INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA):IJPS00]
Journal Homepage: https://www.ijpsjournal.com

Review Article

The Science Behind Burn Injuries And Their Treatment

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ARTICLE INFO

Received: 26 March 2024
Accepted: 30 March 2024
Published: 07 April 2024
Keywords:
Burn injury; burn wound management; pathophysiology.
DOI:
10.5281/zenodo.10937888

ABSTRACT

Burn specialists have always been in charge of managing burn injuries. Both local and systemic treatments have been recommended for treating burn wounds and preventing burn scarring since ancient times. The treatment of burn injuries caused by various physical and chemical agents necessitates unique protocols that differ greatly from those employed for other types of traumatizing injuries. Whereas whole blood loss causes shock in other acute wounds, extensive burns cause shock due to increased capillary permeability and extensive plasma loss. Despite the fact that burn wounds are initially more sterile than most other wounds, the immune compromised state of burn patients makes wound infection and septicaemia the primary causes of death in cases of extensive burns. Escher's and blisters are unique to burn wounds and call for a unique course of care. For deep burns with Escher, antimicrobial creams and other dressing agents designed for traumatic wounds are useless. The microorganisms are found in the sub Escher plane, and many of these agents are unable to pass through the Escher. The remodelling phase lasts longer even after the burn wound has fully epithelised. The maturation of burn scars can take years. This article focuses on the ways in which burn wounds differ from other types of wounds in terms of their path physiology, healing, and management.

INTRODUCTION

The skin is the largest organ in the human body, with an average adult's surface area measuring two square meters. It is made up of the dermis and the epidermis, deep within which are located key skin appendage structures such as sweat glands, sebaceous glands, and hair follicles. Proliferating epithelial cells can be found in these deep structures. These cells migrate into the wound bed and clot and are crucial to the healing process of wounds. When the skin's physical barrier function is compromised, dangerous microorganisms can

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

infiltrate the body, cause an infection, or even cause sepsis (1). Large fluid losses via the wound may also impede the healing process of burn injury, which starts as early as several hours after the traumatic event.Burn injuries are a trauma that is often overlooked but can happen to anyone, anywhere, at any time. Although friction, cold, heat, radiation, chemicals, and electric sources can all result in injuries, heat from hot liquids, solids, or fire is the main cause of burn injuries (2). Even though most burns are not life-threatening, larger burns even ones with partial thickness can still be extremely dangerous if improperly treated. Despite this, minor burns can still result in significant morbidity because they can be extremely painful and cause deformities like contractures, colloids, and hypertrophic scars (3).

While energy transfer causes tissue destruction in all burn injuries, different causes can result in different physiological and pathological reactions. A flame or hot grease, for instance, can result in an instantaneous deep burn, but scald injuries—that is, injuries from hot liquids or steam—usually show up more superficially at first because of the quick dilution of the energy source. Whereas acidic burn produces coagulation necrosis, which preserves the architecture of the dead tissue, alkaline chemicals cause coagulates necrosis, which turns the tissue into a liquid, viscous mass. Although the voltage isfrequently used to describe the circumstances of injury for ease of comprehension, electrical injuries are entirely different because they can cause deep tissue damage that is greater than the visible skin injury. Tissue damage in electrical injuries is correlated with the electric field strength (amperes and resistance of the tissue) (4). The number of Americans seeking medical attention for burn injuries appears to be steady at about 500,000 per year. 60% of these patients receive treatment in specialized burn centres, with about 40,000 of them being admitted to hospitals due to burn injuries (5).

BURN INJURY

When skin comes into contact with a heat source, burn damage occurs.Burn injuries come in different forms, and the patient's mortality and wound morbidity are impacted by the extent of the burn injury on the body. Radiation, chemicals, friction, electricity, and high temperatures are some of the elements that can result in burn injuries. The location of the burn, its temperature, and the length of time it was exposed to the heat source are all significant variables that have a direct impact on the severity of the injury and work in concert (6).

