

# Practical management of neuropathic diabetic foot ulcers

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## Introduction

Diabetic foot problems are a common experience for people with diabetes. Although they are complicated multifactorial entities (with gaps in the evidence base guiding clinical management), a systematic multidisciplinary approach can improve wound healing rate and reduce amputation rates. This article describes a local approach to the management of diabetic neuropathic foot ulcers. The range of factors to be considered, including infection control and pressure relief, are discussed in relation to three typical cases.

The diabetic foot is a complex multifactorial and quite unique entity requiring a multidisciplinary approach. The combined skills of the podiatrist, physician, shoe-fitter, surgeon and nurse can reduce amputation rates by 50% (Edmonds, 1987; Thompson et al, 1991).

Diabetic foot problems are the most common reason for hospital admission in people with diabetes (Williams et al, 1994). Prompt treatment is essential to prevent a small lesion developing into a potentially limb-threatening problem. This article describes our approach to the management of diabetic neuropathic ulceration. This involves:

- Assessment of cause
- Relief of mechanical stress
- Control of infection
- Metabolic control
- Topical treatment
- Oedema and pain control
- Preventive follow-up care.

### Causative factors

Mechanical factors play a major role. Pressure, combined with deformity, and the loss of protective sensation, can lead to tissue damage. With continued trauma, full thickness skin ulcers may develop with the attendant risk of infection. *Table 1* lists the factors that can contribute to abnormal foot pressure.

### Case A: full thickness neuropathic ulcer without infection

Case A had a full thickness neuropathic ulcer over the first metatarsophalangeal joint with no clinical or radiological signs of infection

(*Figure 1*). The causative factor was a marked increase in activity levels at the start of the Speedway season! As in cases B and C, which follow, the vascular supply was good.

### Relief of mechanical stress

Regular debridement is essential to relieve mechanical stress but should only be undertaken by skilled staff, usually the podiatrist. Debridement removes callus, lowers plantar pressures and enables the true dimensions of the ulcer to be reviewed. Drainage of exudate and excision of dead tissue makes infection less likely and enables deeper swabs to be taken for culture. It also encourages healing (Steed et al, 1996).

Pressure relief is also essential. Various methods have been developed to assist this process because total non-weight bearing is

**Table 1. Factors contributing to abnormal foot pressure**

#### Intrinsic factors

- Bony prominences
- Limited joint mobility
- Joint deformity
- Callus
- Altered tissue properties
- Previous foot surgery
- Charcot neuroarthropathy

#### Extrinsic factors

- Poor footwear
- Foreign bodies in shoes
- Walking barefoot
- Falls and accidents
- Activity levels

## ARTICLE POINTS

**1** Diabetic foot disease is common but amputation should be rare.

**2** Relief of pressure and infection control are the most important elements of treatment in the neuropathic foot.

**3** Management requires a multidisciplinary approach.

**4** Education and footwear should be targeted at preventing re-ulceration.

## KEY WORDS

- Neuropathy
- Ulceration
- Cellulitis
- Osteomyelitis

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not an acceptable option to patients.

The aim is a redistribution of plantar pressure, 'resting' the wound site. This is usually best achieved by casting. The Scotchcast boot is removable, allowing easy inspection and redressing, but has the disadvantage that it requires the patient to actually wear it! For case A, being young and active, this was the method that most fitted his lifestyle.

Other individuals might have benefited from a total contact cast which has been considered the 'gold standard' of pressure relief. However, there are risks associated with it (Edmonds and Foster, 1999). In addition, they need to be changed frequently. This calls for adequate time and resources to be available — which are inevitably limited!

Another possibility would be the Diabetic Pneumatic Walker (Aircast), a bi-valved prefabricated cast lined with inflatable air cells, with plasterzote insoles which can be replaced with cradled insoles. Its application requires manual dexterity, limiting its use in some patients. Oedema can also cause problems with fitting.

Alternatively, weight-relief shoes can be used; the Darco Orthowedge shoe for forefoot problems and the Darco Heelwedge shoe for heel problems. However, while being much lighter than a Scotchcast, they need to be used with walking aids as they can be unstable.

