Infection prevention and treatment in patients with major burn injuries


Summary
Infection is a significant challenge in burn care, particularly for those patients who have major burn injuries. This article aims to review the literature and establish best practice in prevention and treatment of infection in patients with major burns. The article considers the causes and clinical features of wound infection, and examines systemic and local methods of prevention and treatment.

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EACH YEAR, 0.5% of the UK population sustain a burn injury, and 10% of these are severely burned (British Burn Association 2004). Sepsis secondary to wound infection is well documented in burn care and associated with poorer outcomes – sepsis accounts for 50-60% of deaths following burn injury (Gallagher et al 2007). Although literature is available to guide wound care of minor burns, there are few recommendations regarding wound management in patients with major burns. Many modern dressings have been developed, several with antimicrobial properties. This article aims to review the available literature and ascertain best practice regarding wound care and the use of topical antimicrobials in patients with burn injuries (Figures 1 and 2).

Burn wound sepsis
Major burns in adults are defined as burns that affect more than 15% of the total body surface area (Herndon 2003). Patients with major burns are at significant risk of infection...
Contamination: The presence of microorganisms in a wound without multiplying or causing a host response.

Colonisation: Microorganisms are multiplying, but host reaction and symptoms of infection are not present.

Critical colonisation: The presence of microorganisms has stopped healing taking place, but they have not yet invaded tissue and signs of infection are still not present.

Infection: Microorganisms have invaded tissue and elicited a host response.

(Adapted from Kingsley 2001)

Bacterial presence in a wound can be a burden to the host, as microorganisms compete for oxygen and nutrients (Stotts 2007). This effect, known as bioburden, is not only the result of the number of bacteria present, but also various factors, including the organisms’ virulence, diversity and interactions with each other (Myers 2008). This is a relatively new concept, as until recently the risk of infection was thought only to positively correlate with the number of bacteria present (Timmons and Bell 2000).

Organisms can be differentiated by their origin; endogenous organisms are those that are already present either on the patient’s skin or normal gut flora, and exogenous organisms are those that are transmitted from the environment or staff to the patient (Timmons and Bell 2000). Limiting microorganism activity is now thought to be enough to prevent the development of sepsis (Gallagher et al 2007). Stotts (2007) suggested that total eradication of microorganisms from the wound is both impossible and unnecessary; best practice is instead concerned with reducing and controlling bacterial numbers at low levels to reduce the severity of septic episodes.

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involving its stage of colonisation, or the presence of inflammation or infection, so as to treat it accordingly (Scanlon 2005).

In addition to this challenge, Percival and Bowler (2004) identified an adverse effect on wound healing caused by the interaction of different microorganisms. Communities of bacteria can combine and attach to the wound surface encased in an extracellular polysaccharide matrix (Myers 2008). This allows bacteria to survive in a hostile wound environment and shields them from certain antimicrobials (Stotts 2007). This is known as biofilms (Percival and Bowler 2004). Biofilms increase resistance and virulence of bacteria, reduce effectiveness of leucocytes and are multimicrobial, which makes them unpredictable (Fletcher 2005). Despite not containing enough bacteria to be considered a true infection, biofilms delay wound healing and contribute to microbial resistance and therefore need prompt treatment (Fletcher 2005).

**Treatment of wound infection**

A patient’s risk of wound infection is determined by numerous factors. Kingsley (2001) suggested eliminating contributing sources of infection where possible. Dealey (2005) listed several factors, including advanced age, obesity, poor nutritional status, diabetes, some medication (corticosteroids, irradiation and some immunosuppressive drugs), length of hospital stay, smoking and pain that increase an individual’s likelihood of contracting a wound infection. Wolf (2007) also highlighted several factors that increase risks of infection specifically in burn care, including stress-induced hyperglycaemia, low cortisol levels, anaemia and low levels of certain trace elements, for example selenium, zinc and vitamin C. Once these factors have been addressed and minimised, Stotts (2007) proposed various methods of wound infection treatment, and divides them into local and systemic treatments. Systemic treatments consist of systemic antibiotics, oxygenation and nutrition. Local treatment consists of cleansing, debridement and topical therapy.

