STUDY OF CONFIGURATION OF HYPERTROPHIC AND KELOID SCARS IN POSTSTERNOTOMY SCARS AND PROPHYLAXIS BY SILICONE SHEETS

Thesis

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 B_y

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LIST OF ABBREVIATIONS

CABG : Coronary artery bypass grafting

HTS : Hypertrophic scar

MIDCABG : Minimally invasive direct coronary artery bypass grafting

NSAIDs : Non steroidal anti-inflammatory drugs

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Abstract

From the study, the following conclusions seem justified: Hypertrophic and keloid scars could occur everywhere in the body; however, the sternum is one of the commonest sites to develop hypertrophic and keloid scars especially following median sternotomy incision. The lower half of the sternotomy wound is more susceptible to develop hypertrophic and keloid scars than the upper half, regardless of the suture materials used in skin closure. This may be due to increased tension and relative mobility of the skin over the xiphoid process. In the upper half of the sternotomy wound, use of nonabsorbable monofilament suture materials like prolene reduces the incidence of development of hypertrophic and keloid scars. So, we recommend the use of this type of suture materials in skin closure to obtain better overall cosmetic results. The incidence of hypertrophic scars is higher than keloids. Both of them can occur at any age. It is apparent that females are much more affected and asking for treatment which may be due to the fact that females are much more concerned about cosmetic disfigurement than males. Keloid is more likely to affect certain races like black and darkly pigmented skinned population, while it rarely affects Caucasians and white people, and this racial factor is the most important risk factor in developing keloid scars. Application of silicone sheet following median sternotomy incisions reduces the incidence of development of hypertrophic and keloid scars. The results that were obtained with the use of silicone sheets might be worse if we didn't use silicone sheets especially in the black race. So, we recommend the use of these sheets following median sternotomy incisions as much as we can.

Keywords:

Hypertrophic Keloid scars Poststernotomy scars Prophylaxis Silicone sheets

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION AND AIM OF WORK

Introduction:

Keloids and hypertrophic scars are abnormal wound responses in predisposed individuals. These fibrous growths result from a connective tissue response to trauma, inflammation, surgery, or burns and occasionally seem to occur spontaneously. Distinguishing keloids from hypertrophic scars can be difficult. Clinically keloids are distinguished from hypertrophic scars in that keloids extend beyond the original wound and rarely regress; whereas hypertrophic scars remain within the confines of the original wound and often spontaneously regress (*Nemeth*, 1993).

The first clear description of a keloid was given by Alibert in 1806. He proposed the term cheloide in 1816, to differentiate keloids from cancerous growths. The term is derived from the Greek word chele, meaning "crab claw", referring to the manner in which lesions grow laterally into normal tissue. In 1825, Alibert entitled a chapter on these lesions Les Cancroides ou Keloides (*Berman and Bieley*, 1995).

Certain body regions show increased susceptibility to keloids. The presternal, deltoid, upper back regions, and earlobes seem to be most commonly affected (*Lanza et al.*, 1992). There are certain races that are more susceptible to keloid scar formation. Individuals with darker skin pigmentation, blacks, and Asians are more likely to develop keloids, while (caucasians and albinos are least affected (*Crockett*, 1964)).

Topical silicone therapy is widely used to improve the signs and symptoms of hypertrophic scars and keloids and to prevent the development of abnormal scarring (*Mustoe*, 2007). The mechanism of action of silicone-based products in scar management has not been completely determined, but the beneficial effects of silicone sheets on scars are not mediated by pressure or by changes in oxygen tension or blood flow (*Musgrave et al.*, 2002).

Studies have shown that silicone sheets decrease evaporation of water from the skin and increases hydration of the stratum corneum (*Gilman*, 2003). A growing body of evidence suggests that the beneficial effects of all silicon-based products on scars are mediated by occlusion and hydration (*Mustoe*, 2007).

Aim of work:

To study the configuration and distribution of hypertrophic scars and keloids in post sternotomy wounds, and the role of silicone sheeting in prevention of hypetrophic scars and keloids in susceptible sternotomy wounds.



DEFINITION AND HISTORICAL

BACKGROUND

Keloids and hypertrophic scars (HTs) are benign fibrous growths that show abnormal wound-healing responses in predisposed individuals from certain ethnic groups (*Kose and Waseem*, 2008).

A clinical definition has often been used to distinguish hypertrophic scars from keloids. Hypertrophic scars remain within the confines of the original wound, whereas keloids extend beyond the boundaries of the skin injury (*Alster and Tanzi*, 2003).

