Davey's Companion to Surgery in Africa

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KELOIDS and HYPERTROPHIC SCARS

DEFINITION

Scar formation usually is the end result of wound healing and it must follow a precise, fine- tuned course for it to be functional and aesthetically acceptable. From antiquity, it has been observed that the wound healing process does not usually give the same result even in the same individual or in different parts of the body[1].

Keloids and hypertrophic scars are abnormal outcomes of cutaneous wound healing following injury. There is much interest about them mainly because of aesthetic reasons for both are benign proliferative connective tissue overgrowth localized in the dermis. Both are often confused hence the need for definitions.

Keloids are defined as scars within the skin that grow beyond the confines of the original wound[2]. This process may be delayed for months, rarely years after the initial injury before starting but once keloids are formed, they do not regress spontaneously. In contrast, hypertrophic scars are raised lesions that stay within the boundaries of the wound. They usually appear within weeks of the causative injury and tend to regress spontaneously over time.

HISTORICAL ASPECT

The existence of surface scarring overgrowth has been recognized almost since the beginning of time. The Edwin Smith papyrus of ancient Egypt gave descriptions of the disease[3]. Also, the Yoruba of Western Nigeria show their familiarity with keloids in their works of Art of facial marks on terracotta sculptures[4]. Some tribes in Africa and South America intentionally made use of the remodelling properties of the skin for centuries inducing keloid and hypertrophic scar formations; primarily as a means of adornment[5].

The first clear description of keloid is usually credited to Jean Louis Albert (1768 1837), a physician born in France. Albert named it "Les Cancroides" in 1806 but changed it to "Cheloide" in 1817 to signify its similarity to crabs' claws. Finally, in 1825, he introduced the term "Keloid"[3,5].

In 1970 Peacock[6] gave the currently acceptable definition when he recognized the importance of the phenomenon where collagen of a scar escapes the original wound edge as found in keloids.

The main distinguishing features of these two diseases are summarized in Table 1.

Table 1: Distinguishing features of Keloid and Hypertrophic Scar

	Feature	Keloid	Hypertrophic Scar
1.	Onset	May be delayed for months or rarely years after injury	Usually within weeks of injury
2.	Age	More common between 10 -30 years.	Any age, common under 20 years.
3.	Sex	Females > males	Equal ratio
4.	Race	Common among the coloured races. Blacks most affected.	Less race predilection
5.	Genes	Predilection inherited as either autosomal dominant or recessive	Less genetic predisposition
6.	Invasion	Invades the surrounding skin	Confined to the area of the original scar.
7.	Regression	Does not regress	Regresses spontaneously in six months to three years.
8.	Location	Commonly affects only certain parts of the body, mostly face, hairline, ear lobes, anterior chest.	May affect any part of the body but common across flexor surfaces.
9.	Aetiology	Multifactorial. Inflammatory process	More probably caused by local factors tension, infection, and timing of closure.
10.	Symptoms	Often asymptomatic	Irritation is common and often severe

EPIDEMIOLOGY

The excessive wound healing in both hypertrophic scars and keloids is found only in humans and occurs in 5- 15% of wounds. The presence of keloids is influenced by race, age, sex and genetic inheritance.

Race

Keloid has been shown over the years to be consistently more common in coloured races compared to the caucasians[7]. This ratio could range from 3.5:1 to 10:1. The hypertrophic scar seems to have no racial predilection. The incidence of keloid in albinos is extremely low[3].

Age

Keloids can occur at any age. The highest incidence is in the younger adults of 10-30 years of age. Young children and older adults are rarely affected.

Sex

There seems to be a slight female preponderance[8], though this may be affected by aesthetic values in-between the sexes and the more frequent facial piercing found in females. Acne keloidalis nuchae, a variant of keloids, affects primarily males[9].

Genetic

Most keloid cases are sporadic although as high as 10 per cent can be familiar. When this occurs, it can either be inherited in an autosomal dominant[10, 11] or recessive[5] fashion. There may be incomplete clinical penetrance and variable expression among those affected. There may be genetic predisposition to hypertrophic scaring too.

