

# Debridement and wound healing using larval therapy

Professor Linda Rafter is Honorary Professor in the Nursing Faculty of Health and Life Sciences, De Montfort University Leicester, and Wound Care Solutions Tissue Viability Nurse Consultant, Burton-on-Trent, Staffordshire

This article, the first in a two-part series, reports on the management of a patient who was newly diagnosed with diabetes and had developed two foot ulcers, which presented clinicians with complicated challenges if they were to save the patient's limb. Management of these complex wounds often requires a careful, multidisciplinary approach.

Biosurgery has been employed and used effectively in wound care for a number of years (Thomas, 2006). Due to the severity of the wounds, the case featured in this article involved a six-week period of debridement with a BioFOAM® Dressing (ZooBiotic Ltd, Bridgend). The larvae used in BioFOAM debride devitalised tissue through sealed net bags that also contain foam chips — the role of the chips is to absorb exudate. The rationale for the use of the BioFOAM maintenance dressing was that the wound had debrided adequately and the author wanted to maintain the healing process through the larval stimulation of the wound tissue and prevent the wound resloughing. The debridement and maintenance BioFOAM dressings were re-applied every five days. This six-week period of debridement was followed by the administration of BioFOAM maintenance dressing to continue the wound healing progress (altogether the treatment programme lasted for 13 weeks).

The second article in the series will explore the wound healing process from the patient's perspective, including how the wound healing process affected quality of life.

## Background

The annual financial burden of diabetic foot disease in the UK is as much as £252m (NHS National Diabetes Support Team, 2008). The rate of lower limb amputation is estimated at 2,600 amputations a year in patients with foot ulcers (there are around 64,000 people with active foot ulcers at any one time) (Posnett and Franks, 2007).



Figure 1. Plantar ulcer on dorsum of left foot.



Figure 2. Left forefoot wound.

Diabetes UK (2009) has developed a pathway of care and service specification, which, if implemented, can ensure that those with acute foot problems have rapid access to healthcare professionals with the necessary skills and experience to manage their condition. This care pathway should be a major step towards reducing foot ulceration and amputation among people with diabetes (Diabetes UK, 2009).

The starting point of treatment for diabetes-associated foot ulceration is a multidisciplinary approach to the management of metabolic control, debridement, offloading/pressure redistribution, vascular control, infection and seamless integrated wound care (White and McIntosh, 2008).

## Aetiology

According to Posnett and Franks (2007), the definition of a diabetic foot is: 'A full thickness wound below the ankle in a diabetic patient'. Irrespective of the duration, this can result in the development of peripheral neuropathy and the loss of sensation can leave the foot vulnerable even to minor trauma. Vulnerability to infection and peripheral vascular disease can inhibit healing after injury and may lead to gangrene and amputation.

An abnormal healing response in a patient with diabetic foot ulceration can have multiple causes — at a cellular level there is often increased blood glucose, thickening of the basement membrane in tissues and changes to cell walls that makes transmission of fluid

and gases less efficient, resulting in abnormal neutrophil function which increases the risk of infection; decreased red cell deformability, which causes decreased tissue oxygenation; and tissue ischaemia (Yue et al, 1987). It is also recognised that patients with diabetes have an increased risk of developing infection, because at cellular level the combination of increased blood glucose, thickening of basement membrane in tissues and changes to the cell wall make transmission of fluid and gases less efficient. All of these factors result in abnormal neutrophil function that increase the risk of infection, with reduced red blood cell deformability causing decreased tissue oxygenation and death of tissue (Yue et al, 1987). The combination of neuropathy, ischaemia and infection often coexists in this population of patients (Boulton, 1994).

## Standard treatment for diabetic foot

The cornerstones of treating full thickness ulcers are as follows (Sibbald et al, 2003; Queen et al, 2004):

- ▶▶ Debridement
- ▶▶ Offloading with orthotics, casting or non-weight bearing regimens
- ▶▶ Local wound care, comprising cleansing with saline and the use of modern wound dressings to promote a moist wound healing environment.

