Overview of Wound Healing and Management

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KEYWORDS

• Wound • Healing • Management • Skin • Soft tissue injuries

KEY POINTS

- Wound repair is a coordinated series of phases that have predictable cell types and microenvironments.
- Wound healing is divided into inflammatory, proliferative and maturation phases.
- The pathway of healing is determined by characteristics of the wound on presentation.
- Wounds can heal via primary, secondary or delayed primary healing.
- Debridement and negative pressure wound therapy (NPWT) are important adjuncts to treat contaminated or chronic wounds.
- Soft tissue injuries should be assessed for blood supply, hypoxia, infection, edema and foreign body contamination; and treated based on these characteristics.

PHASES OF WOUND HEALING

Wound healing is a complex, highly developed chain of events that allows people to interact with their environment. The skin is a protective organ, and it provides vital functions like temperature modulation, moisture regulation, as well as sensation, reception, and transmission. The ability to repair and regenerate is central to these functions. Wound repair is a coordinated series of phases that have predictable cell types and microenvironment preparations.

Inflammatory Phase

The initial event when a wound occurs is a platelet plug that limits bleeding and begins cytokine signaling. This event initiates the coagulation cascade and promotes amplification and recruitment of cells for the debridement of nonviable tissue. The platelets create the plug in response to exposed collagen, which then releases ADP promoting continued platelet aggregation. Aggregation is accompanied by release

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Surg Clin N Am 97 (2017) 189–207
http://dx.doi.org/10.1016/j.suc.2016.08.013 **surgical.theclinics.com**

of platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-β), which is chemotactic for neutrophils in the blood.[1,2]

Neutrophils are drawn to and trapped in the platelet plug in response to PDGF.[1] They are the initial scavengers for debridement. They serve initially to phagocytize dead tissue and bacterial particles as well as create a wound hostile to bacteria by using reactive oxygen species. Neutrophils also provide a key proinflammatory cytokine in interleukin (IL)-1, which has dual effects as a proinflammatory cytokine and a stimulus for proliferation of keratinocytes.[3] The local environment also changes; initially, there is severe vasoconstriction secondary to catecholamine release. This vasoconstriction abates shortly after and there is subsequent vasodilation in response to histamine release from circulating mast cells[4,5] (**Fig. 1**).

As the inflammatory phase progresses, macrophages become the dominant cell type within 24 to 72 hours. Their role in the orchestration of wound healing is critical and changes as wound healing progresses.[6–8] It is widely accepted that macrophages play a central role and their response is key to establishing homeostasis within the wound and downregulating the inflammatory state to avoid pathologic inflammation (**Fig. 2**).

Proliferative Phase

The proliferative phase occurs from days 4 to 21, and is representative of angiogenesis, extracellular matrix (ECM) formation, and epithelialization.[9,10] Although there is considerable overlap between the phases of wound healing, the ability to transition into the next phase can determine whether a wound heals appropriately. ECM formation likely starts with platelet degranulation, because PDGF is a known promoter of proteoglycan and collagen formation. Local fibroblasts respond to PDGF by

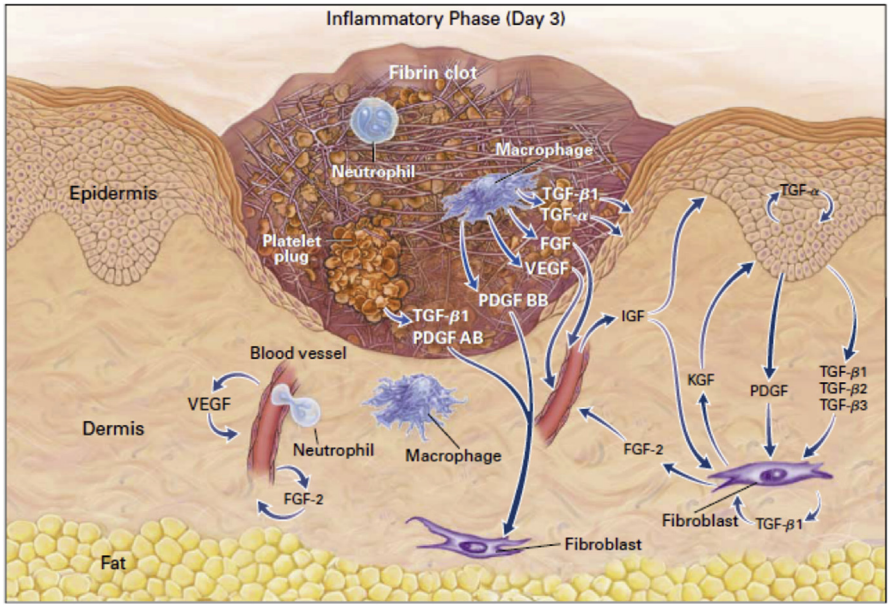

Fig. 1. In the inflammatory phase, the fibrin clot traps the neutrophils which are the first cells in the wound. It invites the macrophage which is involved in orchestrating the process of wound healing. FGF, fibroblast growth factor; KGF, keratinocyte-derived growth factor; VEGF, vascular endothelial growth factor. (*From* Singer AJ, Clark RAF. Cutaneous wound healing. N Engl J Med 1999;341(10):739; with permission. Copyright © 1999 Massachusetts Medical Society.)