AETIOPATHOGENESIS

There is excessive accumulation of collagen in hypertrophic scars and keloids and this is due in equal parts to increased collagen synthesis and decreased collagen degradation.

Histologically, keloids and hypertrophic scars may appear like a foreign body reaction, which in the case of keloid is not reversible. Keloids are hypercellular compared to normal scars, with atrophic or normal epidermis above poorly circumscribed areas of hyaline degeneration. No cutaneous glands, follicles or elastic tissue are present, all having been crowded out by excessive fibrous tissue growth[12].

Though no net increase in fibroblast population has been documented, keloid fibroblasts produce collagen at a faster rate accounting for the higher collagen deposition[13]. Under normal conditions, immature scars show a higher proportion of Type III to Type I collagen which reverts to the usual 1:4 ratio as the scar matures. But in hypertrophic scars and keloids, the type III:I collagen ratio remains closer to 1:2[5].

Collagen organization in both scars is also defective though produced by fibroblasts that looked chromosomally and morphologically normal. Hypertrophic scar also show increased binding of proteoglycans to collagen and abnormally low collagenase production. All these lead to reduced breakdown.

The keloid has three stages of histological evolution:

- **stage of fibroplasia**
- **stage of fibrosis** and
- **stage of hyaline degeneration[12].**

Many theories have been propounded about the aetiology of abnormal scarring and some of the popular ones are discussed below:

MUTATION THEORY

This theory proposes that deleterious mutations in a protein called p53, which is synthesized after the infliction of DNA damage, leads to a hyperproliferative state that can result in keloid formation[14]. Normal p53 causes cell-cycle arrest at the G_i phase until the DNA damage has been repaired. Once the damage has been repaired, p53 is degraded. Mutations in p53 were found only at the keloid sites and not in healthy tissues of the same individuals studied. It should be noted that somatic mutations of p53 have been implicated as causal events in up to 50% of all human malignancies. Mutation leads to impairment of apoptosis and cell proliferation control.

IMMUNE THEORY

Several studies have suggested that alterations in immunity and inflammation may be integral to the development of abnormal scarring. A high level of immunoglobulin G (IgG) and complement levels has been found in keloid tissue and the sera of keloid formers. There was essential fatty acid deficiency of the Omega 3 and 6 series with increased level of arachidonic acid which inevitably led to the over-production of pro-inflammatory metabolites and increased collagen production[15-17].

Other studies have suggested that agents like sebum from damaged sebaceous gland stimulate an autoimmune phenomenon leading to keloid formation[18]. This process may however not be such an important factor in the pathogenesis of keloids[19].

MAST CELL THEORY

In-vitro histamine administration promotes fibroblast replication, particularly in keloid-derived fibroblast strains. Abnormal scars contain an abundant number of mast cells whose degranulated appearance indicates active histamine secretion suggesting that histamine may be responsible for the unchecked growth of the keloid[5].

MECHANICAL THEORY

After observing keloid atrophy following auto-transplantation and successful revision of keloids in cases where the entire keloid base was left intact to splint the wound, it was proposed that increase tension on the wound site caused by altered surface integrity may contribute to keloid formation.

Fibroblasts, along such wound sites with increased tension, were observed to have responded by producing excessive amount of collagen.

HORMONE THEORY

Estrogens have been implicated in the aetiology of keloids on the basis of the frequency of occurrence during puberty and pregnancy, aggravation during pregnancy and resolution after menopause[2]. The growth hormone of the anterior pituitary is said to be essential for keloid formation while androgens have been incriminated because of the increased binding of these hormones by the abnormal tissues.

ISCHAEMIC THEORY

This theory holds that uncontrolled proliferation of perivascular myofibroblasts and endothelial cells causes occlusion in microvessels in wounds, resulting in hypoxia and a tendency for further scar formation.