Keloids were first described centuries ago in the Smith Papyrus (during the pyramid age) ~3000-2500 BC, and later by Retz in 1770 and by Alibert in 1806. It was Alibert who proposed the current name, keloid, which was derived from "Cheloide", meaning crab's claw, to describe the extension of the lesion beyond its original boundary. The ancient Yorubas of Western Nigeria with their scarification rituals had depicted keloids in sculptures as early as the 10th century (*Louw*, *2007*).

SKIN ANATOMY

Introduction:

The skin covers the entire external surface of the human body and is the principal site of interaction with the surrounding world. It serves a protective barrier that prevents internal tissues from exposure to trauma, ultraviolet radiation, temperature extremes, toxins, and bacteria. Other important functions include sensory perception, immunologic surveillance, thermoregulation, and control of insensible fluid loss (*Carlson*, 1994).

The skin consists of two mutually dependent layers, the epidermis and dermis, which rest on a fatty subcutaneous layer, the panniculus adiposus. The epidermis is derived primarily from surface ectoderm but is colonized by pigment-containing melanocytes of neural crest origin, antigen-processing Langerhans cells of bone marrow origin, and pressure-sensing Merkel cells of neural crest origin. The dermis is derived primarily from mesoderm and contains collagen, elastic fibers, blood vessels, sensory structures, and fibroblasts (*Carlson*, 1994).

During the fourth week of embryologic development, the single cell thick ectoderm and underlying mesoderm begin to proliferate and differentiate. The specialized structures formed by the skin, including teeth, hair, hair follicles, fingernails, toenails, sebaceous glands, sweat glands, apocrine glands, and mammary glands also begin to appear during this period in development. Teeth, hair, and hair follicles are formed by the epidermis and dermis in concert, while fingernails and toenails are

formed by the epidermis alone. Hair follicles, sebaceous glands, sweat glands, apocrine glands, and mammary glands are considered epidermal glands or epidermal appendages, because they develop as downgrowths or diverticula of the epidermis into the dermis (*Moore and Persuad*, 1998).

The definitive multi-layered skin is present at birth, but skin is a dynamic organ that undergoes continuous changes throughout life as outer layers are shed and replaced by inner layers. Skin also varies in thickness among anatomic location, sex, and age of the individual. This varying thickness primarily represents a difference in dermal thickness, as epidermal thickness is rather constant throughout life and from one anatomic location to another. Skin is thickest on the palms and soles of the feet (1.5 mm thick), while the thinnest skin is found on the eyelids and in the postauricular region (0.05 mm thick). Male skin is characteristically thicker than female skin in all anatomic locations. Children have relatively thin skin, which progressively thickens until the fourth or fifth decade of life when it begins to thin. This thinning is also primarily a dermal change, with loss of elastic fibers, epithelial appendages, and ground substance (*Burns et al.*, 2004).

Fitzpatrick devised a description of skin types known as the Fitzpatrick skin type classification. This classification denotes 6 different skin types, skin color, and reaction to sun exposure.

Type I (very white or freckled) – Always burn

Type II (white) – Usually burn

Type III (white to olive) – Sometimes burn

Type IV (brown) – Rarely burn

Type V (dark brown) – Very rarely burn

Type VI (black) – Never burn (*Fitzpatrick*, 1988)

Epidermis:

The outermost layer of skin is thin but complex. Melanin, which is responsible for skin pigmentation, is found throughout the epidermis. The epidermis also keratinizes to produce nails, hair and sweat glands. It is the foremost initiator of cell death and regeneration, the final boundary between body and environment. It is useful, however, to itemize the epidermis layer and its progressive keratinization from its innermost sublayer to its outermost, because cell growth naturally follows this pattern. Keratinization, the maturation and migration of skin cells, begins in the innermost layer of the epidermis, the stratum germinativum (Green, 1982). These cells, called keratinocytes, accumulate and move outward toward the next epidermis layer, the stratum spinosum, where they become dense. As they move into the stratum granulosum, skin cells pick up granules that contain lipids. Lipids assist in the formation of water barriers among the cells of the skin, which in turn, help to ensure body moisturization. At this point, the cell also becomes flattened, or horny, and the nucleus disappears; what remains is keratin. In the next layer, the stratum lucidum, the cell is prepared to move into its final sublayer with the addition of melanin granules. Then, sudden changes in