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COMPARISON OF THE EFFECTS OF INTRALESIONAL TRIAMCINOLONE ACETONIDE TREATMENT WITH INTRALESIONAL VERAPAMIL HYDROCHLORIDE IN PATIENTS PRESENTING WITH SCAR

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ABSTRACT

Keloid is an abnormal fibrous tissue development extend beyond the incision. Intralesional triamcinolone acetonide is the most used keloids corticosteroid. Intralesional verapamil is new. Both medications were compared to help us choose the best keloids treatment.

OBJECTIVE: To compare the effects of intralesional triamcinolone acetonide treatment with intralesional verapamil hydrochloride in patients presenting with scar at Jinnah Hospital, Karachi.

METHODS: This Randomized was held in the Department of Dermatology, JPMC, Karachi for Six months after approval from 03 July, 2021 till 03 January 2022. Data was prospectively collected from patients after taking a verbal consent. 100 patients who met the diagnostic criteria were included. Quantitative and qualitative data was collected, presented and analyzed. Post stratification chi square test was applied taking p-value of ≤0.05 as significant.

RESULTS: A total of 124 patients who met the inclusion and exclusion criteria were included in this study. Mean age and duration of scar in our study was 48.21±6.24 years and 14.54±9.78 months in the intralesional triamcinolone acetonide group. Mean age and duration of scar in our study was 49.48±8.41 years and 14.97±9.56 months in the intralesional triamcinolone acetonide group.

CONCLUSION: Both triamcinolone and verapamil intralesional injections can achieve scar flattening in hypertrophic scars and keloids. Intralesional triamcinolone continues to be the gold standard for the first line treatment owing to its rapid and effective response.

KEYWORDS: Intralesional triamcinolone acetonide, intralesional verapamil hydrochloride and scar.

INTRODUCTION

Cutaneous scarring is a common condition. Wound healing and remodeling are challenging and results from several internal and external elements working together. Not all wounds heal well, even under optimal conditions[1-2]. They usually occur on the sternum, shoulders, earlobes, and cheeks. Deviant healing can lead to excessive keloids. Keloids can also arise spontaneously. Collagen buildup in the dermis and subcutaneous tissues is characteristic. Disfigurement, contractures, pruritus, and pain can develop from keloid and hypertrophic scarring, unlike normal wound treatment. No single treatment has been found to work for all patients, including silicone gel sheeting, intralesional injections, surgical manipulation, laser, and radiotherapy[3-4]. Bleomycin and 5-fluorouracil are more effective yet expensive and induce serious side effects. Surgery and laser therapy have drawbacks, while radiotherapy can induce cancer. Currently, steroids are the principal treatment[5-6]. TCA reduces protein synthesis, fibroblast migration, collagen and glycosaminoglycan production, and degeneration while increasing collagen and fibroblast degeneration and anti-inflammatory activity. Ca2+ channel blocker verapamil reduces scar extracellular matrix formation in vitro[7]. Depolymerizing actin filaments increases pro-collagenase release, changing fibroblast shape. In general, verapamil has less side effects than corticosteroids, allowing for extended treatment and use in circumstances where they cannot[7-8]. Saki et al. assessed post-treatment Vancouver scar scale scores for height (0.21±0.56 vs 3.10±1.85), vascularity (0.93±0.70 vs 0.87±0.74), pliability (0.20±0.41 vs 2.07±0.26), and pigmentation (0.13±0.35 vs 0.27±0.75). This study compares intralesional triamcinolone acetonide and verapamil hydrochloride in scar patients to provide a local viewpoint since local data is scarce. Local and international studies indicate mixed results[9-10]. This investigation is justified because these findings were inconsistent and verapamil has not been suggested as a corticosteroid substitute. Keloid and hypertrophic scar treatment are difficult and recurrent. Not all treatments have worked. Clinicians' understanding and scar treatment plans might benefit from this data.

METHODS

This Randomized was held in the Department of Dermatology, JPMC, Karachi for Six months after approval from 03 July, 2021 till 03 January 2022. The sample size for the study will be n=550 patients with 275 in each group. The sample size for was calculated by using the WHO software where, Alpha=5%, Power of the test 1-beta=80, pigmentation (0.13±0.35 vs 0.27±0.75) after 24 weeks of treatment with intralesional triamcinolone acetonide with intralesional verapamil hydrochloride for scars. 14 However, keeping in mind the number of patients with scars we will be able to treat during the study period we will take the sample size of 100 patients 50 in each group for this study and Non-probability consecutive sampling technique was used.

