predisposed to cracks and fissures. The altered autonomic regulation of cutaneous blood flow also contributes.[14]

Charcot neuroarthropathy is a non-infective process occurring in a well-perfused and insensitive foot. It is characterized by bone and joint destruction, fragmentation and remodelling. Although Charcot's neuroarthropathy was first described as a complication of tabes dorsalis, it can develop with any type of sensory neuropathy and currently diabetes is the commonest cause.[1] Charcot foot has been reported to be present in around 16% of patients with diabetes where there is a history of neuropathic ulceration. Bilateral involvement has been reported in up to 30% of patients.[15] The precise mechanism underlying Charcot neuroarthropathy is unclear. The *neurotraumatic theory* attributes bony destruction to the loss of pain and proprioception, combined with repetitive mechanical trauma to the foot, which is largely unperceived by the patient who continues to weight bear.[16] The *neurovascular theory* suggests that joint destruction is secondary to an autonomically mediated vascular response, which causes increased blood flow and periarticular osteopenia by activating osteoclasts.[17,18] Repetitive trauma to the insensitive foot propagates microfractures, with healing of these fractures being prolonged due to continued weight bearing. Motor neuropathy may contribute by leading to intrinsic muscle imbalance, ligament stretching and spontaneous dislocations. The result is eccentric loading of the foot and excessive plantar pressures promoting the development of microfractures and progressive bony destruction. This insensitive deformed foot is at an increased risk of ulceration.[1]

Diabetes is associated with a 2–3-fold increased risk of accelerated atherosclerosis. Subjects with peripheral vascular disease are predisposed to poor wound healing. This underlines the importance of identifying and aggressively managing the associated vascular risk factors such as hypertension, dyslipidaemia and cigarette smoking.[19] Poor diabetes control also contributes adversely on wound healing by impairing collagen cross linking and matrix metalloproteinase function.[20] Furthermore, poor glycaemic control also impairs polymorphonuclear leucocyte function and predisposes to onychomycosis and toe-web tinea infections, all of which may lead to skin disruption.[21,22]

Ulceration of the diabetic foot does not occur spontaneously, but usually follows some form of

Table 2 Assessing the diabetic foot

(A)	Neuropathic assessment
	• History to include neuropathic symptoms
	• Examination to include:
	Testing pressure sensation by 10 g monofilament
	Testing vibration sensation by 128 Hz tuning fork
(B)	Structural assessment
	• Identifying structural abnormalities such as calluses, bunions, hammer toes, claw toes and flat foot
	• Identifying Charcot neuroarthropathy
(C)	Vascular assessment
	• History to include claudication symptoms
	• Identifying cutaneous trophic changes such as corns, calluses, ulcers or frank digital gangrene
	• Palpating pedal pulses
	• ABPI/ TBI/ Arterial Doppler in selected cases

trauma, which may go unnoticed by the patient. This trauma may be inflicted by ill-fitting footwear, walking barefoot, foreign objects or scalding from hot water.[1] Reassuringly, the daily level of physical activity does not in itself predispose to new or recurrent foot ulceration.[23] In patients with diabetes, the local and systemic signs of inflammation may often be reduced as a result of associated peripheral vascular disease and immunosuppression. The infected foot can be painless as a result of neuropathy and this may lead to unnecessary delay in seeking medical attention.[14]

Assessment of the 'diabetic foot'

Assessing the diabetic foot represents a very important element of the annual diabetic review. It is indeed crucial to identify the foot at risk earlier, so as to target preventative and therapeutic measures at the earliest opportunity. This approach does not merely help in reducing the significant morbidity and mortality associated with diabetic foot disease, but also could have major health care-associated economic benefits.

The presence of dry skin, tinea and onychomycosis needs to be identified and treated early. Footwear also needs to be carefully inspected to ensure proper fit. Other factors known to be associated with increased risk of foot ulceration include, past history of foot ulceration, past history of lower extremity amputation, long duration (>10 years) of diabetes, poor glycaemic control, impaired vision and nephropathy. The diabetic foot assessment should include a thorough neuropathic, structural and vascular assessment at least on an annual basis (Table 2).

