TREATMENT OF HYPERTROPHIC SCARS AND KELOIDS

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Hypertrophic scar is just an elevated scar confirmed within the boundaries of the injury(1) while keloids are true benign tumours that extend into skin which was not involved in the original wound (2). Keloids are less common then hypertrophic scars (3). Histologically both usually exhibit the same features and tissue culture studies are inconclusive at this time for both type of lesion (4).

Keloids display a significant familial predilection that can follow either an autosomal dominant or autosomal recessive inheritance pattern. Keloids are far more common in Blacks than in Caucasians, and even though they may be seen at any age, they more common in patients between 10 to 30 years. Hypertrophic scars show less of an inherited and race related pattern (5).

In both hypertrophic scars and keloids excessive accumulation of collagen is proven. This is attributed to increased rate of collagen synthesis and as well as to decreased rate of collagen degradation because of altered production of collagenase enzyme (6). Collagenase apparently originates from fibroblasts in the wound. Its production requires a complex interaction with epithelial elements and its activity is inhibited by local alpha 2-macroglobulin. (7)

Treatment of Hypertrophic scars and Keloids is very limited and remain elusive for both Dermatologist and Plastic Surgeons. Several modalities of treatment do exist.

1. PRESSURE:
   In recent years pressure therapy has involved itself to be as a effective therapy for both the established hypertrophic scars and keloids and as well as a prophylactic measure (8). The exact mechanism of action of pressure is not known but it is postulated that pressure induce hypoxia which results in fibroblast degeneration and decreased collagen metabolism (9).

   Pressure can applied by means of elastic compression bandages or by means of custom made compression garments. Pressure exerted should be atleast 24 mmHg to exceed the inherent capillary pressure (10). It must be maintained day and night for a minimum of 9 months to 1 year (11). Daily discontinuances of pressure should not exceed 30 minutes.

   Pressure is used prophylactically to prevent hypertrophic scars and keloids immediately after burn wound has healed and after surgical excision of these lesions (12).
2. CORTISCOSTEROIDS:

Initial attempts of treatment of hypertrophic scars and keloids with topical hydrocortisone and decadron and intralesional injections with ACTH were unsuccessful (13). In 1965 Maguirie (14) first reported regression of a large keloid following intralesional injection with triamcinolone, which has now become a mainstay of keloid and hypertrophic scars therapy.

The exact mechanism of action of triamcinolone is also not known but inhibition of fibroblasts and subsequent lying down of collagen in the wound is implicated (15).

The method of administration of triamcinolone is fairly well established. Drug should be given intralesional in the upper dermis and not subcutaneously beneath the lesion. Local infiltration of the lesion with a disposable insulin syringe containing a needle that is a part of syringe barrel itself and not detached by high pressure is recommended. The bevel of the injection should be directed down during injection. Local anesthesia is not necessary and only adds to the patient’s discomfort. Dermojet does not appear to the useful in keloid therapy.

Dose in adults is maximum is 120 mg (16). It should be 40 mg (one ampoule of Inj. Kenacort A) for a lesion of 1-2 cm, 80 mg for lesion of 3-6 cm and 120 mg for lesion of 10 cm or large. Injections are repeated once a month for six to nine months.

Dosage in children between ages 1 to 5 year is 40 mg and for ages 6 to 10 years is 80 mg.

Overdose of cortisosterone may result in serious complications. The most common side effects are atrophy, depigmentation and telangiectasis. Symptoms of Cushing's syndrome are rare (17).

Pregnancy or planned pregnancy are contraindications to treatment since corticosteroids may induce birth defects.

Steroids have establish their role both in preventing hypertrophic scars and in resolving them. They are now used preoperatively for 1 to 3 months prior to surgical procedure. One injection into the margins per operatively and thereafter monthly injections are given for at least 6 months to prevent recurrence (18).

