Burn wound management: a surgical perspective

Cameron AM, Ruzehaji N & Cowin AJ

Abstract

Any patient who survives a large burn injury will be left with some degree of scarring. As well as affecting the form and function of the skin, scarring can have severe psychological consequences such as post-traumatic stress disorder and depression [1]. This is particularly the case for hypertrophic or keloid scars, which are common after serious burns. Despite this, the process underlying their formation is incompletely understood and limited effective options are available for their treatment. This paper reviews current understanding of the pathophysiology of the wound healing process in relation to burns and reviews the current management for burn wounds.

Introduction

Major burn injury is probably the most painful and devastating trauma a person can sustain and survive. Burns patients often require multiple surgical episodes and dressing changes followed by prolonged rehabilitation and victims can be left with lifelong dysaesthetic scarring and potential dysfunction. The scale of this clinical problem is demonstrated by the statistics from the AIHW National Hospital Morbidity Database, Australia's Health 2004. During the period of 2001-02, throughout Australia, burns and scalds were responsible for 6,248 hospitalisations in public hospitals with the average length of stay being 7.1 days entailing an estimated cost of $132 million. Despite this, scarring is an area of largely unmet medical need.

Alex Cameron MBBS, B. Med Sci
Women's & Childrens' Health Research Institute, 72 King William Road North Adelaide, South Australia. 5006

Nadira Ruzehaji B App Sc (Podiatry), BSc Hons
Women's & Childrens' Health Research Institute, South Australia

Allison Cowin PhD
Women's & Childrens' Health Research Institute, South Australia

The Wound Healing Process

Any consideration of burn wound healing must bear in mind the general principles of the wound healing process. The fundamental biological and molecular events after cutaneous injury cannot be separated and categorised in a clear-cut way. However, it has been useful to divide the repair process into four overlapping phases of coagulation, inflammation, migration-proliferation (including matrix deposition), and remodelling. These phases are shown in **Figure 1**, which also highlights the main events during each phase and the key types of cells implicated.

Pathophysiology of the burn wound

The pathophysiology of the burn wound is related to the initial distribution of heat onto the skin, which is a function of both the temperature and the exposure time i.e. a high temperature for a short time may cause the same tissue damage as a lower temperature for a longer time [2]. Jackson [3] described three zones of histopathological injury: coagulation, stasis and hyperaemia. The zone of coagulation is comprised of eschar or necrotic tissue and is closest to the heat source. This is surrounded by the zone of stasis, where there is only moderate tissue damage, but slow blood flow and oedema due to capillary leakage and cell membrane disruption [4 5]. Poor blood flow in this zone may lead to local tissue ischaemia and further necrosis [6]. Surrounding the zone of stasis is the zone of hyperaemia, in which cell damage is minimal and blood flow gradually increases resulting in early

