MANAGEMENT OF KELOIDS AND HYPERTROPHIC SCARS

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In certain civilizations, keloids are considered appealing and aesthetically pleasing. In other cultures they are considered unsightly and their occurrence may dictate the need of medical attention. The management of keloids and hypertrophic scars presents a difficult challenge. Keloids and hypertrophic scars may not only have a devastating cosmetic effect, but may also interfere with normal function. The foot is particularly vulnerable to the effects of keloids and hypertrophic scars. Multiple joint surfaces, weightbearing areas and the potential for shoe irritation make the formation of a hypertrophic scar or keloid a potentially disabling condition.

INCIDENCE

Keloids and hypertrophic scars are benign fibrous growths that result from an abnormal connective tissue response during the repair process. They follow trauma, inflammation, or burns and appear as firm, variably pruritic, or tender growths. Keloids usually occur in patients 10 to 30 years of age and are commonly located on the face, earlobes, shoulders, and upper trunk, although they may occur anywhere on the body. (Fig. 1)

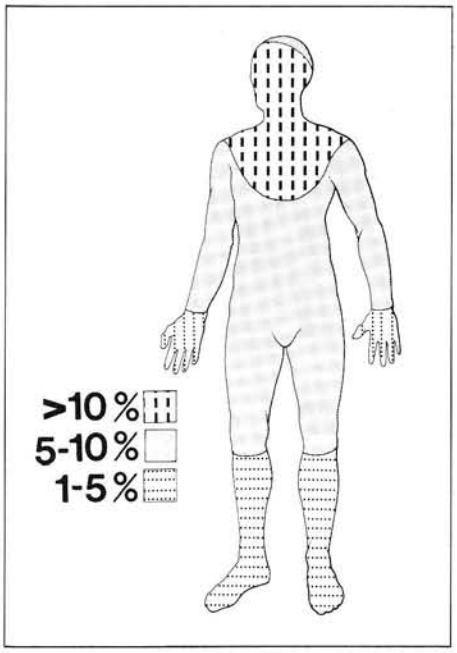

Fig. 1. Relative incidence of keloids. Prevalence by location on the body.

An understanding of normal wound healing is necessary in order to appreciate complications such as keloids and hypertrophic scars. Injury to the epidermis and dermis heals by a coordinated cellular response within the wound. This wound healing process occurs in three phases; inflammation, fibroplasia and maturation. There is a degree of overlap between the different phases. In general, the inflammatory phase occurs in the first to fourth days following injury. The fibroplasia phase occurs from the fifth to the twentieth day following injury. The maturation phase begins approximately three weeks following the initial injury and may continue up to one year.

The inflammatory phase, also called the substrate, lag or cellular phase, is characterized by a vascular response to the injury. Vasoconstriction occurs initially within five to ten minutes after the injury as the body tries to control hemorrhage and wall off the damaged area. Leukocytes are secreted at the injury site and adhere to the area. This initial vasoconstriction is followed by a vasodilatory response. Histamines and

prostaglandins are released and the blood vessel permeability is increased to allow cellular migration which aids in the healing process.

In the fibroplasia phase of wound healing, also known as the proliferative or repair phase, the fibroblasts are trying to make the surface of the wound ready for collagen by walling off the area and removing debris and foreign bodies. Fibroblasts then form a precursor of collagen with production of glycoproteins and mucopolysaccharides (ground substance). Once the ground substance is laid down, the collagen starts to grow into it and scar formation develops. If there is no cessation of this process a tremendous amount of fibroblastic activity will ensue leading to a tremendous amount of collagen formation and increased scar formation. This occurs normally when a wound is allowed to heal by secondary intention. If the balance of formation and degradation of collagen is tipped to formation, a keloid or hypertrophic scar may result.

In the maturation or remodeling phase, the collagen scar changes shape. Fibroblasts migrate to realign wound tissue to approximate that of the surrounding tissues. The normal tensile strength of the skin may not return for several months.

TYPES OF SCARS

There are four basic types of scars: a good scar, poor scar, keloid or hypertrophic scar. A good scar has straight edges, is flush with adjacent skin and has the same color as the surrounding skin. (Fig. 2A) A poor scar has uneven borders or is hyperpigmented. A poor scar is not necessarily a keloid nor a hypertrophic scar, and is not necessarily pathological. (Fig. 2B) When the scar progresses beyond a poor scar it may be classified as a keloid, (Fig. 3A) or hypertrophic scar. (Fig. 3B) Keloids and hypertrophic scars may often appear very similar. The main difference is that a hypertrophic scar is usually contained within the boundaries of the wound margin and a keloid exceeds the boundaries of the wound margin.

