Pathophysiologically Factors Contributing to Keloid Formation

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Keloid formation occurs following an abnormal wound healing. It represents a significant therapeutic challenge to physicians. A better understanding of the pathophysiologic processes underlying keloid formation may help in the development of more successful therapeutic interventions. This article delineates current information on the pathogenesis of keloids. Results reported in the literature and obtained from our studies showed that keloid-derived fibroblasts demonstrated a higher growth potential and produced higher levels of procollagen than those produced by control-derived fibroblasts. In addition, our results also showed a decreased production of inflammatory mediators of MCP-1, PGE2, MMP-1, MMP-2 and TIMP-2 by keloid-derived fibroblasts as compared with normal skin-derived fibroblasts. Furthermore, current literature also indicates that the growth factor TGF-ß is overproduced and poorly regulated in keloid tissue. These alterations in the wound healing process may result in delayed and prolonged activation of injury-induced inflammation as well as imbalance in the extracellular matrix formation and degradation that may contribute to excess deposition of collagen in skin lesions and lead to keloid formation.

Key words: keloid, pathophysiology

Keloids result from aberrant wound healing in predisposed individuals. These elevated abnormal fibrous scars grow continuously beyond the borders of the original wound. They invade adjacent normal tissue and rarely regress over time. The lesions of keloids are characterized by overproliferation of wound fibroblasts and an excessive production and deposition of extracellular matrix (ECM) proteins. To date, there is no single effective modality for keloid treatment. It is a major therapeutic challenge to plastic surgeons. Attempts are being made at a definite understanding of the pathogenic mechanisms behind this abnormal wound healing, which is a prerequisite for the development of better therapeutic strategies for keloids. This article summarizes results obtained from our recent studies and the related update findings with regard to keloid pathophysiology.

Normal wound healing process

Normal wound healing is a complex process that contains the inflammation (first three to 10 days), the proliferation (or granulation, next 10 to 14 days) and the remodeling (or maturation, two weeks to years) phases. Following skin injury, the coagulation cascade is activated which causes the release of inflammatory mediators including the transforming growth factor ß.