Bilateral heel ulcers: a complex case for vacuum assisted closure

Freeman A

Abstract

The heels are the second most common area of the body for pressure wound development, accounting for 20-30% of all pressure lesions [1,2]. Successful treatment, including offloading of heel pressure wounds, can be challenging, particularly in the patient with diabetes and associated complications of neuropathy, peripheral vascular disease and increased susceptibility to infection. Topical negative pressure wound therapy (TNPWT) has the potential to accelerate healing of diabetic foot ulcers and decrease incidence of amputation. This paper discusses one case of the successful use of TNPWT in a complicated case of diabetic foot ulceration.

Introduction

The human foot is the 'sole' weight-bearing structure during ambulation and activities of daily living, and preserving it in cases of complicated ulceration can be significantly enhanced with modern wound management technologies. Topical negative pressure wound therapy (TNPWT) has the potential to accelerate healing of diabetic foot ulcers and decrease incidence of amputation. A retrospective study by Frykberg & Williams [3] found diabetic foot ulcers were less likely to result in amputation when treated with TNPWT compared to 'control' wound care. Similarly, Armstrong & Lavery [4] discovered diabetic foot amputation sites treated with TNPWT were more likely to heal and at a faster rate than similar wounds treated with standard moist wound care.

Case report

Mr Boots [pseudonym] was referred to the diabetic foot unit at the Royal Melbourne Hospital in May 2007, following the development of bilateral Stage III heel pressure ulcers during a hospital admission. Mr Boots was a community dwelling, 68 year old man who was admitted for sepsis from a urinary tract infection leading to delirium. He had type II diabetes mellitus with complications including ischaemic heart disease, peripheral vascular disease, neuropathy, retinopathy and nephropathy.

On assessment, the right and left heel wounds were virtually identical. The wounds measured approximately 40x25mm and were 95% necrotic pre-surgical debridement. Wound swabs indicated infection of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas aeruginosa* organisms. The calcaneal bone could be probed in the wound bases and radiological imagining was positive for bilateral osteomyelitis. Duplex ultrasound indicated three vessel run off to the right foot and one vessel run off to the left foot.

Mr Boots was deemed not a candidate for vascular surgical intervention due to his multiple acute medical problems. The aims of management were therefore to remove unwanted necrotic tissue, eradicate infection, encourage granulation tissue formation, control exudate and offload pressure.

Wound management

Weeks 1-4

The wound bases were surgically debrided of all necrotic tissue to reveal 100% granulation tissue. TNPWT was initiated immediately after the procedure. The device was set to 125mmHg on continuous therapy for 48 hours postoperatively, after which it was changed to intermittent therapy of 5 minutes on, 2 minutes off. This regime is in accordance with evidence-based TNPWT recommendations using a vacuum assisted closure device [VAC® therapy, KCI, San Antonio, Texas] [5].

Pressure relieving ankle-foot orthoses (PRAFOs) were provided to allow complete offloading of the heels. Antibiotic therapy was commenced with intravenous vancomycin and clindamycin and oral metronidazole for the initial 4 weeks of treatment. During this period, the wound beds were approximately 80% granulation tissue and 20% slough. Non-viable tissue was debrided regularly at dressing changes.

Following 4 weeks of TNPWT, the wounds had each decreased to 35x20mm and were superficial with little remaining cavity.

Amy Freeman BPod (Hons)
Podiatrist, The Royal Melbourne Hospital
Grattan St, Parkville VIC
Tel (03) 9342 7134

Weeks 4-16

As TNPWT had addressed the tissue deficit successfully, the wound dressing was changed to silver impregnated hydrofibre (primary dressing) and absorbent pad (secondary dressing). The aims of this regime were to decrease local bacterial load and control exudate levels whilst maintaining a moist wound environment. The wounds were debrided of any slough or other non-viable tissue on a fortnightly basis. This regime, along with the continued use of the PRAFOs, was followed successfully for 12 weeks. At Week 4, intravenous antibiotic therapy was replaced with oral rifampicin, fusidic acid and ciprofloxacin for 2 months.

Sixteen weeks following initial referral, the wound beds had decreased to <10x10mm and contained 90% granulation tissue and 10% slough.

