

Oedema management in a diabetic patient with foot ulceration and peripheral vascular disease: a case study

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Article points

1. Lower limb oedema can delay wound healing in diabetic patients with foot ulceration.
2. Compression therapy can be of benefit in diabetic patients with foot ulceration and wound healing, but requires close monitoring and supervision to prevent complications.
3. Close multidisciplinary working ensures the most appropriate care for the patient.

Key words

- Compression therapy
- Lower limb oedema
- Multidisciplinary working
- Wound healing

Authors

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Peripheral oedema is a recognised complication in prolonging wound healing. Despite this, there is little evidence available on the use of compression therapy in the presence of diabetic foot disease. The case study outlined in this article demonstrates how careful assessment, monitoring and close multidisciplinary working facilitated the use of a reduced compression system to aid wound healing in a diabetic foot.

Peripheral oedema in the lower limb can occur due to a number of different factors and often plays an underestimated part in prolonging wound healing. Injudicious use of compression carries the risk of tissue necrosis due to underlying peripheral vascular disease. Applying rigid guidelines may exclude eligible patients who may benefit from compression. This case study presents a man with long-standing neuroischaemic diabetic foot ulceration, complicated by the presence of peripheral oedema, and highlights the importance of a multidisciplinary approach when addressing oedema management in this complex population.

Case study

A 78-year-old male, Mr G, with a 14-year history of diabetes presented to the diabetic foot multidisciplinary team for a review of a non-healing surgical wound to the right forefoot, 18 months post-surgery.

Background

The patient had originally presented to the diabetic foot clinic in 2012 with dry necrosis of toes 1–4 bilaterally, for which the original cause was unknown. Despite antibiotic therapy and regular dressings, the necrosis became infected and required amputation of these toes. Post-surgery, the left foot achieved full healing, however, the right foot wound was slow to progress.

Mr G's previous medical history included a myocardial infarction and coronary artery bypass graft. He had had recurrent cellulitis in both legs, requiring hospital admission four times in the previous year. At the time of amputation, he had also had a bilateral angioplasty with stenting of the superficial femoral artery and popliteal artery. His diabetes was well controlled on gliclazide alone, with an HbA_{1c} of 56 mmol/mol. He was a non-smoker and was taking a prophylactic dose of penicillin due to recurrent cellulitis.

The presence of oedema in his legs had caused Mr G to lose his independence, as he no longer felt safe to drive. Mobilising was difficult and he was having to rely upon friends and family for day-to-day tasks. He reported that he was 'fed-up' as the current situation was significantly impacting upon his quality of life.

Examination

On examination, the right forefoot wound measured 70 mm × 34 mm, with tendon and slough visible at the wound bed (*Figure 1*). Exudate levels were high, requiring dressings every other day.

Arterial examination revealed crisp monophasic pulses at the dorsalis pedis and posterior tibial vessels bilaterally with non-compressible signals at 140 mmHg. The angioplasty and stenting was patent and the vascular team felt there were no further options for vascular reconstruction. The

Figure 1. Mr G's right foot at presentation to the multidisciplinary team. (a) shows the wound bed and (b) the evidence of lymphovenous disease in Mr G's legs.



patient was known to have neuropathy with 0/10 sites felt using a 10g monofilament and vibration sensory threshold greater than 25 volts.

Common factors for non-wound healing had been addressed: poor diabetic control, footwear, pressure relief and underlying infection — including osteomyelitis — had been ruled out. However, there was found to be significant lower-limb oedema bilaterally with clinical signs of haemosiderin staining, atrophie blanche, lipodermatosclerosis, and hyperkeratosis to the lower legs. These were all features of lymphovenous disease, which had not previously been addressed (*Figure 1*).

After discussion with the vascular team, it was felt that a closely-supervised trial of light compression of 17–25 mmHg would be acceptable. Mr G was commenced in three-layer reduced-compression elastic bandaging system by the tissue viability nurse. An antimicrobial non-adherent contact layer and absorbent pad were used beneath the compression, to manage exudate and prevent infection.

Careful liaison with the community teams was instigated and the patient was reviewed regularly in the diabetic foot clinic. Within 2 weeks, the wound had reduced to 53mm × 31mm. After 8 weeks, the patient drove to clinic for the first time in 18 months and he reported that his legs felt much better. He was pleased that he had regained his independence and was able to mobilise easier.

The wound had also now reduced in size to 37mm × 30mm. Within 4 months, the wound had reduced by 60% and continued to improve, becoming shallower with evidence of granulation and epithelisation tissue throughout (*Figure 2*). Furthermore, the patient had encountered one episode of cellulitis during this time, which was managed with a single course of antibiotics and did not require hospital admission.

Discussion

Peripheral oedema can present in the lower limb as a consequence of a number of different pathologies. Chronic venous insufficiency is the most common cause and is often as a result of venous hypertension due to varicose veins, incompetent deep or perforator veins, or calf muscle pump failure leading to an uncompensated fluid filtration (Gordon et al, 2015; Kelechi et al, 2015). Lymphoedema may often co-exist and is exacerbated by repeated infection, which compromises existing lymphatic function.