White and McIntosh (2008) state that consideration should be given to revascularisation where necessary and the control of serum glucose levels and arterial risk factors such as hypertension and dyslipidaemia.

The International Diabetes Federation (2006) advise implementation of the above care pathway by the multidisciplinary team, appropriate organisations (such as Diabetes UK), close monitoring and education of clinicians and patients in diabetes management.

### Management of bacterial bioburden

There are various methods to debride wounds and manage bioburden, namely:

- ▶ Autolytic: where wound care products are employed, e.g. hydrocolloids or hydrogels are used to donate water to the necrotic tissue. This is a slow method of debridement
- ▶ Chemical: where chemical agents are used to promote debridement, such as iodine, silver enzyme products that are capable of effectively removing necrotic tissue
- ▶ Mechanical sharp debridement: where skilled surgeons in theatre remove all the necrotic tissue until healthy tissue remains (O'Brien, 2003).

Larvae have been seen to reduce the time of debridement when compared with hydrogels (Dumville et al, 2009).

Infected wounds can have a high level of exudate, which requires careful control and frequent dressing changes to prevent maceration (Hilton et al, 2004). Falanga (2000) suggests that debridement is an important stage in the wound healing process, as it forms a key element of wound bed preparation and is an essential precursor to modern wound management. An inadequately prepared wound bed will not promote effective wound healing, wasting both time and resources.

Over the past decade, medicinal maggots have become an acceptable form of treatment for debridement of infected necrotic wounds (Thomas, 2006). Larval therapy is 'used to promote wound debridement' (Horobin et al, 2005) and it has been suggested that the larvae also stimulate wound healing, reduce the bacterial load (Van der Plas et al, 2008) and eradicate methicillin-resistant *Staphylococcus aureus* (MRSA) (Bexfield et al, 2004).

The case report below features the use of sterile bags filled with *Lucilia sericata* larvae and foam pieces (BioFOAM).

### Case report

Mrs J was a 50-year-old woman who was normally fit and well and ran her own successful business. She was admitted with an infected left foot following a fall in her bathroom. The primary wound was the ulcer on the plantar surface (Figure 1), just below the patient's left big toe. Another forefoot wound developed as a result of infection (Figure 2). Mrs J had not sought medical advice as the wounds did not trouble her until she started to feel unwell — she had a raised temperature of 39.5° and felt lethargic. The author was asked by the vascular surgeon to see Mrs J and advise on the appropriate care pathway.

The forefoot full-thickness wound measured 20x8cm with exposed moist tendons. There was 20% brown necrotic tissue and 80% soft yellow sloughy tissue in the wound bed.

The ulcer on the dorsum/planter surface of the foot measured 20x12cm and consisted of 100% yellow tissue. This primary wound was boggy and soft and pus was visible.

Mrs J was able to move her toes and all of her pulses were audible by Doppler: Her foot was warm to the touch. She could feel her foot and had 'sausage' digits, suggestive of osteomyelitis,

but which may also be the result of other foot problems, such as diabetic neuropathy.

Mrs J was admitted on 2 March 2009 and her blood glucose level was 28.5mmols. She was managed by the vascular surgeon until the author first saw Mrs J on 9 March 2009 — she was on antibiotics and her blood results revealed serum albumin of 23, haemoglobin (Hb) 11.1 and a white cell count of 15.9. Her Hb played an important part in the availability of oxygen for wound healing and the white cell count provided information as to how much infection was present in the patient's system.

With the help of the diabetic consultant, Mrs J's blood glucose levels were brought back within normal levels. Using the Lipsky Classification system (Lipsky et al, 2004), the author graded Mrs J's infected foot as a grade 3 ulcer (Table 1).

Mrs J realised the seriousness of her condition and knew that she could lose her foot and lower leg if the correct course of action was not taken.

