

Review Article

Burn wound: How it differs from other wounds?

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ABSTRACT

Management of burn injury has always been the domain of burn specialists. Since ancient time, local and systemic remedies have been advised for burn wound dressing and burn scar prevention. Management of burn wound inflicted by the different physical and chemical agents require different regimes which are poles apart from the regimes used for any of the other traumatic wounds. In extensive burn, because of increased capillary permeability, there is extensive loss of plasma leading to shock while whole blood loss is the cause of shock in other acute wounds. Even though the burn wounds are sterile in the beginning in comparison to most of other wounds, yet, the death in extensive burns is mainly because of wound infection and septicemia, because of the immunocompromised status of the burn patients. Eschar and blister are specific for burn wounds requiring a specific treatment protocol. Antimicrobial creams and other dressing agents used for traumatic wounds are ineffective in deep burns with eschar. The subeschar plane harbours the micro-organisms and many of these agents are not able to penetrate the eschar. Even after complete epithelisation of burn wound, remodelling phase is prolonged. It may take years for scar maturation in burns. This article emphasizes on how the pathophysiology, healing and management of a burn wound is different from that of other wounds.

KEY WORDS

Burn injury; burn wound infection; pathophysiology

INTRODUCTION

By definition, open wound is any traumatic breach in continuity of skin and deep tissue. Contusion is a closed wound and is an exception to the above definition. Classification of wounds is important for medico-legal purpose as well as for management. Wounds are classified mainly on the basis of mode of infliction and causative agent as follows^[1]

Closed wounds -Contusion, closed fracture, etc.

Open wounds

- Sharp cut
- Laceration
- Abrasion
- Avulsion
- Crush wound
- Punctured wound
- Bite wound
- Burn wound

All of the above wounds are different from each other in pathophysiology as well as in management. Bite wound may be clean cut in appearance but is grossly contaminated and has to be managed differently from the clean cut wound inflicted by a knife or glass. Burns or burn wounds are so much different from other wounds that

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a separate medical superspeciality has been designated to manage them. Even though extensive burns primarily involve a single organ; however, almost all systems of the body are affected in this disease making it a generalized disorder. Unlike any other traumatic wound, involvement of intensivist and physician is more in burns.

Classification of burns

Depending on the causative agent burns are classified as follows^[2]:-

Physical

Thermal burns

By dry heat - Flame burns

By wet heat - Scalds

Electrical burns

Contact burns

High voltage

Low voltage

Flash burns

Radiation burns

Laser burns

Chemical

i-Acid burns

ii-Alkali burns

iii-Others

Each type of the burn wound in the above classification is different from the other and so is different from other common type of wounds. Apart from the obvious fact that skin damage is present in all these burns, local and systemic management are not the same for each of the above burns.

PATHOPHYSIOLOGY OF BURN *Vis-a-Vis* OTHER WOUNDS

Heat not only damages skin locally but has many generalized effects on the body. These changes are specific to burn injury and are generally not encountered in wounds caused by other injuries.^[3]

- There is generalized increase in capillary permeability due to heat effect and damage. This causes plasma to leak out from capillaries to interstitial spaces. Increased capillary permeability and resultant plasma leak persists till 48 hours and is maximum in first 8 hours. By 48 hours either capillary permeability returns back to normal or they are thrombosed and are no more the part of circulation. This plasma loss is the cause of hypovolaemic shock in burns. The

amount of fluid loss will depend on extent of burns. Body surface area burns is usually calculated by Wallace's rule of '9' in adults and Lund and Browder's chart in adults and children. Any adult burn more than 15% and pediatric burn more than 10% will land up in hypovolaemic shock if not adequately resuscitated. In burns involving 50% of body surface area, there is maximum possible fluid loss and it remains same even if more than 50% of body surface area is burned.

This generalized increase in capillary permeability is not seen in any other wound. There is only local reaction at the wound site due to inflammation leading to persistent progressive vasodilatation and oedema. Hypovolaemic shock in other major traumatic wounds is usually due to blood loss and requires whole blood replacement immediately. Whereas in extensive burns the whole blood replacement is given after 48 hours.

