Infection in burn wound

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- Infection remains the most common cause of morbidity and mortality in burn patients.
- The diagnosis and management of burn wound infection remains challenging due to the many physiologic features unique to burn injury.

EPIDEMIOLOGY AND RISK FACTORS

- — The incidence of burn wound sepsis has declined from 6 to 1 percent since the practice of early burn wound excision; however, for patients with total body surface area (TBSA) burns >15 percent, the rate has remained the same .
- The highest rates of surgical burn wound infections occur in the lower extremities, but specific organisms or classes of organisms are not confined to one particular anatomic location .
- Patients with burn wounds also have high rates of other types of infection, such as catheter-related infection .
- In a series of 175 patients with severe burns, infections preceded multiorgan dysfunction in 83 percent of patients and were considered the direct cause of death in 36 percent of those who died .

Risk factors :

- — A variety of factors increase the risk of developing invasive burn wound infection (burn wound sepsis).
- TBSA burn >20 percent (however, burn wound infection and sepsis can occur in smaller burns)
- Delays in burn wound excision
- Extremes in age (very old, very young)

  and
- Impaired immunity
- Microbial factors (such as type and number of organisms, enzyme and toxin production, and motility)

- In the management of burn wounds, topical therapy is associated with a higher incidence of burn wound infection and graft loss compared with early excision and grafting.
- Delaying burn wound excision increases bacterial loads and gram-negative colonization.
- When bacterial counts exceed 105 organisms per gram of tissue in the burn eschar, wounds should be considered at risk for developing invasive burn wound infections, even when the wounds are excised.
- Despite the ability of burn wound excision to decrease bacterial counts, burn wounds with high counts are at risk of developing burn wound sepsis both before and after surgery .

- Burned patients lose their protective primary barrier (skin) to environmental microorganisms.
- The burn also consists of avascular necrotic tissue (eschar) that provides a protein-rich environment favorable to microbial colonization and proliferation.
- The avascularity of the eschar impairs migration of host immune cells and restricts delivery of systemically administered antimicrobial agents, while toxic substances released by eschar tissue impair local host immune responses

MICROBIOLOGY

- — The spectrum of microorganisms causing infections in burn patients varies with time and location .

- The organisms causing burn wound infection typically appear at varying stages post-burn-injury .

MICROBIOLOGY

- Immediately after burning, the microbial population of the burn wound is sparse and includes predominantly gram-positive bacteria that survived the thermal insult, such as staphylococci located deep within sweat glands and hair follicles.
- Within the first week post-burn, burn wounds are colonized with other microbes, such as gram-positive bacteria, gram-negative bacteria, and yeasts derived from the patient's normal gastrointestinal or upper respiratory tract flora, and from the hospital environment.
- The predominant gram-positive organisms found in burn wound infections remain Staphylococcus aureus, followed by Enterococcus species, which are found in decreasing numbers .
- Gram-negative pathogens dominate after the fifth day of a typically protracted in-hospital stay and have emerged as the most common etiologic agents of invasive infection by virtue of their large repertoire of virulence factors and antimicrobial resistance traits .
- Pseudomonas aeruginosa remains the most frequent gram-negative microorganism isolated from burn wounds, followed by Escherichia coli

Fungi (Candida, Aspergillus, Fusarium, Mucor species) and multiresistant organism (MRSA, VRE, Acinetobacter) appear late in chronological appearance and typically occur after use of broad-spectrum antibiotics and/or a prolonged hospital stay .

- Candida spp is the most common fungus isolated from burn wounds and the fourth most common cause of burn wound infections overall, and herpes simplex virus type 1 (HSV-1) remains the most common viral organism in burn wounds .

- Infections with MRSA, Stenotrophomonas maltophilia, and Acinetobacter spp have become more frequent and increasingly more difficult to treat as these species are becoming more often resistant to the antibiotics traditionally used to treat them.

CLINICAL FEATURES

- — A rapid change in the clinical condition of the burn patient may indicate that burn wound infection, and potentially burn wound sepsis, is present.
- Patients with invasive burn wound infections (burn wound sepsis) may have fever, other signs of sepsis, changes in wound appearance (purulent drainage, erythema, tenderness), or increased pain.
- Patients who had previously tolerated enteral feedings may show signs of intolerance (eg, abdominal distention, increase in residual volume, uncontrollable diarrhea).