Inclusion criteria:

- Patients presenting with scars as per operational definition were included in the study.
- Either gender.
- Age 20-60 years.

Exclusion criteria:

- Patients with history of receiving treatment for the same keloid and hypertrophic scar in the past 12 months and those who had active inflammation, infection, or ulcer in or around the keloid.

• Immunosuppressed patients.
• Patients with chronic inflammatory diseases.
• Patients with history of melanoma.
• Pregnant patients assessed by history and confirmed by dating scan.
• Patients with history of renal impairment, chronic obstructive pulmonary disease, asthma, congestive heart failure, myocardial infarction and chronic liver disease will be excluded.

The College of Physicians and Surgeons Pakistan approved this study. The study included patients from JPMC, Karachi Outpatient Department of Dermatology with operational scars. Study was conducted with institutional ethical review committee approval. All patients gave informed consent for sample assignment and research use. Age, gender, and scar duration were recorded. A dermatologist with over 10 years of expertise assessed patients with the researcher. The researcher photographed and documented Vancouver scar scale (VSS) scar evaluation before therapy. Scars were scored on height, vascularity, pliability, and coloration. A millimeter ruler assessed scar height correctly. Palpating graded scar pliability subjectively. Visual inspection and blanching refill rate determined scar vascularity. Blanching and comparing scar colour to skin measured scar pigmentation. The sealed opaque envelopes labelled A= Intralesional triamcinolone acetonide and B= Verapamil hydrochloride randomly assigned patients. Group A patients with scars received intralesional triamcinolone acetonide 20mg and Group B patients received verapamil hydrochloride (1ml (2.5 mg)) every 3 weeks for a maximum of 8 sessions or until scar flattening. An insulin syringe and 24-gauge needle were used to inject the lesion at many sites to induce complete and equally distributed blanching at endpoint. No sedation/analgesia was employed during injection. Scar flattening (pigmentation and duration in both groups) and qualitative characteristics (gender, site of lesion, diabetes mellitus type II, hypertension, smoking status, monthly income status, and employment status) were included in the performa attached as an annexure.

Data was analysed using SPSS 20. For both groups, mean and standard deviations were calculated for age, pretreatment and posttreatment height, vascularity, scar pliability and pigmentation, and scar longevity. Data was provided as mean ± SD for normally distributed variables and median (IQR) for non-normal variables. Qualitative characteristics such gender, site of lesion, diabetes type II, hypertension, were calculated as frequencies and percentages. An independent t test compared posttreatment effects on Vancouver scar scale scoring parameters at 24 weeks in the two groups. Effect modifiers were controlled by stratifying age, gender, lesion site, diabetes type II, hypertension, smoking, monthly income, occupation, and scar duration. After stratification, a p-value of < 0.05 was taken as significant in the independent t test.

RESULTS

This study comprised 124 patients who met inclusion and exclusion criteria at JPMC Karachi Department of Dermatology.

In the intralesional triamcinolone acetonide group, 50 patients ranged in age from 20 to 60. In our study, the mean age and scar duration were 48.21±6.24 years and 14.54±9.78 months, respectively. In the intralesional triamcinolone acetonide group, 50 patients ranged in age from 20 to 60. The average age and scar duration in our study were 49.48±8.41 years and 14.97±9.56 months, respectively. As in Table 1.

TABLE-1: DESCRIPTIVE STATISTICS IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) N=100

VARIABLE	MEAN ± SD	STANDARD DEVIATION	MIN-MAX
AGE INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (YEARS)	48.21	±6.24	20-60
AGE INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (YEARS)	49.48	±8.41	20-60
DURATION OF SCAR INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (MONTHS)	14.54	±9.78	8-24
DURATION OF SCAR INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (MONTHS)	14.97	±9.56	8-24

Of 50 intralesional triamcinolone acetonide patients, 26 (52%), and 24 (48%), were aged 20-40 and 41-60, respectively. In the intralesional verapamil hydrochloride group, 27 (54%) and 23 (46%) of 50 patients were 20-40 and 41-60 years old. According to Table 2.

TABLE-2: AGE DISTRIBUTION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50), n=100

AGE	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL
		HYDROCHLORIDE GROUP
20-40 YEARS	26 (52%)	27 (54%)
41-60 YEARS	24 (48%)	23 (46%)
TOTAL	50 (100%)	50 (100%)

The intralesional triamcinolone acetonide group had 50 patients, 26 (52%) male and 24 (48%) female. In the intralesional verapamil hydrochloride group, 28 (56%) and 22 (44%) of 50 patients were male and female. According to Table 3.

