

# Burn wound infection

**Infection is one of the commonest causes of death in burn patients, particularly those with extensive damage.**

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Infection remains a common cause of death in burn patients and is responsible for 75% of all deaths in patients with burns exceeding 40% total body surface area (TBSA). Current techniques of burn wound care and infection control measures have significantly reduced the incidence and mortality resulting from burn wound infection (BWI), changed the bacterial profile, and increased the time interval from injury to the onset of infection.<sup>1</sup> Additional factors associated with improved outcome of infection include early burn eschar excision and grafting.

**Burn wound infection causes a wound to progress from a partial thickness to a full-thickness wound.**

Thermal injury causes instant coagulative necrosis, which rapidly becomes a favourable niche for bacterial colonisation and proliferation. The eschar provides a devitalised, protein-rich environment, which further benefits bacterial proliferation through its exclusion from the systemic circulation and impaired local immune responses – hence the high susceptibility of the burn wound to infection.

Burn wound infection causes a wound to progress from a partial thickness to a full-thickness wound. There is the further possibility of systemic dissemination, especially if the intra-eschar organisms exceed more than 100 000 per gram of tissue. Infection also causes delay or non-healing of wound, gives rise to hypertrophic scars and substantially increases mortality. Burns covering less than 10% TBSA and of partial thickness carry the lowest risk of infection and mortality, but these increase as the percentage TBSA and depth of burn increase.

Other factors that are of importance include:

- the type of organisms

- their numbers
- virulence
- host resistance
- the quality of the wound, as well as
- the presence of devitalised tissue.

Microcolonies of bacteria produce biofilms enveloped within a self-produced matrix, or slime, which acts as an effective barrier against host defences and antimicrobials and are a source of recontamination. These biofilms can form within 10 - 72 hours.

The surface of a burn wound is free of micro-organisms immediately following a burn injury. Thereafter a complex and changing microbial ecology rapidly develops.<sup>2</sup> There is rapid colonisation by predominantly Gram-positive bacteria, which are harboured in the deep unburnt cutaneous structures. Over the ensuing 5 - 7 days,

**Table I. Frequently identified organisms responsible for major episodes of sepsis and mortality**

Gram-positive bacteria: 36% of cultures
<i>Staphylococcus</i> , including MRSA
<i>Streptococcus</i> and <i>Enterococcus</i>
Gram-negative bacteria: 51% of cultures
<i>Klebsiella</i> species
<i>E. coli</i> and <i>Proteus</i>
<i>Serratia marcescens</i>
<i>Acinetobacter</i> species
<i>Pseudomonas aeruginosa</i>
Fungus and viruses
<i>Candida</i> species and <i>C. tropicalis</i>
Herpes simplex virus, cytomegalovirus
Varicella zoster virus
Many of these organisms are multidrug-resistant

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other organisms, including Gram-negative bacteria, will gradually supersede the Gram-positive organisms. These pathogens originate from the upper respiratory, gastrointestinal and urogenital tracts or from the hospital environment. Gram-negative organisms are most common by virtue of their virulent factors and antimicrobial resistant traits (Table I).<sup>3</sup>

Fungal infections, especially *Candida* species, can be present in 30% of burns and are usually seen in the context of extensive burns complicated by delayed healing, following the use of broad-spectrum antibiotics, and in critically sick patients. Fungal infections significantly increase the risk of mortality for all percentages of burns. Viral infections are being recognised more frequently, but are often subclinical and seen in burns greater than 50% TBSA, with little or no effect on the acute course of the burn wound.

The indiscriminate use of antiseptics and antibiotics is largely responsible for increasing prevalence of resistant and opportunistic infections such as methicillin-resistant *Staphylococcus aureus* (MRSA), *Acinetobacter*, multi-resistant *Klebsiella* and *Pseudomonas*. The latter is associated with a mortality rate of up to 80%. Nosocomial infections, whether transmitted from environment to patient, patient to patient or staff to patient, can be very difficult to eradicate. Other frequently identified origins for infections are the respiratory tract, urinary system, indwelling devices and the bloodstream.

