Review

Burn and scald injuries

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الحروق والإصابات السمطية

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الخلاصة: تُعَدُّ الحروق أحد أكثر الإصابات إضراراً من الناحية البدنية والنفسية. وتمثِّل العدوى السبب الرئيسي للمراضة والوفيات التي تحدث في حالات الحروق. وتكون للعداوى المكتسبة من داخل المستشفيات أو من النَّبيت الجرثومي الداخلي المنشأ من المريض نفسه، ذات انتشار يُعْتَدُّ به إحصائياً عقب الإصابة بالحروق. وقد وُجد أن الزائفة الزنجارية *P. aeruginosa* والعنقودية الذهبية *S. aureus*، هي أكثر العوامل المنتجة للمستعمرات تواتراً، في حين أن العقديات الحالَّة للدم بيتا، من النمط "أ"، هي أكثر أنواع الجراثيم المفوَّعة وجوداً. كما تنتشر أيضاً الجراثيم اللاهوائية والفطريات. أما العدوى الفَيْروسية، فهي أقلُّ حدوثاً. ثم إن إجراءات الإنعاش القوي والنشط، والدعم الغذائي، وجودة الاستئصال الجراحي للجروح المصابة بالعدوى، والإغلاق المبكر للجروح، والترقيع، وإعطاء علاج كيميائي موضعي وجهازي مؤثر وفعَّال، تسهم بشكل كبير في تحسين معدلات المراضة والوفيات لدى المرضى المصابين بالحروق.

SUMMARY Burns are one of the most harmful physical and psychological traumas. Infection is the major cause of morbidity and mortality in burns. Infections acquired from hospital or from the patient's own endogenous flora have a significant prevalence after burns. *Pseudomonas aeruginosa* and *Staphylococcus aureus* are the most frequent colonizing agents whereas group A beta-haemolytic streptococci are the most virulent bacteria. Anaerobic bacteria and fungi are also prevalent. Viral infection is less frequent. Aggressive resuscitation, nutritional support, thorough surgical excision of infected wounds, early wound closure, grafting and the development of effective topical and systemic chemotherapy have largely improved morbidity and mortality rates of burn patients.

Brûlures par liquide bouillant et autres

RÉSUMÉ Les brûlures constituent l'un des traumatismes physiques et psychologiques les plus dommageables. L'infection est la cause majeure de morbidité et de mortalité chez les brûlés. Les infections contractées à l'hôpital ou provenant de la flore endogène du patient après des brûlures ont une prévalence non négligeable. *Pseudomonas aeruginosa* et *Staphylococcus aureus* sont les agents colonisants les plus fréquents, les streptocoques bêta-hémolytiques du groupe A étant les bactéries les plus virulentes. Les bactéries anaérobies et les champignons sont également courants. L'infection virale est moins fréquente. La réanimation agressive, la prise en charge nutritionnelle, l'excision chirurgicale complète des plaies infectées, la fermeture rapide des plaies, les greffes et la mise au point d'une chimiothérapie locale et systémique efficace ont permis d'améliorer grandement le taux de morbidité et de mortalité chez les patients brûlés.

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Introduction

The skin is the largest organ of the body. It functions as the first line of defence protecting against the invasion of foreign bodies and organisms. It has specific immune and metabolic functions and is important in regulating body temperature, fluid and electrolytes. Loss of the functional skin barrier after thermal injury results in increased susceptibility to infection, which is the major cause of morbidity and mortality following burns. In addition, factors such as extent and depth of injury, patient age, associated conditions and the presence of inhalation injury can adversely affect clinical outcomes [*1*].

Burns are one of the most harmful and complex physical injuries [*2*]. They often happen unexpectedly and have the potential to cause death, lifelong disfigurement and dysfunction [*3*]. It is also commonly assumed that hospitalized patients for burn treatment will experience some level of depression. It has been found that 1 month after hospital discharge, 54% of patients showed symptoms of moderate to severe depression and 2 years after discharge, 43% of patients still reported moderate to severe depression. Women had higher depression scores than men in both cases [*4*].

It is important to ascertain the cause of the burn because this may be helpful in determining burn depth. Scalds are the most common cause of thermal injury in children. They commonly occur in the kitchen or bathroom, are usually due to brief contact with hot water, and are usually partial-thickness in nature. Tar, grease, or contact burns typically result in deep-partial or full-thickness injury owing to their higher temperature and longer cutaneous exposure. Flame burns may be of variable depth depending on the patient's clothing and level of consciousness at the time of injury, and are often associated with smoke inhalation injury. Electrical burns may be associated with cardiac arrhythmias, neurological damage and significant long and short-term morbidity. Burns caused by household current rarely involve tissues beneath the skin; high voltage (> 1000 volts) exposure may, however, cause damage to deeper tissues such as muscle, nerves, blood vessels and bone despite the absence of a major cutaneous injury. Limb loss is not an infrequent consequence of this type of injury [*1*].