TABLE -3: GENDER DISTRIBUTION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) n=100

GENDER	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP
MALE	26 (52%)	28 (56%)
FEMALE	24 (48%)	22 (44%)
TOTAL	50 (100%)	50 (100%)

Out of 50 intralesional triamcinolone acetonide patients, 56% (28 patients) and 44% (22 patients) developed scars lasting < 12 and > 12 months, respectively. Of 50 patients in the intralesional verapamil hydrochloride group, 52% (26 patients) and 48% (24 patients) developed scars lasting < 12 and > 12 months, respectively. As shown in Table 4.

TABLE-4 SHOWS THE DURATION OF SCARS DISTRIBUTION BETWEEN THE TWO GROUPS

DURATION OF SCAR	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP
≤ 12 MONTHS	28 (56%)	26 (52%)
> 12 MONTHS	22 (44%)	24 (48%)
TOTAL	50 (100%)	50 (100%)

Of 50 intralesional triamcinolone acetonide patients, 32 (64%) had diabetes and 18 (36%) did not. Of 50 intralesional verapamil hydrochloride patients, 26 (52%) had diabetes and 24 (48%) did not. According to Table 5.

TABLE-5: DIABETES MELLITUS TYPE II DISTRIBUTION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) n=100

DIABETES MELLITUS TYPE II	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP
YES	32 (64%)	26 (52%)
NO	18 (36%)	24 (48%)
TOTAL	50 (100%)	50 (100%)

Out of 50 intralesional triamcinolone acetonide patients, 26 (52%) had hypertension and 24 (48%) did not. Of 50 intralesional verapamil hydrochloride patients, 32 (64%) had hypertension and 18 (36%) did not. As shown in Table 6.

TABLE-6: HYPERTENSION DISTRIBUTION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) n=100

HYPERTENSION	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP
YES	26 (52%)	32 (64%)
NO	24 (48%)	18 (36%)
TOTAL	50 (100%)	50 (100%)

Of 50 intralesional triamcinolone acetonide patients, 15 (30%), 11 (22%), 13 (26%) and 11 (22%) developed scars on their faces, pre-sternums, trunks, and extremities. Among 50 intralesional verapamil hydrochloride patients, 14 (28%), 12 (24%), 14 (28%) and 10 (20%) had scars on their faces, pre-sternums, trunks, and extremities. Table 7.

TABLE-7: SITE OF LEISION DISTRIBUTION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) n=100

SITE OF LEISION	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP
FACE	15 (30%)	14 (28%)
PRESTERNAL	11 (22%)	12 (24%)
TRUNK	13 (26%)	14 (28%)
EXTREMITIES	11 (22%)	10 (20%)
TOTAL	50 (100%)	50 (100%)

The intralesional triamcinolone acetonide group had a mean height of 0.85±0.48 while the verapamil hydrochloride group had 0.82±0.49. 0.001 P-value. Table 8.

TABLE-8: POSTTREATMENT HEIGHT IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50)

VARIABLE	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
POSTTREATMENT HEIGHT	0.85±0.48	0.82±0.49	**0.001**

Out of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, mean height was 0.85±0.42 and 0.82±0.33 for those with diabetes mellitus, respectively. The P-value was 0.02. The mean height of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups was 0.85±0.36 and 0.82±0.39 for those without diabetes mellitus, respectively. 0.001 P-value. Table 9.

TABLE-9: POSTTREATMENT HEIGHT IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO DIABETES MELLITUS TYPE II (n=100)

DIABETES MELLITUS TYPE II	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	0.85±0.42	0.82±0.33	**0.02**
NO	0.85±0.36	0.82±0.39	**0.001**

Out of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, the mean height was 0.85±0.29 and 0.82±0.39 for hypertensive patients, respectively. The P-value was 0.02. The mean height of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups was 0.85±0.41 and 0.82±0.41 in non-hypertensive patients, respectively. 0.001 P-value.

TABLE-10: POSTTREATMENT HEIGHT IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO HYPERTENSION (n=100)

HYPERTENSION	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	0.85±0.29	0.82±0.39	**0.02**
NO	0.85±0.41	0.82±0.41	**0.001**

The mean intralesional vascularity was 1.02±0.68 for triamcinolone acetonide and 0.99±0.71 for verapamil hydrochloride. 0.001 P-value. According to Table 20. Out of 50 patients, the mean vascularity in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups was 1.02±0.67 and 0.99±0.59 for the age group 20-40 years, respectively. 0.001 P-value. Age stratification revealed mean vascularity of 1.02±0.62 and 0.99±0.55 in the 41-60 age group for

intralesional triamcinolone acetonide and verapamil hydrochloride patients, respectively, out of 50 patients. 0.001 P-value. Table 11.