Following are the causes of blood loss in burns

- Red blood cells are lost in thrombosed vessels underlying the burned skin in the acute phase. Therefore, deeper the burn more is the blood loss. The blood is to be transfused after 48 hours unless otherwise indicated as in pre-existing anemia or whole blood loss due to any other cause.
- Life span of circulating red blood cells is reduced due to the direct effect of heat and they are hemolysed early. Extensive burn also causes bone marrow depression leading to anemia.
- In chronic stage of burns, blood loss from granulating wound, and infection are responsible for anemia. (B) Unlike most of the other wounds, burn wounds are usually sterile at the time of injury. Heat being the causative agent, also kills all the micro-organisms on the surface. It is only after the first week of burns that these surface wounds tend to get infected, thus making burn wound sepsis as the leading cause of death in burns. On the other hand, other wounds e.g., bite wounds, puncture wounds, crush injury and abrasions are heavily contaminated at the time of infliction yet they are rarely the cause of systemic sepsis.

WOUND HEALING AND BURNS

Healing of burn wound depends on the depth of burns. Burn wounds can be classified according to involvement of skin and deeper tissues as follows:

- First-degree burn or epithelial burns - Skin is erythematic without vesication.
- Second-degree burns - Involving epidermis and variable thickness of dermis. This is again divided into
 - Second-degree superficial –where vesication and inflammation is seen in skin as only papillary dermis is involved.
 - Second-degree deep -eschar formation is seen as it involves deep reticular dermis.
- Third-degree burn - Also known as full thickness burns – eschar formation is present in these burns.^[4,5]

Jackson (1959) has described three zones in the damaged burned tissue^[6] [Figure 1]

- Central zone of coagulation – This is the central part of burns with complete coagulative necrosis.
- Zone of stasis— Zone of stasis is at the periphery of zone of coagulation. The circulation is sluggish in this zone but it can recover after early and adequate resuscitation, and proper wound care.
- Outer most zone of hyperaemia – This is peripheral to zone of stasis. It is the result of intense vasodilatation as is seen in inflammatory phase after the trauma. This eventually recovers completely.

In first degree and second-degree superficial burns, healing is by primary intention. Second-degree superficial burns heal from epithelium of hair follicle remnants, which are in plenty in the superficial dermis. Healing is complete within 5-7 days and is almost scar less. In second-degree deep and third-degree burns, healing is by secondary intention, which involves the process of epithelisation and contraction [Figure 2].



Figure 1: Scald burn in a child showing the Jackson's three zones of damage i.e., (a) Zone of coagulation, (b) Zone of stasis, (c) Zone of hyperaemia

Inflammatory (reactive), proliferative (reparative) and maturation (remodelling) constitute the three phases in wound healing. This is same for all types of wounds, the only difference being in duration of each stage.

Inflammatory phase

This is same in all traumatic wounds. Immediately after the injury, inflammatory response of body begins which has vascular and cellular components.^[7-9]

- Vascular response: Immediately after burns there is a local vasodilatation with extravasation of fluid in the third space. In extensive burn injury increased capillary permeability may be generalized leading to massive extravasation of plasma requiring fluid replacement.
- Cellular response: Neutrophils and monocytes are the first cells to migrate at the site of inflammation. Later on neutrophils start declining and are replaced by macrophages. The migration of these cells is initiated by chemotactic factors like kallikreins and fibrin peptides released from coagulation process and substances released from the mast cells like tumour necrosis factor, histamine, proteases, leukotrienes and cytokines. Cellular response helps in phagocytosis and cleaning of dead tissue and toxins released by the burned tissue.

Proliferative phase

In partial thickness burns re-epithelialization starts in the form of keratinocyte migration from viable skin appendages in dermis few hours after injury, this usually covers the wound within 5-7 days. After re-epithelialization the basement membrane zone forms between dermis and epidermis. Angiogenesis and fibrogenesis help in dermal reconstitution.