Bacterial colonization and invasive bacterial infection are still major problems in the treatment of burn victims. The standard procedures for bacterial monitoring of the burn are swab-culture, which is non-invasive but only detects bacteria at the very surface; biopsy-culture, which gives a more complete view but has the disadvantage of being invasive; and a new technique of dermabrasion of the upper layers of the wound, which is performed using a small rotating carbon-steel disc of defined roughness [*5*]. This procedure is superior to the swab culture in identifying different bacterial species. It can be compared with the biopsy technique, but has the advantage of being less invasive. Nevertheless, several investigators have found that the ratios of various species of organisms from the surface burn wounds were roughly proportional to those from blood specimens or from biopsy cultures [*6,7*]. Because of this, in most centres surface microbial growth is routinely monitored, enabling evaluation of the effect of therapy and prediction of those microbial strains that may become involved in sepsis.

The development of effective topical chemotherapy, prompt burn wound excision, timely closure of burn wounds and the use of biological dressings have significantly decreased the incidence of invasive burn wound infection and have contributed

to the improvement in survival that has occurred over the past 4 decades [*8,9*]. This review discusses the recent advances in burn wound care.

Resources

The Medline database, Biomedical Reference Collection: Comprehensive, and the Index Medicus for the World Health Organization, Eastern Mediterranean Region were searched through 2002 using the keywords "burn, burn wound infection, burn injuries". The type of search used was "standard and all words" and was limited to human studies. Some other related references were manually searched.

Pathophysiology

Tissue loss following thermal injury is a consequence of coagulation necrosis. The depth of injury is related to heat of exposure and tissue conductance. The classic pathologic description considers the burn wound as having 3 concentric zones [*10*]. The zone of coagulation represents the area of most intense thermal injury. It is surrounded by a zone of stasis, or ischaemia, which may or may not survive. The outer layer, or zone of hyperaemia, is the least injured area and may confuse the inexperienced caregiver into thinking that cellulitis is present. This hyperaemia typically resolves within 7 to 10 days post-injury.

The injury process remains dynamic during the 24 to 48 hours after the burning process has been arrested. Capillary occlusion can progress during this time and supports the clinical impression that skin necrosis progresses during the first 48 hours post-burn. This phenomenon is not considered a continuation of the burning process but a pathophysiological event that may be the consequence of tissue oedema, dermal ischaemia or desiccation. This occurrence is clinically very important. If the zone of coagulation is above the level of the dermal appendages, spontaneous healing is expected; if this zone extends below this level, however, a deep, partial or full-thickness wound results and skin grafting is usually required [*1*].

Burns covering more than 10% of the total body surface area are responsible for systemic complications which in very severe cases can represent a vital risk and in all cases affect the wound evolution. Among these general complications, fluid volume and electrolyte changes, leading eventually to burn shock, have the most dramatic consequences. Burn shock is still today a vital risk and can also, in the case of inadequate early fluid resuscitation, results in secondary morbidity and mortality. Fluid replacement during the very first hours after injury is key to the management of severe burn cases [*11*]. Major burn injury is a lesion where the inflammatory reaction is exported to the whole body. After a short period of haemodynamic changes, the inflammation is sustained by necrotic tissues, persistence of an open wound and the pulmonary and gut reactions. When infection starts, it becomes difficult to distinguish its symptoms among the inflammatory signals [*12*].

The burn patient is highly susceptible to infection due to the loss of the skin as a barrier to microorganisms. Immune defences are activated in response to the burn injury; however, some of these defences are altered. Neutrophil chemotaxis is compromised by decreased perfusion caused by hypovolaemia and the formation of microthrombi. Chemotaxis and phagocytosis are dependent on complement components that are reduced in a large burn wound. Neutrophil intracellular killing power is reduced as oxygen delivery to the wound is decreased.

Humoral immunity is altered with the drop in IgG levels. Cell-mediated immunity is depressed and T-cell lymphocyte counts are decreased. Suppressor T-cells are generated.

Specific sources of infection for the burn patient include the patient's own bacterial flora, hospital personnel, respiratory equipment and catheters, both urinary and intravascular [*13*]. It has been shown in children that in extensive burn wounds, bacterial antigens may not be recognized properly owing to the decreased proportion of CD4 cells and increased proportion of CD8 cells, which enhances bacterial growth in these wounds [*14*]. Patients with severe burns have a very high metabolic rate, which can lead to a deep nutritional deficit and immunological suppression. It is then of major importance to provide adequate nutritional support [*11*].

