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REVIEW

Wounds: an overlooked burden (Part 4) – Burn wounds: a searing situation

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Burn injuries are a leading cause of deformities and death globally, and in developing countries, the recurrence of burn incidents increases the financial burden on health systems. Appropriate management of burn wounds with timeous referral of patients to burn wound experts must be prioritised to facilitate recovery and prevent complications. This article describes common characteristics of burn wounds based on their aetiology and severity to help the treating clinician determine the appropriate treatment protocol to be applied. Treatment strategies according to burn wound aetiology have been summarised into downloadable reference tables for use in everyday practice.

Keywords: burn wounds, burn injuries, burn severity, burn treatment, burn depth, referral

Introduction

Burn injuries are a global health issue and a leading cause of deformity and death. According to the World Health Organization (WHO), approximately 180 000 annual deaths can be attributed to burn injuries, with an estimated two-thirds occurring in lowand middle-income countries within the WHO African and South-East Asia regions.¹ In South Africa, burn incidents from kerosene cooking stoves are still a common occurrence, and annual costs of US\$26 million have been reported due to the accumulation of indirect costs.¹ In any burn wound situation, indirect costs such as lost wages, prolonged care for deformities and emotional trauma may arise, which affects patient quality of life.^{1,2}

Structure of the skin and burn wounds

The skin functions primarily as a barrier against the environment providing first-line defence against invading pathogens but also has several important homeostatic functions. Burns, surgical wounds or trauma result in compromised skin integrity and function that can be life-threatening. The skin consists of three main layers: the outer epidermis, the supporting dermis and the hypodermis or subcutaneous adipose tissue layer that connects the skin to the underlying structures (Figure 1).^{3,4}

A burn is defined as the destruction of the epidermal, dermal or deeper tissues due to contact with thermal, chemical, or electrical agents.⁴ The skin loses its functions when it is burned, and the ensuing damage may critically affect the patient's health due to burn depth, location of the burn, fluid loss, the presence of the causative agent, infection and metabolic circumstances.⁴

Burn wound aetiology and severity

Burn wounds can be grouped as thermal, chemical, electrical or radiation burns according to their aetiologies. Knowing the aetiology helps the treating clinician determine an appropriate treatment protocol to be applied.⁴

Thermal burns

Thermal burns are caused by excessive heat, typically from contact with hot surfaces, hot liquids, steam, or flames. Damage to the skin results in cellular death due to temperature and length of contact time. Respiratory burns may occur alone or as a consequence of thermal burns by the inhalation of smoke or steam following exposure to fire, hot air or toxic fumes.⁴

Extremely low temperatures (-22 °C and below) can also cause irreversible damage to the skin. This may be due to prolonged exposure to cold environments, ice packs, dry ice or lengthy spraying with an aerosolised spray.⁵ Damage to the skin is caused by ice crystals forming in the intracellular and extracellular fluid, inhibiting enzyme systems and causing tissue destruction.^{4,6} The associated vasoconstriction, endothelial injury and throm-

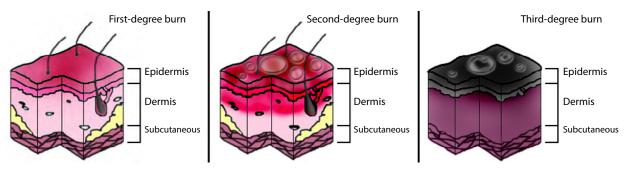


Figure 1: Structure of the skin and degrees of burn wounds³

boembolism contribute to vascular insufficiency and ischaemia. When the skin thaws, cellular damage, loss of endothelial integrity, thrombosis and oedema occur due to melting water crystals and gradually, necrosis develops as evidenced by frostbite.⁶

Radiation burns

Radiation burns include thermal radiation, ultraviolet (UV) light, ionising radiation, and radiofrequency energy. These burns are caused by high radiation doses (8-10 Gy), which cause sudden cell death.⁴ Sunburn is the most common form of radiation burn and is caused by exposure to the UV A and B rays of the sun or artificial sources like tanning beds. UV B rays, in particular, can cause DNA damage which initiates a DNA repair response in the body resulting in vasodilation, oedema, and pain, causing the red, painful skin typically seen in sunburn.7 Another source of radiation burns are those resulting from cancer radiotherapy, a treatment applied in ~ 70% of patients with malignant tumours.8 Radiotherapy results in radiation-induced skin reactions (RISRs) which are caused by inflammatory and oxidative stress responses. RISRs may be acute or chronic, depending on the severity of the reaction. Acute wounds normally present rapidly, whereas chronic wounds may appear several weeks to months after exposure.8

