DEBRIDEMENT IN WOUND CARE

Debridement is the removal of dead tissue from the wound bed. This article examines the different techniques that exist, and how these can be applied in clinical practice.

What is debridement?

Debridement is the removal of dead tissue from the wound bed. There are a large number of debridement options open to the practitioner, the three main types being

- Active
- Autolytic (moisture donation)
- Autolytic (moisture absorption).

Active debridement

Surgical debridement

Surgical debridement is carried out by surgeons/podiatrists and specialist nurses, using surgical instruments such as scalpels and forceps in an operating theatre. Surgical debridement removes all non-viable and compromised tissue until a healthy bleeding wound bed is achieved. It prompts an inflammatory response from the wound, thus stimulating healing (Bale, 1997). Anaesthetic is normally required for this procedure.

Sharp debridement

Sharp debridement is the removal of dead tissue with sharp instruments such as scissors or a scalpel and it is the fastest method of wound debridement. This technique involves debulking the wound of slough and necrotic tissue. The objective is not to create a bleeding wound bed and some slough and necrosis is left. This process is usually carried out at the patient's bedside or in the patient's own home. The skilled clinician trims or pares non-viable tissue away from viable tissue at or above the plain of viability. As live tissue is left intact, additional analgesia is not normally required. Conservative debridement may be undertaken in conjunction with other therapies such as autolysis to enhance healing rates (O'Brien, 2003) and may need to be undertaken serially until the wound bed is clear of debris.

Larval therapy

The use of maggot larvae to debride the wound of dead tissue has become a mainstream therapy in the UK. The larvae liquefy the dead tissue and, where the treatment is successful, can result in rapid debridement (Thomas et al, 1998). In addition, to breaking down dead tissues the larvae are able to ingest and breakdown pathogenic organisms in the wound (Thomas et al, 1999). This can make their use in the infected or heavily colonised wound effective.

Chemical debridement

Chemical debriding agents such as hydrogen peroxide and sodium hypochlorite (EUSOL) have been available for many years. While many of these have been proven to have bactericidal effects, research has shown they can have toxic effects on healthy tissue and fibroblasts and the process can be painful. Use is therefore declining in regular wound care practice in the UK (Leaper, 2002).

Mechanical debridement

Necrotic tissue can be physically 'pulled' from the wound bed. Traditionally this has been achieved by the use of wet-to-dry dressings. This form of therapy is rarely practised in the UK, but more recent developments have led to the use of high-pressure irrigation and hydro-pool cleansing. Jets of warmed solution are used to loosen the bonds between the adherent necrotic material and the viable tissue. In many cases these systems do seem to be clinically effective (Palmier and Trial, 2004), but require expensive equipment and there are issues over equipment cleansing and cross-infection. The addition of ultrasound in pressure cleansing does improve results but also increases costs. This system therefore tends to be reserved for specialist wound care centres.

Autolytic debridement

Autolytic debridement is the process by which the body attempts to shed devitalised tissue by the use of moisture. Where tissue can be kept moist it will naturally degrade and deslough from the underlying healthy structures. This process is facilitated by the presence of enzymes (matrix metalloproteinases) which break down protein bonds and lead to the sloughing away of non-viable tissue, (Thomas et al, 1999). The process can be enhanced by the application of wound management products. These products can be divided into two

categories: those that donate moisture to the dead tissue, and those that absorb excess moisture produced by the body.

Autolytic (moisture donation)

Hydrocolloids, hydrogels, honey and silver sulphadiazine donate moisture to the wound and thus enhance the process of debridement (Cooper et al, 2003). Products such as hydrocolloids and semi-permeable films trap fluid in the wound bed and can lead to rehydration. Hydrogels can be particularly effective as they donate additional moisture, providing a wet wound bed. However, care must be taken to prevent surrounding tissue from becoming macerated, and the time taken for debridement to occur may be protracted (Leaper 2002).

Commercial honey preparations are an alternative to hydrogels. Honey-based ointments (using predominantly manuka honey) are applied to the necrotic tissue and covered with a semi-occlusive dressing. This rehydrates the eschar and draws fluid through the eschar by osmotic action. It is claimed that this process not only enhances the autolytic process, but also provides antimicrobial action against the overgrowth of pathogenic organisms. The use of antimicrobial products should always be based on clinical need and not used as a matter of routine.

Autolytic (moisture absorption)

Alginates, cadexomer iodine and Hydrofiber® facilitate autolytic debridement by absorbing moisture (exudate) from the wound while ensuring that the necrotic tissue does not dry out (Cooper et al, 2003). By absorbing excess exudate, these products avoid damage to the surrounding skin from maceration. As with the moisture-donating products, some of the products within the moisture-absorption group also have an antimicrobial effect.

Enzymatic debridement

Natural autolysis depends on the presence of both moisture and the appropriate enzymes to break down the firm proteinous bonds between the eschar and the wound bed. In the absence of the latter, synthetic enzymes can be introduced to enhance non-viable tissue breakdown. These preparations are applied topically either as a solution or dissolved in a hydrogel and covered with an occlusive dressing. Their effectiveness over simple rehydration with a hydrogel has been questioned, (O'Brien, 2003) and there are concerns that exposure to streptokinase can lead to the development of antibodies, (Vowden and Vowden, 1999) which could subsequently lead to immune reactions should it be required following a myocardial infarction.

When should the clinician leave necrotic material *in situ*?

Generally, the presence of necrotic tissue is seen as a delaying factor in wound healing. However there are exceptions to this. In the absence of adequate vascular supply, tissue regeneration can be inhibited or indeed be absent. Removal of necrotic tissue here will expose underlying structures to the effects of desiccation and bacterial ingress. This can lead to further tissue death and wound extension. In certain circumstances then, the clinician should leave necrotic tissue *in situ* and aim to enhance dehydration. This reduces the possibility of bacterial growth and can lead to successful auto-amputation of the area. Dry or antimicrobial dressings can be helpful in this process. WE

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