Physical examination :

- — Burn wound infections are most often recognized based upon:

- the gross appearance of the burn and/or skin graft donor site
- and
- Alterations in clinical measurements (vital signs, abdominal distention).
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Early diagnosis and treatment

- The most common clinical feature is a rapid change in the appearance of the wound, which may include conversion of a partial-thickness injury to full-thickness injury, or loss of previously viable tissue or skin graft.
- Acute bacterial infection manifests with the development of discoloration, pain, purulent exudate, edema, tenderness, swelling, drainage, or malodor from a burn or burn-related wound (previously reepithelialized grafted burn site, skin donor site).
- The appearance of infection may involve only a portion of the burn wound.
- A surrounding cellulitis can occur and is characterized by erythema involving uninjured skin and may also exhibit localized pain and tenderness, swelling, and warmth.
- Peri-burn cellulitis may be indicative of infection beyond the borders of the burn wound and into normal tissue and signals the possibility of invasive infection.

- Local signs and characteristics of burn wound infections caused by fungi include unexpectedly rapid separation of the eschar, presumably due to fat liquefaction, and rapid spread of subcutaneous edema with central ischemic necrosis .
- Vesicular lesions that appear in healing or healed second-degree burns and the presence of crusted serrated margins of partial-thickness burns of the face, particularly those involving the nasolabial area, are characteristic of burn wound infections caused by herpes simplex virus type 1 (HSV-1) .

Other characteristics of invasive burn wound infection include:

- ●Edema and/or violaceous discoloration at the margin of the burn and/or unburned skin
- ●Hemorrhagic discoloration of subeschar tissue
- ●Separation or discoloration of the burn eschar
- ●Presence of green pigment (pyocyanin) in subcutaneous fat (indicative of Pseudomonas infection)
- ●Presence of initially erythematous and later black necrotic nodular lesions (ecthyma gangrenosom) in adjacent unburned skin.
- ●Exophthalmos may be the first sign of mucormycosis in midface burns (retrobulbar space involvement)

- ●Temperature >39°C or <36.5°C
- ●Progressive tachycardia (eg, adults >90 beats per minute; children >2 standard deviations [SD] above age-specific normal value)
- ●Progressive tachypnea (eg, adults >30 breaths per minute; children >2 SD above age-specific normal values)
- ●Refractory hypotension (eg, adults: systolic blood pressure <90 mmHg or a decrease >40 mmHg, or mean arterial pressure <70 mmHg; children <2 SD below normal)

Laboratory flaboratory alterations in blood tests

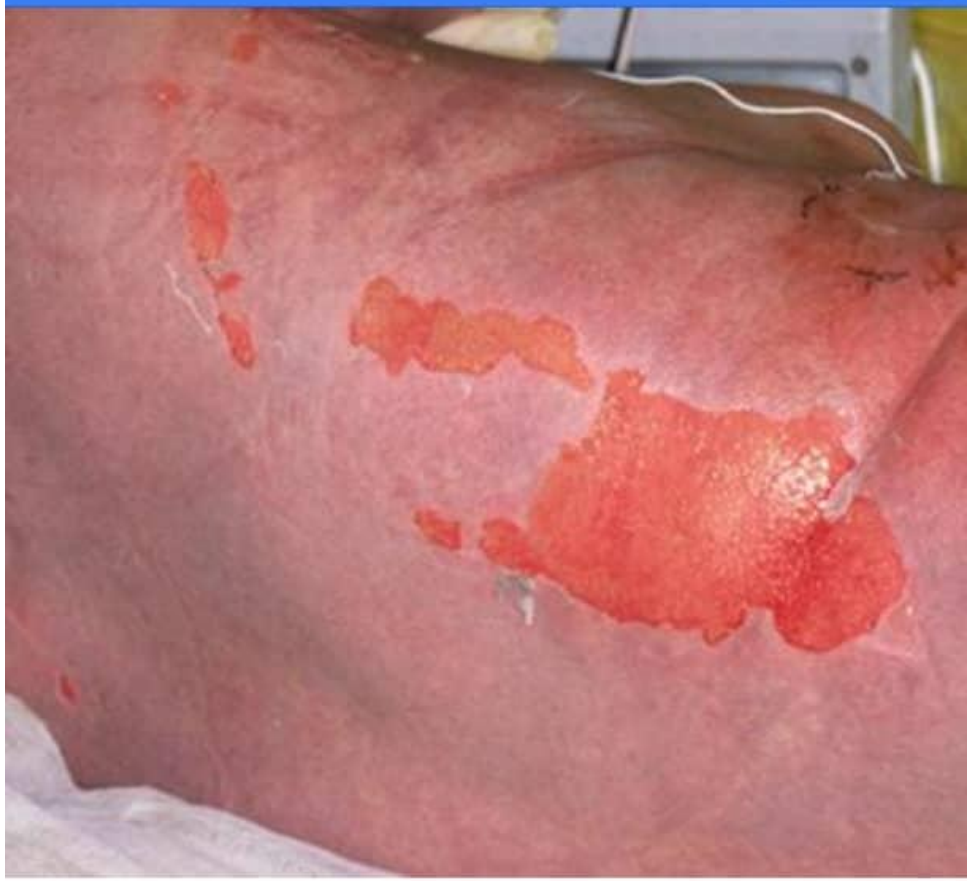
- ●Glucose – Fasting serum blood glucose levels >110 mg/dL (6.1 mmol/L) in the absence of preexisting diabetes mellitus.
- ●White blood count – Leukocytosis (>12,000 cells/microL, adults; >2 SD above the normal level for children (table 3); or leukocytopenia <4000 cells/microL).
- ●Platelet count – Platelet count <100,000/microL (adults), <2 SD below the normal level for children.

