Keloid Scars: Non-Invasive and Invasive Treatments

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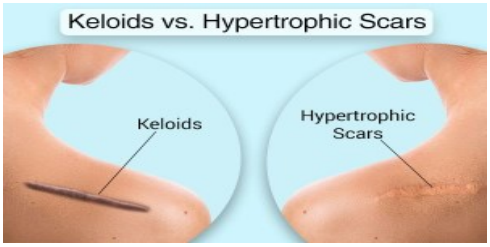
Key Points

- Keloid scar is formed due to abnormal process of wound healing.
- Invasive and non-invasive treatment options are there to treat keloid scar.
- Extensive deposition of collagen leads to keloid scar.

Keloid scars reflect abnormal process of wound healing as they are fibro-proliferative disease entities.[1] These scars cause itching, swelling, pain, stiffness and distress which can affect the quality of one's life. Various methods (invasive and non-invasive) have been advocated to treat these scars for example silicone gel sheeting, compression therapy, intralesional corticosteroids injections, surgery and radiotherapy. Unfortunately, keloids can become significantly challenging to treat.

The pathogenesis is thought due to the excessive deposition of collagen extending beyond the original wound margins.[2] Many factors have been correlated with a predisposition to keloid formation including specific HLA subtypes, all blood type, Fitzpatrick skin types V-VI, and age from 10 to 30 years old. Aspects of initial wound management have also been correlated with the formation of keloid scars including delayed debridement, heavy inflammation and excessive wound tension. Their cause is genetic and it is approximated that about 100 million people worldwide have acquired scars from surgery or trauma, of which 15% of scars are excessive. According to some researches excessive scarring is divided into two subtypes; hypertrophic which stays around the wound and keloid which extend beyond the boundaries of the original wound.



Source: *https://www.google.com/url?sa=i&url=https%3A%2F%2Fwww.emedihealth.com%2Fskin-beauty%2Fmore-skin conditions%2Fremove*

Currently, there is little agreement amongst the medical community regarding the best treatment modality for keloids.[3] Recent studies suggest that a multimodal approach is necessary and gives a successful resolution to decrease rates of recurrence. The following table represents some therapeutic measures taken against keloid scars:

Treatment	*Mechanism of action*
Occlusion	Unknown
Intralesional steroids	Anti-inflammatory, antifibrotic
Pressure	Unknown
Cryotherapy	Unknown
Dye-laser (585,595 nm)	Inhibition of angiogenesis, mast cell deactivation
Ionizing radiation	Antiproliferative
5-fluorouracil	Antiproliferative
Bleomycin	Antiproliferative
Imiquimod	Induction of interferon α and apotosis
Verapamil	Inhibition of collagen synthesis
Anti-TGFβ-antibodies	Inhibition of the TGFβ-pathway
Recombinant TGFβ3	Antagonizing TGFβ
Imatinib mesylate	Inhibition of tyrosine kinase → inhibition of TGFβ signal transduction
Anti-IL-6-antibodies	Reduction of collagen synthesis
Recombinant IL-10	Unknown
VEGF siRNA	Antifibrotic, inhibition of angiogenesis
Activin-like kinase 5	Inhibition of SMAD-activation
Mannose-6-phosphate	Inhibition of TGFβ1 and TGFβ2

Source: *google images keloid scars treatment*

Corticosteroid (TAC) Injection

TAC has been used for years for treatment of keloids. It works by suppressing fibroblast development and suppression of collagen production. It is easily available, cheap and can be administered without any fatal consequences. The only downsides are the most common side effects which include hypopigmentation and dermal atrophy.

Silicone Gel Sheeting

Silicone materials have been recommended as the "gold standard" treatment of keloid scars. Studies have reported up to 90% improvement of keloid scars when using silicone dressings. However, although silicone materials decrease the incidence of keloids after surgical procedures, complete resolution has not been described. Furthermore, the use of silicone materials lacks a scientific underpinning and well-designed studies. There are no adverse effects other than folliculitis and the treatment being expensive.

Fluorouracil

5-Fluorouracil is a fluorinated pyrimidine analogue and a classical chemotherapeutic agent. It functions as a cytotoxic agent, inhibiting cell proliferation in the scar tissue, and has been shown to inhibit fibroblast proliferation and enhance fibroblast apoptosis without causing tissue necrosis. It also inhibits transforming growth factor-β (TGF-β)–induced expression of type I collagen.

Bleomycin

Bleomycin is a cytotoxic anticancer agent with antibacterial and antiviral activities. It induces apoptosis and reduces TGF-β1-induced collagen synthesis. Intralesional injections of bleomycin start at 0.1 mL (1.5 IU/mL) and can be increased to a maximum dose of 6 mL, with two to six sessions per month for keloids that are unresponsive to intralesional corticosteroid injections. Side effects include pain, superficial ulceration and crusting at injection sites, transient hyperpigmentation, and dermal atrophy. No systemic toxicity, such as pulmonary, renal, cutaneous, hepatic, or myelogenous toxicity, has been reported for low-dose subcutaneous injections of bleomycin.

Surgery

Surgical excision of keloids is a popular option and is recommended as the first-line treatment if disabling scar contracture is present. However, it should be used with caution since it often creates even larger lesions, and recurrence rates are high (45%–100%). Adjuvant measures, such as radiotherapy, interferon, bleomycin, cryotherapy, or corticosteroids, should be applied to avoid recurrence. For example, combining corticosteroid treatment with surgery reduced the recurrence rate to less than 50%, and the recurrence rate for surgery with adjuvant radiotherapy ranged from 0% to 8.6%.[4]

References:

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