### Types of burn wound infections

The mere presence of bacteria in a burn wound does not constitute infection and contamination must therefore be clearly distinguished from invasive BWI. From a practical point of view, four types of

burn wound infections can be identified, which will determine the topical treatment modality, possible surgical procedures, systemic intervention and outcome (Table II).

### Risk factors for the development of burn wound infection

Various risk factors have been identified that may increase the prevalence of burn wound infection. These include:

- extremes of age
- burns exceeding 30% TBSA
- the depth of burn
- invasive devices
- prolonged open wounds
- systemic antibiotics
- lengthy hospitalisation
- blood transfusions
- number of days ventilated
- repeated exposure to hospital-acquired organisms, and
- co-morbidities (i.e. obesity, diabetes, immunosuppression, malnutrition, HIV).

### Diagnosing burn wound infection

The burn wound is not the only site for infection and it is important to exclude central line sepsis, septicaemia and pulmonary and urinary tract infections.

### Clinical signs of wound infection

It is imperative that burn wounds be inspected daily, searching for clinical signs of infection. Clinically the wounds have a macerated 'pussy' appearance with an exudative discharge. There may be rapid, purplish, haemorrhagic wound discolouration, deepening of the wound, friable or bleeding granulation tissue, eschar separation and

tissue necrosis, ecthyma gangrenosa and spreading peri-wound cellulitis. Systemic factors include progressive tachycardia and tachypnoea, haemodynamic instability, hyper- (>39°C) or hypothermia (<36.5°C), anaemia, thrombocytopenia, changes in the WBC, hyperglycaemia, ileus, abdominal distension, intolerance to enteral feeding, and mental confusion.

### Laboratory investigations

The purpose of these investigations is to identify the organism(s) (species and strain), quantify the bacterial burden (cfu/gram tissue) and determine the sensitivity and resistance patterns. The results facilitate selection of the appropriate topical therapy and monitoring of response. Many methods have been identified and it is important to select an optimal method(s) suitable to local circumstances.

### Surface swab cultures

This is the most commonly used technique for evaluating wound infections. These cultures allow identification of surface organisms, but cannot distinguish between contamination and invasive BWI. It is important to recognise that a swab or tissue biopsy only represents the bacterial profile at that specific site, and there may therefore be great variation in the number and types of organisms, either colonising or invading different areas of the wound, even if the wound has a uniform appearance. Bacterial identification is vital, and bacterial growth should be expressed as slight, moderate or heavy. Levine described a non-invasive quantitative swab technique which can diagnose infected burn wounds, with a linear numerical relationship between swab and biopsy counts.<sup>4</sup> This technique has been validated and should be the method of choice.

### Quantitative tissue cultures

These cultures are labour intensive but identify and analyse clinical infection more accurately than any surface swabbing technique. Three grams of tissue are required from multiple sites. This is then homogenised, serially diluted, plated out on agar plates and incubated for 24 - 48 hours. A bacterial count of more than 10<sup>5</sup> cfu/g tissue signifies a heavily infected eschar with impending systemic spread.

### Tissue histopathology (biopsy)

Multiple tissue biopsies can also be taken from suspected areas and sent for microbiology and histopathology. Gram-positive organisms are detected with H/E stains and the Sandiford stain is used for Gram-negative organisms. Similarly, special stains are used for suspected fungal infections in conjunction with fungal cultures and