ETIOLOGY

The etiology of keloids is unknown, but certain factors such as trauma, tension, and hormones have been implicated. Keloids have been found

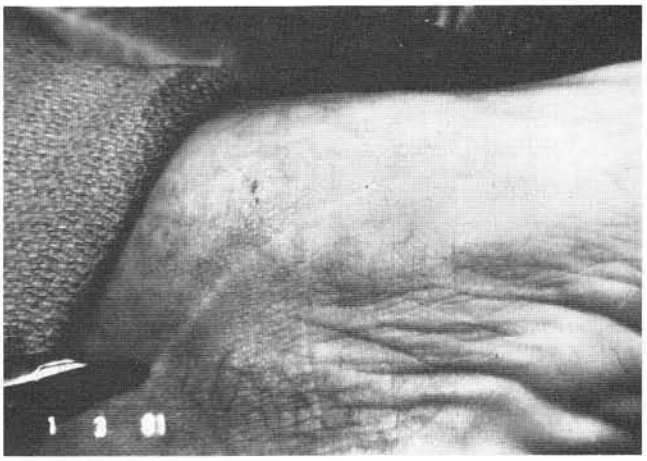

Fig. 2A. Good scar with even surface, fine line, and nearly similar color to surrounding skin.

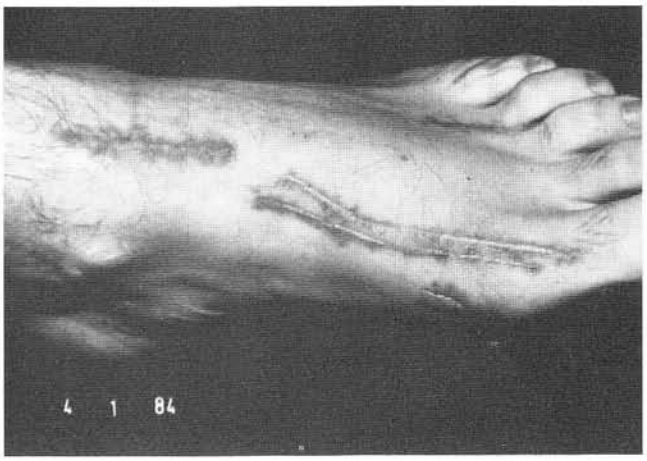

Fig. 2B. Poor scar with hyperpigmentation and uneven surface. Not necessarily pathologic.

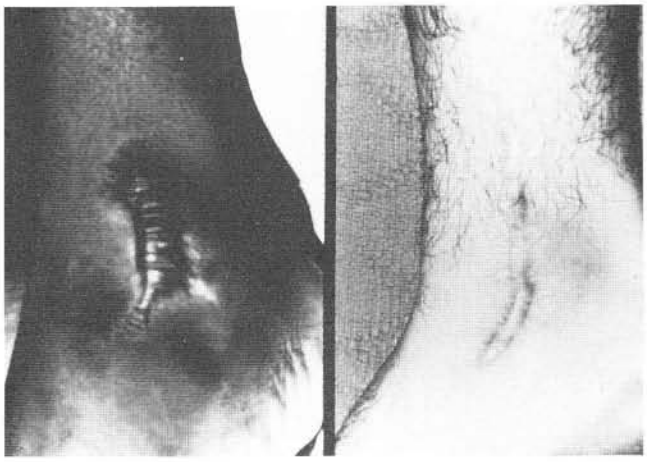

Fig. 3A. Keloids with extension well beyond boundaries of original wound.

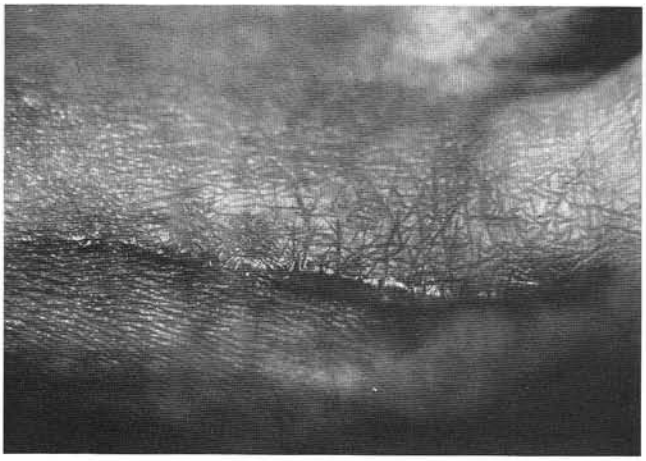

Fig. 3B. Hypertrophic scar with limitation about wound margins.

to have a familial predisposition. Koonin believes that a derangement in the metabolism of a melanocyte-stimulating hormone can be responsible for keloid formation. Osman et al. believe that an autoimmune response to sebum trapped in the deep dermis may lead to keloid formation.