Stability is also a concern with elderly patients. Temporary footwear with cradled insoles is helpful as are other measures including wheelchairs and Zimmer frames. Many patients may benefit from the use of

crutches and younger, more agile patients from the K9 orthopaedic walker.

**Infection control**

There has been much discussion as to whether or not antibiotics should be given prophylactically in situations such as case A's. Published evidence suggests that they are not necessary in the management of ulcers that are clinically clean (Jeffcoate, 2000), although Foster et al (1998) have claimed clinical benefits with more widespread prescribing (particularly in the presence of ischaemia).

Our policy, assuming a superficial ulcer with no underlying structures probed and no signs of infection, would be to avoid the use of systemic antibiotics. It is debatable whether regular superficial wound swabs have a place in this situation. Our standard antibiotic regimes are shown in Table 2.

**Metabolic control**

There is little evidence on metabolic control related to active ulceration. However, it makes intuitive sense to aim for the same standards of glycaemic control, lipid levels and blood pressure as for any person with diabetes who is at risk. Prospective trials in this area are urgently required.

**Topical treatment**

Topical treatment is perhaps less important than the factors above. Today, there is a bewildering array of products available, but the principles of wound healing remain the same, with a warm, moist environment providing optimum conditions for healing.

For case A, the ulcer is clean, superficial and non-sloughy with low exudate (Figure 1). Our choice of dressing would be either a gel (e.g. Granugel, Intrasite) with a non-adherent secondary dressing (e.g. Release); or a hydrocolloid (e.g. Comfeel or Granuflex). Although controversial (Foster et al, 1994), hydrocolloids, in our experience, have not been problematic where used appropriately and carefully monitored.

Novel treatments, e.g. Dermagraft and Regranex, while relatively expensive, may be useful in specific instances. Both have been shown to decrease healing times in chronic non-healing ulcers (McColgan et al, 1998; Young, 1999).



Figure 1. A full thickness neuropathic ulcer over the first metatarsophalangeal joint.

**Table 2. Standard antibiotic regimens used by the authors in the management of diabetic neuropathic ulceration**

Micro-organism	Antibiotics
Staphylococcus aureus	Flucloxacillin 500mg qds Clindamycin 300mg qds Co-amoxiclav 625 mg tds
Streptococcus (including group B,C and G)	Amoxycillin 500mg tds Erythromycin 500mg qds Clindamycin 300mg qds Co-amoxiclav 625 mg tds
Anaerobes	Metronidazole 400mg tds Clindamycin 300mg qds
Gram negatives	Ciprofloxacin 500mg bd

**PAGE POINTS**

- 1 Low intensity laser therapy has been shown to improve wound healing.
- 2 Once healed, the foot is at risk for life.
- 3 Insoles and hospital-provided footwear will be needed to prevent recurrence of ulceration.
- 4 Infection should be considered whenever pain, swelling or ulceration occur.

Low intensity laser therapy (Ashford and Baxter, 1994) has also been shown to improve wound healing (Schindl et al, 1999), and locally we have seen some good anecdotal results in long-standing chronic diabetic foot ulcers — achieving healing in static wounds; although clearly more work is needed in this area.

**Oedema and pain**

Oedema is a common complicating factor. Excessive tissue fluid may compromise the vascular status and nutritional balance, significantly reducing the healing rate of wounds. In some cases, it is directly related to the neuropathy. Diuretics may be helpful, but the patient needs to be educated into keeping the leg and foot elevated (preferably higher than hip level) as much as possible. In the presence of an adequate blood supply (ankle-brachial pressure index greater than 0.9), compression stockings may also prove useful.

Pain is less commonly a problem with neuropathic ulceration, but its presence can indicate a serious infection or other problem. The treatment of neuropathic pain will not be discussed in this article.

**Follow-up**

Once healed, the foot is at risk for life, and will require a cradled insole and prophylactic footwear provided by the hospital. The degree of deformity and level of required cushioning will dictate whether the patient requires bespoke shoes or extra-depth, ready-made orthopaedic shoes. Ideally, an orthotist should be available in the diabetic foot clinic. This enables joint consultations where decisions can be promptly made regarding the most appropriate footwear.