### Care pathway

The author advised that Mrs J's forefoot be treated with Intrasisite® Gel Dressing (Smith & Nephew) and Biatain® Foam Dressing (Coloplast Ltd) every 48 hours — the dorsum was to be treated with Aquacel®

**Table 1**  
Grading the severity of diabetic foot infection

Grade 1 — no infection	No purulence or signs of infection
Grade 2 — mild infection	No systemic illness and evidence of either (a) pus (purulent secretions) or (b) two or more signs or symptoms of inflammation (e.g. erythema, warmth, pain, tenderness, induration). Any cellulitis <2cm around the wound. No evidence of systemic infection
Grade 3 — moderate infection	Either (a) lymphatic streaking, deep tissue infection (involving subcutaneous tissue, fascia, tendon, bone) or abscess, or (b) cellulitis <2cm. No evidence of systemic infection
Grade 4 — severe infection	Any infection accompanied by systemic toxicity (chills, fever, shock, vomiting, confusion, metabolic instability). The presence of critical ischaemia of the involved limb may make the infection severe



**Figure 3.** After six weeks' treatment with BioFOAM the wound bed was covered with granulation tissue.



**Figure 4.** After six weeks' treatment the plantar ulcer demonstrated significant growth of granulation tissue and wound contraction is visible.

(ConvaTec) and Biatain every 48 hours. This was the plan of care until 17 March 2009 when BioFOAM was chosen.

The author discussed the treatment plan with the vascular surgeon and he was happy for Mrs J to start larval therapy on 17 March. Mrs J was supplied with a patient information leaflet outlining larval therapy and consented to proceed.

BioFOAM was ordered in the following sizes and amounts: 10x10cm x 2; 2.5x15cm x 1; 7x7cm x 1.

The author applied the first treatment of BioFOAM to Mrs J's left forefoot on 17 March 2009. On removal of the previous dressing, the author cleansed the wounds with saline before applying the BioFOAM to the forefoot only. The tissue was 100% soft and yellow/brown in colour. The author applied Sudocrem (Forest Laboratories UK Ltd) around the forefoot ulcer to protect the skin from excoriation, as

recommended by the company. (Hydrocolloids are recommended for loose larvae as a method of protecting the skin.) Two bags of BioFOAM debridement larvae were then applied. Two gauze pads and a surgical pad were placed over the bags and secured with toe-to-knee bandaging. The author demonstrated how to care for the larvae (a daily reapplication of clean dry gauze and a surgical pad) until her return the following week.

At this time, the plantar surface had developed into an ulcer that measured 2.5x4cm and comprised 100% yellow tissue. The author redressed the wound with Aquacel and Biatain, which was to be renewed in 48 hours, and took a photograph to monitor the progress of the wound (Figure 1).

On the 20th March, both the wounds were treated with BioFOAM debridement larvae. Despite these measures, when assessing Mrs J on 26 March complications had developed. The forefoot wound had deteriorated and appeared to be 100% yellow tissue. Mrs J had a temperature of 39° and was started on intravenous (IV) antibiotics. The author cleaned the wound with normal saline and, despite the infection, decided to continue larval therapy. The vascular surgeon was informed of the deterioration and was likewise happy to continue with the larvae until his return from leave unless the wounds deteriorated further; in which case he suggested Mrs J be transferred to the local vascular unit for microsurgery.

The author repeated the application of the BioFOAM larvae every five days. Once the granulation tissue had covered the wound bed and the tendons were no longer exposed, the treatment was changed to the maintenance dressing on the forefoot. Mrs J's blood sugar levels were now between 5–7mmols and she was feeling better.

The author contacted the district nursing team who were happy to carry out the daily application of clean, dry gauze. The author agreed that she would see Mrs J as an outpatient twice a week for reapplication of the BioFOAM. Mrs J was told that rest and

elevation of her left foot were essential to a full recovery.

