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Comparative study of intralesional steroid versus intralesional vitamin D3 in patients with alopecia areata

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Abstract

Background and objective: Alopecia areata is a non-scarring type of patchy hair loss accompanied by profound psychological distress. This study aims to assess the effectiveness of intralesional triamcinolone acetonide against intralesional vitamin D3 injection in treating alopecia areata.

Methods: This interventional therapeutic study was conducted on 40 adult patients who were divided into two groups. For a maximum of three sessions, 20 patients in Group I received intralesional injections of vitamin D3 (cholecalciferol aqueous preparation 100 000 IU/2 ml) every three weeks. Twenty patients in Group II underwent three sessions of intralesional injections of triamcinolone acetonide every three weeks. Clinical assessments were conducted both at baseline and following each session.

Results: The largest proportion of the sample (42.5%) were aged 20-29 years, more than two-thirds of the patients (67.5%) were males, about 2.5% of the patients had thyroid disease and 2.5% had atopy. In both groups there was significant increase in McDonald's score, (P <0.001). After receiving three sessions, more than half (55%) of the steroid group had terminal regrowth in all areas, compared with 25% in the Vitamin D3 group. The difference was close to the level of significance (P = 0.063). No significant association was detected between the terminal regrowth on the third session with the following variables: number of patches (P = 0.783), size of the patch (P = 0.152), area (P = 0.319), in both groups

Conclusion: For patchy alopecia areata, both intralesional steroid and intralesional vitamin D3 are easy, safe, and affordable treatment options.

Keywords: Alopecia areata; Intralesional steroids; Intralesional vitamin D3.

Introduction

Alopecia areata (AA) is a chronic, immune-mediated condition characterized by non-scarring hair loss, which can be either acute or chronic. The clinical manifestations of AA vary widely, ranging from patchy hair loss to complete loss of hair on the scalp and body.[1-3]

It is currently believed that the breakdown of the immune privilege of hair follicles, potentially triggered by genetic and external factors, initiates the disease.[1,3] There is a genetic component to alopecia areata (AA), as studies have shown a correlation with positive family history. Genome-wide association studies (GWAS) indicate that AA is a complex polygenic disease, involving multiple genes that encode components of both the adaptive and innate immune systems.[4]

The exposure of anagen hair follicle-associated autoantigens and loss of hair follicle-immune privilege may be increased by increasing IFN-γ secretion in hair follicles, upregulating NK (natural killer) G2D ligands (e.g., MHC class I–related chain A and UL16-binding protein), MHC I and MHC II molecules, and chemokines

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(e.g., IL-15, IL-2, and Chemokine ligand), as well as decreasing local immunosuppressant molecules. Recent research has demonstrated the important role that CD8 + NKG2D + T cells and IFN-γ play in the pathophysiology of AA. Young C3H/HeJ mice may experience AA-like hair loss if they receive an intravenous infusion of IFN-γ. On the other hand, mice lacking IFN-γ were discovered to be resistant to AA. According to reports, CD8 + NKG2D + T cells are both necessary and sufficient for the development of AA. Research on mice models of AA has demonstrated that CD8+NKG2D+T cells stimulated the production of IL-15 in follicular epithelial cells by producing IFN-γ through the JAK1 and JAK2 pathways. After attaching themselves to the surface of CD8+NKG2D+T cells, IL-15 creates a positive feedback loop and increases IFN -γ production through the JAK1 and JAK3 pathways. In both AA patients and mice models, the efficacy of Janus kinase (JAK) inhibitors (JAKi), which block IFN-γ signaling, has been confirmed.[5]

Numerous potential triggers of disease onset and flares have been identified. Epstein-Barr virus (EBV), hepatitis B and C viruses, and swine flu are among the viral diseases linked to the onset and recurrence of AA. Another documented trigger is vaccinations.[6]

Hair loss can manifest in different patterns and different degrees of severity. Alopecia totalis, or entire loss of scalp hair, and alopecia universalis, or complete loss of body hair, are severe forms of AA. Patchy AA, diffuse AA, AA reticularis, AA ophiasis, AA sisaipho, and perinevoid AA are other clinical forms.[4,7]