Figure 2: Healing in deep burns by secondary intention with contraction and hypertrophic scarring

Healing after burn excision and grafting: In deep burns after primary excision and grafting healing is by delayed primary intention. Take of skin graft after primary excision is the part of proliferative phase of wound healing.

Remodelling phase

Remodelling phase is the third phase of healing wherein the maturation of graft or scar takes place. In this final phase of wound healing initially there is laying down of fibrous structural proteins i.e., collagen and elastin around epithelial, endothelial and smooth muscle as extracellular matrix. Later on in the resolution phase this extracellular matrix remodels into scar tissue and fibroblast become myofibroblast phenotype which is responsible for scar contraction.

In second-degree deep dermal and full thickness burns which are left to heal of their own this resolution phase is prolonged and may take years and is responsible for hypertrophic scarring and contractures [Figure 2]. Hyperpigmentation seen in superficial burns is due to overactive response of melanocytes to burn trauma and hypopigmentation seen in deep burns is due to destruction of melanocytes of the skin appendages. In skin-grafted areas once innervation starts, the growing nerves alter the melanocyte control which usually leads to hyperpigmentation of graft in dark skinned and hypopigmentation of graft in white-skinned individuals.

MANAGEMENT

Medico-legal aspect

Every burn injury is considered medico-legal unless proved otherwise. More so when there is an extensive burn injury, irrespective of history told by the patient, it is always considered as medicolegal case because there is always a doubt regarding the actual mode of injury.^[10]

On the other hand in all other traumatic wounds the pattern of injury usually points to the mode of injury which coincides with the history given by the patient.

Burn wound management depends on the severity of burns.

Minor burns are classified as less than 15% TBSA in adults and less than 10% in children

Major burns are burns up to 35% in adults and 30% in children.

Critical burns or life-threatening burns are classified as more than 35% burns in adults and more than 30% in children.

Minor burns of face and perineum require admission for observation. Eyelids and pinna burns require special treatment considering the importance of the underlying structures.

Major and critical burns require hospital admission. As mentioned earlier there is hypovolemic shock due to plasma leak in third space.

Contrary to burn wound hypovolemic shock is usually caused by acute and substantial blood loss in all other traumatic situations.

Fluid requirement in burns

There is no single formula which is universally accepted for calculating quality and quantity of fluids required in extensive burns. All major burn units throughout the world use their own formula or modification of one of the accepted formulae.^[11] This clearly shows that all these formulae are a rough guide for calculating the fluid required in extensive burns for first 24-48 hours. Actual quality and quantity is to be modified according to the hourly urine output and other vital parameters of the patient. Following are few popular and internationally accepted formulae:

1. Evan's Formula –fluid requirement is 2ml/kg/% of burns for first 24 hours in addition to the daily requirement of the patient. Half of this fluid is transfused in first 8 hours and rest half in next 16 hours. As far as the type is concerned half of this fluid will be crystalloids i.e., Ringer lactate and half will be plasma.
2. Brooke's Formula – It is the same as Evan's formula. Here three-fourth of the fluid used is crystalloid and one-fourth is plasma.
3. Parkland Formula –Quantity is calculated as 4 ml/kg/% of burns including daily requirement of fluid. All fluid is given in the form of crystalloids.

We are using modified Brooke's formula in which total calculated fluid is given in the form of crystalloids and colloids are generally not used unless indicated clinically.

Blisters and eschar

These are common in burn wounds. Blisters are present in second-degree superficial burns due to collection of plasma between dermis and dead epidermis. Blister fluid

is plasma-rich protein and very good culture media for bacteria and other organisms and should not be left for long.^[12]

Blisters may also be present in wounds due to repeated friction such as shoe bites which may actually be considered as an extended spectrum of friction burns. Fluid-filled blebs may also be seen in closed wounds which may represent underlying fracture and haematoma.

Skin with full thickness becomes leathery, nonelastic and forms a thick band on drying known as eschar. Circumferential eschar over the extremities may compromise distal vascularity due to compression of the blood vessels proximally.