Classification of burn

Internationally, burn wound depths are categorized into degrees I through III (Table 1). The following figures show clinical images of different burn wound depths along with a summary of histology (Fig.1).

Table 1. Clinical features of burn wounds at different depths are described.

Depth/degree	Layer of skin involved	Aetiology	Appearance	Healing time
Deep 3rd degree	Full thickness of skin and in to the subcutaneous fat or deeper	Flame, electrical, chemical, blast, self-immolation	Leathery, dry, white or red with thromboses vessels	Does not heal by primary intention, requires skin graft
Deep partial 2nd degree	Deeper layer (reticular) dermis	Flame, chemical, electrical, hot liquids with high viscosity	Dry, white, non-blanching, loss of all epidermal appendages	3-6 weeks, with scars

Superficial partial 2nd degree	Superficial (papillary) dermis	Hot liquids, chemical burns with weak acid or alkali, flash	Blister, red, moist, intact epidermal appendages, blanches of pressure	1-3 weeks, long term pigment changes may occur
Superficial 1st degree	Epidermis only	Sun exposure, hot liquids with low viscosity and short exposure	Pink to red, moist, no blisters	3-7 days

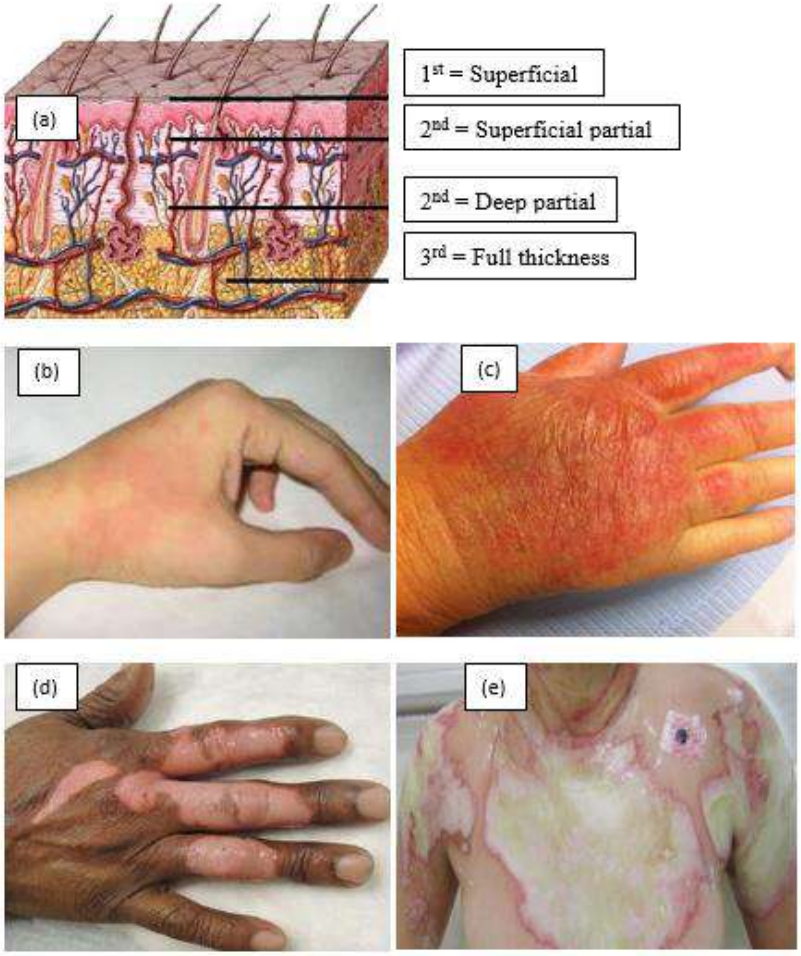

Figure 1.Classification of burn wound depth. (a) histological overview, (b-e) clinical examples of burn degree (b) superficial = 1st degree, (c) superficial dermal = 2nd degree a, (d) deep partial = 2nd degree b, (e) full thickness = 3rd degree.