Table II. Types of burn wound infection

- **Contamination:** Presence of non-multiplying bacteria, transient phenomena, wound healing not delayed
- **Colonisation:** Multiplying bacteria within the wound, with no host reaction or pathogenic effects
- **Burn wound infection:** Multiplying bacteria in the wound, but not deeply invasive, resulting in regional and systemic effects
- **Invasive infection:**
  - More than 10<sup>5</sup> cfu/g of tissue which on histology may show micro-invasion, deep invasion or micro-vascular and lymphatic involvement
  - Rapid change in burn wound appearance
  - Invasion into sub-eschar tissue and systemic spread consistent with sepsis
  - Suppurative separation of the eschar or graft loss
- **Necrotising infection/fasciitis** – an aggressive invasive infection with involvement of structures beneath the skin

electro-microscopy (EM) studies for viral infections. Bacteria identified on histology represent a heavy intra-eschar bacterial burden and this technique allows staging of the bacterial invasive process (Table III).

**Table III. Histological staging – bacterial depth of invasion**

Stage 1
<ul style="list-style-type: none"> <li>• Superficial – bacteria on the surface</li> <li>• Eschar penetration – variable depth</li> <li>• Sub-eschar penetration and proliferation</li> </ul>
Stage 2
<ul style="list-style-type: none"> <li>• Sub-eschar micro-invasion</li> <li>• Deep invasion into deeper tissues</li> <li>• Vascular and lymphatic involvement</li> </ul>

### Treatment – prophylactic and therapeutic

The presence of devitalised tissue is a major predisposing factor for persisting wound infection, and as such it is often impossible to eradicate deep-seated organisms with topical treatments alone. To prevent this, **early wound excision** is required – within the first few days following burn injury. Excision and immediate grafting also restores the barrier function of the skin, removes the inflammatory stimulus and decreases scar formation.

Other methods, such as **autolytic** or **enzymatic** and surgical debridement with low-pressure pulsatile irrigation techniques, are more suitable for chronic wounds and granulation tissue. Enzyme treatment should be combined with topical antimicrobial agents and the dressings changed regularly. These agents should be selected carefully, so as to avoid progression of a superficial

burn to full thickness, and avoid removal of non-devitalised tissue.<sup>5</sup> High-pressure lavage systems should not be used on grossly contaminated tissues, as bacteria may be translocated into the deeper tissues.<sup>6</sup> **Water-jet system** (Versajet) effectively debrides established, infected burn wounds, with the added advantage of controlling the depth of debridement.

Other adjunct measures are also effective. It is imperative that the burn wound be cleaned thoroughly at every dressing change with soap and tepid water, chlorhexidine washes and/or a **sodium hypochloride (NaOCl) solution**.<sup>7</sup> Wounds are soaked daily for 20 - 30 minutes in a tepid NaOCl 0.025%, buffered solution. Heggers' solution is an excellent cleansing and bactericidal agent against most Gram-positive and Gram-negative organisms (resistant *Pseudomonas* and *Klebsiella* infections and MRSA). It can be used in conjunction with other antibacterial agents. Nosocomial transfer of organisms residing in the burns unit often arises from a primary source, **health care professionals** and **immersion hydrotherapy** equipment.

Immersion hydrotherapy equipment is not favoured by modern burns units because it is often contaminated and very difficult to clean. **Showering with a hand-piece** is now the preferred choice for the cleansing and gentle debridement of burn wounds.

### Prophylactic systemic antibiotics

Antibiotics cannot prevent burn wound infection and should be used with caution. They have not been proven to improve outcome in comparison with topical agents. Indiscriminate use can promote the development of other infections and bacterial resistance. Systemic antibiotics as a prophylactic measure are only indicated for a short period peri-operatively and for confirmed systemic infections. However, tetanus prophylaxis is essential, as burns do pose a risk for tetanus infection.

### Antimicrobial therapy

The aim of topical antiseptics is twofold. Firstly, to prevent infection from becoming established and, secondly, to reduce the intra-eschar/sub-eschar bacterial burden to less than 10<sup>5</sup> cfu/g tissue, once the wound is infected, thus preventing invasive burn wound infection.

