UNDERSTANDING WOUND INFECTION

The costs associated with wound infection

The management of wound infection

Identifying risk factors

Suprasorb X+PHMB: a unique HydroBalance anti-microbial dressing

The diagnosis of wound infection in clinical practice

ACTIVA Healthcare LRR Company
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UNDERSTANDING WOUND INFECTION

John Timmons, Tissue Viability Nurse Specialist, Grampian Health Services, Aberdeen & Clinical Manager, Wounds UK

Wound infection is the result of an imbalance of the complex interactions between invading bacteria, the wound and the immunity of the host. In all wounds, chronic or acute, infection has the ability to result in distressing symptoms such as excessive pain, exudate and malodour, which negatively impact upon the quality of life of patients. In addition, wound infection may impact on the health service in terms of increased treatment costs and prolonged length of hospital stay.

The increase in the numbers of elderly patients with wounds and co-morbidities brings more individuals at risk of infection into contact with the healthcare system. Identifying patients at risk of wound infection may help in applying preventative measures and enable the monitoring of individuals, with a view to prophylactic treatment. Wounds which have enough bacteria to impact on healing, may pass through the phases of critical colonisation, local infection and possibly to spreading infection, if not managed appropriately, so the need for vigilance in this patient group is vital.

The early diagnosis of wound infection is essential, and should be underpinned by a systematic approach to the assessment of the patient and their wound, and diagnostic tests if appropriate. There are a number of management strategies which can be employed to deal with wound infection, and an individual treatment plan should be tailored in light of assessment findings. Topical antimicrobial agents, systemic antibiotics and good local wound care such as debridement, are central to the management of this patient group.

When selecting an antimicrobial dressing, it is important to select one that will provide optimum conditions to support rapid healing. The ability of the agent to reduce or eradicate micro-organisms, must also be considered, along with its specificity, cytotoxicity to human cells, its potential to select resistant strains and its allergenicity (Vowden and Cooper, 2006). The ability of the carrier dressing to handle exudate and remove necrotic tissue from the wound is beneficial, since purulent exudate, necrotic tissue and slough are all growth mediums for bacteria (Cutting, 2008). The dressing’s ability to reduce malodour, conform to the site and shape of the wound, perform wound bed preparation functions, satisfy patients’ expectations and to meet treatment goals also need careful consideration (Vowden and Cooper, 2006).

In recent years, the most commonly used topical agents have been silver, honey and iodine-based dressings. The introduction of dressings that contain the antiseptic polyhexamethylene biguanide (PHMB) is relatively new to wound care in the UK, although this antiseptic agent has been used as a successful antimicrobial in other industries for many years with little cytotoxicity and no known cases of bacterial resistance.

Suprasorb X +PHMB (Activa Healthcare, Burton-on-Trent) is a biosynthetic cellulose matrix dressing composed of HydroBalance fibres which give it the ability to provide or absorb moisture from the wound as required. This results in a moist wound environment to promote healing, and also facilitates autolytic debridement to help reduce the focus for infection. The dressing delivers PHMB, its anti-microbial component, direct to the wound bed, killing bacteria and helping to restore the wound bioburden.

All of these elements of care are discussed in this document, so that the management of patients at risk of, or with wound infection can be optimised.

THE COSTS ASSOCIATED WITH WOUND INFECTION

Shila Patel

Infected wounds are associated with unpleasant local symptoms such as an increase in pain, exudate volume and malodour, all of which impact negatively upon the patient and may result in social isolation and depression. In vulnerable patients, wound infection can lead to complications and prolonged stays in hospital. All of these factors can affect an individual’s ability to return to work or to care for their family, resulting in financial and social difficulties, and a reduction in quality of life. Infected wounds also present a considerable financial burden to the NHS in terms of the specialist medical or surgical intervention required and the prolonged stays and nursing time needed to care for patients with wound infection. With prompt intervention, however, some of these costs can be avoided.

**Key words**

Wound infection
Quality of life
Costs to NHS
Costs to society

Wound infection continues to be a challenging problem for clinicians and presents a considerable healthcare burden. The impact on the patient, the NHS and society is considerable and highlights the need for the prompt diagnosis and management of wound infection, so that many of the costs can be avoided. This article will look at the published evidence which considers the extent of the costs associated with wound infection, including both the cost to the individual patient as well as the NHS.

**Implications of wound infection for the patient**

A number of studies have been undertaken which consider the impact of surgical site wound infection on patients. Coello et al. (2005) investigated the impact of surgical site wound infection on mortality. They studied 140 English hospitals participating in the national Surgical Site Infection Surveillance Scheme between October 1997 and June 2001. During this time period, 67410 patients were included in the surveillance scheme following nine different types of surgical procedures, such as limb amputation, abdominal hysterectomy and knee prosthesis. Of the patients, 2832 were found to have a surgical site infection, with superficial infection at the incision site occurring most frequently.

Patients who developed a postoperative wound infection had an increased length of hospital stay and the crude mortality rate was higher for all categories of patients. However, after adjusting for confounding factors only patients with a superficial surgical site infection following hip prosthesis surgery, along with patients with a deep incisional and organ/space surgical site infection following vascular surgery, hip prosthesis and large bowel surgery had a significantly higher adjusted mortality rate. Similarly, Partanen et al. (2006) evaluated the impact of deep wound infection following hip fracture surgery, using both functional mobility and mortality as the study outcome indicators. They followed up 22276 patients over 50 years of age and with non-pathological fractures. Twenty-nine patients were found to have a deep wound infection. Control patients matched for variables such as age, sex, fracture type, treatment method and mobility, but without wound infection were used for comparison. Four months following the initial surgery the patients with deep wound infection had poorer mobility and a greater dependency on walking aids in comparison to the control patients. They also stayed in hospital significantly longer than the control group. One year after the surgery the overall mortality of the patients with deep wound infection was also significantly higher than the control group of patients (34.5% and 24.1%, respectively). Interestingly, the study also found that diabetes was a common risk factor in the patients with deep wound infection.

Delayed hospital discharge as a result of wound infection may in turn delay a return to paid employment and potentially cause financial difficulties, particularly if the patient is the primary wage earner. This may have a knock-on effect on the patient’s self-esteem and overall psychological well-being.
In patients with chronic wounds, recurrent infections may cause severe anxiety and depression, since patients may experience unpleasant symptoms and notice a deterioration in their wound which can act as a visible reminder of their illness and of their vulnerability (Moffatt et al. 2008).

The leakage of exudate and strike-through onto bandages is a source of distress to patients with leg ulcers (Douglas, 2001; Hopkins, 2004) and this can be made worse by the presence of infection. Leaving familiar surroundings and socialising may result in anxiety due to concerns about exudate and how it can be disguised, potentially leading to social isolation (Lindsay, 1999). Unpleasant odours arising from infected wounds can also be distressing to the patient and those nearby (Day and Hayes, 2008).

Edmonds (2006) stated that health-related quality of life for patients with diabetic foot ulceration may be poorer than that of patients who have undergone amputation since they live in fear of recurrent ulcers, repeated bouts of infection and potential life-long disability.

Furthermore, media attention on problems such as hygiene in hospitals and methicillin-resistant Staphylococcus aureus has made some patients anxious about developing resistant or hospital-acquired infections which prevent them from seeking hospital-based care (Moffatt et al., 2008).

While it may be argued that these findings are not applicable to all types of wound infection, they highlight the detrimental effect that wound infection can have on the quality of life and wellbeing of patients.

**Pain**

Wound infection can produce a significant increase in pain (Hofman, 2006). This is because it can give rise to chronic inflammation around a wound site, which in turn can directly excite sensory receptors, such as nociceptors and pain may also arise through sensitisation of indirect pain mediators (Clay and Chen, 2005). For this reason pain is often regarded as one of the most reliable indicators of wound infection.

Dressing choice can also have an impact on pain experienced by the patient. According to Hofman (2004) the use of antimicrobial dressings such as those impregnated with iodine and honey may further increase the patient's pain on application. Dressing removal also has the potential to cause pain. These factors have the potential to make the patient fear wound dressing changes and may further increase the psychological effect of pain on the individual. Conversely it has also been suggested that dressings which contain antimicrobial agents may achieve a reduction in pain in certain types of wound, for example chronic leg ulcers (Yanschel and others, 2003) and burn wounds (Vloemans and others, 2003).

Therefore it is important to consider using dressings that promote moist wound healing and areatraumatic on removal (Moffatt et al., 2008). The suitability of the dressing selected should be reviewed at each dressing change since products should be changed in response to the wound's changing needs.

Unresolved pain negatively impacts upon wound healing and the patient's quality of life and its effects should not be underestimated. Appropriate pain assessment and management is critical to ensuring holistic care and should not be overlooked.

**Cost to the NHS and Society**

Most of the costs of wound care are driven by the small proportion of patients with non-healing wounds whose wound becomes infected and who have specialist medical or surgical intervention in hospital (Posnett and Franks, 2007). These costs can be reduced by ensuring that primary care doctors, community nurses and hospital staff are properly trained in wound infection diagnosis and treatment.

Coello et al. (2005) considered the increased length of hospital stay and the cost of surgical site wound infection in patients undergoing a variety of surgical procedures. They found that surgical site wound infection was associated with increased postoperative length of hospital stay ranging from 3.3 days for abdominal hysterectomy to 2.1 days for limb amputation. The associated additional cost to the NHS ranged from £959 for abdominal hysterectomy to £6,103 for limb amputation, respectively.

These additional costs can further impact upon service provision within the NHS. It may be argued that patients who have delayed discharge due to wound infection can divert resources and create further delays for new patients who need to access healthcare services. This adds to the importance of preventing and managing wound infection correctly.

The total annual cost of diabetes-related foot complications is £252m (Gordois et al., 2003). During their lifetime 1 in 7 patients with diabetes will develop a foot ulcer which is highly susceptible to infection (Reiber, 2001). Such infection can lead to deterioration of the wound with alarming rapidity, resulting in overwhelming tissue destruction and amputation. Indeed, 85% of amputations are preceded by a foot ulcer (Pecoraro et al., 1993).

The Eurodial study followed 1,229 patients with diabetes and new foot ulceration. Of the ulcers, 53% were found to be infected and 83% of all patients admitted to hospital had an infection. Osteomyelitis was diagnosed
in 19% and a life- or limb-threatening infection in 12% of all patients with peripheral arterial disease, the ulcers were more frequently infected compared with non-PAD patients (64% vs 48%, p<0.001). Most deep (77%) and large (>5cm, 75%) ulcers were infected (Promperes et al, 2008). These findings further confirm that infection is a major problem in the diabetic foot, and it is the main cause of tissue destruction which could potentially be avoided by prompt diagnosis and treatment.

IMPACT OF WOUND INFECTION ON THE PRACTITIONER-PATIENT RELATIONSHIP

Gardner and Cook (2004) investigated how patients are informed about having a surgical site wound infection and highlighted the difficulties often experienced by patients in gaining relevant information, along with the way in which healthcare professionals avoid communicating this information to patients. Staff reluctance to discuss the development of a surgical site wound infection with patients may be linked to feelings of guilt and responsibility.

