

Prospective Outcome Assessment of Triamcinolone in the Prevention of Recurrence of Keloids in the Pinna

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Received: 14-10-2022 / Revised: 15-11-2022 / Accepted: 10-12-2022

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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: Total 50 patients who underwent excision of keloid in Department of ENT, Patna Medical College and Hospital, Patna, Bihar, India for 12 months 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 be far 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 nodular lesions that span over the injury site and are a type of pathological scar. They don't go regress on their own, and they keep evolving through time. The lack of control mechanisms that self-regulate cell proliferation and tissue repair causes the pathological wound-healing process that leads to keloid development.[1,2] Keloids can cause both visual and functional deformity,

decreasing one's quality of life. Despite the fact that various treatment options have been reported in the literature, there has yet to be a universally viable cure. The most successful way is to inject corticosteroids into the lesion alone or in combination with other treatment strategies. Triamcinolone acetonide is the most often used intralesional corticosteroid (TAC).

The dark phototypes are highly prevalent. The most commonly affected body parts are the chest, elbows, earlobes, and upper back. Itching and discomfort are typical findings of the condition. Unlike hypertrophic wounds, keloids do not heal over time and frequently reoccur following excision. Large lesions can be disfiguring and functionally debilitating. [3] Symptoms often include itching and pain. Unlike hypertrophic scars, keloids do not improve over time and commonly recur following surgical excision. [4] Large lesions may lead to cosmetic disfigurement and functional impairment, thus affecting the quality of life. [5] The abnormal wound-healing process underlying keloid formation results from the lack of control mechanisms regulating cell proliferation and tissue repair. [6]

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. [7,8] 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. [8-10]

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.¹ This study was done to determine the efficacy of Triamcinolone in preventing recurrence of Keloid.

Methods

Total 50 patients who underwent excision of keloid in Department of ENT, Patna Medical College and Hospital, Patna, Bihar, India for 12 months was included in this study.

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

Methodology

The total 50 patients 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.

Follow-up: Patients were followed up for 1 year at every 3 months intervals.

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 keloid inhibiting. [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. 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. [18,19]

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.

References

1. McCoy BJ, Diegelmann RF, Cohen IK. In vitro inhibition of cell growth, collagen synthesis, and prolyl hydroxylase activity by triamcinolone acetonide. *Proceedings of the Society for Experimental Biology and Medicine*. 1980 Feb;163(2):216-22.
2. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids. *Plastic and reconstructive surgery*. 2010 Feb 1;125(2):557-68.
3. Brissett AE, Sherris DA. Scar contractures, hypertrophic scars, and keloids. *Facial plastic surgery*. 2001 ;17(04):263-72.
4. Mustoe TA, Cooter RD, Gold MH, et al; International Advisory Panel on Scar Management. International clinical recommendations on scar management. *Plast Reconstr Surg*. 2002;110(2):560–571.
5. Robles DT, Berg D. Abnormal wound healing: keloids. *Clin Dermatol*. 2007; 25(1):26–32.
6. Gao Z, Wang Z, Shi Y, et al. Modulation of collagen synthesis in keloid fibroblasts by silencing Smad2 with siRNA. *Plast Reconstr Surg*. 2006;118(6):1328–1337.

7. Wu WS, Wang FS, Yang KD, Huang CC, Kuo YR. Dexamethasone induction of keloid regression through effective suppression of VEGF expression and keloid fibroblast proliferation. *J Invest Dermatol.* 2006; 126(6):1264–1271.
8. Salem A, Assaf M, Helmy A, et al. Role of vascular endothelial growth factor in keloids: a clinicopathologic study. *Int J Dermatol.* 2009;48(10):1071–1077.
9. Bayat A, Arscott G, Ollier WE, Ferguson MW, McGrouther DA. “Aggressive keloid”: a severe variant of familial keloid scarring. *J R Soc Med.* 2003;96(11):554–555.
10. Robles DT, Moore E, Draznin M, Berg D. Keloids: pathophysiology and management. *Dermatol Online J.* 2007 ;13(3):9.
11. Younai S, Nichter LS, Wellisz T, Reinisch J, Nimni ME, Tuan TL. Modulation of collagen synthesis by transforming growth factor- β in keloid and hypertrophic scar fibroblasts. *Annals of plastic surgery.* 1994 Aug 1; 33(2):148-54.
12. Wang X, Smith P, Pu LL, Kim YJ, Ko F, Robson MC. Exogenous transforming growth factor β 2 modulates collagen I and collagen III synthesis in proliferative scar xenografts in nude rats. *Journal of Surgical Research.* 1999 Dec 1;87(2) :194-200.
13. Fedarko NS, Pacocha SE, Huang SK, Lichtenstein LM, Essayan DM. Interleukin-13 modulates collagen homeostasis in human skin and keloid fibroblasts. *Journal of pharmacology and experimental therapeutics.* 2000 Mar 1;292(3):988-94.
14. Metts J. Common complications of body piercing. *West J Med.* 2002 Mar; 176(2):85-6.
15. Wong TS, Li JZ, Chen S, Chan JY, Gao W. The efficacy of triamcinolone acetonide in keloid treatment: a systematic review and meta-analysis. *Frontiers in Medicine.* 2016 Dec 27;3: 71.
16. Muneuchi G, Suzuki S, Onodera M, Ito O, Hata Y, Igawa HH. Long-term outcome of intralesional injection of triamcinolone acetonide for the treatment of keloid scars in Asian patients. *Scand J Plast Reconstr Surg Hand Surg.* 2006;40(2):111–116.
17. Sand M, Sand D, Boorboor P, Mann B, Altmeyer P, Hoffmann K, Bechara FG. Combination of surgical excision and custom designed silicon pressure splint therapy for keloids on the helical rim. *Head & Face Medicine.* 2007 Dec ;3 (1):1-4.
18. Bashir MM, Ahmad H, Yousaf N, Khan FA. Comparison of single intra operative versus an intra operative and two post operative injections of the triamcinolone after wedge excision of keloids of helix. *J Pak Med Assoc.* 2015 Jul 1;65(7):737-41.
19. Anayo N. K., Guinhouya K. M., Apetse K., Agba L., Assogba K., Belo M., & Balogou K. A. Posterior Reversible Encephalopathy Syndrome. A case reports. *Journal of Medical Research and Health Sciences.* 2022; 5(3):1804–1807.