ORIGINAL ARTICLE

KELOIDS AND HYPERTROPHIC SCARS: RESULTS WITH INTRA-OPERATIVE AND SERIAL POSTOPERATIVE CORTICOSTEROID INJECTION THERAPY

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Background: The management of keloids and hypertrophic scars continues to be controversial. Experience of treating 58 such lesions, 58.62% of which were recurrent, is presented.

Methods: Each lesion was subjected to surgical excision with intra-operative local injection of triamcinolone acetonide, followed by repeat injection of the same drug at weekly intervals for 2–5 weeks depending on the symptomatic relief, and then monthly injections for 4–6 months.

Results: Complete symptomatic relief was achieved in all patients within 5 weeks of surgery. Objective response in terms of no recurrence was noted in 91.9% of patients with keloids, and 95.24% of patients with hypertrophic scars at a mean follow-up of 30.5 months. Local or systemic complications were insignificant.

Conclusion: Because of promising results, further use and evaluation of this method of treatment is recommended for large, recurrent and complicated keloids and hypertrophic scars.

Key words: excision, hypertrophic scars, intra-operative steroids, keloids, postoperative treatment.

INTRODUCTION

Keloids and hypertrophic scars are one of the most trying challenges faced by the surgeon. No other cutaneous disease seems to create so much disagreement on the treatment as do these lesions. Many treatment modalities have been described including simple excision, local irradiation, steroid therapy, pressure therapy, lasers, silicon gel and enzyme therapy.^{1–7}

Steroids have been tried in different ways for treatment for keloids and hypertrophic scars.^{3,8,9} These have been used either alone or in combination with surgery and radiation with varying results.^{8,10–12} The present study was undertaken with a view to evaluate the effectiveness of surgery combined with intra-operative and serial postoperative local injections of steroids.

METHODS

The study was conducted on 58 patients over a period of 2 years. There were 37 keloids and 21 hypertrophic scars. The differentiation between the two types of lesions was made on clinical grounds,¹³ and confirmed by histopathological examination. The surface area of each lesion was calculated and recorded.

Seven lesions were excised under general anaesthesia and 51 were excised under local infiltration using 2% xylocain with 1:200 000 adrenaline; the type of anaesthesia depended on the age of the patient and the size of the lesion. Full-thickness excision of the lesion was performed in all the patients. This was followed by injection of triamcinolone acetonide around the margins and base of the wound. The dose of the drug was calculated from the schedule proposed by Ketchum *et al.*,¹⁴ depending

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on the size of the lesion and the age of the patient (Tables 1, 2). Smaller defects were closed primarily after lateral undermining of the edges, and larger defects were resurfaced with a split-thickness skin graft. Wound closure was accomplished using a non-traumatizing surgical technique and meticulous skin edge coaptation with absorbable sutures (3,4-0 chromic catgut) for subcutaneous tissue, and non-absorbable suture (4,5-0 prolene) for skin. Sutures were removed as early as possible depending on the situation, taking particular care to avoid wound dehiscence.

All excised specimens were subjected to histopathological examination for confirmation of clinical diagnosis.

Patients were followed up and each lesion was treated further with 2–5 injections of triamcinolone acetonide in the scar at weekly intervals, depending on the symptomatic relief of the patients from pain, pruritis and paraesthesia. The weekly injections were then followed by monthly injections for another 4–6 months.

After completion of treatment the patients were regularly followed up over a period ranging from 11 to 48 months, with a mean of 30.5 months. Any symptomatic relief obtained by the patient was noted. Patients were examined for local and systemic complications of treatment.

The response of the treatment was judged in terms of any recurrence. A scale consisting of no recurrence, partial recurrence, full recurrence or worse than pre-operative status was established and the results of treatment were analysed.