While it is difficult to say whether these findings are applicable to all wound types, it may be that both patients and staff do experience similar difficulties when faced with other types of wound infection, such as chronic wound infection, particularly if the causative micro-organism is MRSA. Some patients already have a poor perception of MRSA because of the publicity which surrounds it (Hamour et al, 2003). Therefore, where patients are found to have a chronic wound infection due to MRSA, staff may feel reluctant to discuss the full details of the wound's status with the patient. Staff may fear they will be blamed for the MRSA acquisition and infection. Practitioners who do not listen or attempt to understand the feelings of patients with leg ulcers, have been found to negatively affect quality of life, and this can be further compounded when conflicting advice is given by different clinicians (Charles, 2008).

In order to overcome some of these difficulties, adequate and appropriate communication with the patient is vital.

Good communication can help the patient to develop coping mechanisms and a positive, therapeutic relationship with their healthcare professional (Hopkins, 2004). This aspect of care should be not overlooked when managing a patient with wound infection.

Wound infection results in an increased financial burden for the NHS and society, due to associated increased length of stay in hospital, and additional treatment costs.

CONCLUSION

Wound infection results in an increased financial burden for the NHS and society due to associated increased length of hospital stay and additional treatment costs. Therefore, the prevention of wound infection whenever possible is extremely important and where it cannot be prevented, prompt diagnosis and treatment are of the utmost importance. The provision of pain relief, the use of appropriate dressings, effective communication and patient education can all help to prevent wound infection from impacting negatively upon the patient's quality of life and psychological well-being.

REFERENCES


Prediction of outcome in individuals with diabetic foot ulcers: focus on the differences between individuals with and without peripheral arterial disease. The EURODIALE Study


IDENTIFYING RISK FACTORS FOR WOUND INFECTION

Almost all open wounds are contaminated with micro-organisms, with many going on to heal successfully. However, in some wounds the micro-organisms can invade, multiply, and result in infection. There are a number of risk factors which can increase the individual’s vulnerability to a wound infection. The impact of infection is also influenced by the pathogenicity and virulence of the causative micro-organisms. Clinicians need to have a good understanding of all these factors and remain vigilant for early signs of infection, to ensure appropriate wound and patient management is delivered.

**Table 1**
Definitions of wound infection

<table>
<thead>
<tr>
<th>Wound term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Contamination</td>
<td>The presence of micro-organisms on the surface of the wound, without</td>
</tr>
<tr>
<td></td>
<td>microbial multiplication (Sutton, 2004) or signs and symptoms of infection</td>
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<td></td>
<td>(International Consensus, 2008)</td>
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<tr>
<td>Colonisation</td>
<td>The presence of multiplying micro-organisms in the wound do not result in</td>
</tr>
<tr>
<td></td>
<td>damage to the wound tissue, and do not generate a host immune response</td>
</tr>
<tr>
<td></td>
<td>(Ayton, 1995; International Consensus, 2008)</td>
</tr>
<tr>
<td>Critical colonisation</td>
<td>The point at which the host immune response is no longer able to control</td>
</tr>
<tr>
<td></td>
<td>the micro-organisms colonising the wound (Kinglsey, 2001), denoted by a</td>
</tr>
<tr>
<td></td>
<td>delay in healing but not necessarily accompanied by obvious deterioration</td>
</tr>
<tr>
<td></td>
<td>in the wound or other overt signs of infection (Cutting, 2008)</td>
</tr>
<tr>
<td>Infection</td>
<td>The presence of multiplying micro-organisms within the wound that</td>
</tr>
<tr>
<td></td>
<td>overwhelm the host immune response, with associated clinical signs and</td>
</tr>
<tr>
<td></td>
<td>symptoms (Kinglsey, 2001); In locally infected wounds the bacteria</td>
</tr>
<tr>
<td></td>
<td>multiply, healing is disrupted and wound tissue is damaged (International</td>
</tr>
<tr>
<td></td>
<td>Consensus, 2008); In wounds with spreading infection the presence of the</td>
</tr>
<tr>
<td></td>
<td>bacteria may produce problems in the area local to the wound (International</td>
</tr>
<tr>
<td></td>
<td>Consensus, 2008); In wounds with systemic infection the presence of</td>
</tr>
<tr>
<td></td>
<td>bacteria may result in systemic illness (International Consensus, 2008)</td>
</tr>
</tbody>
</table>

Shila Patel

**Key words**
Wound infection  Risk factors  Virulence  Pathogenicity  Quality of life  Economic burden

Wound infection is the result of the complex interaction between micro-organisms and the host environment in which they are found. Infection can be local and contained within the immediate area around the wound or may spread beyond the wound site and may even become systemic (Collier 2004). Therefore wound infection may result in a spectrum of morbidity ranging from superficial infection with uncomplicated healing to deep, severe infection with associated complications, such as extensive cellulitis or bacteraemia and in some cases, death.

Although most wounds contain bacteria not all will become infected. A variety of factors influence the ability of micro-organisms to establish infection, such as the ability of the individual’s immune system to combat the micro-organisms invading (host resistance) and the pathogenicity and virulence of the causative micro-organism(s).
to heal successfully. However, in some people, wound contamination may lead to colonisation, critical colonisation or infection (Cutting, 1998) (Table 1). It is vital to accurately distinguish between these states, particularly as contamination and colonisation are considered normal states with no associated disease, ill health or delayed wound healing. Conversely, critical colonisation is considered to be the precursor to wound infection and critically colonised wounds require a reduction in the level of bacteria present if the wound is to progress towards healing. In chronic wounds, critical colonisation may cause delayed healing in the absence of any indicators of infection, thus the clinician should be alert to this and microbial involvement must be suspected when other causes of indolence have been eliminated (Kingsley et al. 2009).

Infected wounds (Figure 1) require prompt antimicrobial intervention to prevent deterioration and to facilitate wound healing (International Consensus, 2008). In some patients, e.g. those with diabetic foot ulcers or general poor health, wound bioburden can rise quickly from colonisation to infection rapidly thus it is important to be aware of these states and the need for vigilance in patients who are at an increased risk of wound infection.

**RISK FACTORS FOR INFECTION**

The potential impact of wound infection is intrinsically linked to individual susceptibility to infection (Wilson, 2006). Host resistance is influenced by a number of factors which should be considered when managing patients with wounds healing by primary and secondary intention.

**Co-morbidity**

Disorders of the circulatory system, such as cardiovascular or respiratory disease, reduce blood and oxygen supply within a wound which slows the healing process and thus increases the risk of wound infection (Dealy, 2005; Ridgeway et al. 2005). For wounds to heal rapidly the tissue oxygen tension, defined as the partial pressure of oxygen in the wound tissue, needs to be greater than 40mmHg and where levels are below 20mmHg healing is unlikely to occur (Hunt and Hspt, 1997).

Generally acute wounds in otherwise healthy individuals have an oxygen tension ranging between 60–90mmHg compared with tissue oxygen tensions in chronic non-healing wounds which can drop as low as 5–20mmHg resulting in cell destruction and tissue necrosis, both of which provide favourable conditions for microbial multiplication (Sibbald et al. 2003).

Metabolic disorders such as diabetes mellitus can reduce neutrophil activity and specifically interfere with the action of phagocytosis (Slaughter et al. 1993). This can delay the normal inflammatory response and ingestion of microorganisms, preventing the formation of granulation tissue and increasing the risk of infection (Kjersem et al. 1988).

Malnutrition, defined as a state of nutrition where there is a deficiency, excess or imbalance of essential nutrients required by the body which causes a measurable adverse effect within the individual (Abbasi and Rudman, 1993), is associated with a poor immune response and increased risk of wound infection (Dealy, 2005). This is particularly significant given that Stratton et al (2003) found that up to 40% of patients are malnourished on admission to hospital.

**Immunosuppression**

Immunosuppression can arise as a result of concurrent infections and the use of certain drugs such as chemotherapy, long-term corticosteroids and immunosuppressants, all of which predispose the individual to the risk of potentially serious infection (Wilson, 2006).

**Age**

Advancing age is also associated with increased susceptibility to infection, as it is linked to an increased incidence of chronic disease and a slower immune response (Wilson, 2006).

**Psychosocial**

A variety of psychosocial factors can also influence the impact of wound infection on the individual. Hospitalisation increases the risk of cross-infection from other patients, poor personal hygiene means an increase in the overall bioburden of microorganisms on the body, and certain unhealthy lifestyle choices, such as smoking can also have a negative impact.

**Wound characteristics**

Certain types of wound are also
Pathogenicity or the ability of a micro-organism to cause disease is dependant upon its virulence factors; the factors that help micro-organisms to invade, multiply and cause damage to tissues (Wilson, 2006). Virulence factors are any of the genetic, biochemical or structural features of a micro-organism that enable it to produce disease in a susceptible host. According to Todar (2009) virulence factors fall into two main categories:

1. Invasiveness factors
   Micro-organisms are able to produce invasions. These extracellular substances or enzymes facilitate invasion of host cells. For example, hyaluronidase is produced by a number of micro-organisms including streptococci and staphylococci species and can attack connective tissue which can result in significant damage to the wound tissues.

2. Toxigenesis factors
   Micro-organisms are able to produce exotoxins and endotoxins which can cause either a local or systemic effect, dependant on the amount of toxin released.

In addition micro-organisms have a number of structural features that facilitate their pathogenicity for instance the use of capsules which protect them from phagocytosis and complement activation and extending pili which allow the micro-organisms to attach themselves firmly to host cells (Cooper, 2006). Micro-organisms can also encase themselves in a sticky, shiny biofilm on the wound surface and have the ability to intermittently emit single bacteria that can then lead to local infections or weaken the collagen matrix in healing wounds, resulting in further wound breakdown and re-ulceration (Costerton et al, 1999). Micro-organisms can also overcome or bypass host defence mechanisms by producing surface slime as seen with Pasteurella multocida, which enables the bacteria to avoid engulfment (ingestion) during the host phagocytic response.
Aerobic (oxygen tolerant) pathogens such as Staphylococci and Streptococci species are most often found in open wounds (Kingsley, 2003). Increasingly, however, anaerobic (oxygen intolerant) micro-organisms are also regularly isolated from chronic infected wounds, resulting in a polymicrobial wound in which both aerobic and anaerobic micro-organisms are present (Kingsley, 2003). The polymicrobial nature of chronic infected wounds can produce synergistic relationships and thus increase overall virulence; yet in contrast individual microorganisms many have low virulence (Pericival and Bowler, 2004).

**QUANTITY OF BACTERIA**

The overall pathogenicity of a microorganism may also increase when there is a heavy bioburden within a wound site. A quantitative measure of greater than $10^7$ cfu (colony forming units)/cm² is generally accepted as being indicative of infection (White, 2003). However, there are a number of confounding factors in using this quantitative definition. With specific micro-organisms, for example, group A β-haemolytic Streptococci, very few micro-organisms are required for pathogenesis to occur due to their ability to produce a number of exotoxins and toxins which cause and spread infection (Dow et al., 1999). The definition may also be questioned because large numbers of micro-organisms may be identified from wounds without overt signs of infection (Bowler, 2003). Furthermore, in wounds that are polymicrobial it may be difficult to establish which micro-organism(s) are responsible for producing infection, some of which may be present in relatively low numbers but which gain virulence when combined with other micro-organisms within the wound. Therefore, it should not be assumed that a heavy bio-burden always has a greater impact on the individual.