TABLE-11: POSTTREATMENT VASCULARITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO AGE (n=100)

AGE (YEARS)	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
20-40	1.02±0.67	0.99±0.59	**0.001**
41-60	1.02±0.62	0.99±0.55	**0.001**

In individuals with diabetes mellitus, mean vascularity was 1.02±0.62 in the intralesional triamcinolone acetonide group and 0.99±0.58 in the intralesional verapamil hydrochloride group out of 50 patients. The P-value was 0.02. Out of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, mean vascularity was 1.02±0.59 for diabetes mellitus and 0.99±0.57 for non-diabetes. 0.001 P-value. Table 12.

TABLE-12: POSTTREATMENT VASCULARITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO DIABETES MELLITUS TYPE II (n=100)

DIABETES MELLITUS TYPE II	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	1.02±0.62	0.99±0.58	**0.02**
NO	1.02±0.59	0.99±0.57	**0.001**

Stratification for hypertension revealed mean vascularity of 1.02±0.67 in intralesional triamcinolone acetonide group and 0.99±0.62 in intralesional verapamil hydrochloride group out of 50 patients. The P-value was 0.02. In the intralesional triamcinolone acetonide group, mean vascularity was 1.02±0.66 while in the intralesional verapamil hydrochloride group, it was 0.99±0.63 without hypertension. 0.001 P-value. Table 13.

TABLE-13: POSTTREATMENT VASCULARITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO HYPERTENSION (n=100)

HYPERTENSION	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	1.02±0.67	0.99±0.62	**0.02**
NO	1.02±0.66	0.99±0.63	**0.001**

The intralesional triamcinolone acetonide group had a mean pliability of 0.83±0.48 while the verapamil hydrochloride group had 0.83±0.46. 0.001 P-value. Table 14.

TABLE-14: POSTTREATMENT PLIABILITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) (n=100)

VARIABLE	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
POSTTREATMENT PLIABILITY	0.83±0.48	0.83±0.46	**0.001**

Out of 50 patients in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, mean pliability was 0.83±0.44 and 0.83±0.46 for scar duration ≤ 12 months, respectively. The P-value was 0.02. In the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, the mean pliability, out of 50 patients, was 0.83±0.44 and 0.83±0.45 in the rural residence group, respectively, based on scar duration. 0.001 P-value. According to Table 15.

TABLE-15 POSTTREATMENT PLIABILITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO DURATION OF SCAR (n=100)

DURATION OF SCAR	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
≤ 12 MONTHS	0.83±0.44	0.83±0.46	**0.02**
> 12 MONTHS	0.83±0.44	0.83±0.45	**0.001**

Patients with diabetes mellitus exhibited a mean pliability of 0.83±0.45 in the intralesional triamcinolone acetonide group and 0.83±0.44 in the intralesional verapamil hydrochloride group out of 50 patients. The P-value was 0.02. Out of 50 patients divided into intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, mean pliability was 0.83±0.47 for diabetes mellitus and 0.83±0.41 for non-diabetes. 0.001 P-value. Table 16.

TABLE-16: POSTTREATMENT PLIABILITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO DIABETES MELLITUS TYPE II (n=100)

DIABETES MELLITUS TYPE II	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	0.83±0.45	0.83±0.44	**0.02**
NO	0.83±0.47	0.83±0.41	**0.001**

Out of 50 patients, the mean pliability in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups was 0.83±0.47 and 0.83±0.47 for hypertension patients, respectively. The P-value was 0.02. Out of 50 individuals in the intralesional triamcinolone acetonide and intralesional verapamil hydrochloride groups, mean pliability was 0.83±0.46 for hypertension patients and 0.83±0.46 for non-hypertensive patients. 0.001 P-value. Table 17.

TABLE-17: POSTTREATMENT PLIABILITY IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP ACCORDING TO HYPERTENSION (n=100)

HYPERTENSION	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
YES	0.83±0.47	0.83±0.47	**0.02**
NO	0.83±0.46	0.83±0.46	**0.001**

The intralesional triamcinolone acetonide group had a mean pigmentation of 0.88±0.45 while the verapamil hydrochloride group had 0.87±0.44. 0.001 P-value. Table 18.

