

Comparative Assessment of the Efficacy of Triamcinolone in the Prevention of Recurrence of Keloids in the Pinna

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Conflict of interest: Nil

Abstract

Aim: This study was done to determine the efficacy of Triamcinolone in preventing recurrence of Keloid.

Methods: 50 patients who underwent excision of keloid in a Department of ENT, Darbhanga Medical College and Hospital Laheriasarai, Darbhanga, Bihar, India from December 2016 to November 2017. were included in this study. They were divided randomly into two groups of 25 patients each. Surgery alone was performed in 25 patients and surgery with post-operative intra-lesional Triamcinolone injection was given weekly interval for 6 weeks in another 25 patients. Patients were followed up for 1 year at every 3 months intervals.

Results: In the age group of 11-20 years there were 20 patients. In the age group of 21-30 years there were 10 patients. In the age group of 31-40 years there were 16 patients. In the age group of above 40 years there were 4 patients. There were 48 female patients and 2 male patients. Recurrence was present in 7 patients at the end of 1 year. P-value was 0.02 which was significant.

Conclusion: Thus it can be concluded that multi-modality treatment would fare better in preventing recurrence. However the best dosing schedule for steroid injections with regards to the amount of intralesional steroid to be given and the dosing frequency for the best possible results need to be determined.

Keywords: Triamcinolone, Pinna, Intralesional, Recurrence.

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Introduction

Keloids are pathological scars presenting as nodular firm lesions that extend beyond the area of injury. They do not spontaneously regress, often continuing to grow over time. [1] The most frequently affected body areas are chest, shoulders, earlobes and upper back. [2] Symptoms often include itching and pain. Unlike hypertrophic scars, keloids do not improve

over time and commonly recur following surgical excision. [3] Large lesions may lead to cosmetic disfigurement and functional impairment, thus affecting the quality of life. [2]

The abnormal wound-healing process underlying keloid formation results from the lack of control mechanisms regulating cell proliferation and tissue repair. [4]

Histologically, keloids are characterized by haphazardly arranged hyalinized collagen bundles and a tongue-like advancing edge in the papillary dermis. [5] Despite many clinical, histological and in vitro findings, the pathogenic mechanisms underlying keloid formation have not been fully elucidated. [6,7] Histologically, keloids are characterized by haphazardly arranged hyalinized collagen bundles and a tongue-like advancing edge in the papillary dermis. Despite many clinical, histological and in vitro findings, the pathogenic mechanisms underlying keloid formation have not been fully elucidated. [7]

Keloids are defined as pathologically formed scars that exceed the boundary of the original wound. They are also deemed as benign dermal tumors that are unique to humans. Etiologically, keloids may occur because of minor skin injury, such as body piercing and insect bites. In addition, it is widely agreed that the incidence rate of keloid is significantly higher in populations with darker skin, such as Africans and Asians. The external ear is one of the most common sites for keloid formation. Many different treatment modalities such as surgical excision, intralesional corticosteroids, radiotherapy, and pressure earrings have been used for keloids. To date, no specific gene has been linked to the development of keloids, and it is likely that different genes contribute to their formation in different families. [7-10]

Although it has unclear etiology, the development of keloid could be considered

as a process of abnormal wound healing, during which redundant extracellular collagen fibers as well as proteoglycans are deposited. It is known that various molecular factors contribute to this process. Some among them may be the key points that could stop or reverse this pathologic process. However, deeper understanding of the molecular mechanism of keloid formation is still required for detecting critical biological factors and for the further development of effective therapies. This study was done to determine the efficacy of Triamcinolone in preventing recurrence of Keloid.

Methods

50 patients who underwent excision of keloid in a Department of ENT, Darbhanga Medical College and Hospital Laheriasarai, Darbhanga, Bihar, India from December 2016 to November 2017. They were divided randomly into two groups of 25 patients each. Surgery alone was performed in 25 patients and surgery with post-operative intra-lesional Triamcinolone injection was given weekly interval for 6 weeks in another 25 patients. Patients were followed up for 1 year at every 3 months intervals.

Inclusion criteria: Patients presenting with keloids in the Pinna aged >15 years.

Exclusion criteria: Patients with contraindications to surgery – bleeding diathesis, etc. Patients unwilling to participate in the study

Results

Table 1: Demographic details

Age in years	Excision	Excision with Triamcinolone injection	Total
11-20	8 (32%)	12 (48%)	20 (40%)
21-30	6 (24%)	4 (16%)	10 (20%)
31-40	9 (36%)	7 (28%)	16 (32%)
>40	2 (8%)	2 (8%)	4 (8%)
Total	25	25	50
Gender			
Male	0	2 (8%)	2 (4%)
Female	25 (100%)	23 (92%)	48 (96%)

In the age group of 11-20 years there were 20 patients. In the age group of 21-30 years there were 10 patients. In the age group of 31-40 years there were 16 patients. In the age group of above 40 years there were 4 patients. There were 48 female patients and 2 male patients.