All of these factors indicate that the impact of wound infection, on wounds healing by primary and secondary intention, will vary from one patient to another according to the specific pathogenicity and virulence factors of the causative micro-organism(s), coupled with the patient's individual vulnerability.

**CONCLUSION**

A range of factors can influence the impact of wound infection. Essentially there is a fine balance between individual vulnerability to infection versus the pathogenicity and virulence of the causative microorganisms. Where the host immune response is overwhelmed by the causative microorganisms, wound infection will arise and the more susceptible an individual the greater the impact is likely to be.

**REFERENCES**


THE DIAGNOSIS OF WOUND INFECTION IN CLINICAL PRACTICE

Andrew Kingsley, David Leaper

The prompt diagnosis of wound infection is of the utmost importance to enable rapid intervention. Currently, wound infection is clinically diagnosed following an evaluation of the patient, their wound and the surrounding tissues. Identifying the presence of infection is not always easy because the signs and symptoms vary depending on its severity, and according to wound type. Biological tests such as microbiological cultures and blood tests, and imaging procedures can be used to support a clinical diagnosis and to help guide treatment, but findings should always be interpreted in the context of the patient as a whole to optimise clinical outcomes.

KEY WORDS
Diagnosis
Wound infection
Signs and symptoms
Diagnostic tools

All open wounds contain microorganisms, yet the majority do not become infected as the host succeeds in keeping the wound’s bioburden at a level that allows healing to occur normally (contamination and colonisation). However, in some patients, such as the elderly or immunocompromised, the interactions between the host and the invading micro-organisms may become imbalanced, so that the wound bioburden results in impaired healing, or the signs and symptoms of wound infection (critical colonisation through to tissue invasion). When this happens, immediate intervention is required to reduce the wound bioburden and treat the infection. In patients who are at an increased risk of infection, such as those with diabetic foot ulcers, prompt diagnosis is of the utmost importance to prevent deterioration of the wound and its costly implications. Currently, the diagnosis of wound infection is mainly driven by the findings of clinical assessment (International Consensus, 2008), but is sometimes supported by laboratory-based tests. Cooper (2005) remarked that due to the incompletely defined nature of inter-microbial and host and pathogen interactions, the use of clinical assessment in the diagnosis of wound infection remains more reliable than using microbial evidence alone. Indeed, laboratory tests can be of little value if considered without reference to the individual patient. It is imperative therefore that healthcare professionals managing patients with wounds know what the clinical signs and symptoms of wound infection are so that they can be observed, further investigated if necessary and the findings interpreted as soon as possible so that appropriate treatment can be initiated.

CLINICAL ASSESSMENT

A structured holistic assessment of the patient and their wound is essential to establish a baseline against which the success of treatment can be measured. Regular reassessment is vital to enable the progress or deterioration of the wound to be monitored so that the management plan can be adapted as necessary. Systematic history taking can be used to identify any risk factors likely to increase the risk and severity of wound infection (Table 1), while a systematic assessment of the wound and its surrounding tissues enables the early identification of potential indicators of infection (Table 2).

The signs and symptoms of wound infection range in severity from subtle, local symptoms to non-specific indicators of systemic disease (Table 2) and vary according to wound type. For example, infection in a surgical wound is usually obvious (Figure 1), whereas

Figure 1: A surgical site displaying obvious signs of infection: swelling with redness, heat and pain.
in patients with chronic wounds, the diagnosis of infection may rely on the recognition of subtle local signs (International Consensus, 2008).

Cutting and Harding (1994) laid an important milestone in the recognition of the more covert signs of infection because they enable more rapid diagnosis and intervention. These signs have subsequently been subjected to validation (Gardner et al., 2001) and further expanded by expert consensus for different types of wounds (Cutting et al., 2005) (Table 3).

The content and severity of wound infection influence management choices and thus the signs and symptoms of the varying degrees of infection, and how these can vary according to wound types, must be understood. It is sensible for the clinician to integrate these signs and symptoms into their general wound assessment and be familiar with the signs and symptoms of infection in the wound types they manage most frequently (International Consensus, 2008).

However, there remains a need for universally accepted definitions of wound infection because clinical diagnosis remains subjective (Leaper and Snyder, 2008).

Initial assessment may reveal the presence of infection for which appropriate intervention will be required. In some cases, however, clinical findings indicate the need for further investigation in the form of microbiological culture, blood tests or imaging investigations. The tests currently used in clinical practice include:

- **Microbiological culture**
- **White blood count**
- **Erythrocyte sedimentation rate**
- **C-reactive protein**
- **Imaging, such as MRI and X-ray.**

**Microbiological culture**

Microbiological culture takes place as part of routine clinical practice and is used to identify the organisms present in the wound and their particular antibiotic sensitivities (World Union of Wound Healing Societies, 2008) and to aid judgement on the severity of infection. Where spreading infection (cellulitis) is diagnosed, antibiotic interventions should be initiated, and adapted according to the results of the culture. Microbiological culturing is indicated for:

- **Acute wounds with signs of infection**
- **Chronic wounds with signs of spreading or systemic infection**
- **Or in high-risk chronic wounds with signs of localised infection**

Empirical therapy with broad-spectrum antibiotics may also lead to gastrointestinal upset, notably *Clostridium difficile* infection, which can further debilitate the patient and in a minority of cases lead to serious illness requiring surgery or even lead to death. In some cases in the community the time to get a result may be further lengthened as collecting the specimen, returning it to the
The degree of surface preparation required as part of the sampling process is still under debate with some authors suggesting that superficial debridement is necessary to expose the deep wound compartment, and that sampling should only take place on the area of the wound bed exhibiting the most dramatic signs of infection (Sibbald et al, 2001; Dow, 2003). Nurses, especially those practising in the UK, take a large number of wound samples but are unlikely to undertake sharp debridement before doing so because of a competency deficit.

Sampling the whole wound surface in a rolling zig-zagging motion has also been suggested but this has not been validated (Dow, 2003). A standard protocol encompassing this method has been suggested for routine clinical practice (Kingsley and Winfield-Davies, 2003). Theoretically at least it has been suggested that the best technique for swabbing is that described by Levine and which involves pre-cleaning the wound bed and the use of enough pressure from a swab tip to extract fluid from the tissue in a 1 cm area of wound base. (Anon, 2004). This technique, however, is more suited to identifying organisms below the surface of a wound that may be responsible for invasive infection with an accompanying host response, rather than the varied and many species that might be responsible for delayed wound healing.

The most recent guidance to note is from WUWH (2008) which says that ‘sampling should take place after wound cleansing (and, if appropriate, debridement), and should concentrate on areas of the wound of greatest clinical concern... and... beware of interpreting a microbiology report in isolation — consider the report in the context of the patient and the wound...’.

This means that a swab should only be taken when clinical signs and symptoms denote infection. According to Howell-Jones et al (2008) practitioners need to include clinical information with the swab so that laboratories can interpret the microbiology results and then be cautious with the release of antibiotic susceptibility information. They
Table 3
The signs and symptoms associated with wound infection

<table>
<thead>
<tr>
<th>Localised infection</th>
<th>Spreading infection</th>
<th>Systemic infection*</th>
</tr>
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<tbody>
<tr>
<td><strong>Acute wounds</strong> (surgical, trauma and burns)</td>
<td></td>
<td><strong>Sepsis:</strong> documented infection with pyrexia or hypothermia, tachycardia, tachypnoea, raised or depressed white blood cell count</td>
</tr>
<tr>
<td>New or increased levels of wound-related pain</td>
<td>Further extension of erythema</td>
<td>Severe sepsis: sepsis and multiple organ dysfunction</td>
</tr>
<tr>
<td>Erythema</td>
<td>Lymphangitis</td>
<td>Septic shock: sepsis and hypotension despite inadequate volume resuscitation</td>
</tr>
<tr>
<td>Local warmth</td>
<td>Crepitus in soft tissue</td>
<td>Death</td>
</tr>
<tr>
<td>Swelling</td>
<td>Wound breakdown/decubitus</td>
<td></td>
</tr>
<tr>
<td>Pusulent discharge</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pyrexia associated with surgical wounds 5-10 days post-operatively</td>
<td></td>
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<tr>
<td>Delayed or stalled healing</td>
<td></td>
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<tr>
<td>Abscess</td>
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<tr>
<td>Malodour</td>
<td></td>
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</table>

| Chronic wounds (Diabetic foot ulcers, venous and arterial leg ulcers and pressure ulcers) | | **Death** |
| New or increased levels of wound-related pain | As for localised infection plus: | |
| Delayed or stalled healing | Wound breakdown | |
| Peri-wound oedema | Erythema extending from wound edge | |
| Erythema or bullous formation | Crepitus, warmth, induration or discoloration spreading into the peri-wound area | |
| Distinctive malodour or change in odour | Lymphangitis | |
| Dehydrated/eroded granulation tissue | Malaise or other nonspecific deterioration in the patient's general condition | |
| Wound bed discoloration | | |
| Increased or altered/purulent exudate | | |
| Induration | | |
| Pusulent discharge | | |
| Pocketing | | |
| Bridging | | |

Table adapted from International Consensus (2003)

Noted that the release of antibiotic susceptibility data on venous leg ulcer specimens to community clinicians increased the prescription of antibiotics, so inappropriate swab taking may lead to inappropriate care and an increase in healthcare costs.

**Needle aspiration**

Needle aspiration is usually used to obtain samples from closed surgical wounds with signs of infection but without discharge of pus and can be of value in identifying infection in this wound type (Parikh et al, 2007). It can also be used to obtain samples from suspected abscesses, puncture wounds and sinuses. The skin above the site where the sample is to be collected is cleaned with an antiseptic and the needle inserted to withdraw tissue fluid. The use of this procedure can result in pain and needle damage and the operator needs to be aware of structures below the surface in order to minimise harm to the patient. Therefore skill in taking the sample is required meaning it is unlikely to become routine practice for open wounds in the foreseeable future.

While it is an uncommon technique in UK clinical practice, needle aspiration may also be used to obtain a sample from wounds that are deteriorating and not responding to standard antibiotic therapy, as indicated by swab results.

Aspiration cultures have also been used in antimicrobial dressing research (Verdu-Soriano et al, 2004) and along with biopsies may be more suited to this arena where high degrees of accuracy are required rather than routine clinical practice where microbiology requires interpretation of results in the context of the patient rather than direct report of findings.

**Wound biopsy**

Biopsy, in which a sample of tissue is taken from the base of the wound
and processed to identify the pathogens within it, is considered the gold standard microbiological method for research but is not often carried out in clinical practice due to exacting microbiological processing requirements and concerns of causing harm to the patient.

Wound biopsy allows access to bacteria that have penetrated into the tissues below the surface of the wound. These bacteria may be those that are more closely related to invasive or more severe forms of infection which show as cellulitic skin responses and systemic signs such as fever, raised white cells, ESR and CRP counts, and bacteremia. However, taking a biopsy is invasive, requires skill from the collector and in the laboratory, and is more time consuming than a surface swab. It also only samples a small area of tissue, so while it may be free of organisms not considered to be causative pathogens of infection it may miss the very organisms it is trying to identify because bacterial distribution on and in wounds is heterogeneous. As it does not analyse organisms living on the surface and in the wound fluids it may miss out on valuable information regarding the range of isolates and their quantity so is unlikely to be suited as a test in the less severe forms of infection, that is those without clinically apparent host response.