TABLE-18: POSTTREATMENT PIGMENTATION IN INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP (50) VERSUS INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP (50) (n=100)

VARIABLE	INTRALESIONAL TRIAMCINOLONE ACETONIDE GROUP	INTRALESIONAL VERAPAMIL HYDROCHLORIDE GROUP	P VALUE
POSTTREATMENT PIGMENTATION	0.88±0.45	0.87±0.44	**0.001**

DISCUSSION

Keloids are firm, sensitive plaques more common on shoulders, chest, neck, upper arms, and face. They are benign fibrous tissue overgrowths that extend beyond a skin damage caused by trauma, inflammation, surgery, or burns[11-12]. Uncontrolled keloids can cause aesthetic disfigurement and functional disability, lowering quality of life[13-14]. This study includes 124 patients who met inclusion and exclusion criteria. Our study found a mean scar duration of 14.54±9.78 months and an age of 48.21±6.24 years in the intralesional triamcinolone acetonide group Our study found a mean scar duration of 14.97±9.56 months and an age of 49.48±8.41 years in the intralesional triamcinolone acetonide group Another study included 80 patients, 42 (52.5%) male and 38 (47.5%) female. Patients ages ranged from 12 to 40, with a mean of 25.96 (±6.982) years. Most patients were 21–30. The triamcinolone group had a 58.28% baseline score drop after the research, while the verapamil group had 36.75%. Triamcinolone caused pain in most patients, hypopigmentation in five, and abnormal menstrual cycles in two females. Intralesional Verapamil caused just pain[15-16]. Intralesional triamcinolone acetonide is superior than intralesional verapamil for keloids. Combining intralesional verapamil improves results. To assess the effects of i/l triamcinolone (T) (22 scars) and verapamil injections (V) (26 scars), 40 patients (48 scars) were randomized, parallel, and observer blinded. The study protocol set the maximum indicated volume for triamcinolone (40 mg/ml) and verapamil (2.5 mg/ml) at 1.5 ml[17-18]. Patients aged 15–60 with scars 0.5–5 cm (but total area under 2 years) were considered. Keloidal diathesis patients were excluded. The scar was injected every three weeks until it flattened or eight treatments, whichever happened first[19-20]. No massage, silicone gel, or pressure garments were employed. The Vancouver Scar Scale and serial photographic records assessed scars at each level. Kaplan Meier curves were used to compare survival of the two medicines, and Wilcoxon and log rank tests were used to analyse VSS scores[19-20]. Intralesional triamcinolone and verapamil injections were compared in another randomized, single-blind, single-group study with 15 patients (30 scars)[21-22]. Injections and cryotherapy were scheduled every three weeks until the scar flattened or 8 treatments, whichever came first. Both groups lost height and pliability after the research. Triamcinolone improved height and pliability more than verapamil. Neither medication changed vascularity or pigmentation as desired[23-24].

Shanthi et al. found that triamcinolone and verapamil injections reduced scar vascularity, pliability, and height[25-26]. Also, triamcinolone injection speeds this lowering. Neither medication changed pigmentation as desired. Verapamil enhanced scar clinical metrics like triamcinolone, making it a good choice for treating hypertrophic scars and keloids[27].

CONCLUSIONS

Scar treatment is challenging and evolving. Intralesional triamcinolone acetonide is better than intralesional verapamil for scar repair. Intralesional verapamil works better with other methods like surgery. In conclusion, triamcinolone's anti-inflammatory, antimitotic, and vasoconstrictor qualities make it a better scar treatment than verapamil. Finally, verapamil is almost as effective as triamcinolone and can be administered alone or in combination to treat bigger scars. Further studies with more individuals and longer observation periods are encouraged to illuminate this topic.