A biopsy sample is frequently necessary for a conclusive diagnosis of AA incognita, another type that is characterized by acute diffuse shedding of telogen hairs in the areas typically affected by androgenetic alopecia. This condition might be mistaken for telogen effluvium. The prevalence of this subtype is higher in women. Approximately 10% to 20% of individuals may have nail changes, and those with more severe disease are likely to experience these alterations. Changes that may be observed include red lunula, trachyonchia, longitudinal ridging, and regular pitting.[7]

The lifetime incidence of the condition seems to rise at a nearly linear rate, and it can manifest at any age.[2]

The global lifetime prevalence of AA is aro und 2%. AA patients are susceptible to thyroid illness, vitiligo, atopy, depression, anxiety, and other autoimmune disorders.[3,7]

The occurrence and rate of AA seemed to rise during adolescence (i.e., ages 12-17 years) and peak in early adulthood (i.e., ages 18-44 years), with elevated rates seen among females across all age groups.[8]

The response of AA to therapy is uncertain. Certain patients experience spontaneous regrowth without any medical treatment within a year. Therapeutic options vary based on the severity of hair loss and the patient's age.[4] Treating AA can be challenging, yielding inconsistent outcomes. Psychosocial therapy and support play a crucial role in managing this often-disfiguring disease, which can be a significant psychosocial burden.[3]

Steroids are employed in topical, injectable, and systemic forms for treating AA.[6] The most frequent therapy for AA involves administering steroid injections locally in the area of alopecia.[1]

Vitamin D receptors have been identified in hair follicles, and a reduction in their expression has been noted in active patches of alopecia areata. Additionally, intralesional Vitamin D has been investigated as a possible therapy for AA.[6]

Aim of the study

The aim of this randomized comparative study is to evaluate and compare the efficacy and safety of intralesional triamcinolone acetonide versus intralesional cholecalciferol (Vitamin D3) in patients with patchy alopecia areata, based

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on hair regrowth rates, side effects, and patient tolerability.

Methods

This interventional therapeutic study was done in two health facilities Erbil dermatology teaching centre and Erbil teaching hospital in Erbil city Iraq over six-monthperiod. A convenient sample of forty patients were enrolled from patients attending Erbil dermatology teaching centre and Erbil teaching hospital in Erbil city who meet the inclusion criteria and agrees to participate in the study

All 40 participants had localized alopecia areata, who had not received any treatment in the last six months**;** their ages ranged between 10-50 years, both sexes were included. An informed verbal consent was obtained from all participants. Patients were categorized into two groups. Group I involved 20 patients who received intralesional injection of vitamin D3 (cholecalciferol aqueous preparation 100 000 IU/2 ml) by using intradermal needle every three weeks for three sessions. Group II involved 20 patients who received intralesional injection of triamcinolone acetonide 40 mg/mL diluted to 8mg/ml by using intradermal needle every 3 weeks for three sessions.[9] The rationale behind spacing the injections every three weeks is that triamcinolone acetonide is a long-acting corticosteroid and its effect last from 3-4 weeks.[10] The injections were administered by same clinician. Clinical evaluations using Macdonald Hull and Norris scoring system were done at the baseline and at each session: 0—no hair re -growth, Grade 1—vellus hair re-growth, Grade 2—few pigmented terminal hair re-growth, Grade 3—clusters of terminal hair and Grade 4—full terminal hair re-growth.[11]

0	no hair re-growth
Grade 1	vellus hair re-growth,
Grade 2	few pigmented terminal hair re-growth
Grade 3	clusters of terminal hair
Grade 4	full terminal hair re-growth.

Inclusion criteria

Both sexes whom ages were above 10 years old, with patchy alopecia areata who agreed to receive intralesional injection were included, and those who had not received any treatment in the last six months

Exclusion criteria

Patients with other types of alopecia areata other than patchy type, patients who refuse intralesional injection, those on treatment for alopecia areata currently, patients who had treatment in the last six months, those on immunosuppressive medication, patients with any type of malignancy were excluded from the study.

Ethical Consideration

The Ethics Committee of the Kurdistan Higher Council of Medical Specialties has granted its approval. The participants provided informed consent with respect to the objective of our investigation. They have been guaranteed the confidentiality and privacy of the information, which has been verified that it will not be used for any other purpose.