Table 2: Recurrence

Recurrence	Excision	Excision with Triamcinolone injection	Total
Absent	18 (72%)	25 (100%)	43 (86%)
Present	7 (28%)	0	7 (14%)
Total	25	25	50

Recurrence was present in 7 patients at the end of 1 year. P-value was 0.02 which was significant.

Discussion

Keloid is a cutaneous dermal lesion resulting from uncontrolled deposition of collagen and glycosaminoglycan around the wound. Elevated levels of growth factor and cytokines contribute to keloid formation (1–3). Transforming growth factor beta (TGF- β) family is associated with enhanced collagen synthesis in keloid fibroblasts. TGF- β 1 treatment stimulates the production of collagen in keloid fibroblasts but not in normal skin fibroblasts. [11] Observation that anti-TGF- β 1 antibody suppresses collagen synthesis of keloid fibroblasts further confirms the role of TGF- β 1 (1). TGF- β 2 treatment enhances collagen production of xenograft derived from human keloid specimens in athymic rats, indicating a causative role of TGF- β 2 in keloid formation. [12]

Hypertrophic scars rarely recur after surgical excision, and some degenerate spontaneously. In contrast, the recurrence rate of keloid treated by surgery only is high (45-100%), making it important to differentiate keloids from hypertrophic scars in deciding treatment methods. Generally, keloids show a pattern of infiltration beyond primary scars, whereas hypertrophic scars are limited. [13] In addition, hypertrophic scars form within 4 weeks after injury, whereas keloids form later, an average of 30.4 months after injury. Moreover, hypertrophic scars decrease in size within 1 year, whereas

keloids maintain their size for longer than 1 year. Hypertrophic scars are treated by surgery only, whereas keloids are treated by surgery followed by local injection of steroids, which decreases the expression of genes encoding collagen. Due to their recurrence, long-term follow-up in patients with keloids is important. [14]

Since keloids are notoriously characterized by a high recurrence rate after surgical excision, nonsurgical approaches are recommended for primary treatment. [4,15] The most common approach is intralesional corticosteroid injection alone or in combination with other treatment modalities. Triamcinolone acetonide (TAC) is the most commonly used intralesional corticosteroid. Many corticosteroids are available for the treatment of keloids, but the most commonly used is TAC. Clinically, the response to corticosteroid injection alone was variable with 50–100% regression and a recurrence rate of 33% and 50% after 1 and 5 years, respectively. [4,16] Five-year recurrence rates for surgical excision followed by TAC administration were reported to be between 8% and 50%. [4]

Sand et al advocated Surgical excision and postoperative intralesional injection of steroid combined with silicon gel sheeting and compression therapy with an individually designed silicon pressure splint for the helical rim. The procedure combines the advantageous effects of pressure and silicon gel sheeting. Silicon has been described as effective in preventing the development of keloids. It reduces keloid scar formation by 70%

when used consistently. There are several theories of the action mechanism. Although some authors propose that silicon diffuses from the surface of the silicon gel sheets and reduces keloid ground substance it is more likely that retardation of epidermal water loss and a subsequent increase of wound hydration is responsible for the keloidinhibiting. [17] Compression therapy with dressings or devices that apply more than 24 mmHg, the capillary pressure, create a hypoxic microenvironment which results in fibroblast, and, subsequently, collagen degradation. Pressure earrings with compression plates which are available in different sizes are successfully used for ear lobe keloids. [18] It is obvious that the helical rim with its concave anterior and convex posterior surface is not easily amenable for compression. The silicon pressure splint introduced here not only enjoys all the advantages of silicon dressings but also successfully delivers pressure on the helical rim. [17]

Bashir et al advocated that Steroid injection in the residual wound rim can be used as an adjunct following excision of post-piercing ear keloids. It has a low morbidity, is cost-effective, easy to administer, and provides reliable and durable results. Steroids are believed to act by decreasing the level of collagenase inhibitors, thereby increasing collagen degeneration. Early application of steroids in the wound has anti-inflammatory effects which decreases fibroblast and collagen release. Intra-lesional steroids have been used pre-operatively, post-operatively as well as per-operatively. So, timing of steroid with surgery as well as dose frequency in the postoperative period is a matter of question.[19]

Conclusion

Thus it can be concluded that multi-modality treatment would fare better in preventing recurrence. However the best dosing schedule for steroid injections with regards to the amount of intralesional

steroid to be given and the dosing frequency for the best possible results need to be determined. In the treatment of earlobe keloids, pressure devices may play an important role, in combination with triamcinolone intralesional injection. New mechanisms of intraepidermal needle-less delivery of the drug are being explored: they might improve the efficacy and limit the risk of adverse reactions, in particular those related to systemic exposure. However, further preclinical and clinical trials are needed to establish safety and efficacy of this kind of administration.

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