Escharotomy and fasciotomy

Circumferential deep burns may interfere with circulation in extremities or in trunk. Eschar may compromise breathing when present circumferentially over the chest. Escharotomy needs to be done to relieve the patient of such complications. In the extremities longitudinal full-thickness incisions up to the subcutaneous tissue are given on both sides in neutral lines for release. Over the chest a grid pattern is usually used where a combination of vertical and transverse incisions are used anteriorly, laterally and posteriorly to facilitate lung expansion on all sides.

Fasciotomy is commonly performed in injuries which cause compartment syndrome. In this procedure incisions are given dividing the deep fascia and opening all compartments of extremities to facilitate release of pressure and avoid subsequent ischaemia-related complications. Usually all compartments of an extremity need to be relieved of the pressure.

It was previously thought that compartment syndrome in burn trauma is only seen in electrical burns where muscles become oedematous and compartment pressure increases resulting in need for urgent fasciotomies. Thermal burns were considered incapable of damage to the extent of necessitating fasciotomies but recent studies^[13-17] have shown otherwise. It has been documented that there is definite increase in compartment pressure of the extremities even after thermal burns.

Rise in intra-abdominal pressure is another form of compartment syndrome which is a dreaded complication. Studies have shown symptomatic increase in intra-abdominal pressure to a critical

level in burn patients requiring release in the form of laparotomies.

Anaesthesia, surgical excision and closure of wound

For the surgical closure of any other wound the golden period is said to be first 6 hours [Figure 3] where as in extensive burn excision is usually done after 72 hours. The burns wounds are sterile at the onset and it takes few days for micro-organisms to contaminate and invade the wound where as other wounds are usually heavily contaminated from the very beginning and require early debridement and closure as delayed closure may lead to infection and its sequel. More so excision in extensive burns can only be done once patient is fully resuscitated and stable and the physiological changes have been restored back to normal and can withstand another surgical trauma. The ideal time for this is third to fifth postburn period [Figure 4a,b].^[18] In small burns (up to 10%) excision may be planned earlier, as small burns do not alter the physiology or milieu interne significantly.

Anaesthesia is a major concern in burns and anaesthetist is the integral member of burns team.^[19] Anaesthetist in burn team should be well versed with, (a) burn resuscitation in acute burn phase where air way management and intubation are often required, (b) physiological changes in hypermetabolic stabilising phase when excisional surgeries are commonly performed and (c) anatomical changes due to burn contracture and deformity in reconstructive surgical phase. Burn anaesthesia has become a separate speciality in which anaesthetist is intensivist in acute stage of burns and should be able to perform fiberoptic intubation in corrective surgeries



Figure 3: Case of extensive facial trauma in which the repair was done immediately giving excellent postoperative results with minimal scarring



Figure 4: (a) Case of deep dermal burns over thigh – planned for primary excision and grafting after 72 hours, (b) Postoperative result after 10 days

for severe facial and cervical deformities following burn injuries.

Surgical closure of other wounds is mostly done under local or regional anaesthesia and less intensive expertise is required for that [Figure 5a-d]. Physiological changes seen after burns are usually not seen even after major traumas apart from the hypovolaemic shock due to blood loss which can be restored by blood transfusion. In fact at times early surgery is required in many traumatic wounds to stop the bleeding and prevent further deterioration in the general condition.

Primary respiratory tract injury in burn

Injury to respiratory tract in acute burn patients can be either

- Direct damage by heat – This is mainly limited to upper respiratory tract and may cause laryngeal oedema requiring intubation.
- Nonthermal involvement of respiratory tract in the form of inhalation of toxic gases which may be asphyxiant (e.g., Carbon monoxide and cyanide) or irritant (e.g., chlorine, ammonia, etc). In both the cases there is inflammation and damage of respiratory tract mucosa, which if not treated properly leads to secondary damage and infection leading to involvement of lower respiratory tract and alveolus.^[20-23]