Other theories that had been proposed about the aetiology of keloids include the propensity of inflammation and subsequent scarring in dark skinned individuals due to the reduced level of vitamin D-3 production in their skin[20] and more recently, the role of glioma-associated oncogen homologue (gli)-1 in keloid formation. It was observed that gli-1 was inappropriately expressed in granulomatous lesions of the skin such as cutaneous sarcoidosis, granuloma annulare and necrobiosis lipoidica diabeticorum[21].

RISK FACTORS

There are many risk factors for keloid formation, both local and systemic.

LOCAL FACTORS

Site

Certain areas of the body are particularly prone to form keloid scars. In a susceptible individual, a mere insect bite may start a keloid reaction in the pre-sternal skin, while a severe laceration on the leg may heal leaving a flat and supple scar. The varying regional susceptibilities are illustrated in Figs 1 and 2 and recorded in Table 1.

Susceptible areas of the skin will generally be found to have one or more of the following characteristics.

- copious covering with fine downy "lanugo' type hair.
- hairs with restricted growth potential.
- excessive sebaceous activity.

Each of these in its own way will tend to increase the amount of keratin trapped in the wound. Coarse vigorous hair generally has the capacity to grow back to the surface and seldom provokes a keloid reaction.

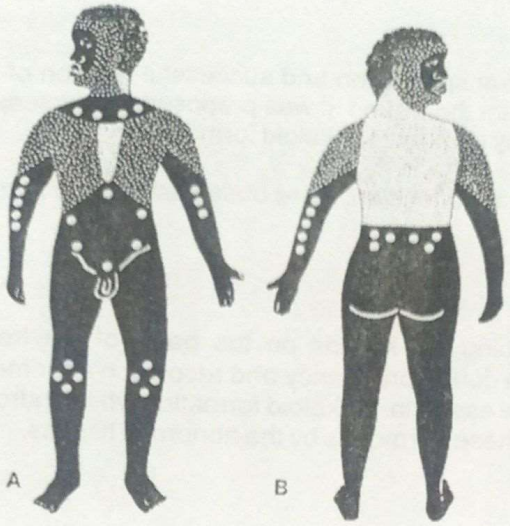

Fig 1. **Regional keloid susceptibility,** indicated in the diagrams by depth of shading. A, Front view. B, Back view.

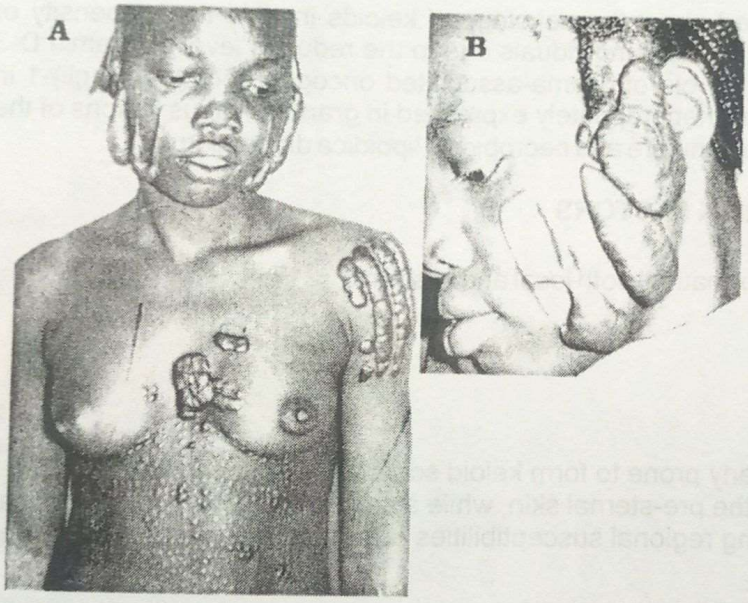

Fig. 2. **Regional keloid susceptibility:** tribal scars in a Sudanese girl. A, Note that all of the presternal scars show severe keloid; the deltoid scars are also severe but a few are spared; abdominal scars are almost unaffected. B, Note severe keloid in pre-auricular and 'beard areas', fading as the centre of the face is approached.