RESULTS

There were 23 males and 35 females in the present study. The ages ranged from 4 to 52 years, with $\sim 80\%$ patients presenting in the second and third decades of life. Anatomical distribution of the lesions and the aetiological factors are shown in Table 3. Of the eight ear lobe lesions caused by trauma, four patients developed this after ear piercing, two developed this after surgical repair for torn ear lobes, and two lesions followed ear lacerations. All four

sternal lesions in our patients developed on sternotomy scars after open heart surgery. In the upper arm eight lesions were located in the deltoid region, four of which were caused by surgical removal of benign lesions and two by acid burns. In the forearm 50% of lesions were due to acid burns (used as a tattoo), and of those in the hand, 50% of lesions followed thermal burns. In the leg all lesions were located on the shin and three were secondary to infections. Cause was not known in seven of our patients. In the trauma group, eight patients gave

Table 1. Dosage of triamcinolone acetonide used in adults

Size of lesion (cm ²)	Dosage (mg)		
1–2	20-40		
2–6	40-80		
6–10	80-100		
> 10	100-120		

 Table 2.
 Dosage of triamcinolone acetonide used in children

Age (years)	Dosage (mg)
1–2	20
2–5	40
5-10	80

Table 3. Distribution and aetiology of 58 lesions

Site of lesion	п	Aetiological factors				
				Infection	Not known	
Head and neck						
Ear lobes	13	8	1	3	1	
Neck	5		3	1	1	
Chin	2	1			1	
Chest						
Sternal	4	4	_		_	
Pectoral	3		1	1	1	
Abdomen	2	2	_	_		
Back	1		_		1	
Upper extremity						
Deltoid	8	4	2	1	1	
Forearm	6	1	3	2		
Dorsum of hand	6	1	3	1	1	
Lower extremity						
Thigh	2	1		1	_	
Leg	5		2	3	_	
Foot	1			1	—	

Table 6. Overall response to treatment

history of healing of their wounds by second intention and in the burn group all patients had sustained deep burns and all were managed by conservative treatment. Eleven lesions were small (0–5 cm²), 33 lesions were medium sized (5–15 cm²) and 14 lesions were large (> 15 cm²). The smallest lesion measured 1.2 cm² and the largest one was 60 cm², with a mean surface area of 11.42 cm². Thirty-seven lesions were confirmed as keloids and 21 as hypertrophic scars on histopathological examination.

Sixteen patients had received topical or intralesional steroids, 10 patients had received pressure therapy, two cases recurred after excision alone, while six patients had been treated by a combination of surgery or topical steroids with pressure therapy.

Primary closure of the wounds was achieved in 44 patients, while 14 patients needed skin grafting after excision of the lesions.

The dosage of triamcinolone acetonide used per sitting ranged from 20 to 120 mg, with a mean of 86.2 mg. The maximum number of initial weekly postoperative injections needed by the patients to get significant subjective symptomatic relief is shown in Table 4. The maximum number of monthly injections received by the patients is shown in Table 5.

On analysing the objective response, partial recurrence was noted in three (5.17%) patients (two keloids on the chest wall and one hypertrophic scar on the face), full recurrence was noted in one (1.73%) patient with a keloid on sternotomy incision, and in 54 (93.10%) patients the lesions did not recur. Of the four patients who showed recurrence, two lesions recurred within 6 months and two recurred within 12 months after completion of treatment. However, no lesion worsened after treatment. The overall response of the treatment is given in Table 6. The examples of treatment results of some patients are shown in Figs 1-3.

Table 4. Maximum number of initial postoperative injectionsneeded (per week) to get significant symptomatic relief

Maximum no. injections (per week)	No. patients	%	
2	2	3.45	
3	15	25.86	
4	24	41.37	
5	17	29.32	

 Table 5.
 Maximum number of monthly injections received by the patients

Maximum no. monthly injections	No. patients	%	
4	15	25.86	
5	34	58.63	
6	9	15.51	

Nature of	No.	Objective response				
lesion	lesions	Symptomatic response n (%)	Full recurrence n (%)	Partial recurrence n (%)	No recurrence <i>n</i> (%)	
Keloids	37	37 (100)	1 (2.7)	2 (5.4)	34 (91.9)	
Hypertrophic scars	21	21 (100)	0 (0.00)	1 (4.76)	20 (95.24)	