Electrical burns

Electrical burns are caused by contact with electrical sources or, in rare circumstances, by a lightning strike. High-energy current travels through the body and injuries occur due to either the flow of current through the body, arc flash, or clothing that catches fire. With the former two, the body converts electricity to heat, resulting in a thermal burn.9 Electrical burns can be divided into high (> 1 000 volts) and low (< 1 000 volts) voltage burns. Low voltage injuries usually affect the skin and seldom cause burns that are more serious than a small deep thermal burn at the site of injury.¹⁰ High voltage burns cause extensive deep tissue damage along the path of the current as it travels through the body. This may cause extensive muscle necrosis and may produce renal shutdown, and severe oedema resulting in "compartment syndrome". Muscle necrosis in the extremities also makes them susceptible to anaerobic infections, such as clostridia. Contractions of the muscles during injury often result in bone fractures, especially in the cervical vertebrae and long bones.¹¹ Electrical injury also affects the conductivity of the

heart, resulting in arrhythmias from the onset of injury, making cardiac monitoring vital.¹¹

Chemical burns

Chemical burns occur when highly corrosive substances such as acids, alkalis, heavy metals, and toxic gases come into contact with skin or mucous membranes, causing localised tissue necrosis. Chemical burns are usually caused by strong acids or alkalis. Acidic compounds result in tissue damage by denaturing and coagulating proteins in the skin.^{12,13} The acid tends to "tan" the skin, forming an impermeable barrier limiting further penetration of the acid.¹⁰ Alkalis combine with cellular lipids in the skin to create a detergent that causes the skin cells to dissolve, resulting in necrosis. This process continues until the alkali is neutralised.^{10,12} Full-thickness chemical burns may appear deceptively superficial in the first few days, causing only a mild brownish discolouration of the skin, after which it will spontaneously slough.¹⁰ The extent of the damage caused by a chemical agent is directly proportional to the concentration and quality, mechanism of action, extent of penetration and duration of exposure to the chemical agent. Limiting the exposure time will reduce damage and improve outcomes.13

Location/depth/degrees

The severity and healing potential of a burn is determined by the depth and the width of the area.4,14 Burn depth is categorised by the extent to which the layers of the skin are injured. This characterisation is done using different zones which correlate to the exposure and extent of damage, namely the zone of coagulation, the zone of stasis and the zone of hyperaemia. The zone of coagulation is the central region of severe burns and the point of maximum damage, where irreversible tissue loss occurs due to destruction or coagulation of structural proteins. The zone of stasis is located along the periphery of the zone of coagulation and is characterised by decreased tissue perfusion and this can be salvageable. However, additional stressors such as hypotension, infection or oedema can convert the zone of stasis into an area of complete tissue loss, leading to an increase in wound depth and width. In the zone of hyperaemia which is the outermost zone, tissue perfusion is increased and there is a high probability for tissue recovery in this region, unless there is severe sepsis or prolonged hypo-perfusion.4,15,16

The depth of burns provides the basis by which burns are classified, and important factors to consider in assessing depth

Table I: Clinical presentation of burns based on classification^{14,17-20}

Degree	Classification	Clinical findings	Layer of skin affected
First	Superficial burn	Redness, pain	Outer layer/epidermis
Second	Superficial partial-thickness burn	Redness, blisters, pain, blisters blanche with pressure	Epidermis Papillary dermis
	Deep partial-thickness burn	Yellow, white or purple-white appearing blisters, redness, oedema, extreme pain	Epidermis Reticular dermis
Third	Full-thickness burn	Black, brown or white tissue, no blisters, no pain, leathery appearance	Epidermis Entire dermis
Fourth	Full-thickness burn	Charred skin, exposure of deep tissue (muscle, bone), nerve destruction	Epidermis, entire dermis, fat, fascia, muscle, bone

include appearance, blanching to pressure, extent of pain and sensation. These factors also indicate thickness of burns which aids in classifying them as either partial- or full-thickness wounds.¹⁷ These are further sub-categorised according to the layer of skin involved from which the different burn degrees are derived as indicated in Table I.¹⁸