3. SURGERY:

The mention of surgery for Keloid is done to just condemn it. Surgery alone for keloids has a high recurrence rate, 100% by one study (19). Surgery for the treatment of treatment of keloids scars has been relegated mainly to a second-line therapy for lesions unresponsive to steroids or pressure and large lesions requiring debulking. Combining surgery with other therapy is usually indicated (20).

Surgery for Hypertrophic scars is often successful when the defect is due to contraction against skin tension lines, and the revision procedure reorients the scar (21).

Several surgical procedures are available. Smaller lesion may be excised and closed primarily if the surrounding tissue is not under excessive tension. Larger lesions may be treated by excision and skin grafting. A full thickness graft that permits primary closure of the donor site is preferred to a split-thickness graft that leaves an open donor site. A primary closed donor site has a lower incidence of abnormal scar formation than a split-thickness donor site (22).
For very large lesions surgical debulking or excision of lesion with leaving behind rim margins can be attempted (23). It must be remembered that surgery is not useful, it must be supplemented by other means to prevent recurrence.

4. RADIATION:
Radiation therapy has a doubtful value. It is only reserve for keloids scars resistance to surgery, corticosteroids and pressure therapy (24). Radiation destroys the fibroblasts and they are not replaced by blood bone cells from distant tissue. By destroying enough cells a balance is reached between collagen synthesis and degradation (25).

The recommended dose is between 300 and 1800 rads given in five to six treatment over a week's time while shielding normal tissues (26). Ortho-voltage low enough radiation is preferred to decrease penetration.

Radiation is most useful if given immediately postkeioidectomy to suppress undifferentiated fibrous and vascular elements and prevent excessive collagen accumulation (27).

Radiation may result in local hyperpigmentation and has been reported to degenerate into basal cell carcinoma (28).

5. LASER SURGERY:
Laser surgery is a developing technique in the treatment of keloid scars. The Laser beam seals blood vessels up to 0.5 mm, seals nerve endings, and causes minimal necrosis of surrounding tissue (29). Argon, carbon dioxide (30) and more recently a flashlamp pumped pulsed dye laser is used (31).

6. MISCELLANEOUS:
A. ZINC OXIDE:
Zinc in the form of ZnO topically has been found to inhibit enzyme lysyl oxidase, a cross-linking enzyme for collagen fiber stabilization, and stimulates collagenase (32). This method holds promise both in the post operative setting of a definitive surgical procedure and as conservative first time approach in keloids management.

B. RETINOIC ACID:
It is theorized that topical 0.05% solution of retinoic acid has an inhibiting effect on DNA synthesis in fibroblastes (33). The main concern about this agent has to do with its method of administration, in a solution containing ethanol and propylene glycol, which may be rather irritating to surrounding normal skin.

C. COLCHICINE:
Colchicine reduces secretion of collagen from fibroblasts, enhances collagenase activity, inhibits wound contraction, and significantly reduces the breaking strength of wound scar (34). This drug is under clinical trials.
D. ANTINEOPLASTICS:
Topically Nitrogen Mustard and thio-TEPA have shown efficacy in limited trials (35).

E. B-AMINOPROPIONITRILE (BAPN):
BAPN inhibits the extracellular enzyme necessary for intra and intermolecular crosslinking of collagen reactions that radically reduce the enzymatic collagenase activity on the collagen molecules (36).

F. PROLINE ANALOGS:
It has been hypothesized that the presence of proline analogs at the time of collagen synthesis would lead to their incorporation into the collagen. Because of their abnormal shape, subsequent hydroxylation of tropocollagen and folding into a triple helix conformation would be inhibited promoting degradation. In this regard no human data are available (37).

G. INTERFERON:
Interferon inhibits cultured fibroblasts and decrease the production of type I collagen mRNA. This finding suggest that interferon decrease the rate at which the procollagen (I) is transcribed (38).

The knowledge of Keloids and hypertrophic scars is expanding but unanswered questions remain. As the various research approaches yield more information, treatment will continue to improve.

REFERENCES