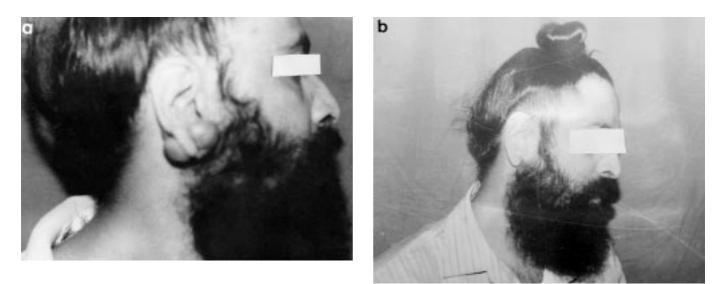


Fig. 1. (a) Recurrent ear lobe keloid following excision alone. (b) Same patient 22 months after completion of treatment.

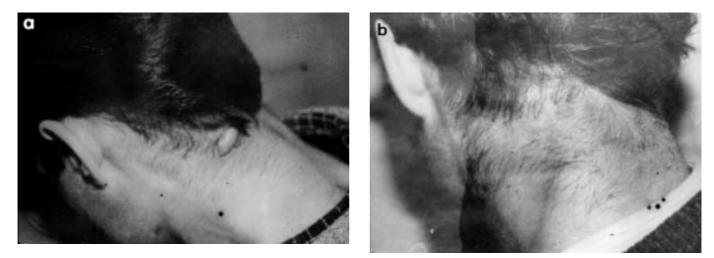


Fig. 2. (a) Small keloid on neck. Patient needed only two (weekly) and four (monthly) injections of triamcinolone acetonide in the postoperative period to achieve significant response. (b) Patient recurrence-free at 18 months following treatment.

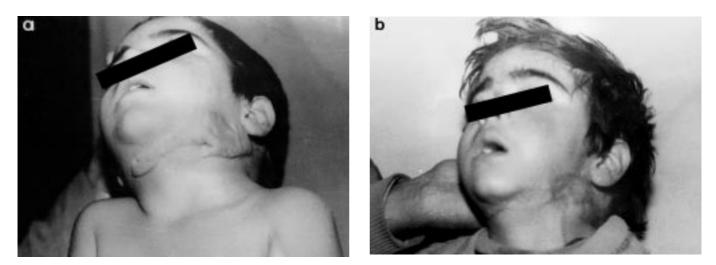


Fig. 3. (a) Hypertrophic scar which had shown no response to topical steroids and pressure therapy. (b) Child is recurrence-free 16 months after excision of the lesion, skin grafting and intra-operative and postoperative triancinolone injections.

Local complications of the treatment included infection in two patients (3.44%), dermal atrophy in one patient (1.72%) and hypopigmentation in one patient (1.72%). However, hypopigmentation recovered completely in 9 months, while dermal atrophy persisted at 26 months after completion of treatment. Seven female patients (12%) developed transitory menorrhagia and all patients attained normal cycles within 3 months after completion of treatment. Cushingoid features were noted in four patients (6.89%) but resolved completely within 2–3 months after cessation of treatment in all patients.

DISCUSSION

Although keloids and hypertrophic scars are known to every surgeon, their aetiology remains obscure and their treatment controversial. The incidence varies from race to race. Black people and Asian people are more likely to develop these lesions than Caucasians, the incidence varying from 5:1 to 15:1.^{15,16} Aberration of the metabolism of melanocyte-stimulating hormone has been suggested to be responsible for the racial difference.¹⁶ Keloid formation is rare at sites with a low melanocyte concentration, such as the palms and the soles, and the incidence of keloid is higher during times of physiologic hyperactivity of the pituitary such as puberty and pregnancy. These lesions have been the subject of an extensive discussion regarding their pathogenesis and treatment. Many treatment modalities have been advocated but none have been universally successful.