Burn management

Assessment

It is important for a burn depth assessment to be conducted timeously and accurately to ensure efficient burn wound management and reduced complications. Burn depth assessment can be challenging in partial-thickness wounds due to progressing inflammation in deep dermal wounds, which may result in inaccurate clinical evaluations by experienced clinicians.^{21,22} In most cases, clinical symptoms are identified and utilised together with tools to determine burn depth and the total surface area of the body that has been injured/burned (TBSA), which aids in evaluating the severity of burns. These tools include the Rule of Nines, palmar method, Lund and Browder Chart and Artz's criteria as depicted in Figure 2 and Table II. Newer tools such as the Laser Doppler Imaging system have become more popular in determining burn depth.^{20,23,24} With this tool, the depth of tissue injury is determined using blood flow as an indirect measure since blood flow is compromised in deep dermal burns where the dermal vasculature is damaged.²⁰ Internationally, mobile 3D application tools are also gaining popularity for burn assessment. This clinical evaluation helps to differentiate superficial burns from full-thickness burns which is important for selection of the appropriate burn management strategy.14

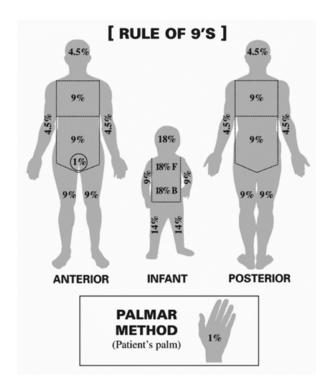


Figure 2: Assessment tools used to determine total body surface area of burns^{25,26}

 Table II: Artz's criteria to determine burn wound severity based on total body surface area^{27,28}

Artz's criteria to determine burn wound severity

Severe burns

- Second-degree burns over at least 30% TBSA
- Third-degree burns over at least 10% TBSA
- Third-degree burns of the face, hands, or feet
- Burns complicated by respiratory tract burns
- · Burns complicated by soft tissue damage or fractures
- Electric shock
- Moderate burns (requiring inpatient care at a general hospital)
- Second-degree burns over 15–30% TBSA
- Third-degree burns over < 10% TBSA (excluding the face, hands and feet)

Minor burns (may be treated on an outpatient basis)

- Second-degree burns of < 15% TBSA
- Third-degree burns of < 2% TBSA

Wound bed preparation and treatment

Burn treatment involves a few simple principles that improve outcomes. These include quick cooling of the burn, adequate cleansing or debridement, fluid resuscitation, addressing nutrition, appropriate dressings and pain management.²⁹ The first step in burn wound management involves immediate appropriate cooling of the burned area to reduce damage. This is achieved by running cool water or saline over the burn for at least 20 minutes. Ice is not used for cooling as this could cause further damage due to vasoconstriction. While cooling the burn, clothing and jewellery should be removed before the burned area becomes inflamed.^{19,30} Once the burn has been sufficiently cooled it can be covered with a transparent film, especially if the patient needs to be transferred to hospital. It is recommended to cleanse the burn wound thoroughly using a non-toxic isotonic solution or a mild antibacterial wash, as this will allow the clinician to establish burn depth and TBSA. To aid the assessment of the depth and extent of the burn, deroofing/debriding of blisters is advised.19

Once the wound is clean, fluid resuscitation is imperative due to the subsequent inflammatory response. This involves intravenous replacement of fluids in burn patients to replenish fluids lost due to hypovolaemia and hypoperfusion resulting from oedema.³¹ The main aim of burn resuscitation is to increase tissue perfusion and prevent irreversible damage.¹⁸ Additionally, patients require nutritional support to meet the demands placed on the body by the hypermetabolic response to a severe burn injury.³² This response is characterised by hyperdynamic circulation as well as metabolic, physiologic, catabolic and immune system dysfunctions. Nutritional support satisfies the increased energy and protein demands which may cause multi-organ dysfunction, increased susceptibility to infection, or death.³³ In severely burned patients who are unable to feed orally, nutrition is provided enterally (nasogastric, gastric, or intestinal tubes) or parenterally (intravenous infusion).32