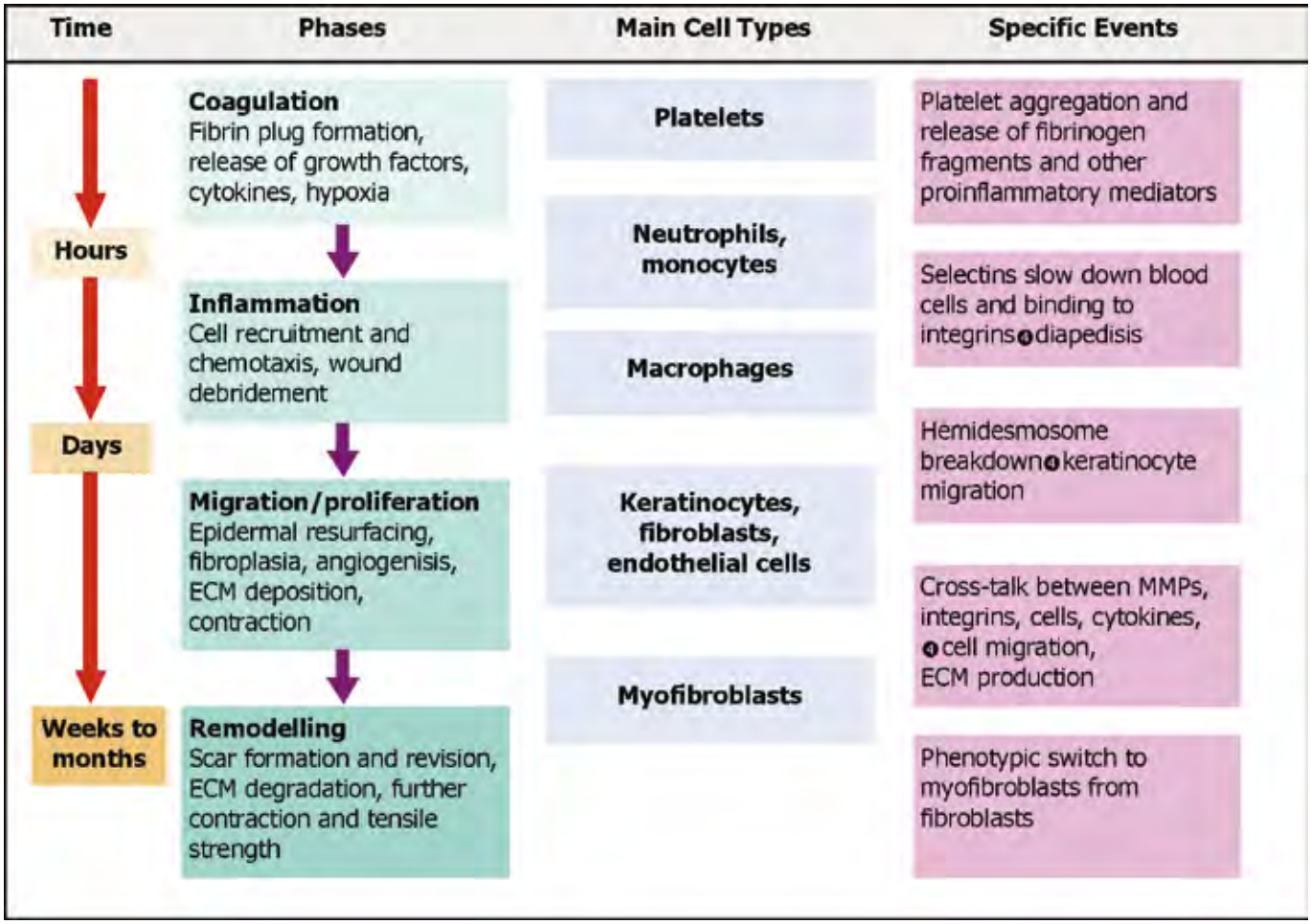

Figure 1. Phases of wound healing, major types of cells involved in each phase, and selected specific event. Adapted from Falanga, (2005).

spontaneous recovery. Burn depth may increase over 48-72 hours, as the zone of stasis becomes necrotic [7].

Superficial burns, such as sunburn, affect the epidermis only and are painful. They will heal completely within 3-5 days.

Partial thickness burns involve the entire epidermis and portions of the dermis. They are divided into superficial partial and deep partial thickness.

Superficial partial burns extend to the superficial dermis and are pink, moist and painful to touch. Blistering is common as a result of serum accumulating between the superficial dermis which has detached from the deep dermis. They will usually heal in two weeks without a scar by regeneration of epidermis from keratinocytes within sweat glands and hair follicles [8]. Burns from water scald are a common example of superficial partial burns.

Deep partial thickness burns involve the entirety of the epidermis and extend to the reticular layer of the dermis. They are a mottled white-pink colour and have variable sensation due to destruction of superficial cutaneous nerves. These wounds may heal within 3 weeks but are at risk of fibroproliferative disorders.

Full thickness wounds involve the entire epidermis and dermis. They are brown-black, leathery and insensate. Healing will be extremely slow and associated with marked contraction.

Management of the burn wound

This article will focus on the management of the burn wound itself; however, it should be noted that a multi-disciplinary approach to burn management is essential for optimal functional and cosmetic outcome [9]. This should include the following team members: anaesthetist, surgeon, nurses, physiotherapist, occupational therapist, psychologist, dietitian, social worker and rehabilitation specialist.

Assessment of burn depth

The depth of the burn wound is the most important determinant of its healing potential and appropriate management [10]. Thus, accurate assessment of this is vital. Clinical evaluation by the clinician using the characteristics described above for different burn thicknesses is the most widely used and least expensive method of assessing burn depth. However, it is accurate in only 2/3 of cases and has poor inter-observer reliability [11]. Other methods such as biopsy, thermography, videography with indocyanine green with laser fluorescence and laser Doppler perfusion imaging are also available [12]. Of these, only laser Doppler has been approved for clinical use. It has been shown to predict with 95% accuracy burns that will heal before 14 days, burns that will heal between 14 and 21 days and burns that will not heal within 21 days [13].