Burn wound depth changes over time, particularly in cases where the wound is partially thickened. Within two to four days following burn injury, wounds that begin as superficial partial or deep partial burns may develop into deep partial or deep burns. Histological studies have demonstrated that burn injury is a dynamic process that peaks at approximately three days. This progression has been proposed to be caused by necrosis in the stasis zone. It has recently been established that apoptosis occurs in the stasis zone and may play a role in the wound's progression (7). Patients with

partial thickness burn wounds require periodic evaluations for wound depth due to their distinct path physiology. Generally speaking, burns of partial thickness that are not expected to heal in three weeks should be removed and grafted. The evaluation of indeterminate burn wound depth may benefit from the use of an inventive multispectral optical system, which allows for the parallel acquisition of spectrally filtered images and the visualization of burn degrees (8).

Aetiology

Burn injuries may result from sunburn, radiation, heat, chemical, or electrical sources, among others (9).

Thermal injuries

About 90% of burns are caused by thermal injuries, and the depth of the injury is influenced by the temperature and length of contact. They are separated into:

- Burn injuries most commonly occur from hot liquids (scalds), which account for almost 70% of burns in children and are also common in the elderly. Partial-thickness burns from scratches typically heal with standard treatment (10).
- Dry heat injuries: these are typically brought on by coming into close contact with radiant heat or a flame. Common in adulthood and frequently linked to issues brought on by smoke inhalation. They typically need surgical intervention and are deep (partial or full thickness) (11).
- Direct touch with a hot object can cause contact injuries. Extended contact with a moderately heated object, such as a radiator, can also result in a thermal injury, which is frequently linked to unconsciousness in certain populations, such as the elderly, epileptic patients, drug addicts, and alcoholics. The majority of contact burns are deep and necessitate surgery (11).

Chemical injuries

Three percent or so of burns are caused by chemical injuries. These kinds of incidents mostly take place in residential and commercial environments. Proteins are denaturised in this kind of damage, and the degree of damage is determined by the concentration, quantity, length of contact, and mode of action of the specific chemical—that is, by reduction and oxidation, corrosion, protoplasmic poisoning, visitation, and desiccation. Chemicals have historically been divided into acids and alkalis despite the fact that all chemical groups exhibit a similar clinical picture. This is because different chemicals may cause different tissue damage mechanisms (12). Damage from acid burns results in protein denaturation and necrosis, which are typically localized and transient conditions. Conversely, alkaline burns result in progressive liquefaction necrosis, which has a longer-lasting effect and deeper tissue penetration. Alkaline burns are caused by cement, which can also create an additional exothermic reaction when combined with perspiration. Furthermore, cement powder severely desiccates the impacted surface due to its high hygroscopicity. Washing with lots of water helps lessen tissue damage by diluting the chemical.Acids and alkalis (sodium hydroxide, potassium hydroxide, calcium hydroxide and lithium hydroxide, sodium and calcium hypochlorite, ammonia, phosphate, silicate, sodium carbonate), oxidizers (bleaches such as household chlorites, peroxides, chromates), and other chemicals (white phosphorus, hair colorants, mustard gas) are the most common causes of burns (13).

Electrical injuries

Less than 5 percent of burns are caused by electrical injuries. Children and male manual labourers are most likely to have them. The voltage and amperage, the kind of current, the length of contact, and the current's path through the body all affect how serious an injury is. Nerves

and vessels in particular are excellent conductors, as are most tissues. Although skin conductivity varies with temperature and moisture content, skin and bones are not good conductors. Because the tissues are poor conductors, the electricity surrounding them generates heat, which damages the nearby tissues. In clinical practice, the "entry and exit points," or locations where the electrical current has passed through the body, are frequently observed. At the entry and exit points, electrical voltages less than 1000 V, which are usually found indoors, result in tiny, deep burns. In addition to impairing heart function, alternating current can cause arrhythmias. Extensive tissue damage is caused by high-voltage injuries (>1000 V), which frequently result in limb loss, systole, cardiac arrhythmia, rhabdomyolysis (breakdown of muscles), and renal failure. Because the injuries are invisible, fluid resuscitation is difficult. 15% of victims sustain additional injuries from falls, and this kind of injury is linked to a high death rate (14).