Following the six weeks of treatment of BioFOAM larval therapy there had been full coverage of granulation tissue over the exposed tendons. Figure 3 clearly demonstrates the increase in granulation tissue and decrease in necrotic tissue, compared with Figure 2.

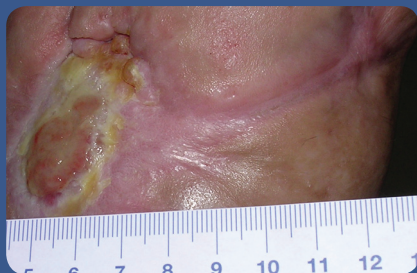
The plantar ulcer had reduced from 4.5x3cm to 9x8cm and the ulcer demonstrated a significant growth of granulation tissue (Figure 4). There was a small sinus measuring 1x1cm, which the author packed with Sorbsan® Ribbon (Unomedical). There was another sinus measuring 1x0.5cm which, although not open, was still palpable. This sinus exhibited 80% pink tissue and the surrounding tissue was beginning to settle down — the skin tone was returning to normal and was far less inflamed.

Having removed the BioFOAM debridement larvae therapy on the forefoot after good results, the author changed to a BioFOAM maintenance dressing, with a reduced number of larvae to promote wound healing and to prevent resloughing. However, the author persisted with debridement larvae on the plantar ulcer as it still had brown necrotic tissue present in the wound bed.

Mrs J's blood sugar measurements were still between 5–7mmols and she was planning to leave her daughter's and return home.

On 1 June 2009, the author removed the larval therapy from Mrs J's forefoot. Some of the maintenance larvae had drowned due to the volume of exudate. The wound had decreased to 4.5x7cm with 90% pink granulation tissue and 10% yellow tissue. There was also infill visible on the granulating tissue. The area on the forefoot nearest the toe measured 1x0.5cm and appeared to be healing as it needed less Sorbsan to pack. Mrs J's blood sugars now measured between 4.6–8.9mmols and she was planning to leave her daughter's and return home.

The ulcer on the plantar surface had also reduced, measuring 1x2cm and exhibiting



**Figure 5.** On removal of the dressing on top of Mrs J's forefoot, the author could see that the wound had decreased in size and now measured 3x2cm with evidence of granulation, contraction and epithelial tissue.



**Figure 6.** The plantar surface had nearly healed and had reduced in size to 0.5x3cm, displaying a small amount of brown tissue, possibly a view of the fatty pad.

pink granulation tissue. The small sinus measured 1x1 cm and had practically healed.

The author cleaned all the wounds with water and decided to change to conventional dressings, using Sorbsan Flat (Unomedical) for the forefoot and Sorbsan Ribbon on the toe and plantar ulcer. These were topped off with Biatain and secured with a bandage every 2–3 days. Mrs J was asked to return in four weeks time for an appointment with the author and the vascular surgeon.

On 14 September 2009 the author reviewed Mrs J at a routine visit — her blood sugars now measured between 7–9mmols and she was feeling considerably better after a holiday in Cyprus. However, she had gained one and a half stone while on holiday and was now trying to lose this weight as she was aware of the impact it could have on her foot. On removal of the dressing on top of Mrs J's forefoot, the author could see that the wound had decreased in size and

measured 3x2cm. There was also infill on the granulating tissue (Figure 5). The area on the forefoot nearest the toe appeared to have healed completely and the sensation and feeling in the bottom of Mrs J's foot and toes had returned.

The plantar surface had nearly healed and had reduced in size to 0.5x3cm, displaying a small amount of brown tissue and possibly a view of the fatty pad (Figure 6). The author cleaned all the wounds with water and advised Mrs J that she should bear as little weight on the foot as possible. Mrs J was also referred to the orthotic department to be assessed for specialised foot wear.

Mrs J was seen by the diabetic consultant who was happy with her diabetic management. The author advised the district nursing team to continue with Aquacel and Biatain on the forefoot, Aquacel Ribbon for the toe track and plantar, and Aquacel and Biatain secured with bandaging every 3–4 days between the toes. The author gave Mrs J an appointment for four weeks time.