Neuropathic assessment

A thorough history should include neuropathic symptoms such as burning, tingling, numbness and nocturnal leg pains. Examination should comprise of careful inspection for muscle wasting, foot deformities such as claw toes, loss of hair and trophic changes. Sensory assessment includes testing for pressure, vibration, joint position and pain or temperature sensation. Pressure sensation is usually assessed by using the 10 g nylon Semmes–Weinstein monofilament. The monofilament is placed at a right angle to the skin on the plantar surface with pressure being applied until the filament buckles, indicating that a specified pressure has been applied. Inability to perceive the 10 g of force applied by the monofilament is associated with clinically significant large-fibre neuropathy.[24] Studies have shown the monofilament test to identify persons at increased risk of foot ulceration with a sensitivity of 66–91%.[25–27] Testing four plantar sites on the forefoot (great toe and the base of first, third and fifth metatarsals) identifies 90% of patients with an insensate foot.[28] Vibration sensation is tested using a 128 Hz tuning fork applied on the bony prominence of the great toe, gradually moving upwards if there is any impairment noted. Sensitivity is around 53% and there is evidence to suggest that the tuning fork is less predictive of foot ulceration compared to monofilament testing.[25] A biothesiometer is a handheld device that assesses vibration perception threshold. A vibration threshold of more than 25 V has been reported to have a sensitivity of 83%.[13]

Structural assessment

Examining the feet for structural abnormalities such as calluses, bunions, hammer toes, claw toes and flat foot is important. Foot ulceration may result from excessive plantar pressures resulting from limited joint mobility, particularly at the ankle, subtalar and first metatarsophalangeal joints. Devices used to identify high plantar pressures include specialized mats that measure barefoot plantar load distribution and transducers in a removable shoe insole that measure pressure inside footwear.[29] It is crucial to identify the presence of Charcot neuroarthropathy as this is likely to go unnoticed by the patient until a grossly deformed insensitive foot results, which is at an increased risk of ulceration (Figure 1). During the acute stage, the affected foot is swollen with pain or discomfort. On examination, the foot is warm, with a temperature differential of >2°C in comparison to the contralateral foot and may appear inflamed and swollen. The temperature of the overlying skin can be measured with an infrared thermometer and may

Figure 1. The Charcot foot.

be helpful in monitoring the disease activity of an acute Charcot foot.[30] Acute Charcot foot may be misdiagnosed as cellulitis, osteomyelitis, inflammatory arhropathy or deep vein thrombosis.[31] Therefore, a high index of suspicion is necessary so as to allow early identification and appropriate treatment of the acute Charcot foot. Once the acute phase of Charcot's subsides, which may take several months; the foot enters a chronic stage. The chronic Charcot foot is painless and deformed, without a temperature differential. The mid-foot is commonly involved in Charcot's neuroarthropathy and can result in mid-foot collapse with a plantar bony prominence and rocker bottom foot. This is associated with a significantly increased risk of ulceration.[1]

Vascular assessment

Atherosclerotic vascular disease is likely to be present in most subjects with diabetes. Palpation of pedal pulses is routine in the diabetes clinic, however this test is subjective and can be influenced by many factors. Intermittent calf claudication is an uncommon presenting symptom in diabetes patients, as the calf muscles derive their blood supply from geniculate arteries that arise proximal to the popliteal trifurcation, a site often spared in diabetes-related peripheral vascular disease. More commonly, the tibio-peroneal trunk and crural arteries are affected, which can lead to foot claudication. However, the symptoms of foot claudication may be obscured by peripheral neuropathy. As a result, the initial detection of peripheral vascular disease is often heralded by the presence of cutaneous trophic changes such as corns, calluses, ulcers or frank digital gangrene.[30] Ankle brachial pressure index (ABPI) is the ratio of systolic blood pressure at the ankle to the systolic blood pressure at the brachial artery and is used to detect the presence of peripheral vascular disease. While, an ABPI of 0.90 or less suggests presence of peripheral vascular disease, an ABPI greater than 1.1 may represent a falsely elevated pressure caused by medial arterial calcification. In patients with symptoms and signs of peripheral vascular disease, ABPI has been reported to have sensitivity and specificity >90%.[32] However, in asymptomatic patients, this sensitivity may fall below 30%, suggesting that ABPI is less accurate for screening asymptomatic subjects.[33] Apart from these limitations, this test is easily performed, non-invasive and reproducible. Furthermore, a large study has shown the ABPI to be strongly related to the risk of foot ulceration.[25] More recently, the toe brachial pressure index (TBI) is being increasingly used as an effective alternative screening tool in diabetics as it is less influenced by arterial calcification than ABPI. However, the influence of peripheral neuropathy on toe blood pressures remains uncertain, thus compromising the accuracy of this tool in the presence of established peripheral neuropathy.[34,35] Doppler arterial waveform is another non-invasive tool used to assess the vascular status. The normal arterial waveform is pulsatile with a positive forward flow in systole, followed by a short reverse flow and a further forward flow in diastole. Even in the presence of neuropathy, the successful demonstration of this triphasic waveform can effectively exclude significant arterial disease in >90% of limbs.[36]