Statistical analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS, version 26). The chi-square test of association was used to compare the proportions of two or more groups. Fisher's exact test was used when the expected frequency (value) was less than 5 of more than 20% of the table's cells. Related-Samples Friedman's Two-Way Analysis of Variance by Ranks was used to compare the McDonald's scores at different periods of the study, and then the post-hoc test (Bonferroni) was used to compare each two time periods. A *P*-value of ≤0.05 was considered statistically significant.

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Results

Intralesional vitamin D was administered to 20 patients (Vitamin D group), and intralesional steroid was administered to another 20 patients (Steroid group). The largest proportion of the sample (42.5%) was aged 20-29 years, but the difference in the age distribution was insignificant between the two groups (*P* = 0.783). More than two-thirds of the patients (67.5%) were males, and the difference was insignificant between the groups (*P* = 0.311). Around two-thirds (60%) of the Vitamin D group had hair loss for 1-4 weeks, compared with 30% of the Steroid group (*P* = 0.061). One patient only (5%) of the Steroid group had a febrile illness, while none of the Vitamin D group had an illness (*P* = 1.000). Regarding symptoms, one patient only of each group had itching (*P* = 1.000). About 2.5% of the patients had thyroid disease and 2.5% had atopy, and the difference was not significant between the groups (*P* = 1.000). One-fifth (20%) of the Vitamin D group had a previous attack, compared with 30% of the steroid group (*P* = 0.465).

No significant differences were detected between the two groups regarding family history (*P* = 0.205), and history of previous treatment (*P* = 0.402). Other details are presented in Table 1.

In the Vitamin D group, there was a significant increase in McDonald's score, starting from a median of 1 and ending with a median of 2.5 (*P* <0.001). In the Steroid group, there was also a significant increase, starting from a median of 1 and ending with a median of 4 (*P* <0.001). More details are presented in Table 2.

At the end of the study, more than half (55%) of the Steroid group had terminal regrowth in all areas, compared with 25% of the Vitamin D group. Around one-third (30%) of patients of the Steroid group had terminal regrowth with patches of alopecia areata, compared with 25% of the Vitamin D group. The difference was close to the level of significance (*P* = 0.063) (Table 3), (Figure 1-3).

Considering the Vitamin D group only; no significant association was detected between the terminal regrowth on the third session with the following variables: number of patches (*P* = 0.783), size of the patch (*P* = 0.152), area (*P* = 0.319), and Vitamin D3 before intervention (*P* = 0.601). More details are presented in Table 4.

Considering the Steroid group only; again, no significant association was detected between the terminal regrowth on the third session with the following variables: number of patches (*P* = 0.850), size of the patch (*P* = 0.816), and area (*P* = 1.000). More details are presented in Table 5.

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Table 1 Basic characteristics

	Intralesional Vitamin D	Intralesional Steroid	Total	*P* value
	No. (%)	No. (%)	No. (%)	
Age				
<20	7 (35.0)	4 (20.0)	11 (27.5)	
20-29	8 (40.0)	9 (45.0)	17 (42.5)	
30-39	3 (15.0)	4 (20.0)	7 (17.5)	
≥ 40	2 (10.0)	3 (15.0)	5 (12.5)	0.783**
Sex				
Male	15 (75.0)	12 (60.0)	27 (67.5)	
Female	5 (25.0)	8 (40.0)	13 (32.5)	0.311*
Duration in Weeks				
1-4	12 (60.0)	6 (30.0)	18 (45.0)	
5-8	2 (10.0)	8 (40.0)	10 (25.0)	
≥ 9	6 (30.0)	6 (30.0)	12 (30.0)	0.061*
Illness				
None	20 (100.0)	19 (95.0)	39 (97.5)	
Febrile Illness	0 (0.0)	1 (5.0)	1 (2.5)	1.000**
Symptoms				
None	19 (95.0)	19 (95.0)	38 (95.0)	
Itching	1 (5.0)	1 (5.0)	2 (5.0)	1.000**
Associated Diseases				
Thyroid	0 (0.0)	1 (5.0)	1 (2.5)	
Atopy	1 (5.0)	0 (0.0)	1 (2.5)	
Others	1 (5.0)	0 (0.0)	1 (2.5)	
None	18 (90.0)	19 (95.0)	37 (92.5)	1.000**
Previous Attack				
Yes	4 (20.0)	6 (30.0)	10 (25.0)	
No	16 (80.0)	14 (70.0)	30 (75.0)	0.465*
Family History				
Alopecia Areata	6 (30.0)	3 (15.0)	9 (22.5)	
Rheumatoid Arthritis	0 (0.0)	2 (10.0)	2 (5.0)	
Diabetes Mellitus	1 (5.0)	4 (20.0)	5 (12.5)	
No Diseases	13 (65.0)	11 (55.0)	24 (60.0)	0.205**
Previous Treatment				
None	18 (90.0)	15 (75.0)	33 (82.5)	
Topical Steroids	0 (0.0)	2 (10.0)	2 (5.0)	
Intralesional Steroid	1 (5.0)	2 (10.0)	3 (7.5)	
Herbal	0 (0.0)	1 (5.0)	1 (2.5)	
Intralesional Steroid + Minoxidil	1 (5.0)	0 (0.0)	1 (2.5)	0.402**
Total	20 (100.0)	20 (100.0)	40 (100.0)	