Burn injuries, by their very nature, tend to produce further ischaemia and infection. Both these factors may mean that what is initially considered superficial damage may ultimately affect deeper levels [*15*]. The mechanism of post-burn pyrexia is not completely understood. Proposed mechanisms include dysfunction of the thermoregulatory system, increased metabolic rate that produces a hypermetabolic state, production of cytokines due to tissue injury, release of endogenous pyrogens and excessive release of endotoxins from the gut or wound [*16–18*]. The most common cause of death in burn patients is multiple organ failure, despite the clinical absence of uncontrolled infection at the time of death [*19*].

The post-traumatic response to burn injury leads to marked and prolonged skeletal muscle catabolism and weakness, which persist despite standard occupational and physical therapy rehabilitation programmes. However, the researchers found that the participation of children with thermal injury in a resistance exercise programme resulted in a significant increase in muscle strength and power and lean body mass relative to a standard rehabilitation programme without exercise [*20*].

Burn wound assessment

Extent

The "rule of nines" is a rough method of estimating body surface area, assuming adult body proportions. The head and neck are roughly 9%, the anterior and posterior chest are 9% each, the anterior and posterior abdomen (including buttocks) are 9% each, each upper extremity is 9%, each thigh is 9%, each leg and foot is 9%, and the remaining 1% represents the genitals. The palmar surface of the hand (excluding the fingers) is approximately 0.5% of body surface area over all age groups [*21*].

Depth

First degree

This type of burn damages only the outer layer of skin (epidermis), which is composed entirely of epithelial cells. These burns are pink or red, dry and painful, sloughing the next day. The skin does not blister, although slight swelling may occur.

Second degree

This injury damages epidermis and a small portion of the underlying dermis, which contains blood vessels, nerve endings, sweat glands, hair follicles, and sebaceous glands. This is also where new skin cells are produced. Blisters are common with this type of burn. This burn blanches slowly and capillary refill is slow. These are red, wet and very painful.

Third degree

This burn completely destroys both epidermis and dermis. The skin is dry, leathery in

consistency, and firm, and can look white, red, brown or black. The burn does not blanch when pressed, is insensitive to touch and looks waxy.

Fourth degree
These wounds involve underlying subcutaneous tissue, tendon or bone. Such burns frequently have a charred appearance [*2,21*].

Causative agents of burn wound infection

The usual cause of burn infection is bacteria, and less frequently fungi or yeasts. Viruses can also cause infection on rare occasions. The surface of every burn wound is contaminated to some degree by bacteria [*22*]. Burn wound infection is, however, defined as burn wound bacterial proliferation at a density $\geq 10^5$ bacteria/g tissue [*23*]. Of the deaths that do occur in adult burn patients, 50%–75% result from infection [*24*]. In previous studies, the most common colonizing organisms were *Pseudomonas aeruginosa, Staphylococcus aureus, Klebsiella* spp., *Proteus* spp., *Escerichia coli*, and other Enterobacteriaceae [*23,25–32*].

Historically, group A beta-haemolytic streptococcal burn wound infection has been a major source of morbidity and mortality in burn patients, and has prompted the prophylactic administration of antibiotics to patients with burns. These infections in burn patients result in severe cellulitis and sepsis. Recent studies have suggested that routine penicillin prophylaxis of burn wounds was not effective in further reducing the incidence of wound infection involving group A beta-haemolytic streptococci [*33,34*]. The L-form of *S. aureus* occurring in burn wounds has been reported where the infection was attributed to a decrease in host immunological function and repeated administration of antibiotics [*35*]. Anaerobic bacteria were isolated from 55.1% of patients with burn wound infection, and were found to have a significant role in burn wound sepsis, especially *Bacteroides* spp. They were also more commonly found in patients who were treated with open wound dressings than those who were treated with occlusive dressings [*25*]. There are many factors that render burn wounds susceptible to infection with anaerobic organisms. The wounds themselves are composed of necrotic, relatively avascular tissue from which anaerobes are frequently isolated in other clinical settings [*36*].