**Case B: full thickness neuropathic ulcer with cellulitis**

Infection in the diabetic foot is diagnosed by the presence of a purulent discharge and/or two or more signs of inflammation (redness, induration, pain, tenderness, and heat). Infection should be considered whenever local symptoms, such as pain, swelling or ulceration, occur; or when systemic problems, such as fever, malaise or poor glycaemic control, develop (Lipsky and Berendt, 2000).

Figures 2 and 3 show the plantar and dorsal views of case B's infected foot. The patient had not responded to low

dose antibiotics initially prescribed by the GP. We immediately started clindamycin 300mg qds, one of our standard regimes. The patient had no recollection of trauma, but on debridement a splinter of glass was removed. One week later, the foot had not shown a significant improvement, and the patient was admitted for intravenous antibiotic therapy (flucloxacillin 1g qds, amoxycillin 1g tds and metronidazole 500mg tds). The surgeons felt that drainage was not required at that time. The cellulitis resolved, and the patient was discharged a week later on oral clindamycin 300mg qds.

Provided the ulcer has been adequately debrided and pressure has been off-loaded, most mild soft tissue infections should resolve within two weeks of appropriate antibiotic therapy. Failure to respond within a week, or deterioration while on treatment, should prompt re-evaluation of therapy and of the need for surgery (Lipsky and Berendt, 2000).

For case B (Figures 2 and 3), the focus of infection could be easily identified, but this is not always the case. Cellulitis may present without any apparent break in the skin, and progress to a limb-threatening infection including abscess formation. X-ray may confirm this and gas may occasionally be seen within the tissues. These severe cases require intravenous broad-spectrum antibiotics, with cover for anaerobes. An urgent surgical opinion should also be sought with a view to drainage and removal of necrotic tissue.

MRSA infections are common (Tentolouris et al, 1999) and the approach should be in conjunction with local microbiologists. A topical approach to this problem may include povidone iodine dressings, e.g. Iodine, and cadexomer iodine dressings e.g. Iodosorb/ Iodoflex (Mertz, 1999).

As with case A, all the other factors above need to be addressed. Case B's ulcer healed with appropriate pressure relief. The patient has been fitted with moulded insoles and footwear and has received intensive education.

**Case C: full thickness neuropathic ulcer with osteomyelitis**

The predominant risk of any soft tissue infection within the foot is the extension of infection into underlying bone. Any foot presenting with infection should be X-rayed to detect: ● Osteomyelitis ● Gas in the



Figure 2. Plantar view of a full thickness neuropathic ulcer with cellulitis.



Figure 3. The dorsal view of the ulcer in Figure 2 (above).



Figure 4. A full thickness neuropathic ulcer with osteomyelitis on the apex of the hallux.



Figure 5. An X-ray of the foot in figure 4 (above) demonstrating bone damage.

tissues ● A foreign body.

However, early bony changes will not be immediately visible on a radiograph, but the ability to gently probe through the ulcer to bone has been shown to be highly predictive of osteomyelitis (Grayson et al, 1995).

Case C had a small apical ulcer on the hallux, with swelling and erythema (Figure 4). Radiological examination (Figure 5) showed destruction at the tip of the terminal phalanx.

Surgical treatment of osteomyelitis involves excision of the infected bone back to solid bleeding bone. However, a number of cases can be treated conservatively with prolonged antibiotic therapy, although the optimum duration of therapy is unclear (Mason et al, 1999). Edmonds and Foster (1999) suggest at least 12 weeks. It is increasingly common for oral antibiotics to be used, reducing the need for admission (Pittet et al, 1999). Our experience certainly bears this out with distal lesions, but less so with lesions in larger bones.

Choice of antibiotics may be guided by the results of deep wound swabs, or in some cases bone biopsy. The choice of antibiotics should include those with proven bone penetration, such as clindamycin 300mg qds or co-amoxiclav 625mg tds (Lipsky and Berendt, 2000). Regular radiological review is used to determine the outcome of treatment, and may include the use of computerised tomography, magnetic resonance imaging and 3-phase bone scans. Other forms of nuclear imaging are available in selected centres. Plain X-rays should be repeated every 6 weeks. If after 3 months of conservative treatment the ulcer persists with continued probing to bone, surgical intervention may be required.

