

V.A.C.[®] Therapy and the diabetic foot

A report from a satellite symposium held on the occasion of the 9th Annual Conference and Exhibition of *The Diabetic Foot Journal*. The meeting took place on 30 September 2008 at The Ibis Hotel, Earls Court, London and was sponsored by KCI Medical.

Introduction

With one of the most substantial, and growing, bodies of clinical evidence in the wound-care industry, V.A.C. Therapy[®] (vacuum-assisted closure; KCI Medical, Kidlington) continues to demonstrate its effectiveness in the promotion of wound healing (e.g. Morykwas et al, 1997; Joseph et al, 2000). The purpose of this symposium was to outline the science behind the success of V.A.C. Therapy, and to look at its use in the podiatric setting. Furthermore, the use of V.A.C. GranuFoam Silver[®] (a silver-impregnated dressing; KCI Medical, Kidlington) in conjunction with V.A.C. Therapy was described. Speakers were Claire Weston (Clinical Marketing Manager, Northern Europe, KCI Medical), Tania Woodrow (Diabetic Specialist Podiatrist, Cornwall) and Frank Bowling (University of Manchester, Manchester Royal Infirmary, Manchester). Helen Tyrer (Diabetes Specialist Podiatrist, Manchester) assisted Frank with his presentation.

V.A.C. Therapy is a non-invasive, topical negative pressure system that helps promote wound healing. The V.A.C. Therapy units deliver negative pressure, continuously or intermittently, to the wound, which decompresses a foam dressing. V.A.C. Therapy is prescribed for a variety wound types including diabetic foot wounds (KCI Medical, 2008a).

The science behind V.A.C. Therapy

The first speaker, Claire Weston, discussed why, from the macro level to the micro level, V.A.C. Therapy delivers quality outcomes in wound healing. Six mechanisms of action were highlighted: provision of a closed, moist wound environment; removal of exudates; increases in local blood perfusion; stimulation of cell proliferation; promotion of granulation; and reduction of localised oedema.

At the macro level, the topical negative pressure of V.A.C. Therapy creates a closed, moist, air-free environment in which the wound healing process can safely take place. Furthermore, V.A.C. Therapy removes from the wound site exudates that inhibit healing. This includes the removal and inhibition of matrix metalloproteinases and proinflammatory cytokines; molecules important in the initial, acute wound healing phase, but ones that need to be modulated and controlled in post-acute or chronic wounds, in which they have been shown to degrade key wound-healing molecules and inhibit

mitosis (e.g. Agren et al, 1992). At a cellular level, V.A.C. Therapy has been shown to promote local blood perfusion in various human and animal models (e.g. Morykwas et al, 1997; Timmers et al, 2005), allowing for the more efficient removal of waste products from the wound and delivery

of antibiotics. Morykwas et al (1997) found that peak blood flow, four times above baseline, could be achieved with V.A.C. Therapy at -125mmHg of negative pressure. More recently however, Timmers et al (2005) achieved a five-fold increase in perfusion from baseline with V.A.C. Therapy at -300mmHg in association with V.A.C. GranuFoam[®] (KCI Medical, Kidlington; $P<0.001$). Claire suggested that the question of which pressure provides the best results is one that remains open, needing further research to clarify.

She also reported on findings that indicate V.A.C. Therapy induces microdeformations in wound surfaces, creating both normal and lateral compressive stress, and thus simultaneously stretching and compressing tissue at the site of foam contact (Saxena et al, 2004). This micro strain or "cell stretch" has been shown in adult dermal fibroblasts to alter cell shape and trigger

signalling pathways for controlled mitosis (McNulty et al, 2007).

Additionally, granulation and oedema have been shown to be positively impacted upon by V.A.C. Therapy. Morykwas et al (1997) found that wounds treated with V.A.C. Therapy filled with granulated tissue at a significantly greater rate than control wounds ($P\leq 0.01$). A reduction in oedema after V.A.C. Therapy was seen in work conducted by Kamolz et al (2004), and is thought to be the result of the mechanical removal of fluid and the therapy's effect on the inflammatory response that causes oedema.