**Systemic treatment of wound infection**

**Systemic antibiotics** Systemic antibiotics are used regularly in wound care to manage infections, but resistance is a concern (Lee 2005). Stotts (2007) suggested using them only when cellulitis, leucocytosis or fever are present. However, these can be difficult to identify in patients with a major burn wound because of inflammation and the hypermetabolic response of the patient following a burn injury (Norbury and Herndon 2007). Timmons and Bell (2000) suggested that prophylactic antibiotics should not be used, and that antibiotics should only be commenced in the presence of positive wound swab cultures, combined with clinical signs of infection. Gallagher et al (2007) discussed the difficulty in identifying infection in burn patients and the rapid onset of infection. They also stated that broad spectrum systemic antibiotics should be commenced (with microbiology advice) when a patient’s condition deteriorates in any way without an obvious cause or when there is a change in the odour, appearance or level of wound exudate. Once a microorganism has been identified, antibiotics should be adjusted accordingly.

**Oxygenation** Some studies have indicated that wounds heal faster and have a lower incidence of infection when the patient receives supplemental oxygen (Grief et al 2000, Stotts 2007). However, the results of this work remain controversial and more research is needed to fully establish a link (Stotts 2007). Most patients with major burns receive supplemental oxygen during the acute phase of treatment, particularly if they require ventilatory support.

**Nutrition** Several authors have noted that poor nutrition may contribute to the development of wound infection (Wissing et al 2001, Mathus-Vliegen 2004), and this relationship has also been identified in patients with burns (Andel et al 2003). Many patients are already malnourished on admission as a result of pre-existing poor health status or lifestyle, which is a predisposing factor to having a burn injury (British Burn Association 2004), and patients with major wounds will require supplemental feeding (Dealey 2005). Liaison with a dietitian is essential to ensure adequate protein intake, as well as vitamins and trace elements required for wound healing (Saffle and Graves 2007). Patients with major burns should be assessed by a dietitian specialising in burns, weighed weekly and receive high protein feed supplemented with glutamine (Zhou et al 2003, Prelack et al 2007). Supplementation of albumin is controversial in the treatment of burn injuries. In some burn centres, patients receive partial or full resuscitation with human albumin, despite several studies stating that it has no benefit in burn care (Greenhalgh et al 1995, Alderson et al 1998). Reduced serum albumin levels are, however, associated with poor wound healing (Dealey 2005). Overall weight loss during admission can be significant – 10% of the patient’s admission bodyweight is the average weight loss following a major burn injury (Saffle and Graves 2007).
Local treatment of wound infection

Wound cleansing: This is a contentious issue in nursing practice (Blunt 2001, Myers 2008). It is now widely accepted that wound cleansing is not necessary if the wound appears clean and contains minimal exudate (Blunt 2001). Therefore, wound cleansing should be considered a method of debridement and not a separate intervention (Myers 2008). Exudate is known to contain growth factors and cytokines beneficial for healing. Removal can delay wound healing by drying the wound surface (Blunt 2001, Bale 2006). However, excessive exudate can macerate or excoriate the wound borders and should be removed (Thomas 1997). Any debris in the wound allows bacteria to adhere to it. Therefore, debris and loose skin should be cleaned; irrigation is the recommended method (Rolstad and Ovington 2007).

Scrubbing a wound to remove debris is no longer recommended as it damages healthy granulating cells and is thought to redistribute bacteria (Blunt 2001). Scrubbing and irrigation are methods of mechanical debridement. Many authors state that scrubbing has no place in wound care unless carried out to appropriate wounds with extreme care (Blunt 2001, Myers 2008). Irrigation should be carried out carefully to ensure that the pressure used is within the optimal range (Myers 2008). Scrubbing the wounds of a patient after a major burn would be extremely painful and may increase the need for analgesia and sedation, which may affect other aspects of recovery (Latarjet 2002). In addition, even relatively mature skin grafts could be damaged by scrubbing and therefore it is probably unsuitable in most cases (Settle 1996).

Other methods of wound cleansing suggested by Blunt (2001) include bathing and showering. Studies have shown no increased risk of infection when using tap water to clean wounds (Angeräs et al 1992, Briggs 1997), and bathing or showering have been advocated in burn care for some time (Lawrence 1987, Gilchrist 1994, Oliver 1997).

Blunt (2001) acknowledged the potential for cross infection when patients share bathing facilities, and the potential of water as a vector for *Pseudomonas aeruginosa* cross infection. Flanagan (1997) stated that bathing is not appropriate for patients with larger wounds, as water can be absorbed into the wound and interstitial spaces. This can increase exudate and damage the ideal wound healing environment. Gordon and Marvin (2007) advocated showering as a suitable method of gentle mechanical debridement for patients with major burns. They suggest that bathing may cause autocontamination and electrolyte imbalances. This may indicate that showering is a preferable option in major burn care, as it removes any debris, without soaking or damaging the wound environment. Several burns intensive care units now have showering facilities for use even with ventilated patients, and outcomes in the United States suggest this contributes to reducing the overall rates of burn-related wound sepsis (Greenhalgh 2007).