Scar Orientation

Keloids tend to thicken preferentially along the axis of greatest skin tension. If the scar is oriented to lie along this axis, the bulk of keloid tissue will be least, and if at right angles to it, greatest (Fig 3). Even a well-placed scar may be marred by keloid developing in marker scratches or stitch tracks running at right angles to it.

Type of Injury

In hypertrophic scar, a shelving wound is a more potent stimulus than one at right angles to the surface, because of the greater number of hair follicles damaged. A deep dermal burn can heal with burial of hair reminants over a wide area and commonly leads to some of the worst instances of scar hypertrophy[22].

SYSTEMIC FACTORS

The role of race, sex, age, hormonal development and genetic predisposition has been discussed. Among those susceptible, even an insignificant almost forgotten injury like vaccination marks of babyhood may become keloid at puberty.

There is also individual susceptibility especially among those with previous history of keloid formation. It should be noted however, that this susceptibility might be present at any time and disappear or reappear again in later life. The severity of keloids also varies from individual to individual, for some may form keloids only in the pre-sternal region while others in scars all over the body.

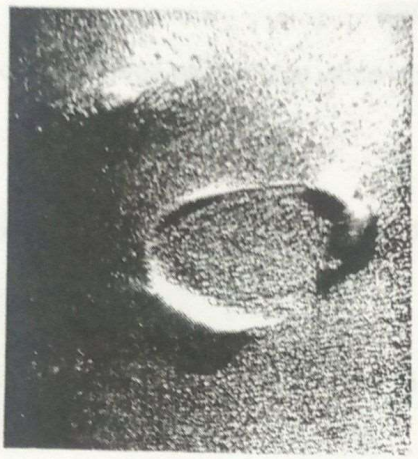

Fig. 3. **Scar orientation:** a circular tribal scar below the nipple of a Sudanese boy. Only thoe parts of the scar which run across the skin tension line show keloid change.

CLINICAL FEATURES

Late presentation and hence advanced disease is quite common in Africa. Many patients present only after some period of unproductive selfmedication or with unsightly scars, the usual complaint being an exuberant scar formation either in recently acquired wounds or in old ones. Occasionally, some patients present with complications like infections (including tetanus) or malignant transformations[23].

Five phases in the natural history of keloids have been identified[12].

HEALING

The growth of keloids may be delayed for months or years after the initial injury. The first phase is the stage of connective tissue inflammatory reaction and epithelialization which is characterized by increased pruritus. The stage is clinically and histologically indistinguishable from normal wound healing.

HYPERTROPHIC SCARRING

The second is indurated lesion raised above the surface of normal skin. The lesion is tender, irritable and accompanied by varying degree of pruritus. The surface is smooth, shiny and pinker than that of the surrounding normal skin, the redness being due to increased vascularization. The hypertrophic scar is confined to the area of injury or incision and continues to grow for months or even years.

KELOIDAL PHASE

After months, even years of hesitation, the keloid reveals its identity by its continued growth in the face of diminished vascularity. With its crab-like processes, it extends beyond the confines of the original scar and invades surrounding normal skin. The pattern of growth at this stage is greatly influenced by its location.

QUIESCENT PHASE

A well-established keloid has no tendency to regress. With age, however, it may shrink in size, soften in consistency and hang out like a mass of redundant skin, its appearance reminiscent of a plexiform neurofibroma.

COMPLICATION

The quiescent phase is the terminal stage in the evolution of an uncomplicated keloid. Rarely, a keloid may undergo ulceration or even malignant transformation. The commonest complication is infection, seen in acne keloids in which bacteria of the hair follicles produce pyogenic infection. Suppurative keloids have been described from Nigeria[24].

CLINICAL VARIETIES

Clinically, keloid disease is not a homogenous biological entity but has site-specific morphologies of scarring[8,25-27].