REFERENCES

1. Andrews JP, Marttala J, Macarak E, Rosenbloom J, Uitto J. Keloids: the paradigm of skin fibrosis—pathomechanisms and treatment. Matrix Biol 2016; 51:37–46.
2. Shah VV, Aldahan AS, Mlacker S, Alsaidan M, Samarkandy S, Nouri K. 5-Fluorouracil in the treatment of keloids and hypertrophic scars: a comprehensive review of the literature. Dermatol Ther 2016; 6:169-83.
3. Ogawa R. The most current algorithms for the treatment and prevention of hypertrophic scars and keloids. Plast Reconst Surg 2010; 2:557–68.
4. Lee SS, Yosipovitch G, Chan YH, Goh CL. Pruritus, pain, and small nerve fiber function in keloids: a controlled study. J Am Acad Dermatol. 2004;51(6):1002–06.
5. Garg AM, Shah YM, Garg A. The efficacy of intralesional triamcinolone acetonide (20mg/ml) in the treatment of keloid. Int Surg J. 2018;5(3):868–72.
6. Robles DT, Berg D. Abnormal wound healing: keloids. Clin Dermatol. 2007;25(1):26–32.
7. Shanthi FM, Ernest K, Dhanraj P. Comparison of intralesional verapamil with intralesional triamcinolone in the treatment of hypertrophic scars and keloids. Indian J Dermatol Venereol Leprol. 2008;74(4):343.
8. Ahuja RB, Chatterjee P. Comparative efficacy of intralesional verapamil hydrochloride and triamcinolone acetonide in hypertrophic scars and keloids. Burns. 2014;40(4):583–88.
9. Jalali M, Bayat A. Current use of steroids in management of abnormal raised skin scars. Surgeon. 2007; 5:175–80.
10. Gauglitz GG, Korting HC, Pavicic T, Ruzicka T, Jeschke MG. Hypertrophic scarring and keloids: Pathomechanisms and current and emerging treatment stratigies. Mol Med. 2011; 17:113-5.
11. Boggio RF, Freitas VM, Cassiola FM, Uryabashi M, MachadoSantelli GM. Effect of a calcium channel blocker (verapamil) on the morphology, cytoskeleton and collagenase activity of human skin fibroblasts. Burns 2011; 37:616–25.
12. Yang JY, Huang CY. The effect of combined steroid and calcium channel blocker injection on human hypertrophic scars in animal model: a new strategy for the treatment of hypertrophic scars. Dermatol Surg 2010; 36:1942–9.
13. Uzair M, Butt G, Khurshid K, Suhail Pal S. Comparison of intralesional triamcinolone and intralesional verapamil in the treatment of keloids. Our Dermatol Online. 2015;6(3):280-84.
14. Saki N, Mokhtari R, Nozari F. Comparing the Efficacy of intralesional triamcinolone acetonide with verapamil in treatment of keloids: a randomized controlled trial. Dermatol Pract Concept. 2019 Jan 31;9(1):4-9.
15. Baryza MJ, Baryza GA. The Vancouver Scar Scale: an administration tool and its interrater reliability. J Burn Care Res 1995; 16:535–8.
16. Muir IF. On the nature of keloid and hypertrophic scars. Br J Plast Surg. 1990; 43:61–9.
17. Niessen FB, Spauwen PH, Schalkwijk J, Kon M. On the nature of hypertrophic scars and keloids: a review. Plast Reconstr Surg. 1999; 104:1435–58.
18. Leventhal D, Furr M, Reiter D. Treatment of keloids and hypertrophic scars: a meta-analysis and review of the literature. Arch Facial Plast Surg. 2006; 8:362–8.
19. Sephel GC, Woodward SC. Repair, regeneration, and fibrosis. In: Rubin E, editor. Rubin's Pathology. Lippincott, Williams & Wilkins; Baltimore: 2001. pp. 84–117.
20. Oluwasanmi JO. Keloids in the African. Clin Plast Surg. 1974; 1:179–95.
21. Moustafa MF, Abdel-Fattah MA, Abdel-Fattah DC. Presumptive evidence of the effect of pregnancy estrogens on keloid growth: case report. Plast Reconstr Surg. 1975; 56:450–3.
22. Deitch EA, et al. Hypertrophic burn scars: analysis of variables. J Trauma. 1983; 23:895–8.
23. Lewis WH, Sun KK. Hypertrophic scar: a genetic hypothesis. Burns. 1990; 16:176–8.
24. Murray CJ, Pinnel SR. Keloids and excessive dermal scarring. In: Cohen IK, Diegelmann RF, Lindblad WJ, editors. Woundhealing, Biochemical and Clinical Aspects. Saunders Elsevier; Philadelphia: 1992. pp. 500–9.

25. Bayat A, et al. Keloid disease: clinical relevance of single versus multiple site scars. Br J Plast Surg. 2005; 58:28–37.
26. Marneros AG, et al. Genome scans provide evidence for keloid susceptibility loci on chromosomes 2q23 and 7p11. J Invest Dermatol. 2004; 122:1126–32.
27. Brown JJ, Ollier WE, Thomson W, Bayat A. Positive association of HLA-DRB1*15 with keloid disease in Caucasians. Int J Immunogenet. 2008; 35:303–7.