Infection and immune profile in burns

The marked decrease in hypovolemic shock and acute renal failure as causes of death due to proper resuscitation and effective fluid management has led to infection as the major cause of concern in burns.^[24] Infection in burns is proportionate to the fraction of body surface

injured.^[25] Improved burn wound management in last decade has significantly reduced and altered occurrence of bacterial burn wound infection. Pruitt *et al.*^[26] in their study of changing epidemiology of infection in burn patients have noticed that apart from bacteria, newer micro-organisms like virus and fungi are also responsible for sepsis in burns. Pneumonia is the most frequent infection as part of systemic sepsis in burns. In study of Pruitt *et al.* *Staphylococcus aureus* accounts for 48% of lung infection whereas *Pseudomonas* accounts for 16% only. Rest of the infection is by other Gram-negative organisms like *Klebsiella*, *Escherichia coli*, *Salmonella* and *Haemophilus*.

There has not been a significant change in the statistics of bacterial burn wound infection in last 20 years after the study of Pruitt *et al.* except for the virulence of organism invading the wound which has increased manifold. *S. aureus* invariably is methicillin-resistant (M.R.S.A.) and *Pseudomonas* and *Klebsiella* are usually extended spectrum β -lactamase (ESBL) enzyme producing species. In last 8 years incidence of multidrug resistant Gram-negative bacilli *Acinetobacter baumannii* is also increasing.

Fungal infection in burns

Presence of fungal infection in burn wounds^[27] was widely reported by Becker WK *et al.* in their study in 1991 and *Candida albicans* was found to be the main causative organism. In a recent study done in 2011 by Sarabahi *et al.*,^[28] on changing pattern of fungal burn wound infection in burns, *C. albicans* has been replaced with *Candida nonalbicans*, mainly *C. krusei* and *C. glabrata* and *Aspergillus*. In the same study fungal infection was found to be associated with very high mortality i.e.,



Figure 5: (a) Case of sharp cut injury over extensor aspect of wrist, (b) Explored under regional anaesthesia and all extensor tendons found divided, (c and d) Extensor tendons repaired and skin closed primarily. Patient required splintage and physiotherapy postoperatively

more than 40% and resistant to the conventional azoles. The organisms were only sensitive to Echinocandins and Amphotericin B.

The major cause of invasive burn wound sepsis is profound immunosuppression. Burn injury affects both nonspecific and specific components of immune system. The nonspecific defences consist of circulating and fixed phagocytic cells and number of plasma proteins that mediate the inflammatory response. In extensive burn patients, polymorphonuclear phagocytes are rendered ineffective in their chemotactic, phagocytic and intracellular killing actions. Similarly mononuclear phagocytic system is also not able to perform its functions of phagocytosis and cytokinin release.^[29-32] In specific component of immune system, cell-mediated immune response is markedly depressed as evident by prolonged survival of homograft in burned patients.

Humoral immune response is also depressed as is evident by significant fall in serum concentration of all classes of immunoglobulins in severely burned patients.^[33,34]

Not only is the quantitative fall of immunoglobulin levels noted in burn patient, the remaining circulating immunoglobulins are also qualitatively inefficient. T-cell-dependent antibody production is suppressed for a long time in extensive burn patients due to deficiency of interleukin-2-regulated secretion and suppression of helper T-cell-derived factors which are necessary for B-cell differentiation into antibody secreting cells.^[35] The highest incidence of septicaemia in burns occurs in first 10 days when serum immunoglobulin titres are markedly deranged. Surprisingly patient with extensive tissue trauma like extensive degloving injury are not as immunocompromised as are the burn patients.

Topical and systemic antibacterial therapy in burn wound *vis-a-vis* other wounds

Burn wounds as mentioned earlier are sterile in the beginning. Only organism present are those which are deep seated in the epithelial appendages of the hair follicles and sebaceous glands. Some of these organisms may multiply and come to the surface of the burned skin. Therefore, dressing of burn wound by antimicrobial agent helps in killing of these organisms and keeps the wound sterile for a longer period. The topical agents used in burn wound dressing also prevent entry of new organisms from the exterior. Bacterial proliferation starts at the deep surface of the eschar of the burn wound after the 5th day. Eschar being a dead tissue acts as a culture media and helps in bacterial growth. This subeschar bacterial multiplication may later invade the deeper tissues and are responsible for burn wound sepsis. Normal antimicrobial agents used on other surface wounds like povidone iodine, mupirocin and neomycin, etc., cannot penetrate the eschar and so are ineffective in control of subeschar bacterial invasion. Therefore, for burn wounds one has to use the antimicrobial agents which can penetrate the eschar and kill the organisms multiplying at subeschar level.