Some researchers have reported a significantly higher rate of collagen synthesis in keloids, as determined by levels of proline hydroxylase. Most keloids develop within one year of the dermal insult. In contrast, hypertrophic scars tend to develop several weeks after surgery and may be exaggerated by local conditions such as inflammation and edema. Hypertrophic scars usually subside with time during the maturation phase of wound healing. (Fig. 4)

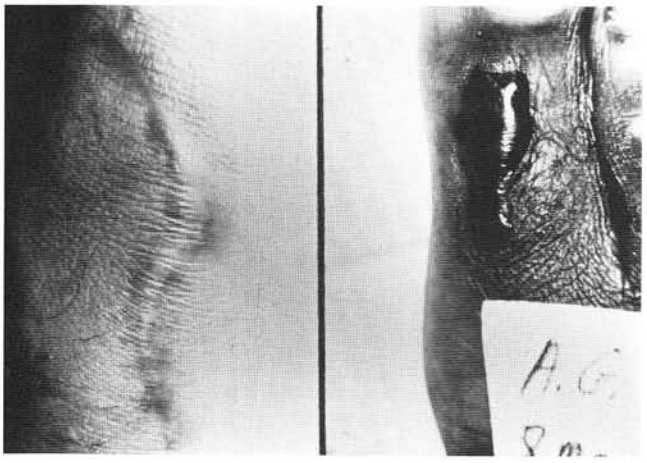

Fig. 4. Hypertrophic scar of keloid? The left figure is difficult to distinguish between an early keloid or late hypertrophic scar. The clinician would need to know how much time has elapsed following the initial dermal insult. The right figure shows a keloid at 8 months following dermal insult.

CLINICAL PRESENTATION

Keloids and hypertrophic scars may appear to be quite similar during the developmental process. The key question that should enter the mind of the clinician is: How long has it been since the initial dermal insult? Keloids may not begin for several months and rarely subside with time. Whereas, hypertrophic scars develop within weeks after surgery, and usually subside with time. Hypertrophic scars remain within the boundaries of the wound and keloids overgrow the boundaries of the initial injury. The size of a keloid can range from two to three millimeter papules to large pendulous tumors.

Keloids are slow growing fibrous tumors near a site of inflammation or injury. The overlying epidermis is thinned. New lesions are usually erythematous and older lesions are usually pale. (Fig. 5)

TREATMENT

The treatment of keloids and hypertrophic scars is a challenging problem despite the many modalities available. Some investigators feel from six to twelve months should have elapsed following injury to the skin to allow complete wound maturation and healing before a keloid or hypertrophic scar is treated. (Fig. 6)

Intralesional injection of corticosteroids, or triamcinolone acetonide, is believed to increase collagen degradation. Concentrations of triamcinolone acetonide may range from 10 - 40 mg/ml not to exceed 40 mg/ml. Injections are performed between three to five weeks apart and are generally repeated three to four times. When corticosteroids are used with surgical excision, the injections should be administered one month before surgery and continued for three to four weeks postoperatively. Local complications of intralesional corticosteroid therapy include atrophy, depigmentation, telangiectasia, necrosis, ulceration, and Cushingoid features. Ceilley and Babin have used cryotherapy before intralesional injections in order to induce edema and cellular disruption. The keloid is then less dense and the injected corticosteroid presumably penetrates more effectively.

Clinical Comparison of Hypertrophic Scars and Keloids

HYPERTROPHIC SCARS	KELOID
Develop soon after wound injury	May not begin for many months.
Usually subside with time (Maturation).	Rarely subside with time.
Limited boundary.	Overgrows its boundaries.
Size commensurate with injury	Minor injury may produce large lesion.
Occurs with motion (Compression)	Independent of motion.
Usually occurs across flexor surfaces (Joints, Abdomen, Etc.).	Areas of high predilection (Earlobes, Pre-sternal skin) Rarely across joints.
Improves with appropriate surgery.	Can be made worse by surgery.

Fig. 5.