Bassak et al (1992) found that 72% and Bill et al (2001) found 79% of wounds considered infected by biopsy culture techniques simultaneously demonstrated infection by quantitative swab cultures. Rutli and Rodeheaver (2002) explored the relationship between the quantitative and semi-quantitative swab methods to determine how accurate the latter easier investigation would be in determining infection. They found that quantitative and semi-quantitative swabs correlated well when used to identify wounds with heavy bioburden (i.e. those with >10^6/g). The level which they reported as being considered by most to be indicative of infection or delayed healing.

The WUWHS (2008) consider biopsying to be accurate but as it is invasive it is often reserved for diagnosis in wounds that are failing to heal despite anti-infective treatment.

**Imaging**

Plain X-rays have value in identifying foreign bodies in traumatic wounds enabling targeted debridement, and for aiding diagnosis of osteomyelitis infection of bone, notably in patients with diabetes who have foot wounds. It is accepted that in this patient group, if bone can be seen or probed that osteomyelitis will be present and will require intervention (Lawrence et al, 2007).

**Blood tests**

Blood test results can support an infection diagnosis and help to monitor the patient’s response to therapy. They are most valuable in cases of spreading infection with or without systemic clinical signs and symptoms.

**Full blood count/white cell count**

White blood cells or leucocytes increase in number in response to the inflammatory cascade following trauma and in response to infection. A full blood count measures five different types of leucocyte: neutrophils, macrophages, eosinophils, basophils, and lymphocytes. Neutrophils are raised early on in response to infection as they are non-specific immune cells capable of indiscriminate killing of invading pathogens. They are also held in reserve in large numbers near the skin and therefore are capable of mounting a rapid response to infection. In patients with spreading infection and where there are systemic clinical signs such as pyrexia or increased leukogram (WBC) provides information on the white blood cell count which in the early stages of wound infection may be elevated (known as neutrophilia) (WUWHS, 2008). Macrophages are part of the non-specific immune system and arrive soon in the wound after neutrophils usually at about 24 hours and continue to attack infection but also phagocytose spent neutrophils thereby preventing the release of toxic antibacterial compounds they contain. Macrophages are also able to remove damaged tissue in the wound bed. Lymphocytes are specific immune cells and so arrive later on in the immune cascade and are targeted at specific pathogens. Eosinophils are small in number but may have a role in dealing with parasitic infections. Basophil function is unclear but may play a role in the ingestion of foreign particles and they produce heparin and histamine which are chemicals of inflammation. Eosinophils and basophils are of no current general interest in clinical wound work in the UK.

The white cell count can also be of value in cases of local infection to help support clinical and microbiological evidence for a diagnosis of infection. In cases of delayed healing where overt signs of infection are absent and response to topical antiseptic therapy is not forthcoming, they may be valuable in assessing if the body is making a subtle systemic response and thereby give some clinical rationale for the use of systemic antibiotics.

White cell counts will reduce in response to effective antinfective treatment but CRP levels tend to be used as this is a more rapid indicator.

**Erythrocyte sedimentation rate and plasma viscosity**

Erythrocyte sedimentation rate (ESR) and plasma viscosity give a non-specific measure of inflammation and tissue damage both of which can be induced by infection. ESR measures the time taken for red blood cells to fall out of solution in a standard tube. This takes longer when the plasma viscosity is higher due to increased levels of proteins in the plasma due to inflammatory process. ESR and plasma viscosity will therefore fall as infection severity decreases. ESR changes more slowly than CRP but is a simpler test to perform and does not require technical laboratory equipment. ESR may also be elevated because of concomitant conditions, e.g. autoimmune disease, so results should be interpreted with care and should
be considered along with clinical signs and symptoms and swab results.

**C-reactive protein**

C-reactive protein is another non-specific marker of inflammation and levels rise dramatically in cases of spreading wound infection. CRP falls rapidly in response to effective antibiotic therapy for wound infection and so could be a valuable monitoring tool and may help to determine duration of intravenous therapy in more severe infections. As with other non-specific tests it should be borne in mind that CRP is elevated in other circumstances including trauma and after surgery.

All the findings of patient and wound assessments and any diagnostic results must be viewed together in order to make appropriate diagnosis of infection and judgements about the efficacy of treatment. Viewed in isolation any one of these indicators may lead to a false diagnosis and inappropriate treatment which may cause harm to the patient, cost more money and extend patient treatment times and length of hospital stay. Remember to treat the patient, not the microbiology report.

**Conclusions**

Clinicians must have a degree of experience and be able to use their clinical skills to diagnose infection using diagnostic tests where possible to confirm enhance their diagnosis. Currently there is no fool-proof approach to diagnosing infection and as each wound and patient is individual, all factors must be considered.

**References**

THE MANAGEMENT OF WOUND INFECTION

John Timmons, David Gray

The management of wound infection is one of the most vital components of wound care. Recent emphasis on healthcare-associated infections and resistant bacteria has further highlighted the need for early assessment and intervention for patients who are at risk, not only of wound infection but of systemic sepsis. Good local wound care such as debridement, combined with topical and systemic antimicrobial agents may be necessary. This article examines some of the approaches to wound management which may be undertaken when a patient has or is at risk of wound infection.

KEY WORDS
Wound infection
Management
Antibiotics
Antimicrobials
PHMB

The complex nature of wound infection requires a multi-professional approach to ensure continuity of care and to maximise the quality of care that the patient receives. Early identification of infection and timely intervention is essential to prevent deterioration of the wound and the systemic spread of invading micro-organisms. The key aims of wound infection management are to reduce the bioburden of the wound locally, and to treat spreading or systemic infection. Improvement of the general medical condition of the host by optimising the management of co-morbidities can help to promote healing, as can the identification and elimination of risk factors for infection. Factors such as wound pain and local wound symptoms such as exudate and malodour should also be addressed, since these can be distressing for the patient and contribute to a reduction in their quality of life. All of these elements of wound management will now be discussed in more detail.

MANAGEMENT OF WOUND INFECTION

The management of wound infection must be guided by the findings of holistic patient assessment and any diagnostic tests, to ensure that the patient is receiving appropriate treatment. Assessment and diagnostic test results also provide a baseline against which the success of chosen interventions can be measured.

Once wound infection is diagnosed, management should focus upon:

- Optimising the host’s ability to fight infection
- Optimising the patient's general health in order to promote wound healing
- Reducing wound and/or systemic bioburden
- Optimising wound bed conditions
- General measures, such as the provision of pain relief, patient education, and psychosocial support
- Psychological needs.

Optimising the host's response
Assessment should identify the individual's risk factors for infection and management should aim to minimise or eliminate these where they exist, if possible. For example, some papers report a reduced immune response in patients who are malnourished (Olde Damink and Soeters, 1997) so addressing the problem may help to reduce their risk of infection, while optimising the patient's wound healing potential by providing the body with the proteins which are required for wound healing (Timmons, 2006).

Optimising the patient's general health
Patients with wound infection may also have co-morbidities such as cardiovascular or respiratory disease, which can impact on their ability to fight infection. The management of concomitant conditions should be optimised as part of an ongoing care plan to improve the patient's wound healing potential.

Patients with wounds may have infection which emanates from other body sites such as in-dwelling urinary catheters, central lines and IV sites. It is necessary to assess all possible sources of infection to rule them out before treating the wound, perhaps wrongly.
Reducing wound/systemic bioburden
Wound bioburden can be reduced locally through the use of debridement and topical antimicrobials. In wounds with spreading and systemic infection, these approaches must be used in conjunction with systemic antimicrobial therapy.

Debridement
Debridement of sloughy/necrotic tissue is one of the cornerstones of good wound practice and is vital when reducing the bacterial burden within the wound (Vowden and Cooper, 2006), unless contraindicated by the patient's overall physical condition or disease process. Sloughy and necrotic tissue can harbour bacteria and is an ideal environment for bacteria in which to multiply, due to the availability of nutrients and oxygen (White et al, 2006). By removing devitalised tissue the clinician can encourage granulation tissue formation by removing the barrier to healing (Case report 1).

For some acute episodes of spreading infection, such as necrotising fasciitis, immediate surgical debridement is necessary to minimise the risk of tissue damage. This treatment combined with systemic antibiotics is necessary to preserve tissue and to reduce mortality and morbidity (Brook and Frazier, 1995).

Sharp debridement can be carried out at the bedside and is a relatively fast method of reducing wound bioburden by the rapid and effective removal of necrotic tissue in wounds with local and spreading infection. However, it needs to be carried out by a skilled practitioner.

Hydrosurgery systems such as Versajet (Smith and Nephew, Hull) use a jet of water and suction handset to remove sloughy tissue from the wound bed and allow large-scale debridement to be undertaken in a ward environment. These devices have revolutionised the debridement process by providing a safe and effective method of wound debridement which can be used outside the surgical theatre. However, training is also required to use hydrosurgery techniques and this may only be available in specialist centres.

Larval therapy can also be used as the larva liquefy the dead tissue and if successful can result in rapid debridement, but may be unacceptable to the patient.

Autolytic debridement is the removal of devitalised tissue from the wound by means of the body's own enzymes operating in a moist environment. These matrix metalloproteinases disrupt the proteins
Case report 2

Figure 1. This patient was 24 hours post-surgery and had had a wound manager in place for this time. The wound bed was heavily contaminated with faeces leaking from the fistula at the base of the wound. At this point the patient was at risk of a wound infection due to the major surgery and high levels of contamination.

Figure 2. After 48 hours of Negative Pressure Wound Therapy (NPWT) the wound bed was infection free and the first growth of granulation has occurred. The removal of exudate using NPWT in combination with PHMB-coated gauze resulted in a healthy wound bed.

Figure 3. Once the NPWT was in place a hole was made in the gauze to allow free flow of the faecal matter into a stoma bag and the hole was then sealed using siamahcsive paste ensuring that the pressure was maintained.

Figure 4. After seven days of NPWT the wound was covered with 100% granulation tissue and this wound that was at high risk of infection remained infection free.

that bind the dead tissue to the body (Schultz et al. 2003). When kept moist, the tissue will naturally degrade and deslough from the underlying healthy structures, so the process can be enhanced by the application of wound management products which promote a moist environment. Moisture-donating products such as honey and silver sulfadiazine (SSD) are best selected for drier infected wounds. In wounds which have moderate to high levels of exudate, Cadexomer iodine is able to assist in the debridement process while providing anti-microbial activity. Other products such as silver-based formulations which are incorporated into alginates or hydrofiber materials are useful debriding agents when used on moderate to heavily exuding wounds. Suprasorb X+PHMB (Activa Healthcare, Burton-upon-Trent) is an antimicrobial wound dressing containing unique biosynthetic HydroBalance fibres, and the antimicrobial polyhexamethylene biguanide (PHMB). The use of these fibres means that...
the dressing is able to regulate the absorption and donation of moisture to the wound and it has been shown to significantly promote wound debridement (Alvarez, 2004).

