Wound healing

Science, Biology

The third phase of wound healing is granulation phase, which takes place to repair the damaged cells by regenerating new cells. This phase consists of different subphases, which can last up to 4 weeks in the clean and uncontaminated wound. These sub phases do not happen in discrete time frames but constitute an overall and ongoing process. The sub phases are " fibroplasia, matrix deposition, angiogenesis and re-epithelialization" (Cho & Lo, 1998). The first sub phase of granulation process is fibroplasia.

In days 5-7, fibroblasts have migrated into the wound, laying down new collagen of the subtypes I and III. In normal wound healing, early type III collagen predominates but is later replaced by type I collagen. Tropocollagen, which is the precursor of all collagen types, is then transformed within the cell's rough endoplasmic reticulum, where proline and lysine are hydroxylated. After tropocollagen transformation, disulfide bonds are established, allowing 3 tropocollagen strands to form a triple left-handed triple helix, termed procollagen.

As the procollagen is secreted into the extracellular space, peptidases in the cell wall cleave terminal peptide chains, creating true collagen fibrils, which mark the hallmark of fibroplasia. After fibroplasia, matrix deposition takes place. In matrix deposition, the wound is first suffused with GAGs and fibronectin produced by fibroblasts. These GAGs include heparan sulfate, hyaluronic acid, chondroitin sulfate, keratan sulfate, and proteoglycans. Then, proteoglycans bond covalently to a protein core and this contributes to matrix deposition.

Later, angiogenesis takes place. Angiogenesis is the product of parent vessel offshoots which is known as new vasculature. The formation of new vasculature requires extracellular matrix and basement membrane degradation followed by migration, mitosis, and maturation of endothelial cells. Basic FGF and vascular endothelial growth factor are also involved in the modulating angiogenesis. Finally, re-epithelization occurs with the migration of cells from the periphery of the wound and adnexal structures.

This process commences with the spreading of cells within 24 hours. Leter, division of peripheral cells occurs in hours 48-72, resulting in a thin epithelial cell layer, which bridges the wound. In addtition, epidermal growth factors play a key role in this aspect of wound healing (Lynch, Colvin, ; Antoniades, 1989). The last phase of wound healing is remodeling. Remodeling process takes place after the third week, whereby the wound is altered constantly. Constant alteration of wound can last for years after the initial injury occurred.

In remodeling, collagen is degraded and deposited in an equilibrium-producing fashion, resulting in no change in the amount of collagen deposited in the wound. In normal wound healing, the collagen deposition reaches a peak by the third week after the wound is created. Then, contraction of the wound takes place following collagen deposition. Wound contraction is an ongoing process resulting in part from the proliferation of the specialized fibroblasts termed myofibroblasts, which resemble contractile smooth muscle cells (Deodhar ; Rana, 1997, para 3).

Wound contraction occurs to a greater extent with secondary healing than with primary healing, whereby it leaves a scar in socondary healing. By the 12th week, maximal tensile strength of the wound is achieved although the ultimate resultant scar has only 80% of the tensile strength of the original skin that it has replaced (Brunner ; Suddarth, 2008, p. 38). In brief, the process of wound healing constitutes an array of interrelated and concomitant events of hemostasis, inflammation, granulation and remodeling.