Dressings and topical agents may be applied depending on the aetiology and severity of the burn wound, to prevent or control infection or enhance wound healing.³⁴ Superficial burns

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Type of wound	Image	Characteristics	Treatment strategy	Dressing
Thermal burr	ns caused by heat			
Superficial thickness		 Involves the epidermis First-degree: burn site appears red and is painful 	 Deroof blisters to aid in assessment of depth Prevent infection Protect skin Manage pain 	 Hydrogel sheet Hydrogel impregnated dressing Polyurethane foam Soft silicone foam Silver containing Hydrofiber*
Superficial partial		 Involves the epidermis and part of the dermal layer of skin (papillary dermis) Second-degree: the burn site appears red, blistered and may be swollen and painful Blisters may blanche with pressure 	 Deroof blisters Debride sloughed skin Prevent infection Promote moist wound healing Manage pain 	 Soft silicone dressings Foam dressings Hydrophobic dressing to control bioload if indicated
Deep partial		 Involves epidermis, dermis (reticular) Second-degree: yellow, white or purple-white appearing blisters, redness, oedema, extreme pain 	 Surgical excision of necrotic tissue if possible Resurfacing (covering exposed wound using grafts, skin flaps, biological substitutes) Prevent or treat infection Promote moist wound healing Manage pain 	 Hydrogel Polyurethane foam Soft silicone foam Silver containing Hydrofiber[®]
Full-thickness		 Epidermis, entire dermis, fat, and may include fascia, muscle, bone Third-degree: black, brown or white tissue, no blisters, no pain, leathery appearance Fourth-degree: charred skin, exposure of deep tissue (muscle, bone), nerve destruction 	 Facilitate wound healing Decrease the risk of hypertrophic scarring Prevent infection and reduce mortality Usually treated with skin grafting Early eschar excision Resurfacing (covering exposed wound using grafts, skin flaps, biological substitutes) 	 Type of dressing dependent on type of graft or resurfacing technique employed Foam, hydrocolloid, alginate, paraffin gauz silicone, antimicrobial (if required), negative pressure dressing (if required)
Thermal burr	ns caused by cold			
Frostbite ⁴³		 Usually seen on ears, nose, and fingers Ischaemia developed in the tissue spreads to the body First-degree frostbite: reflex erythema, vasoconstriction and paleness Second-degree frostbite: erythema, oedema, and subepidermal bullae Third-degree frostbite: blue-black colour changes and hardening 	 Remove wet/cold clothing Assess for hypothermia (core temperature raised to 35 °C) Re-warm frostbitten areas slowly in warm water (40–41 °C) with antiseptic Manage pain Debride or aspirate blisters Elevate and splint affected body part, especially limbs to reduce oedema and promote tissue perfusion Amputation should be avoided unless there is wet gangrene, liquefaction or spreading sepsis Do not rub affected area Tetanus prophylaxis Ibuprofen (prostaglandin inhibition) Topical aloe vera gel/cream to soothe frostbite Tissue plasminogen activator, heparin, thrombolytic agents or prostacyclin analogues (iloprost) have shown to reduce the loss of limbs and digits following severe frostbite Systemic antibiotics only in the 	 Area should be covered with a loose, protective dressing with padding Protective padded dressing to separate affected toes or fingers to promote tissue perfusion

Table III: Treatment of thermal burns^{4-6,14,17-20,39-42}

or cellulitis

presence of proven infection, trauma

can be treated with non-adherent, non-occlusive dressings but prophylactic topical ointments/creams such as silver sulfadiazine are not recommended as they have been associated with less favourable outcomes. However, deep burn wounds require dressings in combination with topical antimicrobial prophylaxis to prevent colonisation of the wound. If wounds do not reepithelialise within 14 to 21 days, surgical interventions should be employed to avoid severe hypertrophic scarring.^{35,36}

Due to the pain that may be experienced by burn patients, adequate pain management is vital to promote healing and

should be in concordance with the treatment plan. Pain can be managed by over-the-counter or prescription medications such as paracetamol, anti-inflammatories, anxiolytics and opiates depending on the pain levels. Prior to administering complex analgesics, simpler ones in appropriate doses and intervals should be used.³⁷ The treatment strategies that are employed in the management of burns are highlighted in Tables III–V and detailed information about the dressings used are discussed in part two of this series.³⁸

