IDENTIFYING AND MANAGING ACUTE INFECTION

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This case study will explore acute foot infection in a patient with type 2 diabetes.

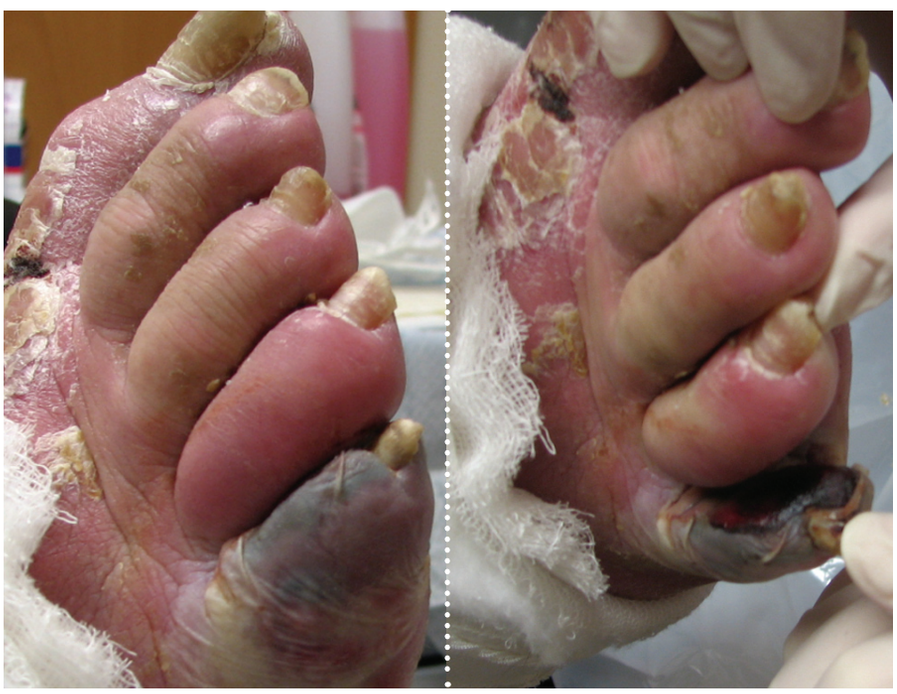
PATIENT DETAILS AND HISTORY

Mr A was a 74-year-old man who was a retired builder who was currently suffering from venous leg ulceration (*Figures 1a and b*). He had smoked for 40 years but had recently given up. He also had a 20-year history of type 2 diabetes. Throughout this time he had hypertension and, for the past 15 years, dyslipidaemia (raised lipid levels in the blood). He had suffered myocardial infarction two years previously and had a previous history of chronic foot ulceration (*Figure 2*). The presence of retinopathy (disease of the retina of the eye due to diabetes) meant Mr A was unable to see his legs and feet which had led to a delay in seeking treatment in the past.

He was on gliclazide and metformin to control his diabetes, low dose asprin, atenolol, nifedipine and ramipril for hypertension and ischaemic heart disease, and simvastatin for dyslipidaemia.

The following questions should be considered when assessing this patient:

1. What has caused this wound?
2. What factors in this patient's history make him susceptible to delayed wound healing?
3. What assessments are required?
4. What management strategies are appropriate in this case?
5. Who should be involved in this patient's care?



Figures 1a and 1b. The patient's foot presentation at the initital consultation.

ASSESSMENT AND ESTABLISHMENT OF TREATMENT OBJECTIVES

Foot ulceration is common in individuals with type 2 diabetes of long duration. This is usually a direct result of the chronic complications of diabetes, namely peripheral neuropathy (nerve dysfunction) and peripheral vascular disease (poor blood supply).

Mr A presented with a painless wound on his right fifth digit (*Figures 1a* and *1b*), caused as a direct consequence of trauma. The gentleman recalls dropping a can of tinned food on his foot but does not remember any pain at the time of the incident. When his wife examined his foot later that evening she discovered discolouration and discharge.

On initial presentation the right fifth digit was swollen, there was visible purulent (thick) exudate under the skin (*Figure 1a*) and on examination of interdigital tissues, haematoma formation (*Figure 1b*).

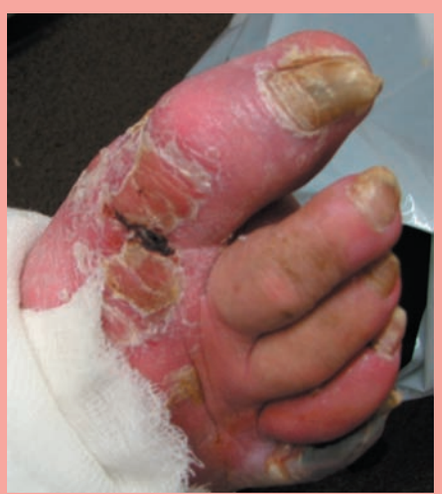


Figure 2. The patient's foot ulceration.

Figure 3. Provision of therapeutic footwear.

When establishing treatment objectives, it is important to undertake a sequenced assessment of known risk factors for delayed wound healing.

Neurological status

The fact Mr A is experiencing no pain suggests sensory neuropathy (loss of sensation). Neurological status was investigated and quantified by use of a neurosthesiometer, an instrument that tests vibration perception threshold (VPT). VPT is a useful measure to identify peripheral neuropathy in patients with diabetes (Garrow and Boulton, 2006). Findings, as expected, revealed loss of sensation in the feet. Additionally a monofilament was used to check for light touch sensation on both feet.