Pressure therapy no doubt is effective for the prevention of scar hypertrophy, and is useful even for established keloids, but has to be used continuously and for months together, and every patient may not comply.¹⁷ Cryotherapy is associated with residual hypertrophy and discoloration, and needs technical expertise.¹⁸ The use of radiation in the treatment of keloids is well established but its hazards (such as atrophy, lack of growth and increased incidence of malignancy) limit its role. Cohan and McCoy strongly recommended that radiation therapy be used only to treat keloids in the elderly and then only when all other methods have failed.¹⁹ Surgery alone has a high recurrence rate.²⁰ It has been recommended as a second-line therapy for lesions that do not respond to steroids and pressure, and large lesions requiring debulking.²¹

The corticosteroids apparently act to diminish collagen synthesis and to decrease X_2 -macroglobulins and X_1 -antitrypsin by potent collagenase inhibiting activity.²² Conway and Stark achieved symptomatic relief by injecting hydrocorticone into the keloids, but no change in contour or texture of the lesion.²³ It was Hollander who was successful in causing regression of the keloid, as well as relief of symptoms while using a fluorinated derivative, triamcinolone.²⁴ Intralesional steroid therapy is now a mainstay of therapy. Nicolai *et al.* recommended steroid injections for narrow lesions, and pressure therapy for broad lesions.²⁵ In the case of no response in 6 months or if the lesions recur after a good response, surgery is indicated.

Various combination therapies have been advocated and surgical excision with steroid injection of the wound rim is now in common practice.21 One of the advantages of this combination therapy is to prevent the wide scar which otherwise results by using intralesional steroids alone without excision. Intralesional steroids have been used before, as well as after, surgery.1,19,26 But we do not think there is any added advantage to using the drug before surgery, as reported by Brown and Pierce.26 The aim of using intensive postoperative steroid injections was to achieve collagen degradation as early as possible, and in our study all the patients were free of symptoms after 2-5 weeks. Similarly, intensive weekly postoperative steroid therapy which was maintained thereafter by 2-4 weekly injections for a period of 5 months, was used by Tang.12 His study included only 11 patients. After symptomatic relief we maintained the patients on monthly injections of triamcinolone acetonide for 4-6 months to prevent recurrence, because this is the period during which scars have a high potential for hypertrophy.1,12,19,26 Some protocols recommend an intraoperative first dose followed by 4-6-weekly injections for 6-10 months.^{1,19} Subsequent doses were given for recurrences. Two of the patients with partial recurrence in our study did not agree to further treatment, the third is responding to pressure therapy. The patient with full recurrence is undergoing pressure therapy combined with monthly injections of steroids and has responded.

The use of intralesional steroid therapy with surgery has been reported in other studies, the results of which are summarized in Table 7,8,10,12,27–35 and most of which have shown excellent objective response rates.

Golladay, in a study of 28 keloids in 19 children, had only one recurrence following treatment involving surgical excision with single intra-operative dilute repository steroid injected at the interface of the lesion with normal tissue.³⁵ He is of the opinion that

Table 7. R	Results of v	arious studies	using surgery	with intralesional	steroid therapy for kel	oids and hypertrophic scars

Author (year)	Treatment method used	No. patients	Significant symptomatic response (%)	Significant objective response (%)
Belisario ⁸ (1957)	Surgery and intralesional cortisone	16		31
Murray ²⁷ (1963)	Surgery and topical/intralesional triamcinolone	67		57
Griffith et al. ¹⁰ (1970)	Surgery and intralesional triamcinolone	31		94
Singleton and Gross ²⁸ (1971)	Surgery and intralesional methylprednisolone or triamcinolone	29		82
Oluwasanmi ²⁹ (1974)	Surgery and intralesional triamcinolone	23	96	15
Moustafa and Abdel-Fattah ³⁰ (1976)	Surgery and intralesional dexamethasone	50		96
Kiil ³¹ (1977)	Surgery and intralesional triamcinolone	15		50
Barton ³² (1978)	Surgery and intralesional triamcinolone	12		95
Shones and Press ³³ (1983)	Surgery and intralesional triamcinolone	20		97
Salasche and Grabski ³⁴ (1983)	Surgery and intralesional triamcinolone	6		100
Golladay ³⁵ (1988)	Surgery and intralesional betamethasone	19		100
Tang ¹² (1992)	Surgery and intralesional triamcinolone	11	100	81.80
Present study (1994)	Surgery and intralesional triamcinolone	58	100	93.10

