Formosan Journal of Surgery (2015) 48, 49—56

Available online at www.sciencedirect.com

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journal homepage: www.e-fjs.com

REVIEW ARTICLE

A critical appraisal of nonsurgical modalities for managing hypertrophic scars and keloids

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Received 10 November 2014; received in revised form 20 January 2015; accepted 26 January 2015
Available online 13 March 2015

KEYWORDS
hypertrophic scar;
keloid;
keloid diathesis

Summary The nature of hypertrophic scars and keloids can be unpredictable even to the most experienced physicians. Predicting the susceptibility and severity of these disorders is difficult. The availability of numerous treatment options which yield various results make deciding on a course of treatment difficult. Further complicating the selection of treatment options are the numerous industry-driven publications that seem biased and are supported by marketing strategies for related products. Physicians often end up using a treatment modality that is not particularly objective or supported by a high level of evidence. Reviewing literature on this topic can be daunting. This study attempts to clarify the complex fibroproliferative disorder of skin wound healing by briefly describing its pathophysiology, categorizing patients into distinct groups based on their clinical behavior, and analyzing relevant evidence for each treatment modality.

1. Introduction

The unpredictability of hypertrophic scars (HTSs) and keloids can confuse the most experienced physicians. In 2014, the International Advisory Panel on scar management published a revision of the recommended practices promoted by the first advisory panel in 2002,[1,2] resulting in new treatment algorithms. However, the numerous treatment options available, combined with contrasting data, continue to make deciding on a course of treatment difficult. Based on a review of existing data, this study attempts to rationalize treatment options after observing the clinical behavior of scars and examining the evidence associated with various modalities, traditional as well as emerging, used to treat excessive scarring.

Conflicts of interest: none.

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http://dx.doi.org/10.1016/j.fjs.2015.02.001

2. Discussion

2.1. Pathophysiology and clinical behavior

Skin damage can result in scars when the wound healing process is disrupted. This can be divided into three distinct but overlapping phases: inflammation, proliferation, and remodeling. A scaffold of the extracellular matrix (ECM) is deposited by fibroblasts during the proliferative phase, forming a structural framework that bridges the wound and enables vascular ingrowth. The recruitment and proliferation of fibroblasts and the production of the ECM are influenced by the following fibrogenic growth factors: platelet-derived growth factor, insulin-like growth factor, transforming growth factors β1 and β2 (TGF-β1 and TGF-β2), and basic fibroblast growth factor.[3] These fibrogenic growth factors upregulate ECM production, increase the rates of proliferation and/or migration of the fibroblasts, and inhibit the production of proteases required for maintaining the balance between production and degradation. The ECM is degraded during the final maturation or remodeling phase, and immature Type III collagen transforms into mature Type I collagen. ECM degradation occurs through the action of collagenases, proteoglycanases, and other proteases released by mast cells, macrophages, endothelial cells, and fibroblasts. Either excessive synthesis of collagens, fibronectins, and proteoglycans by fibroblasts caused by the absence of apoptotic signals or deficient matrix degradation and remodeling may lead to keloid formation and hypertrophic scarring.

In addition, recent evidence suggests that the severity of inflammation or the type of immune response may predispose to excess scar formation.[4] Fibroblasts produce increased amounts of collagen when adverse wound healing factors are present, such as increased skin tension (except ear lobes), delayed wound healing, and wound infection, which prolong the inflammatory response. Experimental evidence suggests that a prolonged inflammatory period with immune cell infiltration increases fibroblast activity with greater and more sustained ECM deposition, leading to keloid formation.[4] In addition, the type of immune response can affect fibrogenesis. Development of a T helper, Th2 response, promotes fibrogenesis, whereas predominance of a Th1 response attenuates tissue fibrosis.[5] This may explain why keloid scars spread beyond the margins of the original wound, whereas hypertrophic scars, in which the immune cell infiltrations decrease over time, remain within the original wound margins and often regress with time.[4]