Ulcer assessment

Once an ulcer develops, it is essential to monitor its progress. Several foot ulcer classifications have been proposed. The simplest classification of an ulcer can be based on the underlying pathogenesis, i.e. neuropathic, ischaemic or neuroischaemic.[37] Figure 2 demonstrates a neuropathic ulcer, whilst Figure 3 highlights an ischaemic ulcer. The commonly used Wagner–Meggitt classification defines wounds by the depth of ulceration and the extent of gangrene.[38] The University of Texas system grades wounds by depth and then stages them by the presence or absence of infection and ischaemia.[38] However, none of these take into account measures of neuropathy or ulcer area. More recently, the International Working Group on the Diabetic Foot

Figure 2. Neuropathic foot ulcer.

Figure 3. Ischaemic foot ulcer.

(IWGDF) has proposed the PEDIS classification, which grades the ulcer on the basis of Perfusion (arterial supply), Extent (area), Depth, Infection and Sensation.[39]

Assessing foot ulcers for the presence of infection is another important issue. All open wounds are likely to get colonized with micro-organisms and it needs to be recognized that even virulent pathogens such as *Staphylococcus aureus* may sometimes represent colonizers. Hence, the presence of infection needs to be defined clinically rather than microbiologically.[19] Clinically the presence of infection is represented by purulent secretions or by presence of inflammation. Other signs suggesting infection include presence of friable tissues, undermined edges and foul odour.[40] Systemic manifestations such as fever or leucocytosis are uncommon, but their presence may suggest a severe infection.[41] Cultures should be sent, preferably from tissue specimens rather than wound swabs.[42] The specimen should be subjected to gram staining and be processed for aerobic and anaerobic cultures. Other investigations include a full blood count, inflammatory markers (ESR/CRP) and a plain radiograph. Plain radiographs can help identify foreign bodies, presence of gas in tissues and bone involvement. More sophisticated imaging modalities such as MRI, bone scans and leucocyte scans may be indicated in certain special situations.[19] The most important pathogens causing diabetic foot infections are the aerobic gram positive cocci such as *S. aureus*, beta haemolytic streptococcus and coagulase negative staphylococcus. They often cause monomicrobial infections, although patients with chronic ulcers or those who have recently been treated with antibiotics often tend to have polymicrobial infections with aerobic gram positive cocci in association with gram negative bacilli.[43–45] Obligate anaerobes may also contribute to this polymicrobial mix, especially in patients with foot ischaemia.[46] Some organisms, such as pseudomonas aeroginosa and enterococci, often represent colonizers and may not need to be targeted specifically.[47]

Recognizing the presence of underlying osteomyelitis is a diagnostic challenge. The presence of underlying osteomyelitis can be expected if bone is visible or palpable on probing. A significantly elevated ESR (>70 mm/h) is also suggestive, although this finding may be less sensitive. For osteomyelitis to produce abnormalities on plain radiographs, infection should be present for at least 2 weeks.[19] It is also important to realize that bony abnormalities on plain radiographs could also represent non-infectious Charcot's neuroarthropathy. Further radiological investigations such as technetium bone scans, leucocyte scans and MRI may be necessary in some patients to define underlying bony involvement. However, it needs to be recognized that diagnosing osteomyelitis in the presence of underlying Charcot's neuroarthropathy can be particularly challenging, especially in the absence of overlying skin ulceration as no form of imaging can reliably exclude osteomyelitis in this setting.[48]

Management of the 'diabetic foot'