In addition to absorbent primary products, a secondary dressing can provide additional absorptive capacity to assist in the management of large volumes of exudate. In wounds with moist sloughy tissue present negative pressure can help in the debridement process (Argenta and Morykwas, 1997; Molnar, 2004).

**Managing excess exudate**

Exudate management is a challenge for the healthcare team as well as for the patient, and despite a number of products being available, patients still report that exudate management is a problem (Jones, 2008). Exudate also affects patients’ quality of life, as the presence of wet dressings or bandages next to the skin is likely to cause discomfort.

Exudate is produced as a normal part of the healing process and is necessary to assist in the chemical interaction which takes place within the wound environment during wound healing (Timmons, 2006). Exudate contains growth factors, nutrients and proteases, as well as cells which are involved in cleaning bacteria. During an infective episode, however, wound exudate is produced at a higher rate than normal which is partly due to the histamine response. If excess exudate is not managed properly there is a risk that it will cause skin breakdown and maceration of the tissues.

Management of exudate involves the use of absorbent dressings such as foams, alginites and hydrofibrers, to prevent maceration and improve patient comfort. Suprasorb X+PHMB has also been shown to effectively protect the peri-wound skin of patients with locally infected wounds (Davis, 2006).

Topical negative pressure (TNP) therapy is also a key therapy for removing and containing exudate when treating chronic wounds where large volumes of exudate are present.

The use of topical antimicrobial agents such as honey dressings, cadexomer iodine, silver dressings and most recently PHMB dressings, such as Suprasorb X+PHMB, can also help to reduce the bioburden and therefore reduce the inflammatory response within the wound, so less exudate is produced. The product chosen will also depend on the tissue types within the wound bed.

Similarly, the removal of sloughy tissue from the wound bed by debridement can help to reduce exudate levels by promoting granulation and reducing the inflammatory response.

**The use of negative pressure in wound treatment**

Topical negative pressure therapy is the use of controlled suction to apply sub-atmospheric pressure to the wound bed to promote faster, more efficient healing. Excess exudate is drawn from the wound bed and stored in a sealed drainage system. TNP is also believed to accelerate wound healing by stimulating angiogenesis, promoting healthy granulation tissue and decreasing the bacterial load (Bale, 2006) (Case report 2). The newer systems of TNP use gauze impregnated with PHMB to line the wound bed, enabling these systems to be used on wounds which are locally infected or critically colonised in order to reduce the bacterial load in the wound as well as managing exudate levels (Bale, 2006).

**Managing wound odour**

Some infected wounds will be malodorous due to the release of chemicals from the bacteria and the breakdown of tissue within the wound (Fletcher, 2008). This odour is unpleasant for patients and their relatives and may add to the patients’ feelings of anxiety (Jones, 2008). The key to treating odour is to remove the underlying cause. By careful...

**Case report 3**

Figure 1. An elderly man presented two weeks post-tibial popliteal grafting and had developed oedema secondary to cardiac oedema and dependency. His long-standing ulcer to the malleolus was infected with Pseudomonas as indicated by distinct blue/green exudate and the periwound area was exoriated, resulting in significant pain and discomfort. The topical anti-microbial dressing Suprasorb X+PHMB was applied and covered with toe-to-knee blue line Comfeel/Softisan and another layer of blue line Comfeel.

Figure 2. After seven days of treatment in which three dressing changes occurred, the peri-ulcer area had epithelialised and there were no signs of Pseudomonas on the dressing or the wound. The oedema had reduced and the wound was proceeding towards healing.
Management of tissue oedema

Local tissue oedema may be present in wounds which have infection present, disrupted vascular networks and/or local lymphatic disruption. The increase in histamine at the wound site due to tissue damage will also contribute to the local swelling. This oedema can be painful for the patient and, depending on the wound site, may affect limb function and mobility (Case report 3).

By reducing local tissue oedema the local blood supply improves which assists healing. In normal physiology there are two blood vessels per cubic centimetre of tissue. When oedema is present, the same blood vessels have to provide blood to a larger area of tissue and this results in a drop in supply of oxygen and nutrients to the wound and in addition the waste products from metabolism are unlikely to be removed efficiently from the wounded area (Molnar, 2004). This inefficient blood supply may impair the delivery of tissue antibiotics and the build up of waste products may provide a reservoir for bacteria if the skin integrity is breached.

Compression therapy can help to reduce oedema by assisting the calf muscle pump, preventing reflux of blood into the lower limbs and reducing venous hypertension. This can be achieved using bandaging systems or compression hosiery garments for venous leg ulcers (Moffatt and O’Hare, 1995).

Topical negative pressure applied to oedematosus wounds results in a reduction of localised swelling by reducing the fluid in the interstitial spaces within the tissue (Argenta and Manywala, 1997). This reduction in oedema has been mentioned in a number of studies examining the impact of VAC therapy. This phenomenon is not witnessed when using traditional wound dressings unless using compression therapy for lower limb wounds.

Antimicrobial therapy

Antimicrobial therapy is usually required in wounds with local infection, or in wounds that have spreading or systemic infection.

Wounds that are critically colonised should be managed with topical antimicrobial agents in order to reduce bacterial burden. For wounds that are locally infected, the main treatment will be with topical antimicrobial agents. Vulnerable patients will require systemic antibiotics in addition to local topical agents. For wounds where infection, either spreading or systemic, has been diagnosed, the patient should be treated with both topical antimicrobials and systemic antibiotics.

The role of topical antimicrobials in treating infection

As a result of emerging antibiotic resistance, there has been a renewed interest in the use of antiseptics in wound care. Antiseptics offer many benefits as they can be relatively easy to use, are widely available, frequently cost less than antibiotics, and can be administered without prescription (Principles of Best Practice, 2008). They are usually used in the treatment of infected open acute and chronic wounds and are indicated for:

- The prevention of wound infection or recurrence in patients at increased risk
- The treatment of local wound infection
- The treatment of spreading and systemic wound infection in combination with systemic antibiotics.

Antiseptics differ from antibiotics in that they are generally active against a broader spectrum of organisms including common pathogenic anaerobic and aerobic bacteria, and fungi. Unlike antibiotics, antiseptics also tend to have multiple target sites, including the bacterial cell wall or membranes, in the organisms on which they exert their effects. This means that the microorganisms are less likely to mount an effective defence and survive as resistant strains (Gilbert, 2006).

However, they too should not be used indiscriminately or indefinitely, as there is also evidence for bacterial resistance to some antiseptics, such as silver (Mallard and Denyer, 2006), and there is a lack of clinical evidence surrounding the cytotoxicity of some antiseptic products (Principles of Best Practice, 2008). Antiseptic therapy should be reviewed regularly, and the clinician should remain alert for the symptoms indicative of spreading or systemic infection, when signs of infection have resolved, or if the wound starts to heal, then therapy should be stopped.

The range of topical antiseptic agents currently in common use in wound dressings in the UK include silver, iodine, and honey. PHMB is a relatively new entrant to the UK wound care market although it is in common use in Europe and US.

Topical antimicrobial agents are available in many formats which gives the clinician a great deal of choice when managing wound infection. Accurate patient and wound assessment should dictate the dressing used, but patients will also have opinions about which products they prefer or find most comfortable, and clinicians must take this on board as this may affect patient concordance.

Choosing the correct antimicrobial dressing will also help the patient feel confident that wound exudate and odour are being managed appropriately (Jones, 2008).

Silver

Silver has been used for centuries in treatment and prevention of
infection. Silver is a broad-spectrum agent which acts on the electron transport system within the bacterial cell and on the DNA within the bacteria (Cervantes and Silver, 1996). Silver is available as a cream (silver sulphadiazine (SSD)), an alginate, in foam and hydrofiber dressings and in nanocrystalline form.

Iodine
Iodine, in the form of povidone iodine and cadexomer iodine, has been used to treat infected wounds. Iodine is a powerful oxidising agent within the bacterial cell and as a result is a powerful bactericidal and bacteriostatic agent which is effective against a number of pathogens (White et al, 2006).

In chronic wounds cadexomer iodine products offer the slow release of iodine which can reduce the risks of infection and toxicity. There is minimal risk of thyroid function toxicity if the product is used as recommended by the manufacturer.

Honey
Honey is a product which has recently been revived as a topical wound care antimicrobial. One type of honey, Manuka, has been of particular importance, as it is clinically effective in a number of chronic wounds (Dunford et al, 2000). As an antimicrobial agent honey is bacteriostatic and contains flavonoids which are known antimicrobial agents. In addition, honey also produces hydrogen peroxide which can affect bacterial growth (Dunford et al, 2000).

Polyhexamethylene biguanide
Polyhexamethylene biguanide (PHMB) is a safe antiseptic and has been used in swimming pools, the brewing industry and as contact lens cleaning solution for many years with no development of resistance (Moore and Gray 2007).

PHMB is an antimicrobial agent which consists of a 12-unit long biguanide chain molecule which is viewed as superior to other biguanide molecules as they have multiple binding sites which bind to the bacterial cell membrane (Gilbert, 2006).

The positive charge on the molecule binds to bacteria and thus disrupts the bacterial cell membrane causing the cell contents to leak out (Gilbert, 2006). PHMB is active against a number of bacterial pathogens which includes methicillin-resistant Staphylococcus aureus, vancomycin-resistant enterococcus, Pseudomonas, Escherichia coli and S. epidermidis.

Antibiotics
Antibiotics have selective toxicity, that is, the ability to impact on bacteria without impacting on the cells of the patient (Douglas Sloigh and Timbury, 1994). The aim of antibiotic use is to treat infection using a compound to which the causative organism is sensitive. In cases where a number of pathogens exist, more than one antibiotic agent may be necessary.

The clinical challenge that resistant bacteria such as MRSA pose to wound management is well known (Guyot and Layer, 2006). Guidelines for the management of MRSA include the avoidance of inappropriate or unnecessary use of antibiotics to reduce the likelihood of emergence and spread of resistant strains (Cox et al, 2006).

However, when used appropriately, systemic antibiotics have an important
role in the management of wound infection (International Consensus, 2008). Their use is indicated for:
- Prophylaxis when the risk of wound infection is high, e.g. contaminated surgery or trauma injury (Case report 4).
- Patient diagnosed with spreading or systemic infection.
- When culture results reveal beta-haemolytic streptococci even in the absence of the signs of infection (International Consensus, 2008).

Guidelines for the antibiotic treatment of infection in the specific wound type should be consulted before prescribing antibiotics. Clinicians should use their judgement to prescribe the most appropriate antibiotic for the suspected causative pathogen(s), and treatment continued or altered in light of microbiology results. Other factors such as allergy, comorbid medication, costs, availability and the patient's ability/willingness to comply with treatment should also be considered (International Consensus, 2008).

Infection control
The principles of infection control include the use of gloves and protective apron when reviewing patients, the use of sterile equipment where possible and continual decontamination of hands when carrying out procedures.

Many moist wound healing dressings are occlusive in nature which helps to prevent bacterial entry into the wound and also assists in preventing the cross-contamination of other patients. All waste products including soiled dressings and used swabs should be discarded in the correct waste bins, and incinerated.

Other precautions include avoiding cool air fans when the wound is exposed to avoid the spread of bacteria around, and for the same reason, bed-making should be avoided at this time.

