Early detection and management of wound infection in the 'at risk' foot:

Why is this important and what options are available to us?

A report from a satellite symposium held on the occasion of the 7th Annual Conference and Exhibition of The Diabetic Foot journal. The meeting took place on 5–6 June 2006 at the Corn Exchange, Edinburgh, and was supported by ConvaTec.

'The complication that patients fear most is amputation, and infection is often the final pathway that leads to this tragic, and often preventable, event.'

'In most cases, diabetic foot ulcers and amputations can be prevented. It is estimated that up to 85% [International Diabetes Federation, 2005] of amputations could be avoided,' said Rachel Mathison (Medical Education Specialist, ConvaTec, Chief Podiatrist, Stockport, and Vice Chair of Foot in Diabetes UK [FDUK]) as she introduced this satellite symposium. The meeting aimed to discuss the merits of the early detection of infection in diabetic ulcers and how best to control and eradicate the infection.

Using clinical indicators to identify early stages of wound infection in the diabetic foot

'We all know that the early detection of infection in diabetic foot ulcers can prevent serious complications such as lower limb amputations and even death,' began Jo Stevens (Medical Education Manager, ConvaTec).

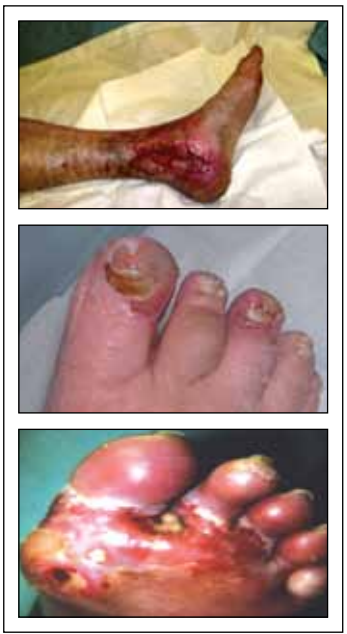


Figure 1. Definitions of the three main stages of wound infection. Top: 'colonisation', refers to a wound which contains multiplying bacteria but that are not causing a host reaction; middle: 'critical colonisation', a wound that is moving through the continuum may be displaying subtle signs of infection, therefore, tipping the host control in favour of the pathogen; bottom: 'infection', a wound in which the pathogenic bacteria are multiplying and causing a host reaction. (These photographs have been kindly supplied by Stockport Primary Care Trust.)

Early and accurate assessment of the diabetic foot for infection allows:

- a prompt diagnosis, which could therefore reduce patient morbidity
- clinicians to differentiate between chronic inflammation and infection
- prompt antimicrobial intervention and more appropriate use of antibiotics.

Jo went on to discuss some classic signs of wound infection – such as pain, malodour, delayed healing and fragile granulation tissue. She said that: 'Practitioners are known to experience difficulties differentiating between colonised and infected wounds. All chronic wounds contain large amounts of bacteria [...] But this should not be confused with a clinical diagnosis of wound infection.' *Figure 1* shows the three key stages of wound infection: from colonisation to critical colonisation and, finally, infection. It also illustrates a 'continuum' of wound infection, the objective of infection control being to create an imbalance in favour of the person (or 'host'), thereby reducing the pathogen load and helping in wound healing, as in a 'wound infection see-saw'.

Characteristics of a critically colonised wound include the following (Davis, 1998):

- an indolent wound that is unresponsive to therapies and demonstrates no cellulitis
- thick slough that returns after sharp debridement
- intransigent malodour.

A possible visible indicator of a critically colonised wound is the appearance of a biofilm. There is controversy as to whether films visible on diabetic foot ulcers are actually biofilms. Biofilms are made up of bacteria which 'exhibit a decreased sensitivity to host immunological defence mechanisms, and decreased susceptibility to antimicrobial agents,' (Costerton et al, 1999).

Conclusions

'Clinicians appear to use their own individualised sets of criteria to assess wound infection that are not based on any working consensus and are not evidence-based. In order to ensure that the balance within infected wounds is moved in favour of the person with the wound, evidence-based guidelines [such as the European Wound Management Association's Position Document, *Identifying criteria for wound infection* (2005)] must be used,' concluded Jo. 'The complication that patients fear most is amputation, and infection is often the final pathway that leads to this tragic, and often preventable, event.'

Antimicrobial dressings in the management of wound infection

In the second talk of the symposium Duncan Stang (Chief Podiatrist, Lanarkshire) considered the use of antimicrobial dressings in the treatment of diabetic foot ulcers. 'What are we [healthcare professionals involved in the care of the diabetic foot] all trying to prevent? Lower limb amputations,' said Duncan.

'Wound healing is a multifactorial event and the application of any antimicrobial dressing is of little value unless all of the factors that may delay wound healing have been assessed and addressed,' said Duncan. 'A holistic approach that makes full use of the whole multidisciplinary team and focuses on all factors of wound management, such as debridement, pressure relief, exudate control and dressing choice, is essential.'

Debridement

There are three types of debridement available to the podiatrist:

- physical
- chemical

- larval.

Duncan explained that the literature shows that centres with high rates of debridement achieve better healing rates than centres that do not debride (Steed et al, 1996).

Pressure relief

There are a number of products available to off-load diabetic foot ulcers. If possible, noted Duncan, the healthcare professional must ensure the individual being treated remains an outpatient and as ambulatory as possible.

Exudate control

The relationship between increased exudate and bacterial load is well documented (for example, Sibbald et al, 2000). A wound with well-managed exudate levels, by dressings that absorb and retain exudate, will provide a moist environment for optimum wound healing, commented Duncan.