General measures

On the basis of the aforementioned assessments, the National Institute for Clinical Excellence (NICE) has

Table 3 Risk stratification of the diabetic foot

Risk stratification	Clinical features	Suggested foot review
At low risk	Normal sensation, palpable pulses	Annual
At increased risk	Neuropathy or absent pulses	3–6 monthly
At high risk	Neuropathy or absent pulses in addition to deformity or skin changes or previous ulcer	1–3 monthly
Ulcerated foot	Foot ulcer	Active multidisciplinary foot care team follow-up

suggested risk stratification of the diabetic foot as shown in Table 3.[49]

Educating patients on issues of correct foot care and the importance of seeking early medical advice is crucial. Managing the diabetic foot should adopt a multidisciplinary approach to manage diabetes and its associated complications. Optimum glycaemic control is important. Although direct evidence linking improved glycaemic control and healing is lacking, there is sufficient agreement to suggest this would help indirectly by a number of mechanisms. First, chronic hyperglycaemia has been shown to impair leucocyte function, a key player in wound healing.[50] Secondly, poor glycaemic control has been shown to be associated with microvascular complications, with nephropathic patients having a 3-fold higher risk of amputations in comparison to those without nephropathy.[1] Smoking cessation is also likely to benefit by virtue of its effects on the vasculature. Addressing other associated cardiovascular risk factors such as dyslipidaemia and hypertension is also important. Lastly, the importance of regular foot care should not be underestimated. Regular foot care includes debridement of calluses as this has been shown to reduce peak plantar pressure by 26%.[51]

Treating diabetic foot ulcers

The importance of seeking timely help to aid healing of diabetic foot ulcers cannot be overemphasized. Treatment of diabetic foot ulcers largely depends on the underlying cause, i.e. ischaemia, neuropathy or a combination of both.

Treating ischaemic ulcers

Diabetes is a vascular disease and hence measures to reduce the overall atherosclerotic risk are essential. Smoking cessation, aggressive treatment of diabetic dyslipidaemia and hypertension and routine use of anti-platelet medications are pivotal in reducing this cardiovascular risk.[52–55] In some patients revascularization to achieve timely and durable healing may be necessary. Patients with supra-inguinal (aorto-iliac) disease may be amenable to angioplasty (with or without stents), with good long-term results being achieved at a low risk.[56] Open bypass surgery may be considered for those patients who do not have an endovascular option.[57] The treatment of infra-inguinal disease is more difficult.[58] The standard treatment for these patients is still femoro-distal bypass with autogenous tissue such as the long saphenous vein.[57,59] If such tissue is unavailable, prosthetic grafts may be used.[60] Most vascular surgeons and interventionalists agree that the multilevel, distal and calcified vascular disease seen in diabetics is unlikely to be amenable to conventional transluminal angioplasty.[61] However, more recently, the BASIL study has for the first time shown that percutaneous angioplasty may be considered as an acceptable option for some patients with severe limb ischaemia.[62] Given the important difference in early morbidity in this study, it appears that angioplasty, when technically feasible may be the favoured initial option. However, a *post-hoc* analysis suggested a possible late-survival advantage (beyond 2 years) in those patients treated surgically in BASIL. These results thus emphasize the need for surgeons and interventionalists to work as a team. Furthermore, the high overall mortality reported in BASIL (37%), suggests that the ischaemic limb is only the tip of an iceberg, reinforcing the importance of multidisciplinary team approach to address the total risk. Lastly, the authors of BASIL rightly emphasize that primary amputation may probably be the best option in some patients, and identifying these patients early may avoid the inappropriate use of these costly and potentially dangerous procedures.

Treating infected ulcers

General management comprises of cleansing the wound, debriding any necrotic material and probing with a blunt sterile instrument to identify any foreign bodies or exposed bone.[63] There is little data from randomized trials to guide the use of antibiotic therapy and hence the initial regime is usually selected empirically based upon clinical experience and local preferences. The antibiotic regime is subsequently modified on the basis of clinical response and wound culture/sensitivity results.[47,64] Commonly used oral antibiotic regimes include