*Calculated by chi-square test. **Calculated by Fisher's exact test.

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Table 2 McDonald's scores at different times of the study in each of the study groups

Time	Median	Mean rank	*P**	Groups	*P*
Intralesional vitamin D					
Baseline	1	1.40		0 X 1	0.050
First session	2	2.20	< 0.001	0 X 2	< 0.001
Second session	2.5	3.03		0 X 3	< 0.001
Third session	2.5	3.338		1 x 2	0.043
				1 x 3	0.004
				2 X 3	0.391
Intralesional steroid					
Baseline	1	1.05		0 X 1	0.007
First session	2	2.15	< 0.001	0 X 2	< 0.001
Second session	3	3.05		0 X 3	< 0.001
Third session	4	3.75		1 x 2	0.027
				1 x 3	< 0.001
				2 X 3	0.086

*Calculated by Related-Samples Friedman's Two-Way Analysis of Variance by Ranks. **Calculated by the post-hoc test (Bonferroni).

Table 3 Comparing the McDonald's scores between the two groups at the end of the study

	Vitamin D	Intralesional steroid	Total	
	No. (%)	No. (%)	No. (%)	*P***
Vellous hair or no hair	4 (20.0)	0 (0.0)	4 (10.0)	
Sparse pigmented or non-pigmented terminal hair	6 (30.0)	3 (15.0)	9 (22.5)	
Terminal regrowth with patches of alopecia areata	5 (25.0)	6 (30.0)	11 (27.5)	
Terminal regrowth in all areas	5 (25.0)	11 (55.0)	16 (40.0)	0.063
Total	20 (100.0)	20 (100.0)	40 (100.0)	

**Calculated by Fisher's exact test.

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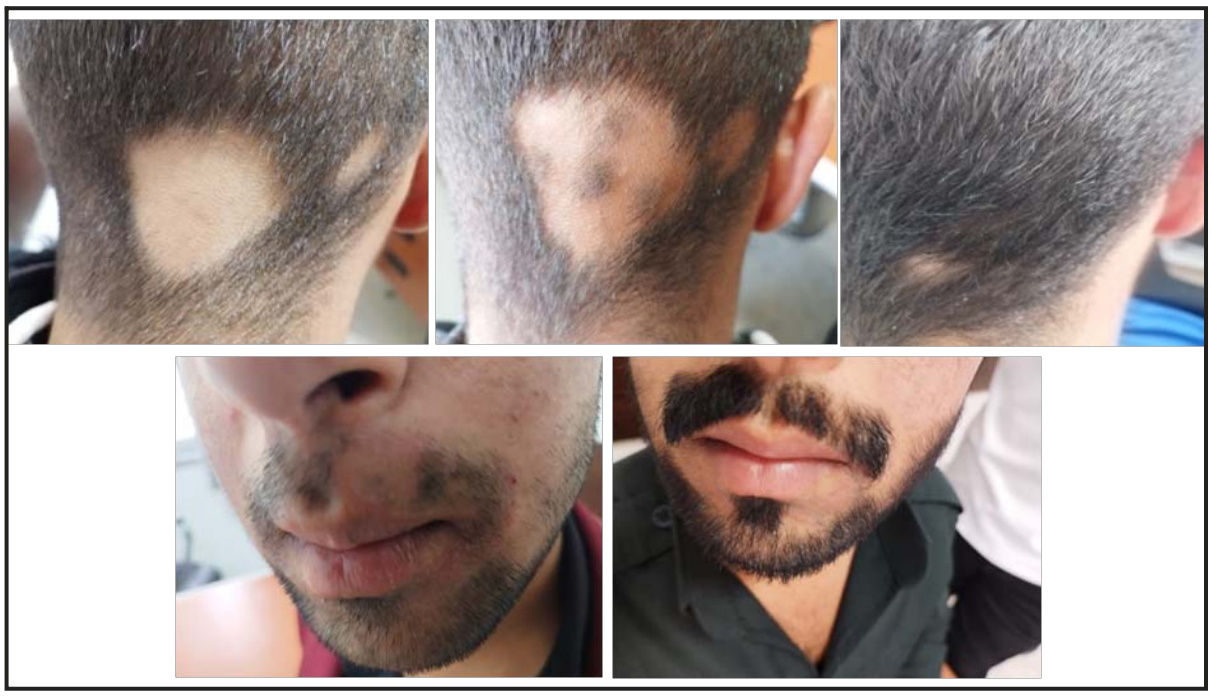