The simple arc flash of a discharge between two high-voltage sources can also result in burns. The arc's heat can burn exposed body parts, such as the hands and face, even though no current flowsthrough them. Generally, the resulting burns are only partially thick, unless the arc ignites the clothing and causes more serious injuries (15).

PATHOPHYSIOLOGY OF BURN

Local effects of burn

Extended exposure to temperatures exceeding 40°C causes proteins to denaturant, ultimately resulting in the loss of their plasma membrane integrity. When exposed to temperatures higher than 60°C, this process is quick and can happen in just a few seconds, resulting in flame burns. The clinical manifestation of coagulation necrosis is the outcome of the local alterations (16). As will be discussed later, temperature and contact time have a synergistic effect (Table 2).

The earliest cooling applied topically following a burn maximizes epithelisation and minimizes scarring. When it comes to late scarring and early re-epithelisation, room temperature water (15°C) is superior to ice water (2°C) (17).

Table 2. Relationship between full thickness burn occurrence and temperature exposure duration (adapted from Moritz and Henrique (7))

Temperature in Degree Celsius	The exposure time in seconds
45.0	3600
54.4	30
60.0	10
69.0	1

Burn shock path physiologic effects are caused by the release of toxic metabolites, antigens, and immune modulator agents through molecular structural alterations. Histamine, serotonin, bradykinin, nitric oxide, oxygen free radicals, pro- and thromboxane producing products of the eicosanoid acid cascade, TNF, and interleukins are among the local mediators released. The mediator most likely in charge of the early stage of elevated micro vascular permeability observed right after burn is histamine. Venial endothelial cells contract in response to histamine, resulting in the temporary formation of large endothelial gaps. Research has indicated that the relationship between histamine, xanthenesoxidise, and oxygen radicals may play a role in the path physiology of burn oedema in the skin. Jackson divides the local variations in burn injuries into three zones (18). The general consensus is that devitalized tissue makes up the zone of coagulation at the central

focus of injury. The zone of hyperaemia, which is the most peripheral zone, is defined by vasodilatation and inflammatory changes without structural damage. The term "zone of stasis" refers to the intermediate area of uncertain prognosis that develops between these zones. The general consensus is that devitalized tissue makes up the zone of coagulation at the central focus of injury. The zone of hyperaemia, which is the most peripheral zone, is defined by vasodilatation and inflammatory changes without structural damage. The term "zone of stasis" refers to the intermediate area of uncertain prognosis that develops between these zones (19). The zone of stasis is often best identified in mid to deep dermal burns and represents a region of vascular stasis and ischemia. From a clinical perspective, it is this region which poses some of the greatest challenges for the burn team. This tissue has the potential to heal or alternatively to progress to a full thickness lesion. Clinically this ischemic area can only be salvaged, if revascularization is achieved within a few days. Otherwise the irreversible tissue death is inevitable. The phenomenon of ischemia reperfusion events in the zone of stasis has been described in the past (20). This area is vulnerable to reperfusion-related oxidative stress, especially following significant partial thickness burns. Cell death caused by reperfusion injury is mostly apoptotic. Progressive tissue loss could be attributed to apoptosis in the stasis zone. Prior research on partial thickness burn wounds demonstrated a decrease in the apoptotic rate following inducible NO syntheses inhibition (21).