perilesional injection just before excision allows a uniform layer of material to be available for inhibiting collagen synthesis in the area of excision for several weeks. The avoidance of postoperative injections certainly has an advantage in children. We do not find any reason for not achieving uniform distribution of the drug by injecting it after excision of the lesion. In fact, drug loss with the excised specimen can be prevented by using this technique. However, for comparing the results of single intra-operative with multiple postoperative injections of steroid, further studies need to be carried out to settle the issue.

Steroids should be used with caution because an overdose may result in serious infections. Local side effects of the drug in the form of infection, atrophy, depigmentation, and telangiectasia have been reported in the literature.^{10,12,26} The complications have a low incidence rate overall and most of these are transitory and resolve after cessation of therapy. Menstrual disturbances and cushingoid symptoms are rare.²⁶ To minimize the occurrence of most of the local complications, the drug should be injected into the lesion only, and not into the subcutaneous tissue. Ketchum *et al.* reported that 95% of these effects result from incorrect use of the drug.¹⁷

Since all of our recurrences occurred in the first year of completion of treatment, we think that a mean follow-up of 30.5 months for our patients was adequate. Kiil noted a 33% recurrence in 1 year and a 50% recurrence at 5 years but he was using an air injection technique.³¹

Because 58.62% of our patients had been treated previously with other modalities, we support the view of other authors of using surgical excision with intra-operative and postoperative steroid therapy for recurrent, complicated and large keloids and hyper-trophic scars.^{12,21} But for small and simple lesions, other measures such as mechanical pressure and steroid therapy alone should be the first line of treatment.

REFERENCES

- Ramakrishnan KM, Thomas KP, Cheyyur R. Study of 1000 patient with keloids in South India. *Plast. Reconstr. Surg.* 1974; 53: 276–80.
- DeBeurmann R, Gougerot H. Cheloides des maquescies. Ann. Dermatol. Syplilol. 1906; 7: 151–8.
- 3. Conway H, Stark RB. ACTH in plastic surgery. *Plast. Reconstr. Surg.* 1951; **8**: 354–7.
- Mercer DM, Studel DMMM. 'Oyster Splints': A new compression device for the treatment of keloid scars of the ear. *Br. J. Plast. Surg.* 1983; 36: 75–8.
- Henderson DL, Cromwell TA, Mes LG. Argon and carbondioxide laser treatment of hypertrophic and keloid scars. *Lasers Surg. Med.* 1984; 3: 271–7.
- 6. Mercer NSG. Silicone gel in the treatment of keloid scars. *Br. J. Plast. Surg.* 1989; **42**: 83–7.
- Lawrence WT. In search of the optimal treatment of keloids: Report of a series and a review of literature. *Ann. Plast. Surg.* 1991; 27: 164–78.
- Balisario JC. The treatment of keloids. Acta Derm. Vanereol. 1957; 37: 165–85.
- Ketchum LD, Smith J, Robinson DW, Masters FW. The treatment of hypertrophic scars, keloids and scar contracture by triamcinolone acetonide. *Plast. Reconstr. Surg.* 1966; 38: 209–17.
- Griffith BH, Monroe CW, McKinney P. A follow-up study on the treatment of keloids with triamcinolone acetonide. *Plast. Reconstr. Surg.* 1970; 46: 145–50.