Oedema is a widely acknowledged risk factor in prolonging and complicating wound healing, by reducing capillary blood flow and increasing the diffusion distance of oxygen to the wound site, thus reducing the oxygenation and nutrition required for wound healing at a cellular level (Rhou et al, 2015). Furthermore, accumulation of fluid in the interstitial space increases exudate levels from the wound and consequently periwound maceration is a common feature, which can lead to further tissue breakdown (Young et al, 2011).

The presence of oedema often leads to the wound remaining in a chronic inflammatory phase. High neutrophil levels and the presence of matrix metalloproteinases at the wound site are common features of a chronic wound, which are known to delay healing. Therefore, in order to achieve wound healing, the chronic inflammatory phase must be overcome (Diegelmann and Evans, 2004; Ho et al, 2012).

In the case presented here, there may have been a number of reasons why oedema developed. Mr G had become immobile due to oedema in his legs leading to calf muscle pump failure, had experienced recurrent episodes of cellulitis and the long saphenous vein had been harvested for

his coronary bypass, all of which are factors in the development of chronic venous insufficiency and oedema (Kelechi et al, 2015). However, patients with long-standing venous insufficiency are at a higher risk of lymphatic involvement and it was felt the presence of mixed lymphovenous disease was likely to be the predominant cause of the oedema in this case (Williams, 2009).

Diabetes is frequently cited as being a risk factor for chronic venous insufficiency and oedema, however, there is limited literature describing oedema management in the presence of diabetic foot disease.

Neuropathy was identified in a study by Lefrandt et al (2003) to be associated with a significant reduction in the degree of vasoconstriction in the arteriovenous anastomoses and precapillary arterioles in the dermis of the foot. Additionally, when compared to matched participants with diabetes and no neuropathy and a control group, there was an increase in



capillary permeability ($P < 0.02$). The absence of a response by the sympathetic nervous system, a consequence of autonomic neuropathy, leads to the loss of vasoconstriction of the pre-capillary arterioles, causing hydrostatic loading of the capillary bed, increasing capillary permeability. This consequently alters the capillary exchange rate leading to oedema (Ho et al, 2012).

Figure 2. Notable improvement in the wound bed 7 months after compression therapy was started.

Two studies have shown oedema is directly related to poorer outcomes in diabetic patients with foot ulceration. In a prospective study by Apelqvist et al (1990), the presence of oedema was found to be more prevalent in people with diabetes requiring an amputation (58%) or in patients who died (55%) when compared to patients who achieved healing (26%) ($P < 0.001$). The double-blinded randomised control trial carried out by Armstrong and Nguyen (2000) found that aggressive oedema management, by the use of a pneumatic pedal compression system, increased healing rates in post-surgical debridement wounds of the foot, in patients with diabetic neuropathy. Seventy-five per cent of participants in the active arm of the study achieved healing compared with 51% in the placebo group. Moreover, the active arm showed greater oedema reduction compared to the placebo group.

The gold standard for the treatment of venous insufficiency and lymphovenous disease is compression therapy (Williams, 2009), however, many healthcare professionals are reluctant to treat oedema in patients with diabetes, due to the well-described risk of peripheral arterial disease in this population. In the presence of moderate peripheral arterial disease and venous disease, a reduced elastic compression therapy system is effective in the management of oedema (Dowsett, 2006). This system omits the highly elastic third layer of a four-layer compression system, enabling a constant resistance of 17–22mmHg to be applied to the limb, and was used in the case presented. Bowering (1998) found reduced compression effective in the healing of diabetic patients with venous leg ulcers and inadequate circulation, with 67% achieving healing. This was compared to 81% of patients who had adequate circulation and were treated with a full four-layer compression system. There were no further arterial compromise or complications reported in the group of patients with reduced circulation. However, to date there is little literature on the use of compression and diabetic foot ulcerations.

Conclusion

Oedema management in people with diabetes can be complex and is often complicated by the presence of peripheral arterial disease. The role of the multidisciplinary team, in this case vascular

surgeons, tissue viability specialist nurses, podiatrists and endocrinologists was imperative to ensure a careful assessment and appropriate treatment plan. Although it was acknowledged that vessel calcification contributed to an erroneously high ankle pressure in this case, it was the good quality of the arterial signal that led to the decision to use reduced compression, but only under very close supervision.

The role of oedema management and reduced compression is widely reported in the literature. However, further research is required regarding the role of oedema and its management in diabetic patients with foot disease. This case highlights the important role oedema management can play. This is not just in wound healing in the diabetic foot, but also in improving quality of life for the patient by increasing mobility and reducing hospital admissions for cellulitis.

The early identification of patients with venous disease allows early intervention, to prevent further complications, such as venous leg ulcerations and highlights the need for oedema management to be considered as an adjunctive therapy in the treatment of selected diabetic foot wounds. ■

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