Prior to the advent of topical antimicrobials, invasive fungal infections were not common. With the use of these agents, 25% of burn wounds seen post-mortem will harbour fungi [*37*]. Many fungal species were isolated in previous studies, but the most frequent ones were *Aspergillus* spp. [*25,38–40*]. Fungal infection was more frequent in patients treated with open dressings than those treated with occlusive dressings [*40*]. It was established that there was a correlation between fungi infecting burned patients and fungi which were located in burn care units. This indicated there is a potential risk that fungal infection could be acquired from these units [*41*]. Several species of yeasts, such as *Candida tropicalis, C. krusei, C. albicans* and *C. rugosa*, have been reported in burn wound infection and were regarded as significant pathogens [*42,43*]. Yeasts from burn wounds may invade deep organs via the bloodstream, leading to severe complications [*43*].

Type I herpes simplex virus has been reported as a cause of burn wound infection or activated elsewhere in non-burned skin or, rarely, systemic visceral dissemination. Many people harbour herpes simplex virus, often where there is a known history of cold

sores. During the relatively immunosuppressed state associated with a serious burn, reactivation of such infections can occur. Herpes simplex virus infection is thought to occur relatively frequently in the burn patient. Most commonly, children with significant burns, particularly involving the head and neck, are affected [*44–46*].

Management

Appropriate first aid treatment of burns can lessen the physical and psychological impact of injury. Eliminating the heat source is the single most important action to be taken at the scene of the injury. A secure airway with adequate ventilation is an absolute priority. Any clothing involved should be removed, as well as rings, watches, and other jewellery. Cold water is regarded as the only suitable substance to stop the burning process, remove heat or chemical agents from the skin and relieve pain. Cooling and/or neutralization with tepid water may also be appropriate to stop the initial burning process; however, once the heat source has been removed, cooling is no longer of benefit and may result in significant hypothermia.

Chemical burns should be copiously irrigated with water; dry chemicals should, however, be gently brushed off the skin before irrigation is begun. Tar burns should be cooled but no attempt should be made at removing the tar from the wound until the patient is evaluated. A petrolatum-based ointment or solvent may be applied to facilitate tar removal. During the resuscitation period, it is also important to assess circumferential burns since tissue perfusion may become restricted as oedema forms beneath the eschar. A surgical escharotomy, properly performed in a timely manner, may be limb-saving or prevent the development of a peripheral neurovascular compromise.

All patients with high-voltage exposure should be admitted for cardiac monitoring and careful observation of the wound for at least 24 hours [*1,47*].

The most frequent mistake of prehospital management of burn patients was ina-dequate airway management (no intubation) and a lack of an intravenous line and vo-lume resuscitation [*48*]. Fluid resuscitation with a balanced salt solution by continuous infusion is the mainstay of burn treatment. Extravasation of intravascular plasma into the soft tissue in the form of oedema produces a relative hypovolaemia. Oedema accumulates rapidly during the first 8 hours post-burn and continues more slowly for the next 16 hours. If the loss of intravascular volume is inadequately treated, tissue perfusion pressures diminish and multiple organ system function may become impaired.

It should also be remembered that inhalation injury is associated with increased fluid loss and may have a significant impact upon resuscitation requirements [*49*]. Intravenous replacement therapy should be initiated promptly via one or more large-bore peripheral intravenous lines if the burn exceeds 10% of total body surface area in children, or 15%–20% in adults. Urine output is a useful tool to monitor the adequacy of resuscitation and should be maintained at 0.5–1.0 mL/kg body weight per hour in children and 30–50 mL/hour in adults. A Foley catheter may need to be inserted if hourly measurements are necessary [*1*].

Traditional management of the burn wound involves careful debridement of loose necrotic tissue, gentle cleansing of the wound with a bland soap and the application of dressings [*50*]. The periphery of the burn wound should be shaved to remove hairs that can harbour bacteria. Controversy still exists over the management of blisters. Blisters can be debrided, left intact, or

aspirated (leaving the epithelium to act as a biological dressing). In general, however, blisters greater than 1 to 2 cm in diameter should be debrided. Many investigators have found that occlusive dressings are beneficial in speeding the rate and quality of wound healing [*25,51–53*]. In contrast, in open dressing there is drying of the burn wound which leads to progressive thrombosis of previously intact vasculature, tissue dehydration, cell death and decreased breakdown of dead tissue and fibrin [*53,54*]. These changes tend to produce an environment that will encourage the growth of anaerobic bacteria and fungi [*25,40*].

Early, aggressive and thorough surgical excision of infected burn wounds followed by sound and complete covering of the area with grafting at a suitable time play a crucial role [*11–13,27,55–58*]. Early covering of the open wound is essential to limit bacterial colonization and prevent infection, and to reduce fluid and electrolyte and heat loss [*57*].