Debridement: This is another element of local wound infection treatment (Stotts 2007). The cleansing methods of scrubbing and irrigation can be considered mechanical debridement (Myers 2008). Other debridement methods include sharp debridement, biologic debridement (larval therapy), autolytic debridement (promotion of moist wound healing to encourage endogenous enzymes to remove devitalised tissue), enzymatic debridement (using topical exogenous enzymes to remove devitalised tissue) and surgical debridement (Ramundo 2007). The full-thickness areas of major burns are ideally surgically debrided (excised) within 48 hours of admission (Ramundo and Tompkins 2007). Sharp debridement is only indicated for wounds with significant eschar (Ramundo 2007), and initial surgical excision can be considered an equivalent treatment. Autolytic debridement relies on the use of appropriate dressings to create a moist wound healing environment, and this should be considered during dressing selection (discussed later). Biologic and enzymatic debridement are methods often used in burns centres, and these methods are gaining in popularity in modern wound care (Ramundo 2007). Myers (2008) suggested that these are more appropriate strategies for chronic, non-healing wounds, which would not apply to a major burn patient in the initial phase of treatment, and further research needs to be done to ascertain their role in acute burn care.

Topical antimicrobials: The third local element of wound infection treatment is topical antimicrobial therapy. The expression antimicrobial is given to a substance that destroys unicellular microorganisms, and may be one of several types (Box 2). Much controversy surrounds the use of antimicrobials (Timmons and Bell 2000). Antiseptic solutions for wound cleansing are generally not recommended (Timmons and Bell 2000, Stotts 2007, Myers 2008) as they are associated with cellular toxicity (Stotts 2007), increased inflammatory response with delayed epithelialisation (Myers 2008), and damage to healthy tissue (Timmons and Bell 2000). However, several authors advocate their use on specific wounds that have a significant likelihood...
of infection or wounds that appear to be critically colonised (Blunt 2001, Collier 2003, Dealey 2005, Percival and Cutting 2009). Dealey (2005) suggested that topical antiseptic washes are of little use when wiped across the surface momentarily, and advocates the use of soaks or impregnated dressings that are applied for more than 20 minutes to ensure optimal effect. Chlorhexidine is thought to have relatively low toxicity to living cells, but becomes inefficient when in contact with wound exudates; also large concentrations are needed to affect certain Gram-negative bacteria (Dealey 2005).

Topical antibiotics are even more controversial in modern wound care literature as their widespread prophylactic use has been associated with resistance problems (Gallagher et al 2007, Greenhalgh 2007, Stotts 2007). Percival and Cutting (2009) suggested that they should only be used when specific bacteria are isolated in a wound, and even then they should be used with caution because of the multimicrobial nature of many wounds (Collier 2003).

Topical elemental antimicrobials, such as silver and iodine, have been used for many years in burn care (Bailie and Wilson 2003, Evans 2007, Gallagher et al 2007). However, they have only recently become part of general wound management (Dowsett 2004, Leak and Johnson 2007). Dowsett (2004) described silver as a noble metal, which has a broad antimicrobial action against a range of organisms; has some degree of anti-inflammatory action; and comes in a range of preparations, such as creams, impregnated dressings and ointments. Dowsett (2004) stated that no resistant organisms have been found.

Gallagher et al (2007) described silver sulfadiazine 1% cream (such as Flamazine) has been proven to reduce inflammatory cell migration, vascular margination and bacterial density. Fumal et al (2002) and

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**BOX 2**

**Classification of topical antimicrobials**

**Antiseptic:** Solutions that destroy or inhibit microorganisms on external body surfaces, such as chlorhexidine.

**Antibiotic:** Creams or ointments that target specific bacteria, such as mupirocin.

**Elemental:** Substances that inhibit microorganisms that are derived from naturally occurring elements, such as silver or iodine.