amoxicillin–clavulanic acid, ciprofloxacin, cephalexin and clindamycin.[64] Topical antibiotics may often be effective in mildly infected ulcers, whilst the presence of severe infection may warrant use of parenteral antibiotics. Intravenous regimes commonly used include amoxicillin–clavulanic acid, imipenem–cilastin, ampicillin–sulbactam, peperacillin–tazobactam and broad-spectrum cephalosporins such as cefuroxime.[64] Suspicion of anaerobic infection may warrant the addition of metronidazole to this regime. The optimal duration of antibiotic treatment remains largely unknown. For mild infections, a 7–10 day course of antibiotics is usually considered to be sufficient, whilst more severe soft tissue infections may need up to 2–3 weeks of treatment.[42] It is important to bear in mind that the aim of antibiotic treatment is to cure the infection and not to heal the wound, which usually takes a much longer time. Extended antibiotic treatments not only increase the likelihood of antibiotic-related side effects, but also may lead to the development of antibiotic resistant strains.

Lastly, treating underlying osteomyelitis is an important therapeutic challenge. The presence of osteomyelitis warrants long-term treatment of at least 4–6 weeks duration with antibiotics that are capable of penetrating well into bone such as fluoroquinolones, clindamycin or fusidic acid.[65] Surgical resection still remains the most definitive treatment for osteomyelitis especially for patients not responding to antibiotics.[66]

Offloading

In simple terms, offloading refers to interventions aimed at relieving pressure from the wound area and redistributing it to healthy areas. Armstrong[67] rightly said that '*it is not what you put on these wounds that heals them, but rather what you take off*'. The simplest way of offloading is through strict bed rest, but this is inefficient, first in view of poor compliance and secondly due to fear of complications such as deep vein thrombosis and osteoporosis.[1] The best time-tested and evidence-based offloading technique is total contact casting (TCC) because compliance is assured and the bulk and weight of the cast reduces patient activities. TCC has been shown in various studies to aid in the accelerated healing of non-infected neuropathic ulcers.[67,68] TCC involves a padded cast moulded to the shape of the foot with a heel for walking. This relieves the pressure from the ulcer and distributes it over the entire foot, allowing more rapid wound healing. Although it allows mobility, the main disadvantage is that it needs expertise in applying it and needs changing at least once weekly. Also, TCC can limit the patient's daily activities such as bathing, besides not permitting daily wound inspection.[1] The later has led some centres to design a cast with a window to permit daily wound inspection and dressing.[69] Furthermore, TCC is contraindicated in patients with significant peripheral vascular disease, infected ulcers or osteomyelitis.[1] Other techniques of offloading include the use of removable offloading devices. This is more likely to be accepted by the patient, but is disadvantaged by poor compliance as patients can easily remove this device. To circumvent this, a new technique called 'instant total contact cast' is being used. In this, a removable cast is wrapped with a bandage or plaster of paris to make it difficult for the patient to remove, but allowing the health care professional to remove it easily when needed. This way compliance can be improved and the wound can be regularly reviewed.[70] Lastly, ambulatory braces, splints and modified shoes with rigid rockered soles may be used to offload and/or immobilize the foot in some patients.[67,71,72]

Preparing the wound bed and use of dressings

The wound bed needs to be prepared with a view to aid endogenous healing and to facilitate the benefits offered by other wound healing techniques.[73] Debridement is a crucially important process of this phase and includes the removal of necrotic, unhealthy and infected tissue from the wound bed. This is commonly achieved by sharp debridement, which is usually carried out by using a scalpel and forceps. Studies have confirmed that regular weekly sharp debridement is associated with more rapid wound healing.[74] The last decade has seen resurgence in the use of larval therapy to promote healing in chronic diabetic ulcers. Medicinal maggots used in larval therapy, secrete enzymes capable of selectively digesting the necrotic tissues and stimulating wound healing.[75] Furthermore, a recent small study by Bowling *et al.*[76] in 13 diabetic subjects with MRSA colonized ulcers treated with larval therapy for a mean duration of 3 weeks successfully eliminated MRSA in 12 patients and was associated with a significantly reduced slough and increased granulation tissue. However, larval debridement therapy at the present time suffers from a lack of a large-scale randomized control trial (RCT) evidence.