Claire also highlighted recent findings by Blume et al (2008) who specifically examined V.A.C. Therapy in relation to diabetic foot ulcers. Use of the therapy in this controlled study resulted in a significant increase in the number of ulcer closures ($P=0.007$), a significant decrease in the median time to ulcer closure ($P=0.001$; closure was not achieved within the study period for the control group), and a significant reduction in the number of people who experienced a secondary amputation ($P=0.035$). These results are encouraging, showing that V.A.C. Therapy can help diabetic foot wounds move along a more acute healing

pathway, achieving positive outcomes.

Provision of local care

Tania Woodrow opened her discussion with results from Armstrong et al's 2005 study on V.A.C. Therapy in the treatment of the diabetic foot, a paper she says changed her professional life. Tania found Armstrong's data compelling, and moved to establish a diabetic foot clinic at the Royal Cornwall Hospital, Triliske. The diabetic foot clinic team looked to establish pathways to healing, including the use of V.A.C. Therapy.

Several challenges faced Tania in achieving funding for the use of V.A.C. Therapy in the community setting of their foot clinic. These included convincing the PCT executive board that this was the way to treat wounds, and that it could be undertaken by podiatrists. Tania achieved initial, case-by-case funding for V.A.C. Therapy, but the delay this caused in using the treatment prompted her to seek a more permanent funding arrangement.

Tania stressed that when securing funding, it is important to build a case based on previous successful examples, and findings from the literature. Especially when presenting a case to non-clinicians, Tania found that photographs illustrating the reality of the results can be very persuasive. Building such a case had the desired effect for Tania's clinic; a budget stream was created for V.A.C.

Therapy, allowing the foot clinic team to directly order units and consumables, and to determine the length of therapy by patient need, rather than financial considerations.

During the first 12 months of funding, 25 people with post-surgical diabetic foot wounds were treated by Tania's foot clinic team. Of the 25 people, 12 had healed, 8 were progressing to healing, and, at the time of the meeting, 5 had failed to heal. The community contract with KCI Medical ended in September 2008 and funding was to return to a case-by-case assessment process. However, Tania negotiated a small contract with the manufacturer independently, allowing treatment to continue.

Tania stressed that the results achievable with V.A.C. Therapy as part of an advanced wound care programme for the diabetic foot are significant, and while securing funding can be a difficult and ongoing process, it is certainly worth the effort.

Prevention of wound deterioration

The third speaker, Frank Bowling, discussed the use of V.A.C. GranuFoam Silver dressings as an adjunct to V.A.C. Therapy. He began by reminding the group that people with infected diabetic foot wounds are at higher risk of amputation (Armstrong et al, 1998) and must be carefully managed so as to prevent deterioration.

When looking at infected

wounds, it is important to (i) determine the pathogen causing the infection, and (ii) distinguish between contamination, colonisation and true infection of the wound. As many normal flora benignly coexist with their human hosts, or even provide protection against other invading microbes, the presence of a given pathogen is only significant when injury and tissue invasion result. Thus, frequent wound surface cultures are central in identifying an infecting microbe, and determining a course of treatment.

Frank outlined the two-fold action of the V.A.C. GranuFoam Silver dressings, used in conjunction with V.A.C. Therapy. First, the silver ions impregnated in the dressing create a protective barrier on the wound surface, reducing the presence of a range of pathogens. Second, application of topical negative pressure via GranuFoam Silver promotes cell stretch and microdeformations on the wound's surface, which in turn triggers signalling pathways for controlled mitosis.

During question time, it was suggested that a less-expensive fenestrated silver dressing could achieve the same deformation results; Frank stressed that GranuFoam Silver acts not only at the macro level to create deformation, but also at the micro level, and that this microdeformation, in conjunction with the antibacterial properties of silver ions, reduces the risk

of abscess formation (KCI Medical, 2008b).

"With appropriate interventions, at appropriate times," Frank told the group, "we can prevent wound deterioration." ■

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