CHEST SCARS

Could be butterfly or non-butterfly shaped found most commonly in the mid-sternal line. The scar tends to be bilaterally symmetrical if it crosses the midline.

BACK SCARS

Single scars are usually well demarcated botryoid or pedunculated but multiple scars may be butterfly shaped, spheroidal and irregular.

UPPER LIMB SCARS

These occur mostly in the deltoid region and may be propeller shaped. Scars elsewhere may be nodular, linear or irregular.

EAR

Commonest site being the lobe. Here, scar may present as reniform to bulbous shaped. It can sometimes stretch the whole pinna to give it a "question mark" configuration (fig. 6.5) or hang down to give it a pedunculated appearance (fig. 6.7).

HEAD AND NECK

Scars here can be firm, nodular to hard. Posterior auricular scars are oblong shaped or vertical and reniform in outline. Scalp scars are commoner in the occipital region but generally tend to occur in areas involving the "short hair" region. Occipital scars vary from small papules to big plaques.

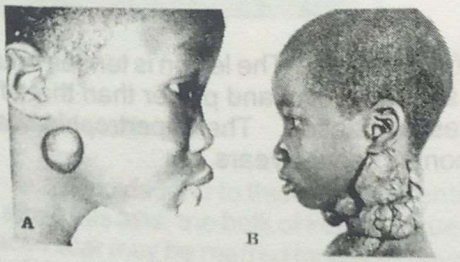

Fig. 4. **Keloids of the 'beard areas' in males:** keloids of the chin and neck are not uncommon in boys, but seldom develop after the beard has started to grow.
A, Jur boy from Southern Sudan with keloid following incision of an abscess. B, Nigerian boy with multiple keloids following incision of abscesses by a 'native doctor'.

Fig. 5. **Massive pedunculated keloids.** A. Arising from pre-auricular tribal scars in a Sudanese girl. The keloid on the chest developed from a small vertical scar and infiltrated axially on both sides along skin tension lines. B, Tribal scars of the scalp and forehead in a Dinka boy from Southern Sudan. Severe keloid is limited to the areas where the scar crosses the 'short hair' regions.

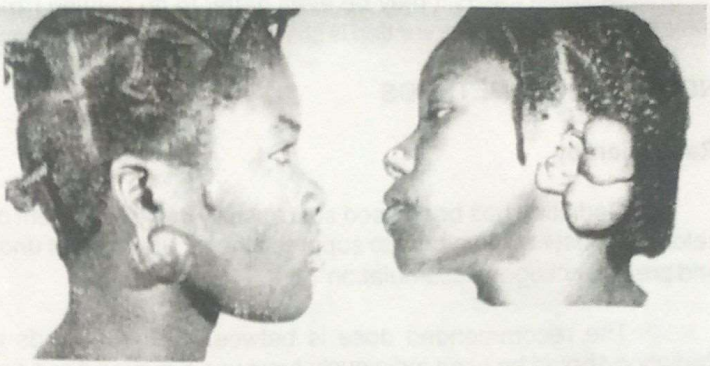

Fig. 6. **Keloids of the ears.** A, Yoruba girl from Nigeria, showing keloid of pierced ear lobule. B, In the Sudan it is more usual to pierce the rims of the ears, which may become keloid; but the pierced nose never does.

Fig. 7. **Severe keloid changes in presternal tribal scars** inflicted in infance. Note the typical lateral extension infiltrating the skin on either side. Same patient as in Fig. 6A

LOWER LIMB SCARS

May vary from propeller, butterfly, petalloid to dumb-bell shaped. Plantar keloids, though rare, can also occur.

OTHERS

Keloids have been known to occur in the cornea[28, 29] and rare sites like the pubic region. Pubic keloids can take on giant proportions.