Weeks 16-18

By this time, exudate levels had decreased significantly and all signs of clinical infection had resolved. Antibiotics were ceased. A silicone backed adhesive foam was considered appropriate in the final stages of healing as the exudate level had decreased and the aim was to protect the fragile newly formed skin.

As the wounds healed and rehabilitation became a priority, the PRAFOs were replaced with protective postoperative footwear. Both the right and left heel wounds healed during the 18th week after initial referral. To date, 11 months following complete healing, Mr Boots remains wound free and independent. He wears custom-made insoles in extra width and depth footwear and sees a community podiatrist in a diabetes foot service regularly.

Discussion

This case illustrates the successful use of TNPWT in contributing to healing of bilateral necrotic heel pressure ulcers complicated by osteomyelitis. In addition to the successful salvation of both lower limbs, the wounds were able to heal without residual deformity. Wounds on the feet managed with other dressing regimes often heal with deformation that goes on to be a site of future tissue fragility and high risk of breakdown. The open pore structure of the VAC® granufoam applies an equal distribution of negative pressure over the entire wound base, encouraging the formation of granulation tissue and preventing epithelialisation over a deformed cavity [6].

Initial surgical debridement of Mr Boots' wounds allowed thorough wound investigation and provided a clean and healthy wound bed favourable for TNPWT. Guidelines regarding TNPWT in the diabetic foot recommend all neuropathic diabetic foot wounds must be debrided and be sufficiently offloaded before TNPWT begins [7]. Other features of TNPWT include the ability to control high levels of wound exudate and remove excessive waste and infectious material without significantly increasing frequency of dressing changes. TNPWT has also been shown to promote angiogenesis with maximum local blood flow achieved at a local negative pressure of 125mmHg [6].

The specifically-designed VAC® heel foam with prefabricated bridging piece allows the TRAC® pad and tube to be placed on the dorsal surface of the foot instead of directly over the wound at the posterioplantar aspect of the heel. This design prevents pressure related complications that may occur with the patient lying supine with the heels on the mattress surface with a TNPWT dressing in situ. Further, offloading pressure to the heels with PRAFOs in the initially healing stages was vital to the successful outcomes achieved. Similarly, appropriate protective footwear is desired through all stages of wound healing and when the wound has healed in patients with high-risk feet.

Wound dressings utilised following cessation of TNPWT included a silver hydrofibre and absorbent pad. Although the true antimicrobial effects of silver impregnated hydrofibre dressings remain unclear, motives for this dressing choice were to decrease local bacterial contamination and to control high exudate levels.

TNPWT can appear at first as an overwhelming dressing choice to untrained clinicians; however, it needs to be utilised more frequently as the first line of wound management in cases such as this. Chronic foot ulcers complicated by osteomyelitis often sadly lead to amputation and, in certain cases, death. The successful healing of a deep foot ulcer with its anatomical shape preserved more than justifies the time and resources invested in healing these wounds with TNPWT technology – just ask Mr Boots.

References

1. Amlung S, Miller W & Bosley L. The 1999 National Pressure Ulcer Prevalence Surgery: a benchmarking approach. Adv Skin Wound Care 2001; **14(6)**:297-301.
2. Cheneworth C, Hagglund K, Valmassoi B & Brannon C. Portrait of practice: healing heel ulcers. Adv Wound Care 1994; **7(2)**:44-49.
3. Frykberg R & Williams D. Negative-pressure wound therapy and diabetic foot amputations. J Am Pod Med Assoc 2007; **95(5)**:351-359.
4. Armstrong D & Lavery L. Diabetic Foot Study Consortium. Negative pressure wound therapy after partial foot amputation: a multicentre randomised control trial. Lancet 2005; **366(9498)**:1704-1710.
5. Banwell P (Ed). VAC Therapy Clinical Guidelines. London. KCI Europe Holding, 2007.
6. Armstrong D, Boulton A & Banwell P. Topical Negative Pressure: Management of Complex Diabetic Foot Wounds. Oxford: Oxford Wound Healing Society, 2004.
7. Andros G, Armstrong D, Attinger C, Boulton A, Frykberg R, Joseph W *et al.* Consensus statement on negative pressure wound therapy (VAC therapy) for the management of diabetic foot wounds. Ostomy Wound Manage 2005; **June(Supp 1).**