Superficial burns

Superficial burns do not require any specific therapy to aid healing but topical non-steroidal anti-inflammatory drugs or aloe vera may be used to reduce discomfort.

Superficial partial thickness burns

Superficial second-degree burns should be treated with a topical antimicrobial agent or an absorptive occlusive dressing.

The general principles of wound management with dressings are as follows [14]:

- Treat and prevent infection (reduce colonization by micro-organisms);
- Cleanse the wound to remove debris and facilitate the body's repair process;
- Debride the wound to remove necrotic or dead tissue and foreign matter;
- Provide an optimal healing environment by ensuring a degree of moisture at the wound surface; and
- Relieve pain and discomfort

Burn Wound Dressings

The range of dressing options is innumerable and differences between trade names in different regions can cause confusion. The Australia-New Zealand Burns Association (ANZBA) provides the following overview:

1. **Retention Dressings**: Low profile dressings used to assist adherence of other dressings or as primary dressing for superficial (minimally exudating) wounds e.g. *Fixomull®*
2. **Hydrocolloids**: Low profile, waterproof, highly conformable, wound interactive dressing e.g. *Duoderm®*
3. **Alginates**: Haemostatic dressing for moderately exudating or bleeding / oozing wounds e.g. *Sorbsan®, Algisite®.*
4. **Topical Anti-microbial Dressings**: Very important for burn wounds – used for control of or elimination of infectious organisms in the wound. May be used in various forms such as creams e.g. SSD, dressings / sheets e.g. *Acticoat®, Aquacel Ag®*, formulations e.g. Betadine® soaked gauze, AgNO3 soaks, sulfamylon soaks.
5. **Foams**: Used to control moderate to highly exudating wounds or protect fragile healed or almost healed areas e.g. *Mepilex®, Allevyn®*. Provided in thick and thin varieties.
6. **Hydrogels**: Used to maintain or introduce moisture into a wound. May be used to protect and hydrate exposed tendons or bone. Provided in liquid e.g. *Intrasite®* or sheet e.g. *Clearsite®* forms.
7. **Combination Dressings** e.g. *Combiderm®, Acticoat Absorbent®*
8. **Absorbent Dressings**: Used to mop up heavily exudating wounds e.g. combine, *Zetuvit®*, gauze, *Exu-Dry®*.
9. **Non-stick Dressings**: Used as anti-shear layers in dressing systems e.g. *Jelonet®, Melolin®.*
10. **Wound Growth Factor Impregnated Dressings**: Low profile, interactive, (relatively expensive) dressings / films which are often used acutely to reduce / prevent wound progression e.g. *Transcyte®, Biobrane®.*
11. **Biological Dressings**: Preparations used to provide another (potentially less expensive) option for introducing growth factors onto the wound e.g. xenograft (commonly pigskin), allograft (cadaver skin).
12. **Films**: Low profile, waterproof, highly conformable, adhesive dressing. Often used to secure IV cannulae.

Deep dermal burns/Full thickness burns

For any burns that extend to the deep dermis or beyond, excision and split skin grafting is required. Excision of the eschar removes necrotic and inflamed tissue which would otherwise act as a nidus for infection and retard wound healing [15]. Grafting minimises fluid loss and protects the wound against infection. New substances are now available to augment the traditional surgical approach. These include cultured epithelial autograft (CEA) and synthetic dermal templates such as *Integra*®.

Management of hypertrophic and keloid scars

It is difficult to differentiate between hypertrophic and keloid scars in their early phases. Phenotypic differences between the two will not become evident until the keloid has invaded adjacent normal tissue [16]. In terms of natural history, hypertrophic scars will tend to regress over time, albeit leaving a widened gap of thinned dermis between wound edges. Keloid scars will not regress, but rather grow to a certain size at which they will remain indefinitely [17].

Management of these scars is a major challenge in burn care. Treatments include:

Local administration of corticosteroid or non-steroidal anti-inflammatory drugs (NSAIDS) which interfere with the inflammatory process.