Compression therapy along with intralesional injections of corticosteroids are commonly used as first courses of treatment. Compression therapy must be maintained approximately four to six months for best results. Some researchers have stated compression therapy merely decreases moisture within the lesion and thus does not truly reduce the lesion.

Topical silicone gel has recently been useful in the reduction of hypertrophic scars and keloids. The silicone gel is held in place over the lesion by adhesive tape or an elastic bandage. The silicone gel bandage must be worn at least twelve hours a day and for best results only removed for washing. Increased elasticity of scars has been seen after one month. An 86% success rate has been reported.

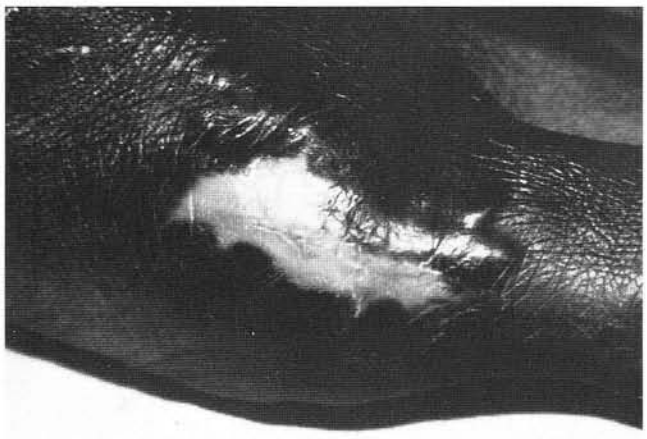

Fig. 6. Hypopigmentation following intralesional corticosteroid injection of hypertrophic scar.

Peacock hypothesizes that inhibiting the amount of cross-linking in newly produced collagen should increase its susceptibility to collagenase degradation. He refers to this technique as lathyrism. Lathyrism was induced in patients by administering Baminopropionitrile fumarate of penicillamine and administering colchicine. This treatment is undergoing further study.

Surgical excision of the lesions is more applicable to hypertrophic scars than to keloids. Keloids commonly recur following simple excision, and this recurrence often results in a larger lesion. Surgery for a hypertrophic scar involves resection with primary closure. Directional change of the scar by Z-plasty or W-plasty technique is generally planned as part of the excisional procedure. Excision of keloids alone has had poor success. Full-thickness grafts when necessary are preferred over split-thickness grafts because primary closure of the donor site is possible.

Surgical excision with carbon dioxide laser has been used with varied results. The Argon laser has also been used with poor results.

Radiation therapy has been reported to best prevent keloid recurrence if administered early after surgical excision. Ionizing radiation in low doses inhibits fibroblastic activity. Also, vascular bud ingrowth may be slowed by irradiation in the early stages of wound repair. Nevertheless, many investigators oppose radiation therapy for benign lesions such as keloids because of the carcinogenic effects.

When predisposing factors are present, certain measures may help prevent keloid and hypertrophic scar formation. Surgical technique and skill along with careful dissection and meticulous soft tissue handling will reduce trauma and inflammation to the surgical site. Care should be taken to avoid dead space, hematoma formation, infection, and wound dehiscence. The incision should follow relaxed skin tension lines where possible. The suture material should be as nonreactive as possible and strong enough to keep the wound margins opposed without tension while healing. Skin sutures alone should never be relied upon to keep the wound closed. Proper dissection, along with appropriate plastic surgical technique and instrumentation should be utilized to approximate the wound edges without tension. Sutures should be removed after seven days. A good compression dressing after surgery will reduce edema and skin tension. Pressure therapy after surgical excision has been reported to be beneficial; although pressure therapy must be maintained for at least four to six months following surgery.

CONCLUSION

Keloids and hypertrophic scars present difficult problems to manage. Success depends on accurate diagnosis to differentiate the two types of lesions and knowing when to initiate treatment. A careful history and physical examination will aid in both the proper diagnosis of these lesions and identification of patients with the potential for developing these lesions.

There are many courses of treatment, none of which offer consistently successful results. The mainstays of treatment are intralesional injections of triamcinolone acetonide and compression therapy. New treatments such as silicone gel may be helpful and improved upon with further study. Surgical excision may be considered in combination with the above treatments or with irradiation therapy if more conservative treatments are not producing a desired effect. The potential hazards of surgical excision must be appreciated by the physician and patient alike.

RISK MANAGEMENT CONCERNS

The surgeon should possess a good working knowledge of wound healing and its complications. Early diagnosis and treatment of potential wound healing problems is essential. A through history and physical may identify potential at-risk patients.

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