## Conclusion

Effective debridement in the diabetic foot is challenging. In any multifaceted condition it is essential that a multidisciplinary approach is adopted and that each area is addressed. Treating the underlying issues and selecting a suitable care pathway is vital to recovery and, in this case, saved the patient's foot.

This case report provides insight into how a six-week period of debridement with larval therapy, followed by the application of BioFOAM maintenance dressing, can contribute to successful wound healing. **WUK**

## References

- Bexfield A, Nigam Y, Thomas S, Radcliffe NA (2004) Detection and partial characterisation of two antibacterial factors from excretions of medicinal maggots *Lucilia sericata* and their activity against methicillin-resistant *Staphylococcus aureus* (MRSA). *Microbes Inf* 6(14): 1297–1304
- Boulton AJM (1994) The pathway to ulceration: aetiopathogenesis. In: Boulton AMJ, Connor H,

Cavanagh PR, eds. *The Foot in Diabetes*. 2nd edn. John Wiley and Sons, Chichester: 27–48

Diabetes UK (2009) *Putting Feet First*. NHS Diabetes, London

Dumville JC, Worthy G, Bland JM, Cullum N, et al (2009) Larval therapy for leg ulcers Venus II: randomised control trial. *Br Med J* 338: 1047–53

Falanga V (2000) Classification for wound bed preparation and stimulation of chronic wounds. *Wound Rep Regen* 8(5): 347–52

International Diabetes Federation (2006) Costs of Diabetes. Available online at: [www.eatlas.idf.org/costs-of-diabetes](http://www.eatlas.idf.org/costs-of-diabetes) (accessed 29 October, 2009)

Hilton JR, Williams DT, Beuker B, et al (2004) Wound dressings in diabetic foot disease. *Clin Inf Dis* 39(2 suppl): S100–3

Horobin A J, Shakesheff KM, Pritchard DI (2005) Maggots and wound healing: an investigation of the effects of secretions from *Lucilia sericata* larvae upon the migration on humal dermal fibroblasts over a fibronectin-coated surface. *Wound Rep Regen* 13: 422–33

Lipsky BA, Berendt AR, Deery HG (2004) Infectious Diseases Society of America guidelines: diagnosis and treatment of diabetic foot infection. *Clin Infect Dis* 39: 885–910

NHS National Diabetes Support Team (2008) *Improving Emergency and Inpatient Care for People with Diabetes*. NHS Diabetes, London

O'Brien M (2003) Exploring methods of wound debridement. *Trends in Wound Care*. Vol II. Quay Books, MA Healthcare Ltd: chap 8, 95–107

Posnett J, Franks P (2007) *Skin Breakdown*. The Smith & Nephew Foundation, London

Queen D, Orsted H, Sanada H, Sussman GA (2004) A dressing history. *Int Wound J* 1(1): 59–77

Sibbald RG, Torrance G, Hux M, et al (2003) Cost effectiveness of becaplermin for non-healing neuropathic diabetic foot ulcers. *Ostomy Wound Management* 49(11): 76–84

Thomas S (2006) Cost of managing chronic wounds in the UK, with particular emphasis on maggot debridement therapy. *J Wound Care* 15(10): 465–9

Yue DK, McInnon S, Marsh ME, et al (1987) Effects of experimental diabetes, uraemia and malnutrition on wound healing. *Diabetes* 36(3): 295–9

White R, McIntosh C (2008) Topical therapies for diabetic foot ulcers: standard treatments. *J Wound Care* 17(10): 426–35

Van der Plas MJA, Jukema GN, Wai SW et al (2008) Maggot excretions/secretions are differentially effective against biofilms of *Staphylococcus aureus* and *Pseudomonas aeruginosa*. *J Antimicrob Chemother* 61: 117–22