The systemic effects of burns

After thermal injuries, various organs and body systems experience systemic pathophysiology changes that can result in clinical manifestations such as immune suppression, shock, intestinal changes, respiratory and renal failure, and others. After a burn injury is successfully resuscitated from the shock phase, the primary metabolic manifestations associated with major thermal injury are extreme hyper metabolism and catabolism. Burn patients experience a biphasic metabolic response, with an initial phase and a hyper metabolic and catabolic flow phase of injury following.The evaporative heat loss from trauma victims' wounds contributes to the increased oxygen consumption and metabolic rate, but inflammation's direct central effect on the hypothalamus is also probably a contributing factor (22). The gastrointestinal tract (impairment of gastrointestinal motility and absorption, splanchnic vasoconstriction, loss of mucosal barrier function with bacterial translocation, increased intra gastric pH), the haematopoietic system (anaemia, immune depression), the pulmonary system (local vasoconstriction, oedema), and the renal system (splanchnic vasoconstriction) are among the path physiologic changes that follow severe thermal injuries (23).

CLINICAL MANAGEMENT OF BURN WOUNDS

Superficial and medium partial-thickness burns

The preservation of viable dermal appendages is the cornerstone of treatment for wounds with superficial and medium partial thickness. After cleaning the wound, any loose, nonviable tissue is extracted. Washing the wound and using a topical ointment and light, no adherent dressing twice a day until the wound has fully reepithelialised are the steps in the follow-up care protocol. For superficial wounds, this process typically takes 5 to 10 days, and for medium-depth wounds, it takes 10 to 14 days. The wound needs to be re-evaluated once or twice a week if the patient is being treated as an outpatient. The wound should then be massaged four or five times a day with moisturizing cream after epithelialisation has taken place. The patient is told to come back in four to six weeks so that any pigment changes and hypertrophic scars can be evaluated (24).

Deep partial-thickness burns

Deep partial-thickness burns are more likely to form hypertrophic scars and usually reepithelialise after 3–4 weeks. For ten to fourteen days, wounds of "indeterminate" depth can be treated in the same way as superficial wounds. The wound can be monitored for the duration of epithelialisation if the epithelial buds are dense and the area is not functionally or aesthetically significant. Pressure garment therapy is probably necessary for these wounds in order to reduce the formation of hypertrophic scars. Elective excision and skin grafting should be done if the wounds are larger than 2.5 cm, affect significant body areas, and have not healed after 14 days. Reduced scarring and a quicker return to function are the outcomes of this treatment plan, which also gives deeper wounds more time to heal and demarcate (25).

Full-thickness burns

Full-thickness injuries are characterized by the destruction of all dermal appendages, making spontaneous epithelialisation impossible. These wounds can only heal by contraction and migration of epithelial cells from the wound edges in the absence of skin graft coverage. The location and size of the wound determine the best closure strategy for full thickness wounds. Greater burns typically cause the surgeon to shift their attention to wound coverage and survival, whereas smaller to moderate-sized burns (less than 35% TBSA) allow the surgeon to concentrate on the functional and cosmetic outcome. Before being surgically closed, full-thickness wounds are cleaned, debrided, and treated with topical antibiotics twice a day (26).

Surgical management of burn wounds

Numerous medical developments over the last 20 years have allowed surgeons to modify their surgical strategy for treating deep bum wounds. These include the creation of the Tanner-Vandeput mesh dermatome, enhanced critical care monitoring tools, safer and more effective blood and skin replacements, and a deeper comprehension of postpartum nutritional requirements (27).