Topical antimicrobial therapy remains the single most important component of wound care in hospitalized burn patients. The goal of prophylactic topical antimicrobial therapy is to control microbial colonization and prevent burn wound infection. In selected clinical circumstances, topical agents may be used to treat incipient or early burn wound infections. Silver sulfadiazine is the most frequently used topical prophylactic agent; it is relatively inexpensive, easy to apply, well tolerated by patients and has good activity against most burn pathogens. In patients with large burns the addition of cerium nitrate to silver sulfadiazine may improve bacterial control. Mafenide acetate has superior eschar-penetrating characteristics, making it the agent of choice for early treatment of burn wound sepsis. However, the duration and area of mafenide application must be limited because of systemic toxicity associated with prolonged or extensive use [*59*]. Other agents, such as nitrofurazone or chlorhexidine preparations, may be useful in isolated clinical situations. The undesirable side effects of silver nitrate solution limit its use by most clinicians [*59*]. Systemic antibiotics are a valuable therapeutic modality in the burned patient when properly used. Injudicious use, however, may not only fail to be beneficial to the patient but also may produce harmful effects, either through direct toxicity or by contributing to the emergence of resistant strains of microorganisms. General guidelines and principles for systemic antibiotic use include the following:

- The burned patient, despite all efforts, will be exposed to microorganisms.
- No single agent or combination of agents can destroy all the organisms to which the burned patient is exposed.
- Treatment involves first identifying the organism responsible for clinical sepsis, then choosing appropriate agents.
- Combinations of antibiotics are not always synergistic, or even additive, in effect.
- Multiagent therapy may have the untoward effect of predisposing to superinfection by yeast, fungi or resistant organisms.
- Antibiotics should be used for a long enough period to produce an effect, but not long enough to allow for emergence of opportunistic or resistant organisms.

In general, prophylactic systemic antibiotics are indicated in only a few clinical situations including the immediate preoperative and postoperative periods associated with excision and autografting, and possibly in the early phases of burns in children [*60*]. A study in 1999 revealed that ciprofloxacin was the most effective

antibacterial agent: 42.9% of bacteria which were encountered in burn sepsis were susceptible [*31*]. Metronidazole, if administered to patients with burns, besides offering protection against anaerobic infections, might also protect the patients from some aspects of burn-induced oxidative stress, and has promoted healing in partial-thickness burn wounds [*61*]. Vancomycin-resistant enterococci are multi-resistant microorganisms that have emerged as important nosocomial pathogens during the past decade. Emergence of these organisms has been blamed mainly on the increased and inappropriate use of antibiotics, in particular, the cephalosporins and the glycopeptide, vancomycin. Linezolid has a spectrum of activity against Gram-positive bacteria, including methicillin-resistant *S. aureus* (MRSA) and vancomycin-resistant enterococci, and can provide a useful treatment alternative to the glycopeptides [*62*]. Treatment of an established herpes simplex virus infection includes use of intravenous acyclovir, meticulous wound care, and efforts to prevent nosocomial spread. The vast majority of cases resolve without sequelae unless complicated by systemic, multiorgan herpes simplex virus infection [*46*].

Advances in burn wound care

Thirty years ago, someone with a 40% burn had little chance of survival. Today, burn care can save 50% of those who sustain an 80% burn [*63*]. The development of effective topical and systemic chemotherapy, nutritional support, early burn wound excision and closure, and the use of biological dressings have significantly improved survival rates of burn patients [*8,64*]. The currently available skin substitutes are imperfect, and research endeavours continue in an effort to develop a nonantigenic, disease-free, readily available, physiologically effective tissue that will promptly effect wound closure, reduce scar formation (and thereby improve cosmetic results) and reduce the need for reconstructive surgery. As monitoring and physiological support techniques improve and additional advances in wound care occur, morbidity and mortality in burn patients will be further reduced [*8*].

In the field of technological advances in the area of plastic surgery, burn surgery may be the most progressive, with the evolution of biological, tissue-engineered skin substitutes and the research into growth factors in healing. Further improvements in tissue engineering and technology should result in even more effective skin substitutes and hence more functional and aesthetic outcomes in large burns, in a more economically efficient way [*57*]. An encouraging therapeutic effect of the silicone elastomer TopiGel® was noticed by a group of investigators. They found that it had a positive effect on the reduction, stabilization and normalization of hypertrophic scars. The study revealed no positive therapeutic effect of the silicone sheet on the painfulness of scars or on old, mature, hypertrophic scars [*65*].

On the other hand, the increasing number of new antimicrobial agents has presented a new dilemma to the practising clinician because many of these agents have not been evaluated thoroughly in the burned population. With further studies, the armamentarium of the burn treatment team will inevitably increase. It is in this manner only that so many of the unanswered questions will be solved, and that infection will start to decline as the major cause of death in the burned population [*60*].

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