Wound cleansing with normal saline or tap water may be necessary to remove old dressing material from the wound bed, but may be of little value in the removal of bacteria (Collier, 2004). Some antiseptic wound irrigation preparations are currently being trialled but there is insufficient data to support its use at the present time.

**GENERAL MANAGEMENT**

**Pain management**
The increase in pain which a patient is likely to experience when wound infection is present is thought to be a result of local swelling in the tissues and the inflammatory reaction to bacteria in the wound (Cutting and Harding, 1994). Management of this pain may be achieved by using oral or intramuscular (depending on severity) analgesics combined with non-steroidal anti-inflammatory drugs, if not contraindicated, which can help to relieve soft tissue pain. Dressings should be chosen which do not adhere to the wound bed and/or the surrounding skin. Analgesia should be given 30 minutes before dressing changes to ensure maximum benefit during painful procedures (King, 2003).

It is also vital to ensure that dressings used are absorbent enough to handle exudate from the wound and are atraumatic on application and removal to minimise the risk of maceration and skin irritation. The surrounding skin should be protected using a barrier film which will help to minimise trauma during dressing removal or due to exudate.

If the patient is experiencing high levels of pain, it may not be possible to carry out procedures such as sharp debridement until the pain is effectively controlled.

**Management of pyrexia**
Patients with pyrexia will need their temperature restored to normal and this can be achieved by using antipyretics such as paracetamol. The room temperature should also be adjusted to avoid overheating. Fans can be used but should be decontaminated before and after use and switched off during dressing change to prevent wound contamination.

**Key Points**
- Wound infection can impact on patients' quality of life.
- Without optimal treatment, systemic illness can develop.
- A multi-disciplinary approach to care is vital.
- Monitoring of the patient; vital signs, bloods and cultures can help to monitor effectiveness of interventions.
- Local wound therapy such as debridement is often necessary.
- Topical and systemic therapies should be considered.
- Regular reassessment of the patient and the wound are necessary to monitor change.

**Pain education/communication/psychosocial support**
Patients and relatives require information about their illness and must be involved where possible when wound treatment decisions are being made. Practitioners who do not listen or give conflicting advice have been found to negatively affect quality of life (Charles, 2008). Conversely, good communication can help the patient to develop coping mechanisms and a positive, therapeutic relationship with their healthcare professional (Hopkins, 2004).

**Reassessment**
Wounds which are critically colonised or infected need to be monitored regularly in order to detect deterioration such as spreading infection or improvement in the state of the wound. Changes in the wound condition must be recorded accurately in the patient’s case and nursing notes.
If the wound is not responding to treatment and deterioration is noted, alternative therapies, both topical and systemic, may be required. If there are concerns about the wound status, dressings may be changed more frequently, i.e. every 48 hours, however; there are no supported recommendations in the literature, and so this should be done on a per patient basis.

The European Wound Management Association guidelines recommend complete review of the effectiveness of therapies every 10 days (Moffatt, 2006). Photographs or tracking changes in inflammatory markers such as CRP, ESR, and WBC count may also help to identify the success or failure of treatment, especially in chronic wounds where changes can be subtle (Timmons, 2006).

CONCLUSION

Wound infection is the most troubling wound care complication for many reasons, not least the potential threat to life. Patients with multiple pathologies presenting with wounds are likely to be at risk of infection and are less likely to produce a sufficient immune response (Gray et al., 2005). The management of the patient and the wound are inextricably linked, and attention must be paid to the systemic pathologies with which the patient presents. Patients such as those with diabetes are at higher risk of infection and should be closely monitored for changes in the wound site and also systemic signs of inflammatory response.

The management principles of infected wounds are to reduce the bioburden using systemic and topical treatment. In addition the removal of devitalised tissue from the wound bed is essential as it can impede healing and provides a bacteria-friendly environment. The increase in exudate, pain and odour caused by wound infection can affect the quality of life of the patient and must be treated with appropriate products to minimise their impact.

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Suprasorb X+PHMB is a new antiseptic dressing that combines Suprasorb X, a unique HydroBalance dressing that is able to both absorb and donate moisture, with PHMB, an antiseptic compound with no known cytotoxicity or resistance. Studies have shown that Suprasorb X+PHMB effectively reduces wound bioburden, promotes autolytic debridement, improves the rate of wound closure through an increase in granulation and epithelialisation, and effectively reduces wound-related pain. A series of case reports which evaluated the dressing’s performance on a variety of wound types support these findings and are presented in this article.

**KEY WORDS**
Suprasorb X+PHMB
Antiseptic
Wound infection
Case report evaluation

Antiseptics have been in use for much longer than antibiotics yet resistance to antiseptics presents much less of a problem. This may be because antiseptics differ from antibiotics in that they are generally active against a broader spectrum of organisms including common pathogenic aerobic and aerobic bacteria and fungi. Unlike antibiotics, antiseptics also tend to have multiple target sites, including the bacterial cell wall or membranes, in the organisms on which they exert their effects. This means that the micro-organisms are less likely to mount an effective defence and survive as resistant strains (Gilbert, 2006).

Once the need for topical antiseptic intervention has been identified, it is important to select a product that will provide optimum conditions to support rapid healing. The ability of the agent to reduce or eradicate micro-organisms, must also be considered, along with its specificity, cytotoxicity to human cells, its potential to select resistant strains and its allergenicity (Vowden and Cooper, 2006). The ability of the carrier dressing to handle exudate and remove necrotic tissue from the wound is beneficial since purulent exudate, necrotic tissue and slough are all growth mediums for bacteria (Cutting, 2008). The dressing’s ability to reduce malodour, conform to the site and shape of the wound, perform wound bed preparation functions, satisfy patients’ expectations and to meet treatment goals also need careful consideration (Vowden and Cooper, 2006).

The range of topical antiseptic agents currently in common use in wound dressings in the UK include silver, iodine, and honey. Polyhexamethylene biguanide (PHMB) is a relatively new addition and provides an alternative to the anti-microbials currently available.

**POLYHEXAMETHYLENE BIGUANIDE**

PHMB is a synthetic compound which is structurally similar to naturally occurring antimicrobial peptides (AMPs). AMPs are produced by the majority of living organisms and have a broad spectrum of activity against bacteria, viruses and fungi (Moore and Gray, 2007). AMPs are positively-charged molecules that bind to bacterial cell membranes and induce cell lysis by destroying membrane integrity, in a similar way to penicillin and cephalosporin antibiotics. AMPs are produced by many cells within the wound such as keratinocytes and inflammatory neutrophils, where they are thought to play a role in protection against infection (Sorensen et al., 2003).

The structural similarities between AMPs and PHMB mean that the latter can interact into bacterial cell membranes and kill bacteria in a similar way to AMPs (Moore and Gray, 2007). Some bacterial cells use an efflux pump to protect themselves from the effects of some antiseptics. However, the effect of PHMB on the bacterial cell membrane mean that the pump is unable to remove antiseptic, so bactericidal concentrations are maintained in the cell. This mechanism of action is quick and means that bacteria are unlikely to develop resistance to PHMB (Seipp and Korber, 2008).

**PHMB IN WOUND MANAGEMENT**

Polyhexamethylene biguanide (PHMB) is a...
Acrobacter xylinum. The bacteria produce fibres of cellulose which are 300 times finer than cotton, giving the material an exceptionally high surface area. The same microbes weave a mesh structure of fibres that enhances both its moisture handling capabilities and its tensile strength.

As a result of the biosynthetic HydroBalance fibres, Suprasorb X is able to regulate the absorption and donation of moisture at the wound dressing interface (Figure 1). Depending on the status of the wound, surplus exudate can be absorbed by the dressing or donated to the case of lightly exuding wounds. This moisture absorbing and donating capacity can also be exerted within the same wound, removing exudate and donating moisture to drier areas.

It also protects the wound against abrasion, desiccation and external contamination. These unique fluid-handling capabilities of the dressing mean that Suprasorb X can be used on moderately exuding, non-exuding and dry wounds. The moist environment also has a cooling effect that has demonstrated a significant reduction in pain (Alvarez, 2004; Davis, 2006; Wild and Eberlein, 2009).

In a 24-patient multicentre randomised controlled study carried out by Alvarez et al. (2004) to determine effectiveness of Suprasorb X compared with care already being received in patients with venous leg ulcers, Suprasorb X was found to significantly promote autolytic debridement and significantly reduce wound pain at weeks three, six and eight of the 12-week study. An improved rate of wound closure, in terms of increased epithelialisation and granulation tissue, was also noted (Alvarez, 2004). Results of decreased pain (Gailit et al., 2004), increased granulation and epithelialisation and an improved rate of wound closure have also been observed by Vijfberg et al. (2007), Eberlein et al. (2007) and Baikuturashvili et al. (2009).

THE SUPRASORB X DRESSING RANGE

Suprasorb X dressings have a unique structure made up of biosynthetic HydroBalance fibres. These fibres are the products of a cellulose fermentation process using a proprietary strain of Suprasorb X + PHMB, combines the proven efficacy of Suprasorb X with the antimicrobial action of PHMB (0.33%) and is indicated for use on lightly to moderately exuding superficial and deep infected wounds in all phases of wound healing. The PHMB component exerts its antimicrobial effect both within the dressing but also at the wound-dressing interface (Figure 2). As the PHMB is not bound to the HydroBalance fibres of the dressing it is released into the surrounding fluid along a concentration gradient.

The presence of fluid in the dressing means that antimicrobial activity is possible even on dry wounds, unlike silver-containing dressings which require the mechanical action of wound fluid to initiate antimicrobial activity.

SUPRASORB X + PHMB IN CLINICAL PRACTICE

A clinical case series performed by Mulder (2007) to determine the antimicrobial effects of Suprasorb X + PHMB showed that PHMB effectively reduced wound bioburden and had a positive effect on wound healing. Twelve patients with a total of 26 wounds were evaluated. 11 of whom had previously been unresponsive to silver or iodine-containing dressings.

Wound swabs were taken before and after treatment with Suprasorb X + PHMB. Before treatment, organisms were identified in the wounds of eight patients, most commonly Pseudomonas aeruginosa and Staphylococcus (including MRSA). At the end of the evaluation, levels of bacteria were decreased in five of the eight patients (two patients were lost to follow-up, and one patient experienced no change in bioburden). For the eight patients, there was a mean reduction in wound size from 6.79 cm² to 4.57 cm² in a mean of 25 days. Two wounds healed during the study and 13 showed improvement.

An evaluation of Suprasorb X + PHMB in the treatment of four patients with wounds which had previously been treated unsuccessfully with various silver-containing dressings was undertaken by Davis (2006). Although two wounds were locally infected, application of Suprasorb X + PHMB healed three of the four wounds, protected peri-
wound tissue and resulted in a decrease in wound pain (Davis 2006).

Similarly, an evaluation of Suprasorb X + PHMB in the treatment of 79 wounds of varying etiology by Cavosio (2006) revealed that healing or clinical improvement was achieved in >80% of the cases receiving treatment with Suprasorb X and PHMB. In a subset of wounds that had not been responsive to prior treatment with silver dressings, a decrease in wound size of 33% was observed after 3 weeks.