Dressing choice

'Why not just blanket-prescribe antibiotics?' asked Duncan. He provided the answer himself: 'Because they can cause side effects, they are known to upset diabetes' control, they can cause microbial resistance, they can be expensive, there is little evidence to support prophylactic use, and the vast majority of diabetic foot ulcers have an ischaemic element.'

He said that 'as an alternative, topical antimicrobials can be used.' Examples include iodine- and silver-releasing products. Such products are increasingly being used because of bacteria becoming resistant to antibiotics.

Duncan went on to explain how silver-based dressings work: 'Silver is an inert metal and does not react with human tissue in its non-ionised, or 'pure', form. In the presence of moisture, such as wound exudate, silver readily ionises to produce silver ions (Ag^+) which, by binding to proteins on cell surfaces of bacteria and fungi, kill them. 'Studies (both on animals and humans) have shown that silver released from dressings promotes or "kick-starts" wound healing by promoting haemostasis, reducing inflammation and increasing epithelialisation and neovascularisation' (Kjolseth et al, 1994; Lansdown et al, 1997; Karlsmark, 2003).

Conclusions

'There are many challenges in managing diabetic foot ulcers. Choosing the appropriate dressing at the appropriate time is only one element. The correct use of a silver dressing in the management of diabetic foot ulcers that are progressing along the infection continuum from critical colonisation to wound infection meet all the requirements to control the wound bioburden and absorb and retain the wound exudate. This in turn creates an optimal environment to support wound healing.'

Clinical case studies

In the third talk of the symposium Graham Bowen (Chief Podiatrist, Southampton) presented three case studies on the use of silver-containing dressings. In all of the cases AQUACEL Ag (a silver-containing dressing; ConvaTec, Uxbridge) was used as the primary dressing from the outset or was introduced when other dressings failed to help in infection control. 'As the diabetic foot ulcer is a multifactorial wound, AQUACEL Ag did not heal the ulcers on its own but it certainly helped by controlling the symptoms at the wound bed,' said Graham. One of the case studies is detailed on the right. ■

Costerton JW, Stewart PS, Greenberg EP (1999) Bacterial biofilms: a common cause of persistent infections. *Science* **284**(5418): 1318–22

Davis E (1998) Education microbiology and chronic wounds. *Journal of Wound Care* **7**(6): 272–4

European Wound Management Association (2005) *Position document: Identifying criteria for wound infection.* Medical Education Partnership Ltd, London

International Diabetes Federation (IDF; 2005) *Diabetes and Foot Care: Time to Act.* IDF, Brussels

Karlsmark T, Agerslev RH, Bendz SH et al (2003) Clinical performance of a new silver dressing, Contreet Foam, for chronic exuding venous leg ulcers *Journal of Wound Care* **12**(9): 351–4

Kjolseth D, Frank JM, Barker JH et al (1994) Comparison of the effects of commonly used wound agents on epithelialization and neovascularization. *Journal of the American College of Surgeons* **179**(3): 305–12

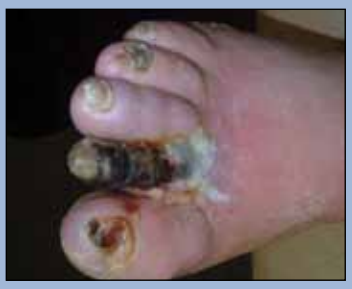
Lansdown AB, Sampson B, Laupattarakasem P, Vuttivirojana A (1997) Silver aids healing in the sterile skin wound: experimental studies in the laboratory rat. *British Journal of Dermatology* **137**(5): 728–35

Sibbald RG, Williamson D, Orsted HL et al (2000) Preparing the wound bed–debridement, bacterial balance, and moisture balance. *Ostomy/Wound Management* **46**(11): 14–28

Steed DL, Donohoe D, Webster MW, Lindsley L (1996) Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. Diabetic Ulcer Study Group. *Journal of the American College of Surgeons* **183**(1): 61–4

Patient details

Mr S is a 65-year-old male with type 2 diabetes diagnosed in 1994. His average HbA_{1c} was 7.5% in 2004, it rose to 12.0% in July 2005. After smoking for approximately 40 years he stopped in 2005, after being asked to do so as a prerequisite of his procedure. His oral medications included metformin and gliclazide (later replaced with glipizide). He presented to the podiatry team with shallow ischaemic ulcers on the apex of his right second and lateral fifth metatarsal head and plantar surface of his right heel in 2004. He was admitted twice in 2004 for infection in his right foot that needed intravenous antibiotic treatment. He underwent a pop fem bypass in 2004. Subsequent admission in January 2005 was required for the amputation of his right first, second and third toes. These treatments were undertaken over 16 months.



Time 0. *Foot is ischaemic and pulseless. The first, third and fourth toes have dry necrosis. The second toe is necrotic with some maceration at its base. Infection is apparent.*

58 Days. *Following bypass and amputation of Mr S's right first to third toes, the wound bed is very sloughy and exuding heavily. The fourth toe is dry and necrotic and awaiting auto–amputation. AQUACEL Ag was used as the dressing of choice from this moment onwards.*

6 months. *The amputation site is healing well. The fourth toe has auto–amputated; however, the proximal phalanx is still attached with some necrotic tissue. There is still a small amount of slough present at the amputation site. AQUACEL Ag is still being used.*

7 months. *Following debridement of the necrotic tissue and removal of the remaining proximal phalanx within clinic, the site is healing well with minimal exudate and no slough.*

9 months. *The amputation site has healed well. There is minimal callus formation over the scar line, which requires regular debridement.*