In extensive burn, the amount of topical antibacterial cream to be used is often large. These agents get absorbed from the skin into the circulation. So the antimicrobial agent to be used in burns should be non toxic with minimal side effects on systemic absorption.

Burn wound may take several weeks to heal completely and will require dressing change several times during this period with same antibacterial cream. There should be minimal emergence of resistance amongst the organism against the antimicrobial agent. Considering all these criteria, 1% silver sulfadiazine cream is one of the best available cream for extensive burn dressing.

Systemic antibiotics have a very limited role in burns except in burn wound sepsis. In any other infected wound systemic antibiotics are used for therapeutic or prophylactic purposes according to surface bacterial culture and sensitivity. In burn injuries there is no role of prophylactic antibiotic as it will not reach the subeschar level, the place where these micro-organisms multiply.^[36-41]

Nutrition

Extensive burns is a severe catabolic injury sustained by

the body which extends over a long period of time till all the burn wounds heal unlike other traumas where the catabolic phase lasts for a shorter period as the wounds most often are not that extensive and closure is achieved early. Therefore, nutrition is a very important aspect to be looked into during the management of burns. The requirement of protein and calories is very high and supplementation has to be done accordingly.

Scars following burns and trauma

An individual sustaining burns is labelled as a burn patient forever as he or she is scarred for life. Scars and contractures are two inevitable sequel of burns. Only way to avoid scar in burns is not to sustain burns. Scar maturation in burn is akin to remodelling phase of wound healing. Deep dermal burns, if not excised and grafted primarily are bound to get hypertrophic scarring.^[42]

Compared to other traumatic wounds, surface involved in burns is more, hence the extent of scarring is also significantly more, signs are obvious and symptoms of itching and pain are severe enough to hamper daily routine. As a result most of these patients require long-term treatment in the form of pressure garments, silicone gel sheets and massage to relieve them of their symptoms. Therefore, rehabilitation and physiotherapy are very essential in the management of extensive burns. All deformities and contractures are preventable. Splintage and physiotherapy of burn areas crossing joint line has to be started as early as possible and continued till the scars mature. Upper extremity along with hand and neck are two most important areas where functional disability due to contractures is most common. Deep burns of hand are to be dressed and immobilised in cock-up position to prevent Z deformity. Cervical collar is used over the burn dressing in neck burns or pillows are placed below the shoulder to keep the neck extended. Similarly for inferior extremity splints are used to prevent equinus deformity and knee joint contracture.

Physiotherapy and splints are mainly required in wounds involving tendons and nerves. Scar management for other wounds is usually done for aesthetic reasons as there may be no functional limitations.

Postburn psychosis is a well known entity as a burn patient is scarred not only physically but also mentally. Extensive scarring due to burns especially those involving the face and exposed areas of the body is considered to be a taboo in the society even today as the patients are not accepted

for employment or marriage proposals especially girls. As a result a individual with facial burn scars usually stay indoors and hardly interact with the society as they are most often depressed. They, therefore, need constant encouragement and counselling.