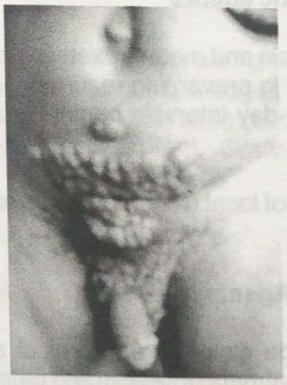

Fig. 8. **Keloid of suprapubic prostatectomy scar** (courtesy Dr O A Awojobi)

DIAGNOSIS

This is often clinical. A thorough history and examination may be all that is needed. In cases where there may be other pre or co-existing skin diseases or suspected malignant transformation, a skin biopsy should be taken for histopathologic diagnosis.

MANAGEMENT

The presence of myriads of treatment options is a proof of how notoriously difficult it is to treat keloids. The use of combination therapy is now the mainstay of treating established keloid because of the high recurrence rate associated with monotherapy[30]. Non-surgical means of treatment should be considered first and surgery should be a last resort.

In some cases it may be even better to do nothing ("masterly inactivity"), apart from reviewing the patient periodically[1]. Prevention is still the gold standard for managing keloids.

NON-SURGICAL METHODS

Radiotherapy

Radiation has been used successfully in the treatment of keloids but seems best used on immature keloids or post keloidectomy to suppress the proliferation of undifferentiated fibrous and vascular elements and prevent collagen accumulation[3,31].

The recommended dose is between 300-1800 rads in five to six treatments over one week[5]. Radiation should be used judiciously however, because it can produce local hyper pigmentation and trigger malignant change. Efforts should be made to protect the growth plate of paediatric patients if radiation must be used in them.

Pharmacotherapy

- **Steroids**

These can be topical or systemic[32,33]. Topical steroid use is not as widespread as the use of potent systemic steroids that are usually injected directly to the keloid. The most commonly used systemic steroid is triamcinolone. Steroids seem to be more effective in preventing rather than resolving abnormal scar formation, and are therefore usually used in combination with surgery to prevent recurrence of keloids.

Triamcinolone should be used as a 10mg/ml formulation, injected intralesionally using a 25 to 27-guage needle on a Luer-lock syringe to prevent spillage because of the considerable pressure sometimes needed to inject the tough tissue of the keloid. Steroid injection can be repeated monthly for four to six months[3, 5]. A maximum dose of triamcinolone of about 2mg/kg body weight should not be exceeded per session given as 20mg/sq.cm of lesion.

The side effects of intralesional steroid injection appear minimal though Cushing's syndrome and local skin changes have been known to occur. The beneficial effects of steroids in inhibiting collagen synthesis can be reversed with Vitamin A administration[5].

- **Cytotoxic agents**

Bleomycin and methotrexate have been used for the treatment of keloids. Methotrexate has proven quite successful in preventing recurrences post-surgical excision. Oral methotrexate (15-20mg) stat and repeated at four-day intervals beginning a week prior to surgery and continued for about four months is recommended[5].

The risk of local ulceration and systemic side effects of chemotherapy should be considered before initiating therapy.

- **Topical Agents**

Zinc oxide ointment and topical application of 0.05% retinoic acid have been used with some success in the treatment of keloids and other abnormal scars.

- **Others**

Many other agents such as tetrahydroquinone, proline analogs, antihistamines, asiatic acid, colchicines and penicillamine have all been tried in the treatment of keloids with variable success.

- **Newer Agents**

One of the newest therapeutic modalities on the horizon is intralesional injection of interferons. They act by inhibiting the steady state level of mRNA and are best given immediately post operatively. Imiquimod cream (5%), a local interferon production inducer, can also be used. It is started immediately after surgery and continued daily for eight weeks. Patients with large wounds or grafts should not start imiquimod therapy for four to six weeks. Major side effects are local irritation and hyper-pigmentation.

Recent research into the role of gil-1 oncogen in keloid formation prompted the introduction of Tacrolimus. It is given twice a day and believed to mute the gil-1 oncogene[3].