An analysis of the performance of Suprasorb X + PHMB in 6 patients with 5 different wound types supported these study findings. The evaluations found that Suprasorb X + PHMB promoted autolytic debridement which, in addition to the antimicrobial effects of the PHMB component of the dressing, helped to reduce the bioburden of the wound. The unique Hydrobalance element of the dressing promoted a moist healing environment which saw the rapid development of granulation and epithelial tissue, despite some of the patients being elderly and/or having co-morbidities which would make wound healing difficult. The dressing also protected the pen-wound skin from the effects of exudate and importantly, its use resulted in a decrease in wound-associated pain, with one patient describing the dressing as having a cooling effect. The case reports described here are presented on page 29–34 of this document.

CONCLUSIONS

The ideal antibiotic dressing will reduce wound bioburden while providing a moist wound environment that promotes wound healing. Such a dressing however must be used wisely to minimize the cytotoxic effects of the cells needed for wound healing and to reduce the selection of resistant bacterial strains (Vowden and Cooper 2006). Suprasorb X + PHMB is able to effectively reduce the number of pathogen in the wound. Currently, PHMB does not have a history of resistance or cytotoxicity making it a good alternative to antibiotics for which development of bacterial resistance and toxicity is an issue. Suprasorb X’s unique ability to absorb and/or donate moisture depending on the needs of the individual wound provides a moist environment that will allow the wound to progress towards healing and leads to a reduction in pain.

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MANAGEMENT OF AN INFECTED BURN IN A HIGH DEPENDENCY UNIT

Sean Fumaraio is Clinical Nurse Specialist and Tissue Viability Lead, University Hospital of North Staffordshire

The patient was a 38-year-old woman who was an intravenous drug user and had tested positive for both hepatitis C and human immunodeficiency virus (HIV). She had collapsed at home following an overdose sustaining a cerebral bleed and extensive burns to her shoulder and upper back from a radiator. She was admitted to the neurosurgical high dependency unit.

The patient was assessed by the plastic surgeons and prescribed topical silver sulfadiazine and similar dressings daily. In view of her comorbidities, no sharp debridement was attempted in the high dependency unit.

The wounds became colonized with Methicillin-resistant Staphylococcus aureus, and the plastic surgeons referred the patient to the tissue viability nurse for ongoing wound management.

At presentation the wound dimensions were 20x15cm and 6x6cm (Figure 1). The larger wound was approximately 40% necrosed and 10% sloughy and the remainder of the wound bed was dusky. The smaller wound was 60% sloughy tissue. There were high levels of exudate and malodour and although the patient was ventilated she was able to indicate that the wound was painful. The periwound skin was healthy in appearance.

The wounds were dressed with Suprasorb X + PHMB (Figure 2) (Activa Healthcare, Burton-upon-Trent) and a secondary foam dressing. This regimen was chosen to promote autolytic debridement while reducing the wound bioburden in a safe and controlled manner. In view of the patient's comorbidities, consideration was also given to cross-contamination. BlisterTech adhesive foam secondary dressing (Coloplast, Peterborough) was chosen to provide a high level of absorption during autolytic debridement and subsequent high volumes of exudate. The combined dressings controlled exudate for 48 hours and protected the periwound skin from its destructive effects. The dressing was changed on alternate days.

After 48 hours there was a significant improvement to the wounds. A large area of necrosed had been debrided and the wound bed appeared healthier (the larger wound consisted of 40% slough, with no necrosed and improved vascularity to the wound bed, while the smaller wound was covered with approximately 30% surface slough). There was less malodour and the periwound skin was healthy despite high exudate levels (Figure 3).

At the third dressing change there was no malodour and the wounds continued to debride. There was no evidence of epithelialisation at the wound margins and wound cultures showed no significant growth (Figure 4).

The wounds were swabbed on 2 March (after six days of treatment), and the patient was able to move into the main ward area because she was no longer considered a risk to other patients in terms of cross-contamination (the wound was to be swabbed weekly). This move proved to be a turning point in her recovery because, as the patient had suffered a cerebral bleed and sustained neurological deficit, it was vitally important that she be nursed in a stimulating environment with more social interaction than could be achieved in an isolated area.

After four weeks of treatment the wounds had made significant progress and its dimensions were 15x7cm and 3x3cm, respectively (Figure 5). There was no significant bacterial growth, no malodour and there had been a good progression towards healing (50% healthy granulation tissue, 10% superficial slough).

This regimen was particularly successful because rapid and safe wound debridement was achieved while managing wound pathogens and preventing cross-contamination to other vulnerable patients. Following stabilisation of the wounds, the patient was able to participate more actively in a rehabilitation programme which facilitated transfer out of secondary care to a rehabilitation unit for ongoing care in a timely manner.

The nurses in the rehabilitation unit were able to manage the wounds effectively and no further IVN support was necessary.
MANAGEMENT OF AN INFECTED SKIN TEAR

Sian Fumarela is Clinical Nurse Specialist and Tissue Viability Lead, University Hospital of North Staffordshire

The patient was a 97-year-old woman who lived at home independently with her husband. She had fallen on the stairs and sustained injuries requiring admission to the emergency care centre. She presented with multiple abrasions, bruising and a skin tear to the lower leg which required sutures. She was admitted overnight for observation, but became unstable and suffered a myocardial infarction. She was subsequently diagnosed with pulmonary oedema, cardiomyegy and pneumonia.

The patient was cared for on a medical ward for two weeks where she remained very unstable. A decision was taken with the family not to actively resuscitate in the event of a cardiac arrest.

The patient was referred to the tissue viability nurse because the wound deteriorated and developed a clinical infection. At the first wound assessment by the tissue viability nurse the wound measured approximately 40mm and was 1cm deep. The wound bed was unhealthy with approximately 20% necrotic tissue, 40% slough and exposed tendon (Figure 1). The periwound skin was fragile with surrounding cellulitis. There was moderate exudate and the wound was very malodorous. No local antimicrobials were used at this stage, and the wound was treated with a foam dressing and bandage.

Microbiology results indicated that clinical infection was present with a wound culture identifying a heavy growth of Klebsiella and scanty growth of gram positive Staphylococcus 0 – the systemic antibiotic amoxicillin/clavulanate potassium was prescribed as a result. The patient’s C-reactive protein (CRP) level was 21.5, white cell count (WCC) 13.6 and Hb 9.0. Low Hb indicates a reduced oxygen-carrying capacity of the blood, increasing the risk of infection and delayed healing as a result of possible low oxygen tension at the wound site. Raised CRP and WCC are inflammatory markers secondary to infection.

The patient’s pain score was 8 using a visual analogue scale of 0–10 with 10 being extreme pain, she was unable to stand and had a poor appetite. She said of her wound: “My leg smells. I think everyone can smell me.” She clearly found this distressing.

The wound had been managed with a foam dressing with a buffer bandage (Siliband) and crepe retention. The prescribed management was to use Suprasorb X + PHMB (Activa Healthcare, Burton-upon-Trent™), with a secondary adhesive foam dressing. Suprasorb X + PHMB was chosen to aid autolytic debridement while reducing the wound bacteria and protecting the tendon. The wound dressing provided a semi-occlusive, absorbent secondary dressing. No bandage was applied at this stage so that the cellulitis could be monitored. The dressings were changed on alternate days and the patient was monitored for clinical signs of infection.

Poor nutrition was also identified and she was referred to a dietitian for nutritional support. Nutritional supplements were advocated to support wound healing and her legs remained elevated in an attempt to alleviate the oedema.

Six days later, following three dressing changes there was significant wound progress. The wound dimensions were now 36mm and 6.5cm deep (Figure 2). Necrosis and slough had debried to reveal healthy granulation tissue (approximately 40% slough and 60% healthy wound bed). The periwound skin was healthy and, while the tendon remained exposed, it was hydrated and healthy. There was moderate exudate, no cellulitis and no malodour. The patient’s pain score had reduced to 3 and she was standing with assistance.

The nurses found the dressing easy to apply and the patient said that the dressing was comfortable and easy to remove. The patient continued the prescribed treatment with an added buffer bandage (Siliband) and crepe retention to aid venous return and improve the mild lower leg oedema. Compression therapy was not considered due to her unstable heart condition.

After 11 days of treatment there was extraordinary progress, particularly when considering the patient’s co-morbidities. There was little change to the wound’s dimensions, however, there was robust granulation tissue to the tendon and wound bed with no malodour, low exudate and no significant growth on wound culture (Figure 3). The oedema to the lower leg had also reduced after application of the support bandaging and increased mobility.

The patient clearly had an identified wound infection and was treated with systemic antibiotics accordingly. The wound was simultaneously autolytic debrided while controlling the surface microbes with Suprasorb X + PHMB. While this proved effective in protecting the tendon and promoting rapid granulation, we cannot attribute resolution of the infection solely to the antimicrobial dressing in this situation, but should consider it an adjunct to promoting healing in this complex wound.

Shortly after this, the patient was able to be transferred to a rehabilitation unit for a short stay before discharge home.

Figure 1. A debried wound following skin tear softening. It was infected and malodorous and contained necrosis and slough surrounding exposed tendon.

Figure 2. The wound following six days of treatment with Suprasorb X+PHMB. The wound now had no malodour and the patient’s pain levels had reduced.

Figure 3. The wound at final assessment with robust granulation tissue covering the tendon and surrounding wound bed.
TREATING A DEHISCED LAPAROTOMY WOUND

David Gray is Clinical Nurse Specialist, Department of Tissue Viability, Grampian Health Services, Aberdeen

The patient was a 58-year-old woman with a history of bowel disease who presented with a dehisced laparotomy wound following the formation of a stoma. The wound was 28.5x2cm wide and 3cm in depth, with the wound bed consisting of 30% sloughy tissue and 70% subcutaneous tissue. There was no infection evident and exudate was low in volume and viscosity (Figure 1).

She was immediately commenced on topical negative therapy (TNP) using AMD Gauze (Colidien, Mansfield MA). The dressing was changed every 48 hours.

She continued with this regimen for eight weeks. On review, the wound measured 26x2.5 and 1.5cm in depth and had a wound bed consisting of 100% granulation tissue. However, the wound appeared to be critically colonised as no progress had occurred for the preceding two weeks (Figure 2).

The patient was also being discharged home from convalescence care and did not feel able to deal with the TNP system at home as she lived by herself.

At this point the patient's wound was managed using Silverol ribbon (Systagenix, Crawley) secured with an Aiken Adhesive dressing (Coloplast, Peterborough) and was changed as exudate dictated.

The wound continued to be treated with topical antimicrobials for a number of weeks but started to show signs of improvement.

At the first treatment review after four weeks, the wound measured 13x2cm and was 1cm in depth. The tissue present was 100% granulation, no infection was evident, and the exudate was of medium volume and viscosity (Figure 3). As the silver dressing had failed to produce consistent improvement, it was decided to switch the primary dressing to Suprasorb X+PHMB (Activa Healthcare, Burton-upon-Trent).