Vascular status

To establish the likelihood of wound healing it is crucial to establish vascular status. Pulse palpation, Doppler examination and non-invasive tests such as the ankle:brachial pressure index (ABPI) or toe brachial pressure index (TBPI) are useful clinical assessments to quantify arterial perfusion. In this case, tests revealed the presence of mild peripheral vascular disease.

Glycaemic control

To promote the healing of diabetic foot ulcers (DFU), it is imperative to achieve tight glycaemic control. The diabetologist was consulted to discuss the patient's current control as his HbA_{1c} results (measurement of glucose levels in the blood) were high (12%). Medication was reviewed and Mr A commenced insulin therapy to try to achieve better glycaemic control and dietary advice issued.

Identification of infection

Identification of in infection in the diabetic foot can prove challenging; the classic signs of infection are frequently absent due to arterial disease and neuropathy. However, in this case, the digit was swollen with spreading erythema (redness) extending to the forefoot and purulent exudate was visible. These are all signs of bacterial infection.

TREATMENT PROVIDED

Wound management strategies addressing the TIME acronym (Watret, 2005) were employed.

T: Tissue non-viable or deficient: The wound underwent sharp debridement by a specialist podiatrist to remove dead and devitalised tissue. It is imperative practitioners undertaking sharp debridement are appropriately trained in the technique, having undertaken appropriate training. If practitioners are not sufficiently skilled then a referral should be made to a member of the diabetes foot care team who is competent in sharp debridement.

I: Infection and/or inflammation: The primary objective in this case was prompt management of infection. Infection in the diabetic foot can spread rapidly causing extensive tissue necrosis (death). A wound swab was taken to identify infecting organisms. In the short term while awaiting swab results, a broad-spectrum systemic antibiotic was prescribed. Due to the presence of mild peripheral arterial disease, an antimicrobial dressing (a silver-impregnated product) was also applied direct to the wound, as antibiotic delivery to the site of infection can be impaired in the presence of peripheral arterial disease (Sheppard, 2005).

M: Moisture imbalance: Assessment of a DFU should include the volume and consistency of wound exudate. In this case the consistency was purulent and the volume of exudate was moderate to low.

E: Epithelium advancing or undermining: Healing of DFU can be impaired due to the presence of necrotic tissue. Frequent debridement by a specialist podiatrist was undertaken to reduce the bacterial bioburden, which could delay healing, and promote epithelialisation.

DRESSINGS USED

There is currently no optimum wound dressing for DFU. However, general consensus agrees a moist environment is required for optimum healing. A silver-impregnated hydrofibre dressing was selected as a primary contact layer to provide antimicrobial properties, absorb exudate and maintain a moist environment for healing. Silver dressings have been shown to have effective antimicrobial properties (Cooper, 2004). At present there are limited clinical studies that evaluate its effectiveness in DFU, hence the need for antibiotic therapy (Bergin and Wraight, 2006). A secondary foam dressing was selected for thermal insulation, conformability and cushioning properties around the digit.

INTERVENTIONS

Achieving effective offloading (pressure relief) is fundamental to promote wound healing in DFU. Total contact casting is considered to be the 'gold standard' method in offloading. A total contact cast is a specially moulded, padded cast that is applied to the lower limb and foot to evenly redistribute pressure across the entire plantar aspect of the foot. However, in this case, total contact casting was contraindicated due to the presence of infection. Casting prevents regular visual inspection of the wound, an essential aspect of patient care when managing infection in the diabetic foot.

A referral was made to the orthotist to request therapeutic footwear. Therapeutic footwear can be a useful adjunct to assist the healing of DFU by offering extra width and depth (Maciejewski et al, 2004). In this case a soft, neoprene upper reduced pressure on the digit thus facilitating healing (*Figure 3*).

OUTCOMES TO BE REVIEWED

Regular review of clinical outcomes is essential to monitor wound healing. This should include:

- Establishing the effectiveness of wound dressing regimens
- Monitoring infection closely
- Reviewing footwear
- Advocating tight glycaemic control
- Encouraging the patient to optimise his own diabetes control and foot health through health promotion
- Communicating with and involving all members of the multidisciplinary team to achieve optimum outcomes.

CONCLUSION

The management of DFU is often complex and challenging. Mr A had a long duration of diabetes mellitus which placed him at risk of diabetes-related complications such as peripheral neuropathy and peripheral vascular disease both of which are known to delay wound healing. In addition his glycaemic control is poor (HbA_{1c} 12%) which increases ulcer risk and can impede healing. A past and present history of chronic lower-limb ulceration further increased the likelihood of more episodes of non-healing wounds. In this case the wound was caused by inadvertent trauma in an insensate foot.

Rapid management of infection in the diabetic foot is essential. Systemic antibiotics were prescribed and topical anti-microbial dressings initiated. Localised wound care based on TIME principles ensured wound bed preparation and off loading was achieved with cushioning dressings and provision of therapeutic footwear. A team approach is essential to optimise this patient's care. In this case the diabetologist, specialist podiatrist, diabetes specialist nurse and orthotist were key to implementing immediate management strategies. As Mr A is also receiving treatment from district nurses for chronic leg ulceration it is imperative the diabetes MDT liaise directly with the district nursing team. While treatment objectives may differ for leg ulceration compared to DFU the common goal in Mr A's care is to promote optimum wound care and achieve healing. **WE**

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