CONCLUSIONS

Burn wounds are part of a phenomenon rather than an isolated wound. Although the healing process of the burn wound progresses in a fashion similar to other wounds there is a large interplay of systemic factors in this process. The burn wound is directly affected by the general condition of the patient and also has a direct impact on the same leading to conditions such as septicaemia and death. Burn wounds are a direct reflection of the management of the burn patient. Healing in these wounds is reciprocative of the general condition of the patient. Burn wounds are similar to other wounds in the fact that the basics of wound healing and care remain the same but are dissimilar in the fact that they have a more profound impact on the general status of the of the patient playing a key role in the ultimate survival, development of deformity and rehabilitation of the patient

REFERENCES

- Mohil RS. Classification of wounds. In: Sarabahi S, Tiwari VK, editors. Principles and practice of wound care. 1st ed. New Delhi (India): Jaypee Publishers; 2012. p. 42-52.
- Bhattacharya S. Burns: Etiology and classification. In: Sarabahi S, Tiwari VK, Goel A, editors. Principles and practice of burn care. 1st ed. New Delhi (India): Jaypee Publishers; 2010. p. 25-36.
- Vartak A: Pathophysiology of Burn shock. In: Sarabahi S, Tiwari VK, Goel A, editors. Principles and practice of burn care. 1st ed. New Delhi (India): Jaypee Publishers; 2010. p. 37-41.
- Deodhar AK, Rana RE. Surgical physiology of wound healing: A review. J Postgrad Med 1997;43:52-6.
- Ethridge RT, Leong M, Phillips L. Wound healing. In: Touensend CM, Beauchamp RD, Evers BM, Mattox KL, editors. Sabiston Textbook of surgery. The biological basis of modern surgical practice. 18th ed. Philadelphia: Saunders; 2009. p. 191-216.
- Arturson G. Cross reference from 'Local effects: Principles and Practice of burn management. 1st ed. Settle JAD, editor. New York: Churchill Livingstone; , 1996.
- Werner S, Grose R. regulation of wound healing by growth factors and cytokines. Physiol Rev 2003;83:835-70.
- Kumar V, Abbas AK, Fausto N, Aster JC. Tissue renewal, repair and regeneration. In: Kumar V, Abbas AK, Fausto N, Aster JC, editors. Robbins and Cotran. Pathologic basis of diseases. 8th ed. Pennsylvania: Saunders; 2009. p. 191-216.
- Sephel GC, Woodward SC. Repair, regeneration and fibrosis. In: Rubin E, Gorstein F, Rubin R, Schwarting R, Strayer D, editors. Rubin's pathology. Clinicopathologic foundations of medicine. 4th ed. Philadelphia: Lippincott Williams and Wilkins; 2001. p. 85-116.
- Chandrakant SS. Medicolegal aspects in burn injuries. In: Sarabahi S, Tiwari VK, Goel A, editors. Principles and practice of burn care. 1st ed. New Delhi (India): Jaypee Publishers; 2010. p. 516-23.
- Caison's JS. Treatment of burns. London: Chapman and Hall; 1981. p. 14-57.
- Williams WG, Phillips L. Pathophysiology of burn wound. In: Herndon DN, editor. Total burn care. WB Saunders Co Ltd.; 1996. p. 64.
- Jensen AR, Hughes WB, Grewal H. Secondary abdominal compartment syndrome in children with burns and trauma: A potentially lethal complication. J Burn Care Res 2006;27:242-6.
- Greenhalgh DG, Warden GD. The importance of intra-abdominal pressure measurements in burned children. J Trauma 1994;36:685-90.
- Ivy ME, Atweh NA, Palmer J, Possenti PP, Pineau M, D'Aiuto M. Intra-abdominal hypertension and abdominal compartment syndrome in burn patients. J Trauma 2000;49:387-91.
- Hobson KG, Young KM, Ciraulo A, Palmieri TL, Greenhalgh DG. Release of abdominal compartment syndrome improves survival in patients with burn injury. J Trauma 2002;53:1129-33; discussion 1133-4.
- Latenser BA, Kowal-Vern A, Kimball D, Chakrin A, Dujovny N. A pilot study comparing percutaneous decompression with decompressive laparotomy for acute abdominal compartment syndrome in thermal injury. J Burn Care Rehabil 2002;23:190-5.
- Kumar P. Surgical excision of burn wound and skin grafting. In: Sarabahi S, Tiwari VK, editors. Principles and practice of wound care. 1st ed. New Delhi (India): Jaypee Publishers; 2012. p. 196-207.
- Verma PK. Anaesthesia for the thermally injured. In: Sarabahi S, Tiwari VK, editors. Principles and practice of wound care. 1st ed. New Delhi (India): Jaypee Publishers; 2012. p. 208-22.
- Gamer JP, Jenner J, Parkhouse DA. Prediction of upper airway closure in inhalation injury. Mil Med 2005;170:677-82.
- Gueugniaud PY, Carsin H, Bertin-Maghit M, Petit P. Current advances in the initial management of major thermal burns. Intensive Care Med 2000;26:848-56.
- Haponik EF. Respiratory injury. In: Haponik EF, Munster AM, editors. Smoke inhalation and burns. New York: McGraw-Hill Inc.; 1990.
- Haponik EF, Summer W. Respiratory complications in burned patient: Pathogenesis and spectrum of inhalation injury. J Crit Care 1987;2:49.
- Pruitt BA Jr. Advances in fluid therapy and the early care of the burn patient. World J Surg 1978;2:139-50.
- Pruitt BA Jr. The diagnosis and treatment of infection in the burn patient. Burns Incl Therm Inj 1984;11:79-91.
- Pruitt BA Jr, McManus AT. The changing epidemiology of infection in burn patients. World J Surg 1992;16:57-67.
- Becker WK, Cioffi WG, McManus AT. Fungal burn wound infection. Arch Surg 1991;126:44-8.
- Sarabahi S, Tiwari VK, Arora S, Capoor M, Pandey A. Changing pattern of fungal infection in burn in a large burn unit in Asia. Burns 2012;38:520-8.
- Arturson G. Neutrophil granulocyte function in severely burned patients. Burns Incl Therm Inj 1985;11:309-19.
- Schmidt K, Bruchelt G, Kistler D, Koslowski L. phagocytic activity of granulocyte and alveolar macrophages after burn injury measured by chemiluminescence. Burns Incl Therm Inj 1983;10:79-85.
- Stephan RN, Ayala A, Harkema JM, Dean RE, Border JR,