Occlusive dressings, such as elastic pressure wrap or silicone gel sheeting, whose exact mode of action is unknown, but is thought to involve accelerating scar degradation rate.

Calcium antagonists (e.g. verapamil) increase collagenase activity and scar tissue degradation.

Surgery: only indicated to under certain conditions e.g. very large scars unlikely to respond to medical therapy or scars impairing musculoskeletal function. It should be noted that the rate of recurrence of both hypertrophic and keloid scars is high following surgery.

Summary

In summary, burn wound management remains an area of great clinical challenge. Understanding the processes involved in burn wound healing and applying the appropriate wound management response is imperative to aid healing of the burn and reduce the risk of scarring. The remodelling stage of wound healing is associated with the formation of fibrous tissue or the scar and is considered to be the longest and least understood stage of healing. Pathologic fibrosis and scarring often lead to poor functional and aesthetic results, hence understanding the underlying cellular and molecular mechanisms involved in scar formation allows the surgeon and other health care practitioners to better address these issues, and suggests new avenues to explore therapeutic modalities designed to target the various biologic causes of dysregulated wound healing and abnormal scarring. As Australian health care becomes increasingly focused on providing multidisciplinary burn wound care, which in turn has been associated with dramatically improved outcomes for burn victims, we can look forward to more optimal burn care and improved patient outcomes.

References

1. Van Loey NE, Faber AW, Taal LA. Do burn patients need burn specific multidisciplinary outpatient aftercare: research results. Burns 2001;27:103-10.
2. Moritz AR, Henriques FC. The relative importance of time and surface temperature in the causation of cutaneous burns. Am J Pathol 1947;23:695-720.
3. Jackson DM. The diagnosis of the depth of burning. Br J Surg 1953;40:588-96.
4. Despa F, Orgill DP, Neuwalder J, Lee RC. The relative thermal stability of tissue macromolecules and cellular structure in burn injury. Burns 2005;31:568-77.
5. Baskaran H, Toner M, Yarmush ML, Berthiaume F. Poloxamer-188 improves capillary blood flow and tissue viability ina cutaneous burn wound. J Surg Res 2001;101:56-61.
6. Zawacki BE. The natural history of reversible burn injury. Surg Gynecol Obstet 1974; 139: 867-72
7. Deutsch A, Braun S and Granger C (1996): The FIM and the WeeFIM: 10 years ofdevelopment Critical Reviews in Physical and Medicine Rehabilitation 8: 267 – 281
8. Papini R. Management of burn injuries of various depths. BMJ 2004 329: 158-60
9. Serghiou M, Evans EB, Ott S, Calhoun JH, Morgan D, Hannon L (2002) Comprehensive rehabilitation of the burned patient. Chapter 45. In Hemdon DN (Ed) Total Burn Care (1st ed). London: Saunders.
10. Monstrey S, Hoeksema H, Verbelen J, Pirayesh A, Blondeel P. Assessment of burn depth and burn wound healing potential. Burns. 2008. 761-769
11. Monstrey S, Hoeksema H, Verbelen J, Pirayesh A, Blondeel P. Assessment of burn depth and burn wound healing potential. Burns. 2008. 761-769
12. Devgan L, Bhat S, Aylward S, Spence RJ. Modalities for the assessment of burn wound depth. J Burns Wounds 2006;5:e2.
13. Monstrey S, Hoeksema H, Verbelen J, Pirayesh A, Blondeel P. Assessment of burn depth and burn wound healing potential. Burns. 2008. 761-769
14. David, J.A. (1986). Wound management: A comprehensive guide to dressing and healing. Practical Nursing Handbook. London, Martin Dunitz.
15. Ong YS, Samuel M, Song C. Metaanalysis of early excision of burns. Burns 2006;32:145-50.
16. Santucci M, Borgognoni L, Reali U. Keloids and hypertrophic scars of Caucasians show distinctive morphologic and immunophenotypic profiles. Virchows Arch. 2001 ;438:457-463.
17 Roseborough IE, Grevious MA, Lee RE. Prevention and treatment of excessive dermal scarring J Natl Med Assoc. 2004;96:108-116
18. Ketchum LD, Cohen IK, Masters FW Hypertrophic scars and keloids. A collective review. Plast Reconstr Surg. 1974;53: 140-154.