Figure 1 Regrowth in patient after receiving three intralesional injection of vitamin D3

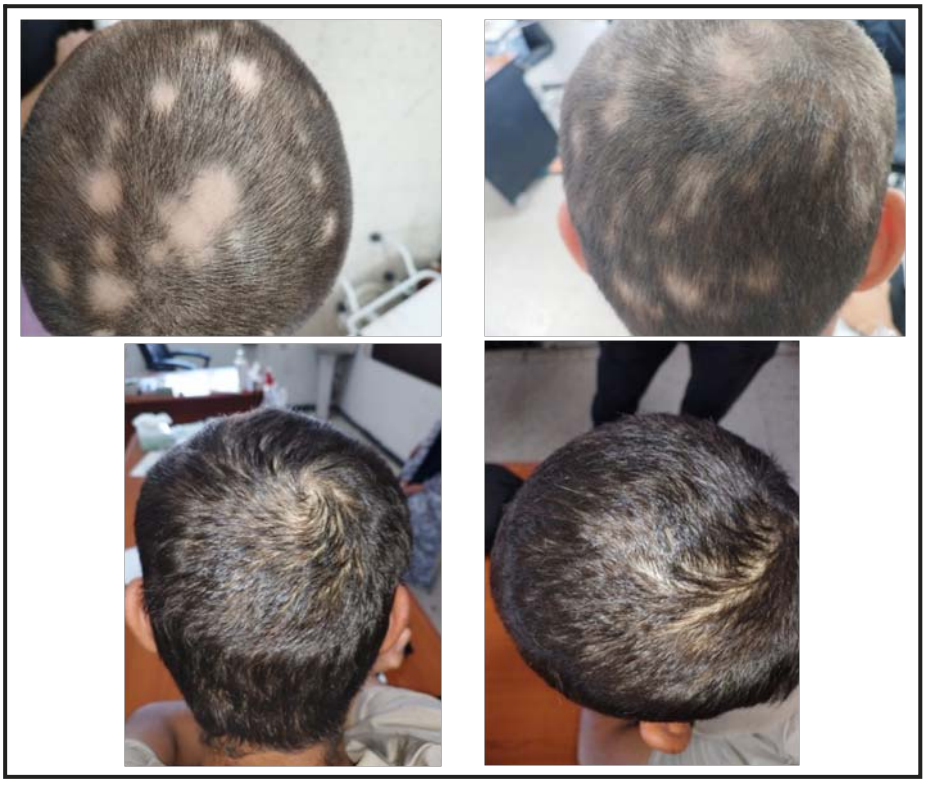

Figure 2 Regrowth in the steroid group

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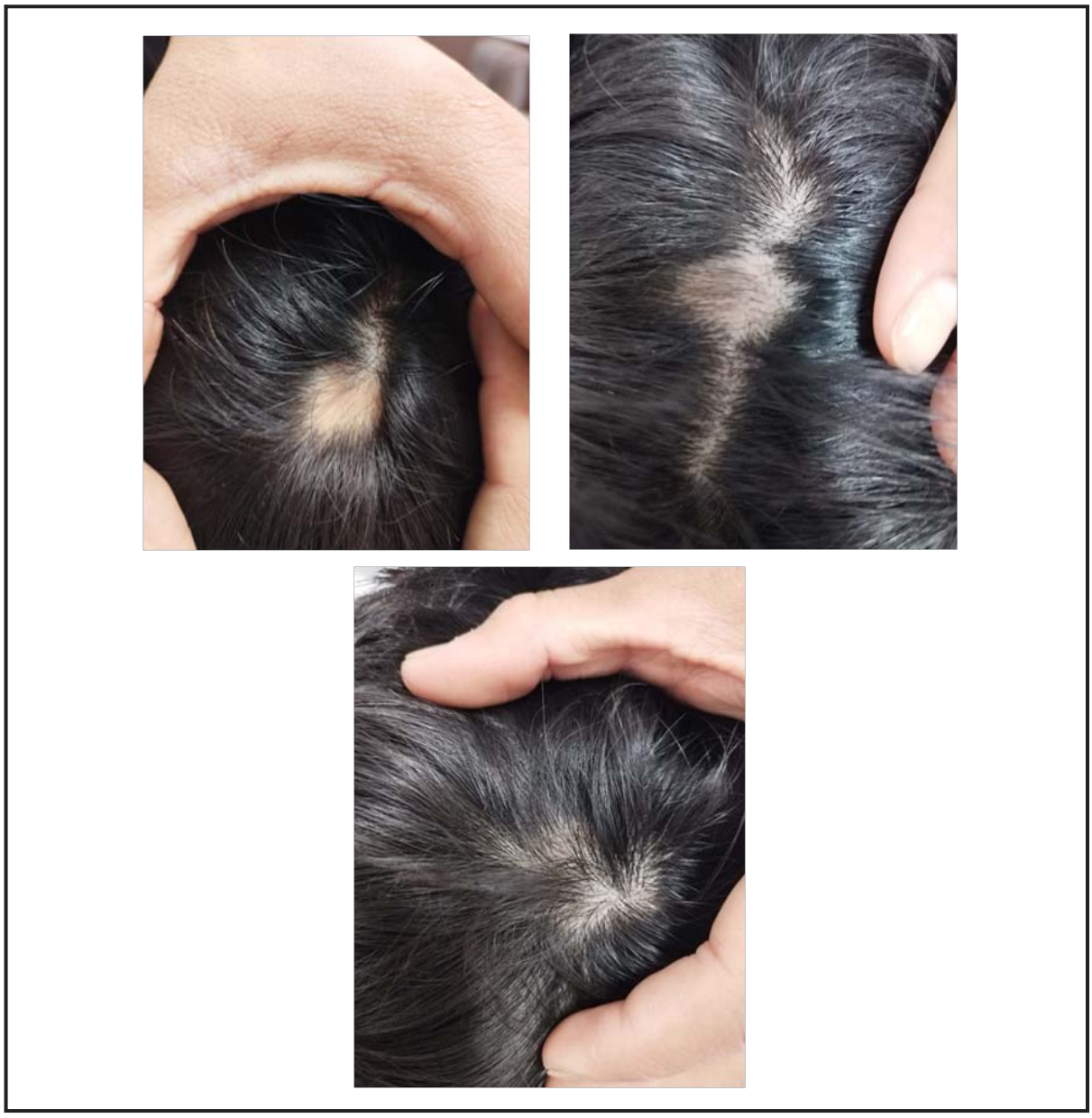

Figure 3 Regrowth in patients received intralesional steroid injection

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Discussion

Alopecia areata is a prevalent non-scarring hair condition that adversely affects the health-related quality of life for both children and adults. AA can impact individuals of both genders and can manifest at any age. Regrettably, it is estimated that nearly 10–15% of individuals with AA may advance to total scalp (AT) or scalp and body (AU) hair loss types.[12] This study aim is to evaluate and compare the efficacy and safety of intralesional triamcinolone acetonide versus intralesional cholecalciferol (Vitamin D3) in patients with patchy alopecia areata. In this study the mean age was 25.75 years, and more than two-thirds of the patients were males and almost one third were females, and one third of the patients had previous attack of alopecia areata, while in another study were a total of 1,641 patients with alopecia areata were included, The mean age was 29.86 ± 14.48 years, and nearly half of the patients were female and the rest were males.[13] In another study the mean age at diagnosis was 35.0 years and was notably lower in men (31.7 years) vs women (37.3 years). Most cases were diagnosed between the ages of 25 and 30 years old.[12]