- Chaudhry IH. Mechanisms of immunosuppression following hemorrhage: Defective antigen presentation by macrophages. *J Surg Res* 1989;46: 553-56.
32. Zembola M, Uracz W, Ruggiero I. Isolation and functional characteristics of FcR+ and FcR- human monocyte subsets. *J Immunol* 1984;133:1293-9.
 33. Daniels JC, Larson DL, Abston S, Ritzmann SE. Serum protein profiles in thermal burns. *J Trauma* 1974;14:137-52.
 34. Munster AM, Hoacland HC, Pruitt BA Jr. The effect of thermal injury on serum immunoglobulin. *Ann Surg* 1970;172:965-9.
 35. Teodorczyk JA, Sparkes BG, Peters WJ. Regulation of IgM production in thermally injured patients. *Burns* 1989;15:241-7.
 36. Saffle R, Schnebly WA. Burn wound care. In: Richard RL, Staley MJ, editors. *Burn care and rehabilitation: Principles and Practice*. Chapter 7. Philadelphia: FA Davis Company; 1994. , p. 119-67.
 37. Monafro WF, Fredman B. Topical therapy for burns. *Surg Clin North Am* 1987;67:133-45.
 38. Moncreif JA, Lindberg RB, Switzer WE, Pruitt BA. Use of topical antibacterial therapy in the treatment of burn wound. *Arch Surg* 1966;92:558-65.
 39. Pruitt BA Jr. Diagnosis and treatment of infection in the burn patient: Presidential address. *Arch Surg* 1986;121:13-22.
 40. Lindberg RB, Moncreif JA. The successful control of burn wound sepsis. *J Trauma* 1965;5:601-16.
 41. Stone HH, Kolb LD, Petit J, Smith RB. The systemic absorption of antibiotic from the burned wound surface. *Am Surg* 1968;34:639-43.
 42. Goel A. Post burn sequelae and their management. In: Sarabahi S, Tiwari VK, editors. *Principles and practice of wound care*. 1st ed. New Delhi (India): Jaypee Publishers; 2012. p. 468-515.

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