Surface therapy can be used either as a prophylaxis, or as a therapeutic measure, to reverse or control invasive BWI. For the effective use of topical antiseptic/antimicrobial agents (Table IV), the following factors are important:

- They are not universally effective and differ in their ability to penetrate the eschar, their period of bio- and antibacterial activity and side-effects.
- They cannot sterilise the burn wound.
- They can prevent the infective progression of a partial to a full-thickness burn.
- They are often used as an adjunct to early excision of the burn wound.
- Their individual pharmacokinetic properties must be taken into account for optimum usage.
- Antiseptics should routinely be rotated to reduce the risk of bacterial resistance.
- The bacterial profile of the wound will determine which topical agent is best suited.
- Some antiseptics can be used in combination to improve the bacterial spectrum, i.e. silver sulphadiazine and nystatin, mupirocin (Bactroban) and chlorhexidine.
- A closed dressing technique should be used to ensure continued contact between the agent and the burn wound.

### Infection control measures

Modern infection control measures are essential and include regular bacterial surveillance, individual isolation rooms

**Table IV. Profile of topical antimicrobials**

Agent	Application frequency	Eschar penetration	Action
Sodium hypochloride NaOCl 0.025%	Daily for 20 - 30 minutes	Surface action	Bactericidal to Gram (+) and (-) organisms including MRSA and <i>Pseudomonas</i>
Silver-sulphadiazine 1%	12-hourly liberal application	Limited	Bactericidal for many Gram (+) and (-) organisms including yeasts
Povidine iodine	6 hourly	Limited	Bactericidal, broad-spectrum bacterial and antifungal activity
Mupirocin	Daily	Excellent	Bactericidal, broad-spectrum bacterial and antifungal activity
Chlorhexidine	Daily		Bactericidal, especially against <i>Pseudomonas</i>
Nanocrystalline silver	3 - 4 days	Excellent	Bactericidal to more than 150 bacteria and yeasts

Topical agents can be used in combination to enhance their antibacterial and antifungal spectrum, i.e. silver-sulphadiazine with nystatin and mupirocin with chlorhexidine.

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for patients, universal aseptic techniques, rotation of antiseptics/topical antimicrobial agents and strict environmental control.

### Conclusion

Current techniques of burn wound care have reduced the prevalence of burn wound infection substantially. Prevention, early excision and grafting, identification and effective topical antiseptic strategies are critical for successful burn care. Topical antiseptics are used to prevent infection from becoming established and reduce the bacterial burden in the wound. Systemic antimicrobials do not have a significant

role in the general management of BWI and must be used discerningly, where indicated. However, the primary focus remains to minimise exposure to infection and therefore its consequences.

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### In a nutshell

- Infection remains a common cause of death in burn patients and is responsible for 75% of all deaths in patients with burns exceeding 40% total body surface area (TBSA).
- Thermal injury causes instant coagulative necrosis, which rapidly becomes a favourable niche for bacterial colonisation and proliferation.
- Burn wound infection causes a wound to progress from a partial thickness to a full-thickness wound.
- Microcolonies of bacteria produce biofilms enveloped within a self-produced matrix, or slime, which acts as an effective barrier against host defences and antimicrobials and is a source of recontamination. These biofilms can form within 10 - 72 hours.
- The mere presence of bacteria in a burn wound does not constitute infection and therefore contamination must be clearly distinguished from invasive BWI.
- Burn wounds should be inspected daily for signs of infection.
- Laboratory investigations are used to identify the bacterial organism and to quantify the bacterial burden.
- A non-invasive quantitative swab technique is the method of choice for diagnosing infected burn wounds, with a linear numerical relationship between swab and biopsy counts.
- Multiple tissue biopsies can also be taken from suspected areas and sent for microbiology and histopathology.
- Early wound excision is needed to prevent the build-up of devitalised tissue, which is a major factor predisposing to burn wound infection.
- Other methods, such as **autolytic** or **enzymatic** and surgical debridement with low-pressure pulsatile irrigation techniques, are more suitable for chronic wounds and granulation tissue.