(Adapted from Stotts 2007)

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Gallagher et al (2007) mention its ease of use and pain reducing ability, as well as being effective for up to 24 hours and penetrating the epidermal layer. It is also active in the presence of exudate, blood or pus and achieves a moist wound healing environment (Pankhurst and Pochkhanawala 2003). Silver sulfadiazine 1% is associated with neutropenia in patients with large wounds (Settle 1996), but this is rare, and resolves spontaneously even if treatment is continued (Gallagher et al 2007). There are several other preparations using silver as a contact layer in ionic or nanocrystalline form that allow slow release of silver and ensure antimicrobial action over several days, for example Acticoat (Myers 2008). There are also varieties of hydrofibre dressings containing ionic silver, such as Aquacel Ag (Dealey 2005). These dressings are appropriate for burns that have not yet been debrided and are suitable for skin grafted areas (Figure 4), providing the contact layer is non-adhesive. The principles of dressing selection remain the same for undebrided and grafted areas (Dealey 2005), and antimicrobial dressings have been shown to accelerate skin graft healing (Livingston et al 1990).

Silver dressings have been used in burn care for many years. Thomas and McCubbin (2003) identified several factors that influence the effectiveness of a silver dressing. These include the distribution of the silver through the dressing, the physical and chemical form of the silver, and the ability of the dressing to absorb moisture (several dressings require moisture to release their antimicrobial action). Leak and Johnson (2007) suggested that the donation of the silver to the wound bed is also critical to the performance of the dressing. Their research has shown that dressings that contain silver on the surface, rather than woven through the dressing, perform better and that ionic silver is more effective than metallic forms. Dowsett (2004) suggested that ionic silver in nanocrystalline sheets (for example, Acticoat) is more effective. This preparation gives a rapid
influx of silver into the wound, but also maintains a steady release of silver thereafter. In addition, nanocrystalline sheets maintain a moist wound environment, which ensures the best conditions for healing. These dressings are gaining in popularity in burn care and are available in large sizes. Alternatively, 1% silver sulfadiazine cream is the standard treatment in many UK burn centres, in conjunction with a non-adherent contact layer (such as Telfa Clear (permeable polyethylene membrane), Mepitel (silicone membrane), or Jelonet (paraffin gauze)), used with absorbent padding (Pankhurst and Pochkhanawala 2003). Wright et al (1998) found that 1% silver sulfadiazine cream was less effective than a silver-coated dressing. However, it is still recommended by Evans (2007) in conjunction with a non-adherent contact layer, as the limited sizes of other silver dressings – even though some are now available in larger sizes – make this combination more adaptable and conformable for large wounds.

Another widely used elemental antimicrobial is iodine (Stotts 2007), which, in its traditional povidone-iodine form was found to be cytotoxic (Dealey 2005). However, cadexomer iodine products have recently been developed and allow slower release of iodine and have a broad antimicrobial effect (Myers 2008). However, iodine is indicated for partial thickness wounds only as it is unable to penetrate eschar sufficiently to have an antimicrobial effect on the wound bed (Scanlon 2003).

Most authors do not recommend general use of topical antimicrobials, instead advocating their use only when a wound is critically colonised or infected (Collier 2003, Dealey 2005, Stotts 2007, Myers 2008). However, Dowsett (2004) stated that an exception to this is burn injuries, which should always be treated with topical antimicrobials because of the higher incidence of infection associated with these wounds. This is supported in literature surrounding burn care. Topical antimicrobials are recommended after the first 48 hours once the depth of the burn has been established (Pankhurst and Pochkhanawala 2003, McCahill 2006, Evans 2007, Gallagher et al 2007).

References


Greenhalgh (2007). Topical antimicrobial dressings should be the primary prophylactic treatment of infection, and they also help to prevent sepsis (Gallagher et al. 2007).

Psychosocial factors

The psychosocial effect of burn injury is well documented (Blunt 2001, Gordon and Marvin 2007). Many patients sustain extensive scarring to the body. This may lead to poor reintegration into society and sometimes psychological problems, such as depression and post-traumatic stress disorder (Noronha and Faust 2007). Sepsis in burn patients is known to contribute to wound breakdown, which causes more severe scarring (Sheridan and Tompkins 2007). Any interventions that can prevent sepsis or reduce scarring should be undertaken to ensure the best possible outcome for the patient (Gallagher et al. 2007).

Conclusion

After an examination of the literature, it is clear that there are many implications for practice. Many of the dressings available have not been specifically trialled on patients with burn injuries, and research is still required to develop practice further. Many issues need to be dealt with, for example showering major burns patients, and introducing alternative dressings when appropriate. With recent advances in burn care, patients are surviving more severe injuries, and any intervention that can reduce complications and improve outcomes should be trialled and implemented.

The overall aim in burn care is timely excision of eschar and coverage of the wound, but effective wound management throughout the patient’s stay has a beneficial effect on overall outcome and subsequent scarring. Patients with burn injuries are at high risk of developing infection, and contributing factors should be minimised or eliminated where possible NS.

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