By combining immediate auto grafting with tangential wound excision, burn care outcomes for deepdermal bums were dramatically improved in the 1970s (28). This technique of wound excision has been shown to reduce hospital stays and bum morbidity in multiple groups. More clinical data, however, did not start to suggest better outcomes following prompt wound closure until the early 1980s. Though genuine prospective, randomised trials comparing early excision therapy to the standard procedure of grafting following wound separation (roughly 4 weeks) have not been conducted, early excision and grafting have become standard practises and are currently the preferred treatment for all deep burns. It has been reported that enzymatic debridement can remove the need for Escher surgical removal. But this method hasn't become very well-liked in homeless shelters (29). One important factor in excision therapy is blood loss. About 200 ML of blood are lost for every 1% of TBSA that is removed and grafted in adults.55 Blood loss in children is roughly 3% to 4% of the volume of circulating blood for every 1% of TBSA removed and grafted (30). This appears to help achieve haemostasis during skin grafting and allows for the restoration of blood volume and body temperature. Using a Padgett electric dermatome, split-thickness skin grafts are harvested between 0.008 and 0.016 inches. Although it is frequently necessary to use a 4:1 mesh to expand the skin in extensive bums greater than 60% TBSA, every attempt is made to use sheet or minimally expanded 1.5: 1 mesh grafts on the hands. On the face, sheet grafts are always utilized (31). Use the least expanding mesh and sheet grafts for both cosmetic and functionally significant areas,like the hands, for bums with 40 to 60% TBSA. The best cover for bums with less than 40% TBSA is a sheet graft. These grafts produce the best cosmetic outcome and are linked

to a 50% decrease in pressure garment therapy until wound maturation is fully achieved (32).

Donor sites

Patients with large bums may need to use all unburned areas, so choosing a donor site is less important in these cases. The back and scalp are preferred donor sites because they cause the least amount of scarring from donor sites, despite the fact that the anterior thighs are commonly used. When doing face grafts, the scalp in particular is a great donor site because of the possibility of getting a very good colour match in the mature graft. The scalp's quick healing and near-invisibility after hair grows are two more benefits of using it as a donor site (33). For the purpose of caring for the skin graft donor site, several novel dressing materials have been developed. If a topical medication is used, it should be changed twice a day in a fashion similar to how superficial partial-thickness wounds are treated. It could be preferable to apply a topical antimicrobial agent to the entire wound if the donor sites are close to deep bums. It might be feasible to cover the wound with a synthetic dressing or another occlusive device if the donor site is tiny. Kaltostat, Bobbin, Dodder, and other more recent dressings are becoming more and more well-liked. For many of these materials, careful consideration of application techniques and donor area selection are necessary. According to reports, these dressings have been linked to quicker epithelialisation and less discomfort. In the modern management of skin graft donor sites, dry gauze dressings are largely unnecessary. Last but not least, donor sites especially those that are used repeatedly may experience hypertrophic scarring and pigmentation changes. Therefore, careful monitoring is necessary to determine whether pressure garment therapy is necessary as these wounds mature (34).

REHABILITATION

The excision of bum wounds has significantly reduced hospital stays and produced better cosmetic outcomes, but the rehabilitation team must be included in the bum patient's care. As soon as possible following injury, bum patients must start comprehensive rehabilitation. Proper positioning and early active range-of-motion exercises are essential for a successful functional outcome. To get the intended long-term results, pressure face masks, pressure garments, and appropriate splinting techniques are also crucial. Pressure needs to be applied to healed wounds for twenty-three hours a day until the scars have fully developed, which should take about a year (35).

CONCLUSIONS

Burn injuries don't exist in a vacuum; they are a part of a phenomenon. A multitude of systemic factors interact to influence the healing process of burn wounds, even though it proceeds similarly to other types of wounds. Both the patient's overall health and the burn wound are directlyimpacted by each other, which can result in fatalities and other conditions like septicaemia. The treatment given to the burn patient directly affects their wounds. The patient's overall health is correlated with the healing of these wounds. Burn injuries are similar to other wounds in that the fundamentals of wound healing and care are unchanged, but they differ in that they have a more significant effect on the patient's overall condition, which is crucial for the patient's eventual survival, the development of deformity, and their rehabilitation.

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HOW TO CITE: Vikas Dewangan, Nikita Sinha, Tajuddeen Khan, Chirag Jain , Yogesh Kumar Dewangan, Bhumika Chandrakar, The Science Behind Burn Injuries And Their Treatment, Int. J. of Pharm. Sci., 2024, Vol 2, Issue 4, 417-426. https://doi.org/10.5281/zenodo.10937888