Procalcitonin :

- For patients with severe burns, daily measurements of serum procalcitonin (PCT) may be useful for monitoring the effectiveness of antibiotic therapy. The value of serum PCT concentration for differentiating bacterial infection from other causes of postoperative fever has been explored in patients with burn wound sepsis.
- However, postoperative PCT concentrations are quite variable, particularly in postoperative patients, and as a result, a specific threshold value is difficult to determine .
- In a systematic review that identified 566 PCT samples among burn patients in nine trials, cutoff values above which a positive result was defined ranged from 0.53 to 3 ng/mL. For similar cutoff values, the sensitivity and specificity of PCT also varied widely .

Burn wound categories

- Wound colonization
- Noninvasive infection
- Medication-related mucocutaneous reactions(TEN,SJS)
- Hypermetabolic response to thermal burn



Other cause of sepsis

- catheter-related infection
- urinary infection
- Pneumonia
- others in those with combined burn trauma injury)

Treatment:

- Burn wound care (ie, cleansing, dressings)
- Antimicrobial therapy (topical with or without systemic agents)
- and
- Burn wound excision or debridement
- For patients found to have only wound colonization (no burn wound infection), treatment is conservative and aimed at preventing infection.

Noninvasive burn wound infection

- treatment consists primarily of topical antimicrobial therapy and burn wound excision for unexcised wounds, and possibly reexcision for excised wounds.
- If there is significant peri-burn wound erythema (even in the absence of invasive infection and/or empirically while awaiting burn wound cultures and histopathology), we initiate treatment with intravenous cefazolin or clindamycin, or vancomycin if there is suspicion for MRSA, with or without an oral fluoroquinolone for burns involving the lower extremity or feet or burns in patients with diabetes.
- Burn wound impetigo is also treated using systemic antimicrobial therapy and possibly topical glucocorticoids .
- Contact precautions should be used until 24 hours after the start of antibiotic therapy to avoid spread of impetigo.

Antimicrobial therapy:

- Antibiotic choices depend upon the antibiogram of the individual institution.
- initiate empiric antimicrobial therapy with piperacillin/tazobactam or carbapenem, +/- vancomycin if there is suspicion for methicillin-resistant S. aureus (MRSA), +/- an aminoglycoside if there is suspicion for multidrug-resistant (MDR) P. aeruginosa.
- A suspicion for MRSA or MDR organisms is based on the typical flora found in each particular burn center.
- In addition, other patient factors such as the length of hospitalization, recent prior hospitalization, institutional status, immunocompromise, and use of empiric broad-spectrum antibiotics increase suspicion.
- The duration of therapy is based upon clinical response.

- Specific antimicrobial therapy is guided by the results of burn wound culture and histopathology.
- Whenever possible, antimicrobial therapy should be directed toward the specific organisms isolated from the wound, blood, and/or urine cultures.
- When systemic antimicrobial therapy is needed, awareness should be heightened for the possibility of super-infection with resistant organisms, yeasts, or fungi.
- Phycomycotic infections benefit from both topical and systemic therapy.
- However, systemic antifungal or antiviral agents should only be administered based on a proven diagnosis.

Diagnosis and treatment of burn wound infection and sepsis