- Darzi MA, Chowdri NA, Koul SK, Khan M. Evaluation of various methods of treating keloids and hypertrophic scars: A 10 year follow-up study. *Br. J. Plast. Surg.* 1992; **45**: 374–9.
- 12. Tang YW. Intra and post-operative steroid injections for keloids and hypertrophic scars. *Br. J. Plast. Surg.* 1992; **45**: 371–3.
- 13. Muir IEK. On the nature of keloid and hypertrophic scars. *Br. J. Plast. Surg.* 1990; **43**: 61–9.
- Ketchum LD, Robinson DW, Masters FW. Follow-up of treatment of hypertrophic scars and keloids with triamcinolone. *Plast. Reconstr. Surg.* 1971; 48: 256–9.
- Alhady SMA, Sivanantharajah K. Keloids in various races: A review of 175 cases. *Plast. Reconstr. Surg.* 1969; 44: 564–6.
- 16. Koonin AJ. The aetiology of keloids: A review of the literature and a new hypothesis. S. Afr. Med. J. 1964; **38**: 913–16.
- Ketchum LD, Cohen IK, Masters FW. Hypertrophic scars and keloids: A collective review. *Plast. Reconstr. Surg.* 1974; 53: 140–54.
- 18. Muti E, Ponzio E. Cryotherapy in the treatment of keloids. *Ann. Plast. Surg.* 1983; **11**: 227–32.
- Cohen IK, McCoy BJ. The biology and control of surface overhealing. World J. Surg. 1980; 4: 289–95.
- Cosman B, Wolff M. Correlation of keloid recurrence with completeness of local excision: A negative report. *Plast. Reconstr. Surg.* 1972; **50**: 163–6.
- Rockwell WB, Cohen IK, Ehrlich HP. Keloids and hypertrophic scars: A comprehensive review. *Plast. Reconstr. Surg.* 1989; 84: 827–7.
- McCoy BJ, Diegelmann RF, Cohen IK. Invitro inhibition of cell growth, collagen synthesis and polyhydroxylase activity by triamcinoline acetonide. *Proc. Soc. Exp. Biol. Med.* 1980; 163: 216–22.
- 23. Conway H, Stark RB. ACTH in plastic surgery. *Plast. Reconstr. Surg.* 1951; **8**: 354–7.
- Hollander A. Intralesional injections of triamcinolone acetonide: A therapy for dermatoses. *Antibiot. Med.* 1961; 8: 78–83.
- Nicolai JP, Bos MY, Bronkhorst FB, Smale CF. A protocol for the treatment of hypertrophic scars and keloids. *Aesthetic Plast. Surg.* 1987; 11: 29–32.
- Brown IA, Pierce HE. Keloids: Scar revision. J. Dermatol. Surg. Oncol. 1986; 12: 51–6.
- Murray RD. Kanalog and the treatment of hypertrophied scars and keloids in Negroes and whites. *Plast. Reconstr. Surg.* 1963; 31: 275–80.
- Singleton MA, Gross CW. Management of keloids by surgical excision with local injections of steroid. *South. Med. J.* 1971; 64: 1377–8.
- Oluwasanmi JO. Keloids in the African. *Clin. Plast. Surg.* 1974; 1: 179–95.
- Moustafa MFH, Abdel-Fattah AMA. Keloids of the ear lobe in Egypt: Their rarity in childhood and their treatment. *Br. J. Plast. Surg.* 1976; 29: 59–60.
- Kiil J. Keloid treatment with topical injections of triamcinolone acetonide (Kenalog): Immediate and long-term results. *Scand. J. Plast. Reconstr. Surg.* 1977; 11: 169–72.
- 32. Barton RPE. Auricular keloids: A simple method of management. Ann. R. Coll. Surg. Engl. 1978; 60: 324–5.
- Shones AR, Press BHJ. The treatment of earlobe keloids by surgical excision and post-operative triamcinolone injections. *Ann. Plast. Surg.* 1983; 10: 480–2.
- 34. Salasche SJ, Grabski WJ. Keloids of the earlobes: A surgical technique. J. Dermatol. Surg. Oncol. 1983; 9: 552–6.
- Golladay ES. Treatment of keloids by single intra-operative perilesional injection of repository steroid. *South. Med. J.* 1988; 81: 736–8.