Hypertrophic scars (Fig. 1) and keloids (Fig. 2) that develop following skin damage represent the ends of a spectrum of healing by scarring (Fig. 3). However, patients who develop keloids spontaneously and have a family history of keloids may be presumed to have a "keloid diathesis" (Fig. 4) a term introduced by Burd and Huang.[6] Patients with a keloid diathesis may have a history of multiple scarring, and the high recurrence rate makes such scarring appear as a benign fibroproliferative tumor. Structurally and biochemically, hypertrophic scars have more Type III collagen compared with keloidal scars, which contain a higher Type I:Type III collagen ratio. Hypertrophic scars have fine collagen fibers with more α-smooth muscle actin-containing myofibroblasts, whereas keloidal scars have coarse collagen fibers with fewer α-smooth muscle actin-containing myofibroblasts.[6]

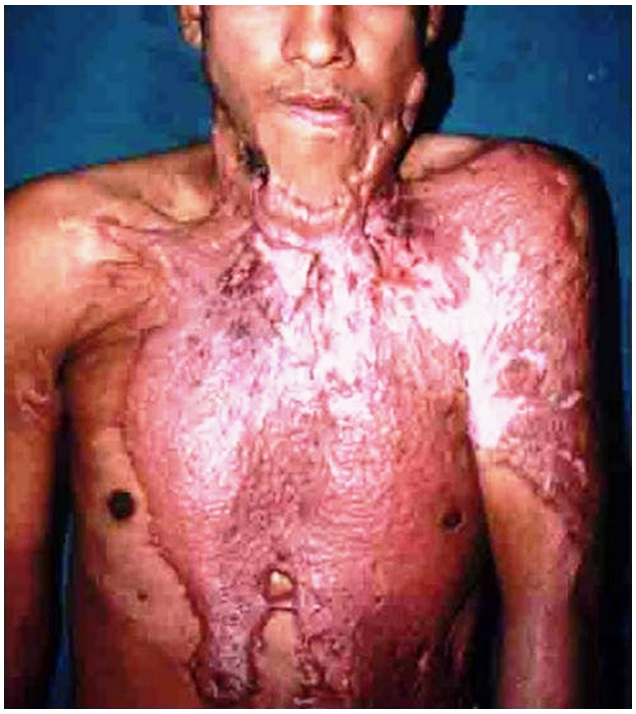

Figure 1 A patient with extensive post-burn hypertrophic scarring of trunk and neck with contracture.

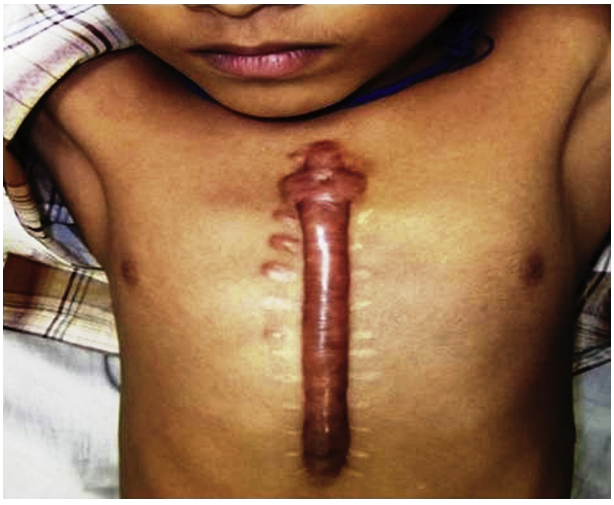

Figure 2 A child with postsurgical keloid on sternum following cardiac surgery.

Skin pigmentation is one of the major risk factors for the development of keloids. Keloids are observed in people of all races, except albinos. People with dark skin are more susceptible to keloid formation, with a reported incidence rate of 6—16% in African populations.[7] The fact that this condition never occurs in albinos, or on the palms or soles, is testimony to the fact that keloids are associated with increased skin pigmentation. There is increased sensitivity to melanocyte-stimulating hormone (MSH) which leads to a