Type of wound	Image	Characteristics	Goals	Dressing
Burn caused b	y radiation			
Superficial thickness	J.	 Affect only the epidermis, or outer layer of skin (superficial thickness) Red, painful, dry, with no blisters Mild sunburn is an example Long-term tissue damage is rare and usually causes change in skin colour 	 Usually heal on their own within a week Cold compresses - cold running water 10-15 min Cooling lotion that contains aloe Pain medication, i.e. NSAIDs Treatment is determined by: Age and patient profile Extent of the burn Location of the burn Cause of the burn Tolerance for specific medications, procedures, or therapies 	• Hydrogel
Superficial partial		 Redness (erythema) with pruritis and dry desquamation 	 Protect skin Prevent infection 	• Hydrogel
Deep partial⁴		Blisters and moist desquamation	 Protect skin Prevent infection Manage moisture 	 Hydrogel sheet Soft silicone dressings
Electrical burn	l			
Flash injury		 Entry and exit points with possible tissue damage between these points due to the flow or current Third- or fourth-degree burns with complete necrosis of skin and sometimes necrosis of the tissues below skin such as fascia, nerves, muscles, tendons, vessels and bones Surface areas usually not more than 1% of TBSA 	 Intravenous antibiotics to treat anaerobic infection and early debridement to create aerobic environment Skin necrosis should be evaluated every 24–48 h and debrided as required Debridement should be conducted every 48 h until tissue is revitalised; thereafter wound coverage is recommended Minor electrical burns can be treated by application of cream or ointment to soothe skin High voltage shocks require cardiac monitoring 	 Avoid using wet dressings Dressings should be chosen according to severity of the electrical burn See thermal burns

Table V: Treatment of chemical burns ^{10,12,13,45,46}				
Type of wound	Image	Characteristics	Goals	Dressing
Chemical b	urn caused by acid			
Acid burn		Acids cause coagulative necrosis	 Immediate treatment is more important than establishing the type of chemical Lavage with a sufficient volume of water Remove all contaminated clothing Acids can be neutralised with sodium bicarbonate If contact substance is in powder form or a metal, it should be removed prior to irrigation with water 	 Neutralisation is vital Debridement of necrotic tissue Hydrogel sheet
Chemical b	urns caused by alkali			
Alkali burn⁴⁵		 Alkali burns result in coagulative or liquefication necrosis 	 Alkali can be neutralised with 0.5% acetic acid or ammonium chloride Irrigation with copious amounts of water could aid in reducing damage Wound may require a surgical debridement and grafting 	 Dressings should be chosen according to severity of the burn Wound may be loosely covered with a sterile gauze bandage following irrigation

Table V: Treatment of chemical burns^{10,12,13,45,46}

Referral criteria

Burn wounds that are more severe than minor superficial wounds should be considered for referral to a specialist, as this is associated with improved outcomes for the patient. Burns meeting the following criteria should be referred to a specialist burns unit based on the aetiology, severity, and location of burns.¹⁹

- 1. Burns greater than 10% of TBSA
- 2. Burns in special areas face, hands, major joints, feet and genitalia
- 3. Full-thickness burns
- 4. Electrical burns to allow full assessment
- 5. Chemical burns to allow full assessment
- 6. Circumferential burns of limbs or chest
- 7. Burns in children and elderly
- 8. Burn injury in patients with pre-existing medical disorders (or disability) which could complicate management, prolong recovery, or increase risk of mortality
- 9. Burns with associated inhalation injury
- 10.Any burn patient with concomitant trauma

Patient education or health dialogue

Patient education is vital to ensure optimal wound healing. Clinicians should discuss appropriate diets and patient lifestyle changes, while highlighting the importance of continued medical surveillance. Patients and their families should be educated on wound characteristics and warning signs that would warrant a visit to their specialist or the emergency room.

Conclusion

Early response and accurate assessment, appropriate treatment and dressing choices as well as pain management are key principles in burn wound care. Timeous referral to a burns unit or wound care specialists is important, as wound healing is a multifaceted process that takes time and dedication from the clinical support team and the patient with improved outcomes for serious burns or wounds requiring specialised care. Continuous monitoring is essential to ensure effective wound healing and the importance of monitoring and patient involvement should be discussed with burn patients to prevent wound regression.

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Conflict of interest

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