The patient presented to the clinic two weeks later and the wound had reduced in size to measure 12x1.5cm and 0.5cm in depth (Figure 4). The wound bed consisted of 100% granulation tissue, exudate levels had increased and viscosity was low. There was no odour present and a wound smear confirmed the existence of Pseudomonas infection. Management with Suprasorb X+PHMB was continued but due to the increased exudate volume, dressing changes became daily rather than every 2–3 days as before. Wound swabs identified the presence of methicillin-resistant Staphylococcus aureus and Pseudomonas aeruginosa. No antibiotic therapy was commenced.

When the wound was reviewed a week later it had continued to improve and now measured 11x1.9cm and 0.5cm in depth (Figure 5). The wound remained granular, exudate had reduced in volume, and no evidence of P. aeruginosa or MRSA was present. It was decided to continue with the current treatment regimen as it was providing positive clinical outcomes; the wound was only colonised with MRSA and no infection or critical colonisation was evident.

The wound continues to be treated with Suprasorb X+PHMB and secured with Aiken adhesive as the secondary dressing. Dressing frequency was reduced to alternate days. The results from this case would suggest that the wound had stopped healing due to the presence of bacteria critically colonising the wound. Suprasorb X+PHMB appeared to kick start the wound into healing and was effective in managing the development of a local infection and facilitating healing.

The ongoing treatment with Suprasorb X+PHMB reclassified the wound, reducing the level of bacteria present and allowing the wound to continue to heal.

Figure 2. The granulation development and reduction in exudate indicate that the NPWT can be stopped.

Figure 3. Static, non-healing wound with evidence of previous healing in the form of reduction in depth and epithelium at one margin.

Figure 4. In this image the wound has a thin layer of slough across its surface.

Figure 1. Deep dehisced stoma site with underlying negative pressure wound therapy (NPWT) via the Ventur system (Vallance Medical).
TREATING A NON-HEALING LEG ULCER

David Gray is Clinical Nurse Specialist, Department of Tissue Viability, Grampian Health Services, Aberdeen

The patient was an 87-year-old woman who had a long-standing history of mixed ulceration in her left leg. Seven months before presentation the patient underwent an unsuccessful split skin graft and had suffered recurrent infection.

At first presentation the ulcer was located primarily on the inner aspect of the lower left leg and on the Achilles area extending to the outer aspect of the limb (Figure 1). The wound bed showed evidence of hypergranulation and was bleeding during the examination. Wound swabs had been previously taken revealed the presence of Streptococcus G and Pseudomonas. There was evidence of maceration on some of the wound margins and a medium volume of high viscosity exudate across the wound bed and dressing (Figure 2). The wound measured 18x12cm at its widest points.

In the four weeks before presentation the patient had been treated using a silver dressing which was changed every two days. The patient had found the dressing to be painful for the first few hours after application at every dressing change.

It was decided to switch to Suprasorb X+PHMB (Activa Healthcare, Burton upon Trent) with a 2-3cm overlap at the wound margins with a Mepilex (Mölnlycke, Gothenburg) dressing to cover and two to three blue line Tubifast (Mölnlycke) with Soft Ban to secure. This would provide an opportunity to deliver an antimicrobial in a form that suited the patient. This regimen was changed every two days and the leg washed in warm water with 50/50 ointment applied to the non-broken skin from the knee down.

After one week of treatment with Suprasorb X+PHMB the wound bed had begun to heal on the inner aspect with epithelium evident across the lower portion of the wound bed (Figure 4). Some hypergranulation remained on the outer aspect (Figure 5). Overall, the wound exudate had become low in volume and viscosity and an examination, the granulation tissue did not bleed. The wound was measured at its widest points and was found to be 10x10cm. The patient described no pain but a cooling effect when the dressing was applied.

This regimen was continued for a further three weeks during which time the wound continued to reduce in size to 5x5cm with no evidence of a recurrence of infection (Figures 6, 7 and 8).

CONCLUSION

While the patient was being treated with Suprasorb X+PHMB the wound changed from a non-healing critically colonised wound with hypergranulation which was bleeding when touched to a healing wound with healthy granulation and epithelialisation. The dressing reduced the wound biofilm and facilitated healing without causing the patient any pain.
A NON-HEALING SURGICAL WOUND IN A PATIENT RECEIVING CHEMOTHERAPY

Pam Cooper is a Clinical Nurse Specialist, Department of Tissue Viability, Grampian Health Services, Aberdeen

A 64-year-old man with a history of bowel cancer presented with a non-healing surgical wound. He had undergone extensive surgery for his cancer which led to a laparotomy wound. He also presented with large abdominal hernias which could not be operated upon due to his underlying condition. He was advised that because of the extent of surgery undertaken he would not be a candidate for further surgical intervention but would require ongoing chemotherapy.

The patient initially presented in April 2009 with a wound that measured 4.5x2.5cm and which undermined by 15cm around the circumference. On examination surgical mesh was evident from previous surgery, but buds of granulation tissue were forming over the mesh. The wound was colonised and there were medium volumes of low viscosity exudate.

The wound was initially treated with topical negative pressure (TNP) both in the hospital and by the district nurses in the community. Over a period of 10 weeks the wound improved significantly reducing in size to 5.5x5cm and 0.3cm in depth. No undermining was evident, there was 100% granulation tissue present with no infection and moderate volumes of low viscosity exudate.

The patient was then treated with a number of interventions including PolyMere® Foam (Apiron Medical, Redditch), Versiva® XC (Convatec, Ickenham) and Prisma (Sydenham Wound Management, Crawley) with the wound almost healing. By December 2009, the dimensions of the wound were 0.9x0.2cm. During this period the patient underwent a number of treatments of chemotherapy, with multiple blood transfusions. At this point he was discharged from the department of tissue viability.

The patient returned in April 2009 with a non-healing wound (Figure 1). He had been treated at home with Versiva XC dressing. The wound now measured 2.6x2.6cm with no depth and it was pale and fibrinous. No infection was evident and there was no exudate. The dressing was changed to Suprasorb X-PhMB (Activa Healthcare, Burton-upon-Trent) because its moisture-donating ability provided a moist wound healing environment coupled with antimicrobial properties to reduce bioburden, and was secured with Versiva XC. The patient felt comfortable with this secondary dressing as no surrounding skin issues had arisen while using it previously. Dressing changes were recommended every 3-4 days. He was also having fortnightly chemotherapy treatments.

During this time the patient went on holiday with his wife. He returned for review three weeks later and for more chemotherapy, reporting that dressing changes had taken place every 3-4 days. The wound measured 2.5x1.8cm with no depth, and was made up of 50% granulation tissue and 50% delayed fibrinous tissue (Figure 2). No infection was evident, and there was minimal exudate. The patient continued with the regimen and was reviewed two weeks later while admitted for chemotherapy. His wound measured 1.4x2cm with no depth, no infection and no exudate (Figure 3). At this stage, the wound bed consisted of 100% granulation tissue.

This patient’s wound had been non-healing due to his underlying cancer, ongoing chemotherapy and the presence of a large abdominal hernia. However, the wound is improving against all the odds with the wound bed covered in granulation tissue. The ongoing treatment of Suprasorb X-PhMB with a secondary dressing of Versiva XC continues and it is hoped that this regimen will result in the wound being fully healed.
CARE OF A PATIENT WITH A LARGE VENOUS LEG ULCER

Anna Coulbourne is a Tissue Viability Nurse and Cathie Bree-Aslan is Tissue Viability Consultant, Wound Healing Centre, Eastbourne

A 58-year-old woman was obese and presented with bilateral leg ulcers. The patient was using two sticks to mobilise but was still active. She owned a florist shop and spent many hours standing making bouquets but was able to rest at home with her legs elevated.

She had suffered with bilateral leg ulcers for many years and her wounds were particularly difficult to treat because she was unable to tolerate compression therapy and many dressings caused her extreme pain. Her wound was subsequently managed rather than treated.

Many different types of dressings and bandaging regimens had been previously used to help her wound to heal but few had been tolerated and often had to be removed within a short period of time. Maggot therapy had been tried at a cost of £300 for one application, but they were only in situ for four hours as the pain became intolerable. The patient was currently managed using a hydrofiber dressing with silver which was changed twice weekly. A system of bandaging with a layer of granufoam, unbleached bandage and a wound layer followed by reduced compression was also being applied, which the patient was able to tolerate.

On initial assessment, her wound was yellow, sloughy, extremely malodorous and producing large volumes of exudate (Figure 1). This had resulted in necrosis around the wound edge. The peri-wound area was over-hydrated but intact (Figure 2), suggesting colonisation of bacteria although there were no signs of clinical infection.

Doppler assessment showed the ankle brachial pressure index (ABPI) to be 0.9 in the right leg. The ankle measured over 25cm and the calf was large due to obesity. The patient was unable to tolerate full compression which had been attempted on many occasions but the pain had been too great. The aim was to reduce the pain through cleansing and healing the wound, so that compression would be tolerated.

Management of exudate was extremely important as the patient often stood for long periods of time and this increased the fluid loss, making her uncomfortable and embarrassed. Thus, to control the exudate levels in the wound, Aquacel® (Convatec, Ilkisonham) was applied as a primary dressing and covered using Fivascorb® (Active Healthcare, Bunravon-Tork), a highly absorbent dressing. After one week the hydrofiber dressing was changed to Suprasorb X +PHMB for a further week to control the colonisation of bacteria. The Fivascorb was then continued as the primary dressing and very successfully locked the exudate in its centre (Figure 4). Reduced compression was continued and the stockinette was placed over the Fivascorb.

On removal of the dressing after the first 24 hours, the Fivascorb appeared to have drawn the exudate away from the wound surface leaving it moist but not wet. Although there was strike-through when the hydrofiber and stockinette was placed under the Fivascorb (Figure 3), it can be clearly seen in Figure 4 that Fivascorb successfully retained the fluid in the centre of the dressing and there was no strike-through to the bandages.

Fivascorb and Suprasorb X +PHMB successfully controlled the exudate and were both acceptable to the patient who was freed from maceration. At no time did the nurse experience strike-through in this lady’s dressings while Fivascorb was used as a primary or secondary dressing.

The patient’s wound is unlikely to heal, nevertheless, the symptoms can be reduced by the use of appropriate dressings and this can increase the quality of life for the patient with a difficult and complex wound.

Figure 1. The wound is full of slough and extremely malodorous.
Figure 2. The surrounding tissue is slightly over-hydrated but the slough continues to lift and there are areas of granulation showing through. The wound is less malodorous because the dressing locks the bacteria away from the wound.
Figure 3. On removal of the patient’s bandaging, there was strike-through of exudate from the dressing onto the stockinette and primary dressing.
Figure 4. Fivascorb had absorbed the exudate and locked it away within the centre of the dressing.
Suprasorb® X + PHMB

For critically colonised & infected wounds

Powerful and gentle

- PHMB - powerful against bacteria, gentle with healthy cells
- Easy to apply, soft, supple and moulds to the shape of any wound
- wide antimicrobial spectrum - effective against MRSA & VRE
- reduces pain

How Polyhexanide (PHMB) works

PHMB interferes with the bacterial cell metabolism, inhibiting the bacteria, yeast and fungi cells ability to absorb any nutrients or dispose of waste products, effectively killing the bacteria without damaging surrounding healthy cells.

1. Supernatant is taken up from the wound into the dressing.
2. Microorganisms are killed by the released PHMB.
3. Moisture is released from the dressing into the wound.

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