The importance of dressing wounds is well established, although the optimal type of dressing still remains unclear. Dressings commonly used are the standard wet and dry saline dressings, but they do not provide a sufficiently moist environment and may lead to non-selective tissue destruction.[1]

Semipermeable polymeric membrane dressings allow absorption of extravasated fluid from the wound bed, promoting wound healing and can be useful in uncomplicated chronic diabetic foot ulcers.[77] Kerraboot is a boot shaped dressing made up of a super-absorbent, polyacrylate-derived pad that can absorb exudates. It promotes a warm and moist environment that encourages granulation tissue formation and growth factor production. Studies have shown that it is easy to use and acceptable to both patients and health care professionals.[78] Promogran dressing consisting of a matrix composed of collagen and oxidized regenerated cellulose is believed to help by binding and inactivating proteases such as matrix metalloproteinase in the wound.[79] However, in a recent RCT, Promogran has been shown to be comparable to moistened gauze in promoting wound healing.[80] Hyaluronan dressings have also been tried in diabetic foot ulcers and helps by slowly releasing hyaluronic acid, which can speed wound closure by promoting keratinocyte migration.[81] Alginate dressings have the ability to activate macrophages within chronic wound beds and the subsequent pro-inflammatory response generated is believed to promote granulation tissue formation and early wound healing.[82] Unfortunately, none of these dressings have been tested in a large, well-designed RCT.

Sub atmospheric pressure dressing using vacuum assisted closure (VAC) can be achieved by placing foam dressing into a wound cavity and applying sub atmospheric pressure with the help of the VAC device.[83] This technique helps by reducing oedema, improving local blood flow and enhancing formation of granulation tissue. Furthermore, recent studies have confirmed its safety and efficacy in treating complex diabetic foot wounds and has been shown to lead to a higher proportion of healed wounds, faster healing rates and potentially fewer re-amputations than standard care.[84]

Use of custom footwear

Prescription shoes for the high-risk patient may help by reducing high plantar pressures and friction, besides accommodating foot deformities.[85] Patients at low risk may safely wear well fitting, good quality over the counter walking shoes.

Prophylactic foot surgery

The last decade has seen a dramatic interest in reconstructive foot surgery for the diabetic foot. Non-vascular foot surgery in diabetes may be classified into elective surgery (to alleviate pain), prophylactic surgery (to reduce risk of ulceration), curative surgery (to heal an open wound) and emergency surgery (to control limb and life threatening infection).[86] A short Achilles tendon may be associated with an elevated forefoot plantar pressure and hence may benefit from Achilles tendon lengthening surgery.[87] Tenotomy of toe extensors may reduce toe deformities, thus preventing recurrent ulcerations in this group of patients.[88] Metatarsal osteotomy may reduce the risk of ulcer recurrences in subjects with prominent metatarsal heads.[89] Similarly, patients with a mid-foot prominence may benefit from surgical removal of the prominence, with a view to create a more plantigrade (anatomical) foot.[90] However, currently there is no RCT evidence comparing surgery with medical therapy.

Treating Charcot's neuroarthropathy

This depends largely on the stage during which the disease is diagnosed. During the acute phase, there is evidence to suggest that offloading the affected foot by using a TCC is the most effective therapy. Use of TCC should continue until the swelling and hyperaemia has resolved. If the skin temperature is being monitored, the temperature difference between the affected and non-affected foot should be less than 1°C before the cast can be removed.[1] Once the cast is removed, custom-made footwear should be used.[1] Bisphosphonates are potent inhibitors of osteoclast activation and may be used in the acute phase of Charcot's neuroarthropathy. In this regard, intravenous pamidronate therapy has been shown to reduce disease activity as measured by markers of bone turnover.[91] Patients with Charcot's neuroarthropathy remain at an increased risk of future foot problems and hence need continued follow-up.

Conclusions

Disease of the foot is among one of the most feared complications of diabetes and comprises of varied pathologies such as, neuropathy, vasculopathy, neuroarthropathy, foot ulceration, infection and the potentially preventable endpoint, amputation. As the majority of amputations are preceded by foot ulceration, it is crucial to identify those at an increased risk. Once identified, specific interventions can be directed to reduce this risk. As these patients are also likely to harbour other associated complications of diabetes, they are best managed by a multidisciplinary team. The last decade has not only seen the emergence of new therapies, but also has confirmed the effectiveness of existing interventions. Each of these interventions, when used

appropriately, may reduce the risk of foot ulceration and with it the risk of amputation.

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