Burns are one of the most common and devastating forms of trauma. Burn wounds are especially prone to infection. Thermal destruction of the skin barrier and concomitant depression of local and systemic host cellular and humoral immune responses are pivotal factors contributing to infectious complications in patients with severe burns. The burn wound surface provides a protein-rich environment consisting of avascular necrotic tissue (eschar) that provides a favourable niche for microbial colonization and proliferation. The avascularity of the eschar results in impaired migration of host immune cells and restricts delivery of systemically administered antimicrobial agents to the area, while toxic substances released by eschar tissue impair local host immune responses.

Patients with serious thermal injury require immediate specialized care in order to minimize morbidity and mortality. The survival rates for burn patients have improved substantially in the past few decades due to advances in modern medical care in specialized burn centres. In patients with severe burns over more than 40 per cent of the total body surface area (TBSA), 75 per cent of all deaths are currently related to sepsis from burn wound infection or other infection complications and/or inhalation injury.

Although burn wound surfaces are sterile immediately following thermal injury, these wounds eventually become colonized with microorganisms. Wound colonization by yeasts and fungi usually occurs later due to the use of broad-spectrum antibiotic therapy. Microorganisms transmitted from the hospital environment tend to be more resistant to antimicrobial agents than those originating from the patient’s normal flora.

Prior to the antibiotic era, Streptococcus pyogenes (group A beta-haemolytic streptococci) was the predominant pathogen implicated in burn wound infections and was a major cause of death in severely burned patients. Staphylococcus aureus became the principal aetiological agent of burn wound infections shortly after the introduction of penicillin G in the early 1950s, which resulted in the virtual elimination of S. pyogenes as a cause of infection in thermally injured patients. Although S. aureus remains a common cause of early burn wound infection, Pseudomonas aeruginosa from the patient’s endogenous gastrointestinal flora and/or an environmental source is the most common cause of burn wound infections in many centers as was also found in the study by Ekrami & Kalanter in this issue. Many centres from India have also reported the same.

The emergence worldwide of antimicrobial resistance among a wide variety of human bacterial and fungal burn wound pathogens, particularly nosocomial isolates, limits the available therapeutic options for effective treatment of burn wound infections. MRSA, methicillin-resistant coagulase-negative staphylococci, vancomycin-resistant enterococci, and multiply resistant Gram-negative bacteria that possess several types of beta-lactamases, including extended spectrum beta-lactamases (ESBL), ampC beta-lactamases, and metallobeta-lactamases (MBL), have been emerging as serious pathogens in hospitalized patients. The study by Ekrami & Kalanter highlights the high levels of antimicrobial resistance wherein they found P. aeruginosa to be 100 per cent resistant to amikacin, gentamicin, carbenicillin and ciprofloxacin. Fifty eight per cent of S. aureus and 60 per cent of coagulase negative Staphylococci were methicillin resistant in this study. In a prospective study conducted at the Burns unit at Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh a tertiary care centre in north India, S. aureus and P. aeruginosa were the most frequent organisms causing wound as well as blood stream
infection. Ninety per cent of *P. aeruginosa* were resistant to amikacin and ceftazidime, 45 per cent to ciprofloxacin and 25 per cent to piperacillin, 43 per cent *S. aureus* were MRSA.

Modern burn centres are designed to minimize the unnecessary traffic of health care workers and visitors alike through the unit. Cross-contamination is further diminished within the unit by housing burn patients in individual nursing units composed of individual isolation rooms, each with its own laminar airflow. Modern burn unit designs should allow all intensive and burn care procedures, including ventilation and operative procedures, to be done within the burn centre itself, or, as a minimum, the facility design should minimize the need to transfer patients out of the burn unit for different aspects of their care. A study showed that the rate of cross-colonization with resistant organisms in 66 critically ill children with severe burns and inhalation injury on ventilator support during a 5 yr period was extremely low (3.2 cases per 1,000 patient-days) in such a facility. Such centres are the need of the day to control burn wound infections in our country.

An effective infection control policy is very much required to reduce or eliminate endemic pathogenic and/or antibiotic resistant organisms, prevent the establishment of antibiotic-resistant organisms as the predominant nosocomial flora of the burn unit, and prevent cross-contamination.

The infection control programme for burn centres requires strict compliance with a number of environmental control measures that include strictly enforced hand washing and the universal use of personal protective equipment (*i.e.*, gowns, gloves, and masks). Health care personnel must be gowned (including use of disposable or reusable gowns and disposable plastic aprons to prevent soiling of health care workers’ clothing during wound care procedures) and gloved at each entry to the burn patient’s isolation room. Monitoring and diagnostic equipment is housed in each burn patient’s room to prevent cross-contamination between patients. All equipment in the isolation room must be regularly cleaned with appropriate disinfectants. Procedures that may predispose burn patients to cross-contamination, such as exposure hydrotherapy, are to be kept to a minimum. Overcrowding, inadequate sterilization and disinfection practices, gross contamination of the environment, lack of isolation facilities, inadequate hand washing and barrier nursing are some of the reasons for high infection and sepsis rates in burn centres of resource poor settings in many developing countries.

Burn wound infections should be rigorously monitored according to the standard definitions in order to generate accurate epidemiological data about infection rates. Routine surveillance should also be carried out for other types of nosocomial infections commonly diagnosed in burn patients, including catheter-related infections, pneumonia, and urinary tract infections. In all cases, published standard definitions should be used in identifying these types of infection complications. National Nosocomial Surveillance system definitions (NNIS) developed by the Centers for Disease Control and Prevention (CDC), Atlanta are very useful in this regard as used in this study also.

Laboratory surveillance cultures (*e.g.*, culture of nasal, rectal, or groin swabs for MRSA and culture of rectal swabs for vancomycin-resistant enterococci) as well as routine microbial surveillance cultures of the burn wound and other sources (*i.e.*, blood, respiratory, and urine samples) should be monitored to rapidly identify epidemic pathogens and/or antibiotic-resistant strains so that control measures can be immediately implemented. Antibiotic utilization should be rotated or changed based on monitoring of antibiotic resistance trends (antibiograms) within individual burn centres. Finally, adverse outcomes, including morbidity and mortality due to burn wound infection, sepsis, or another nosocomial infection complication, should be monitored in burn patients according to the extent of burn injury in order to assess the effectiveness of existing infection control practices within the centre’s modern burn therapy programme.

Infection control programmes need to document and report burn wound infections according to the recently established definitions of the classification system. Future studies of burn wound infections should use this standardized burn wound classification system so that clinical outcomes can be compared for burn patients with a specific condition (*e.g.*, burn wound cellulitis). More research is required to determine the best methods for sampling excised and unexcised burn wound areas over the course of a severe deep partial-thickness and/or full-thickness injury. Reproducible standardized methods should be developed so that clinical microbiology laboratories can routinely test burn wound bacterial isolates for susceptibility to the
topical antimicrobial agents on formulary at a particular burn centre. A strict antibiotic policy should be followed by all centres. A rotation programme for topical antimicrobial use may also retard the development of resistance. Laboratory surveillance should include the reporting of burn unit-specific antibiograms for topical antimicrobial agents once standardized methods are available for performing susceptibility testing.

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