Comparative Evaluation of Two Techniques for the Management of Traumatic Linear Atrophic Scars: Surgical Revision versus Combined Resurfacing and Surgical Revision

Thesis

Submitted for Partial Fulfillment of Master Degree
in Dermatology , Venereology and Andrology

Presented by

Hanaa Ahmed Ali Ahmed Hussein
(M.B., B. Ch)

Under Supervision of

Prof. Dr. Ahmad Ibrahim Rasheed
Professor of Dermatology , Venereology and Andrology
Faculty of Medicine-Ain Shams University

Dr. Al-Hassan Mohamed Hassan El-Hefnawy
Lecturer of Dermatology , Venereology and Andrology
Faculty of Medicine-Ain Shams University

Faculty of Medicine
Ain Shams University
2012

List of Contents

List of Figures

List of Figures (Cont.)

List of Tables

List of Abbreviations

AFR.....Ablative Fractional resurfacing:

CROSS .....Chemical reconstruction of skin scars

CO_2 .....Carbon dioxide

Er:**YAG**.....Erbium-yttrium- aluminum-garnet laser

FP .....Fractional photothermolysis

GA.....Glycolic acid

IPL .....Intense pulsed light

LHA.....Lipohydroxy acid

Mg .....Miligram

MJ.....Microjoul

Mm.....Milimeter

MMPs .....Matrix matalloproteinases

Ms .....Milisecound

Nd:YAG .....Neodymium:Yttrium-Aluminum-Garnet

NAFL.....Nonablative fractional laser

Nm.....Nanometer

PMNLs.....Polymorph nuclear leukocytes

PRP.....Platelet-rich plasma

RSTL.....Relaxed skin tension lines

SA .....Salicylic acid

TCA.....Trichloroacitic acid

TIMPs.....Tissue inhibitors of metalloproteinases

Acknowledgment

*First and foremost, I feel always indebted to **Allah** the most Merciful the most Gracious who gave me the power to accomplish this work.*

*It is an honor to express my gratitude and cardinal appreciation to **Prof. Dr. Ahmed Ibrahim Rasheed,** Professor of Dermatology, Venereology and Andrology, Faculty of Medicine, Ain Shams University, for his kind guidance, careful supervision, endless cooperation, valuable comments and meticulous revision.*

*I am also profoundly grateful to **Dr. Al Hassan Mohamed Hassan El-Hefnawy,** Assistant Professor of Dermatology, Venereology and Andrology, Faculty of Medicine, Ain Shams University, for his supervision and encouragement throughout this work.*

Finally I would like to thank all patients treated in this work for their trust and I would like to express my best regards and thanks to all staff members and colleagues for their support and to all those who gave me a hand to accomplish this work.

Hanaa Ahmed Ali

Introduction

Traumatic facial atrophic scars are common and severely distressing cosmetic problem that affect millions of people every year ***(Khatri et al., 2011).***

A successful scar treatment can dramatically improve a patient's quality of life ***(Batra, 2005)***. Treatment options of scars are subdivided into surgical & nonsurgical ***(Kaplan et al., 1997).***

Current available surgical treatment options of traumatic linear atrophic scar include fusiform excision ***(Shockley, 2011)***, w-plasty, z plasty, serial excision, punch techniques (punch excision, punch grafting & punch elevation) and subcision ***(Batra, 2005; Haradi et al., 2011).***

The non surgical treatment techniques employed include dermabrasion, chemical peels, fillers, outologous blood injection ***(Chang and Ries 2001; Cooper and Lee, 2010)***, microneedling, Radiofrequency ***(Majid, 2009; Sachdev et al., 2011)*** and lasers ***(Sawcer et al., 1999).***

Factors limiting the former techniques include incomplete scar removal, transmission of infection and pigmentary alteration ***(Alster, 1996; Cupp, 1998).***

In punch grafting some parts of the atrophic scar or acne scars are removed by a coetaneous punch and replaced with

slightly larger full thickness skin grafts (Donor grafts) which are obtained from the postauricular skin ***(Johnson, 1986)***.