Once again, assessment of cause, pressure relief, metabolic control and topical management need consideration, as discussed.

### Conclusion

The treatment of patients with diabetic foot ulceration remains difficult and challenging, but often rewarding. There is a lack of evidence on which to base the management of this problem. However, it is known that a multidisciplinary and systematic approach to the management of diabetic foot problems can and does reduce the rate

of amputations (Edmonds, 1987; Thompson et al, 1991). Importantly, ulcers do not heal if treated in isolation; social and other factors should be considered. It is important to treat the whole person, not just the foot. All neuropathic ulcers will heal, unless something is preventing them from doing so. The footcare team must identify and remove these obstacles and facilitate healing in the shortest possible time. ■

Ashford RL, Baxter GD (1994) Low intensity laser therapy: an introduction for podiatrists. *Journal of British Podiatric Medicine* 49(4): 51–5

Edmonds ME (1987) Experience in a multidisciplinary foot clinic. In: Boulton AJM, Connor H, Ward JD (eds) *The Foot in Diabetes*. John Wiley, Chichester

Edmonds ME, Foster AVM (1999) *Managing the Diabetic Foot*. Blackwell Science, Oxford

Foster AVM, Spencer S, Edmonds ME (1994) Deterioration of diabetic foot lesions under hydrocolloid dressings. *Practical Diabetes International* 14(2): 62–4

Foster A, McColgan M, Edmonds M (1998) Should oral antibiotics be given to 'clean' foot ulcers with no cellulitis? *Diabetic Medicine* 15 (suppl 2): S10

Grayson ML, Gibbons GW, Balogh K, Levin E, Karchmer AW (1995) Probing to bone in infected pedal ulcers: a clinical sign of underlying osteomyelitis in diabetic patients. *Journal of the American Medical Association* 273: 721–3

Jeffcoate W (2000) Use of antibiotics in uninfected ulcers may do more harm than good. *The Diabetic Foot* 2(4) 132–5

Lipsky BA, Berendt AR (2000) Principles and practice of antibiotic therapy of diabetic foot infections. *Diabetes Metabolism and Research Reviews* 16 (suppl 1)

Mason J, O'Keefe C, Hutchinson A (1999) A systematic review of foot ulcers in patients with type II diabetes mellitus II: treatment. *Diabetic Medicine* 16: 889–909

McColgan M, Foster A, Edmonds M (1998) Dermagraft in the treatment of diabetic foot ulcers. *The Diabetic Foot* 1(2): 75–8

Mertz PM (1999) The evaluation of a cadexomer iodine wound dressing on methicillin resistant *Staphylococcus aureus* (MRSA) in acute wounds. *Dermatol Surgery* 25: 89–93

Pittet D, Wyssa B, Herter-Clavel C et al (1999) Outcome of diabetic foot infections treated conservatively. A retrospective cohort study with long term follow-up. *Archives of Internal Medicine* 159: 851–6

Schindl M, Kerschman K, Schindl A et al (1999) Induction of complete wound healing in recalcitrant ulcers by low-intensity laser irradiation. *Photodermatology Photoimmunology and Photomedicine* 15: 18–21

Steed DL, Donohoe D, Webster MW, Lindsley L (1996) Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. Diabetic Ulcer Study Group. *Journal of the American College of Surgeons* 183(1): 61–4

Tentolouris N, Jude EB, Smirnof I, Knowles EA, Boulton AJM (1999) Methicillin-resistant *Staphylococcus aureus*: an increasing problem in a diabetic foot clinic. *Diabetic Medicine* 16(9): 767–72

Thompson FJ, Veyes A, Ashe H et al (1991) A team approach to diabetic foot care: The Manchester experience. *The Foot* 2: 75–82

Williams DD, Anthony P, Young RJ, Tomlinson S (1994) Interpreting hospitals admissions data across the Korner divide: the example of diabetes in the NW region. *Diabetic Medicine* 11(2): 166–9

Young MJ (1999) Becaplermin and its role in healing neuropathic diabetic foot ulcers. *The Diabetic Foot* 2(3): 105–7