Mechanical Therapy

- **Compression**

Mechanical compression has been shown to be very effective in treating keloid scars especially those involving the ear lobes. Such devices are also very useful in managing extensive hypertrophic scars that may result from burns[34]. Compression devises should be started once re-epithelialization occurs and continued to be worn until scar maturation. It is worn for 24hours/day and custom built for the patient. Pressure range should be between 5 - 25 mmHg. Button devises are available for the earlobe. The mechanism of action is still unknown.

- **Occlusive dressing:**

Silicone gel sheets and occlusive dressings have been used with varied success[35]. It is worn for 24hours/day for up to a year. Its anti-keloid action appears secondary to occlusion, hydration and increase in the temperature of the scar with possible increase in collagenase activity.

SURGICAL METHODS

Surgical excision can be partial or complete.

- **Complete Excision**

This is followed by a recurrence rate of almost 100% in keloids making the resulting scar bigger than the previous one.

Surgery is indicated for debulking and recontouring while recurrence must be prevented by other means. Surgery should generally be avoided in areas of high tension especially the pre-sternal region[36]. A hypertrophied scar may be totally excised and resulting wound closed without tension either primarily or with the aid of grafts.

- **Partial Excision**

Partially excised keloid scar are not usually cosmetically acceptable but it is of great value in the treatment of earlobe keloids. Core excision is usually done preserving the keloid skin edges[37] which should be closed without tension with non absorbable sutures. Favorable outcomes had been reported when the base of the keloid was left in situ to splint the wound which was subsequently grafted. Grafts could be from the keloid itself.

General guidelines for the surgical management of abnormal scars are as follows[5]:

Combination therapy (e.g. surgery and intralesional steroid injections) is more effective in preventing recurrence than any single treatment modality.

Pressure and irradiation can be useful as surgical adjuvants but have limited role in established lesions.

Small lesions should be excised with triamcinolone injected to the skin edges at the time of closure. Pressure should be added to excision/triamcinolone combination when treating large lesions. Skin grafts should be harvested where pressure could be applied remembering that thick split-thickness grafts is good for the recipient site but increases the risk of abnormal scar formation for the donor site.

SURGICAL EXCISION MAY PROVOKE UNCONTROLLABLE KELOID FORMATION.

- **Laser Surgery**

Carbon dioxide (CO_2) laser and Neodymium: YAG (Nd:YAG) laser have been used for treating keloids. Better outcome is achieved however if intralesional or topical steroid application is combined with laser therapy. Laser therapy provides precise, haemostatic excision with minimal tissue trauma thereby reducing inflammatory responses[3].

- **Cryosurgery**

Cryosurgery uses liquid nitrogen to cause cell damage and eventual cell anoxia. Cryotherapy involves freeze-thaw periods of about 30 seconds in 1-3 sessions repeated every 20 to 30 days. As with laser therapy, better response rate is achieved if combined with intralesional steroids..

PREVENTION

Abnormal scars are best prevented. In the approach of an existing wound or that inflicted surgically, all efforts must be made to prevent abnormal scar formation. All unwarranted foreign bodies should be removed keeping in mind that sutures are foreign bodies too! Sharp instruments should be used in surgery to limit crush injury to tissues. The least-sized appropriate suture should be used for deeper tissues and the skin closed with a small-size monofilament non-absorbable suture like nylon or prolene. The following principles of wound closure should be followed[1,3,5].

- Incisions should follow tension lines and natural folds in the skin
- Tissues should be handled gently and debrided only as much as necessary to ensure an adequate clean bed
- There should be minimal handling of skin edges with instruments
- Haemostasis should be painstakingly complete.
- Electrocautery should be used with caution to limit tissue damage
- Use fine sutures set to evert wound edges and remove them early
- Wounds should be closed without tension
- Allow scar maturation before attempting revision.
- It should always be borne in mind that all wounds warrant meticulous closure whether the patient is keloid prone or not.

FOLLOW UP

This should be for at least a year post-therapy in order to institute management in cases of recurrence and to evaluate long term success. It is usual, however, for patients to default on follow up and only present with full keloid recurrence.

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