The laser may be ablative or nonablative, fractional or non-fractional ***(Westine et al., 2005)***. When properly used, laser can achieve superior clinical responses in scar improvement ***(Alster, 1996).***

Dermatologists can employ a number of combination therapies in order to achieve the optimal results ***(Batra, 2005).*** Often a combination of scar resurfacing with other techniques (e.g., punch grafting, subcision or filler injections) will yield a superior result compared to resurfacing alone because less aggressive resurfacing will be practiced ***(Goodman, 2000; Fife, 2011).***

Aim of the Work

Is comparative evaluation of two techniques for the management of traumatic linear atrophic scars: punch grafting alone versus combined Erbium: YAG laser resurfacing and punch grafting.

Wound Healing and Scar Formation

Normal Wound Healing

Wound is described as disruption of tissue integrity. Wound healing is a complex and dynamic process to restore cellular structures and tissue layers. The wound healing process can be divided into 3 phases; which are the inflammatory phase, the proliferative phase, and the remodeling phase ***(Matory, 1998).***

1) Inflammatory phase:

This phase lasts for five to seven days in uncomplicated wounds ***(Su et al., 1998)***.When injury activates the clotting cascade, the kinin cascade and the complement cascade, there is release of vasoactive mediators and chemotactic factors which together stimulate the migration of inflammatory cells as polymorphs and macrophage ***(Steenfos, 1994)***.

Polymorphs are the predominant cells for 48 hours (i.e. early inflammatory phase). Within a short time, the PMNLs begin to adhere to the endothelial cells in the adjacent blood vessels through a process called margination and start to move through the vessel wall, a process known as diapedesis. They phagocytose bacteria and other foreign particles and kill them by releasing degrading enzymes and oxygen-derived free

radical species. In fact, the main function of PMNLs is to prevent infection while contributing little to the normal wound healing process beyond this stage ***(Clark, 1996).***

Subsequently, there is influx of monocytes and their conversion to macrophages (after 72 hours) which seem to be critical for initiation of tissue repair. As the neutrophilic infiltrate resolves, macrophage accumulation continues as the late inflammatory phase of wound healing. The macrophages release chemotactic and growth factors that influence wound healing by promoting fibroblastic migration, proliferation and matrix deposition ***(Dierickx et al., 1995).***

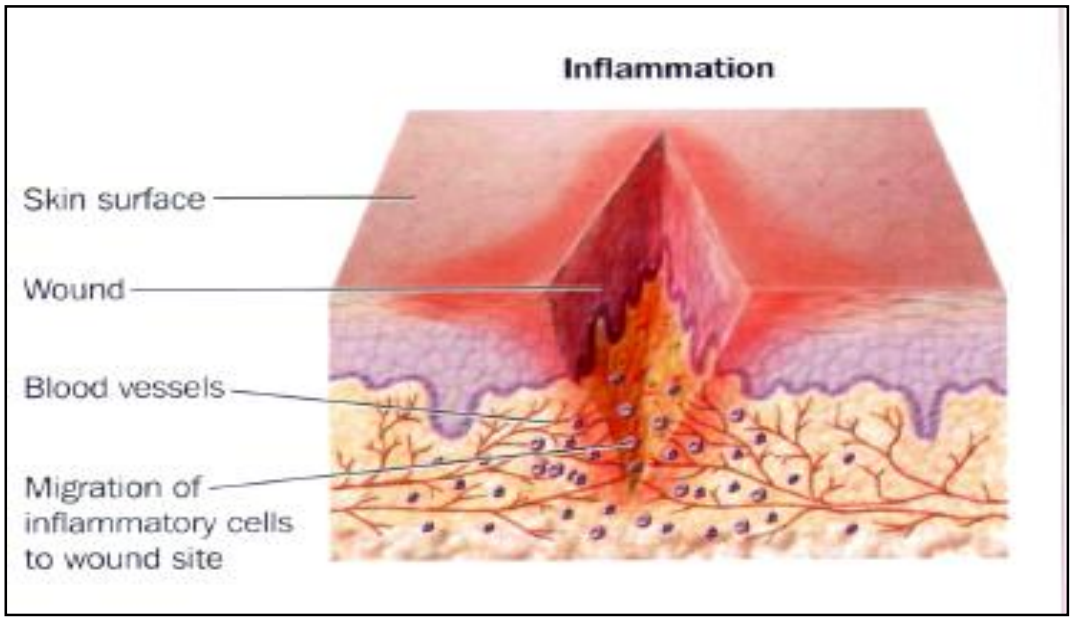

Figure (1): Inflammatory phase ***(Karen, 2006).***

2) Proliferative Phase:

This phase takes place from the seventh day till the third week after wounding. The generation of granulation tissue is the first major event and the characteristic finding during this stage of healing ***(Su et al., 1998).*** Early granulation tissue is highly vascular and densely cellular. Granulation tissue consists of a dense population of macrophages, fibroblast and neovasculature embedded in a loose matrix. Endothelial cells respond to angiogenic stimuli by capillary bud formation that is directed toward the center of the wound. As granulation tissue evolves, an abundant collagen matrix is deposited. Connective tissue matrix formation provides a substrate on which macrophage, new vessels and fibroblast can migrate ***(Dierickx et al., 1995)***. During epithelization, the basal cell layer at wound margins migrates into the open wound area, which is easy in healing by primary intention but in those healing by secondary intention the cells migrate over a greater distance and require more time **(Fig. 2)** ***(Monaco and Lawrence, 2003).***

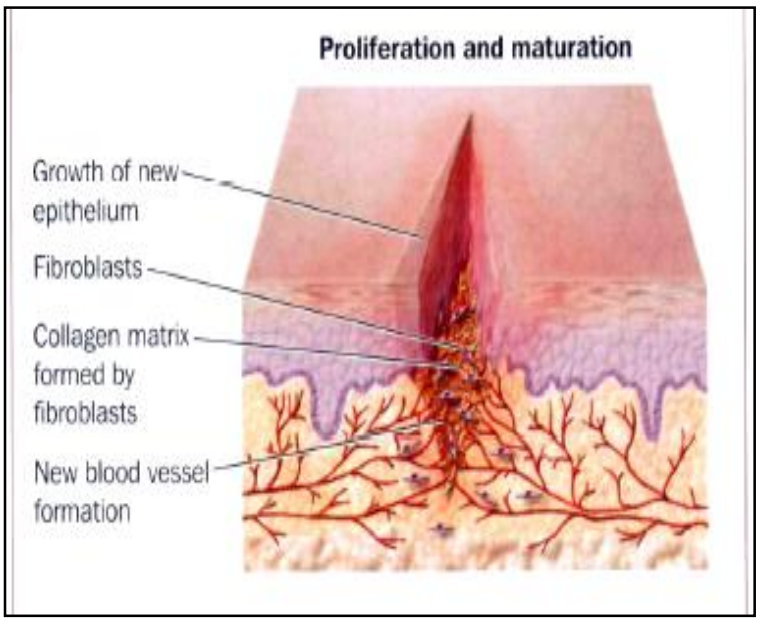

Figure (2): Proliferative phase ***(Karen, 2006).***

The second major event in the proliferative phase is wound contraction to reduce wound size. This occurs around the ninth day of wound healing ***(Monaco and Lawrence, 2003).***

Fibroblasts migrate to the wound and predominate by the seventh day. Fibroblasts are the most important cell in this process as they undergo a series of phenotypic changes. First, fibroblasts show migratory phenotype then profibrotic phenotype. Around the ninth day of wound healing they gradually develop into the final phenotype which is myofibroblasts. The latter are characterized by large bundles of α smooth–muscle-actin-containing microfilaments. When contraction stops and the wound is fully epithelized, the

myofibroblastic phenotype would disappear ***(Nissen et al., 1999).***

The final event in proliferative phase is production of substances of connective tissue repair as collagen and connective tissue matrix (fibronectin, elastin and ground substances). They synthesize collagen in a linear fashion which takes place from two to three weeks ***(Carlson and Longaker, 2004).***

3) Remodeling phase:

This phase takes place after wound closure (21 days after wounding). It is the phase of scar maturation. Scar maturation involves a balance of matrix degeneration and collagen biosynthesis ***(Dang et al., 2003).***

After deposition of the early extracellular matrix, the matrix starts to remodel its collagen framework collagen degradation is achieved by specific matrix matalloproteinases (MMPs) that are produced by many cells at the wound site, including fibroblasts, granulocytes and macrophages. As remodelling of the wound continues, (MMPs) activity decrease and tissue inhibitors of metalloproteinases (TIMPs) activity increase thus obtaining the ultimate skin strength which is about 70%-80% of that of normal skin ***(Dierickx et al., 1995).***