In this study family history of alopecia areata was found in about one third of the patients while in another study (6.6%) had a family history of AA.[12]

In this study, no significant association was found with other diseases, including atopy and thyroid diseases, while in another study 7.5% had a personal history of atopy, notably almost a third of the patients had a history of skin diseases.[12] Other investigators observed thyroiddisease in 8%–28%, vitiligoin 1.8–16%, and atopy in 1%–52%.[2]

Glucocorticoids have vasoconstrictive, anti-inflammatory, and immunosuppressive properties and are primarily engaged in metabolism. Corticosteroids' anti-inflammatory effects are multifaceted. They lead to granulocyte redistribution at the cellular level, which decreases tissue pools and increases circulating granulocytes. Moreover, they induce lymphopenia. The transcriptional effects of glucocorticoid receptor agonism, which modifies the transcription of many genes in leukocytes, both up and down, are directly or indirectly responsible for the majority of the anti-inflammatory and immunosuppressive effects of glucocorticoids. Blood vessels are the site of corticosteroids' most noticeable pharmacological effects. Because of prostaglandin E and bradykinin, they induce non-competitive antagonism of vasodilation and adrenergically mediated vasoconstriction. Corticosteroids prevent the synthesis of prostaglandins.[14]

Vitamin D receptors (1,25-(OH)2) are known to be expressed in hair follicles, with a decrease in this expression reported in active alopecia areata patches.[5] Vitamin D exerts various biological effects that engage with both the innate and adaptive immune systems, primarily resulting in its downregulation. Vitamin D directly influences T and B cells and alters their activation response.[15] Research indicates that 1,25-dihydroxyvitamin D suppresses T helper (TH) 1 cytokine production and promotes TH2 cytokine release. The immune system's polarization toward the TH2 phenotype inhibits autoimmune diseases driven by TH1. Furthermore, 1,25 -dihydroxyvitamin D suppresses the activity of TH17 cells, which strongly promote autoimmune disorders. Moreover, it boosts regulatory T cells, which are crucial in inhibiting autoimmune reactions.[16] Vitamin D has been demonstrated to suppress antibody release and the formation of autoantibodies in B cells.[15]

Intralesional injection of corticosteroid and vitamin D3 injection are two different modalities used in the treatment of alopecia areata. In this study, after three sessions of therapy, patients who received intralesional steroid achieved McDonald's score 3 and 4 more than those who received vitamin D 3 injection. The result was near significant which could be due to

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the small sample size. Concerning the application of vitamin D3, several studies have noted the effectiveness of topical vitamin D3 analogs for treating alopecia areata. Narang et al. observed a notable response rate of AA treated with calcipotriol, achieving approximately 59.1% effectiveness.[17] Furthermore, another study observed complete hair regrowth with topical calcipotriol in cases of AA that were resistant.[15]

A comparable study found no statistically significant difference in the level of clinical improvement between the two groups under investigation. (P = 0.8). But in the same study, there was a statistically significant difference between the two groups in terms of the reported side effects, with the intralesional vitamin D3 injection resulting in noticeably higher patient satisfaction.[13] While in this study there was no significant difference between intralesional vitamin D3 and intralesional steroid regarding the adverse effects which could be due to dose difference, frequency of administration or clinician technique in administering the injections.

In a randomized controlled clinical trial involving 60 adult patients suffering from localized alopecia areata, researchers compared intralesional injections of vitamin D3 to intralesional normal saline. A statistically significant difference with a P-value <0.001 was noted between the two study groups concerning the level of improvement.[18]

In this study, no significant association was detected between the level of Vitamin D3 before intervention and the terminal hair regrowth on the third session (P = 0.601).

In another study, there was no statistically significant difference between the serum vitamin D3 and the improvement of alopecia (P >0.05).[18]

Limitation of the study:

The small sample size (40 patients), limits the statistical power and makes it harder to detect small but meaningful differences. Also, it limits the generalizability of the results to the wider population, and because of the short duration of follow-up, long-term relapse rates and sustained efficacy cannot be adequately assessed and lack of blinding.

Conclusion

While both therapies showed efficacy, intralesional steroids demonstrated a stronger trend toward terminal regrowth, warranting larger trials to confirm superiority. Vitamin D3 remains a viable steroid-sparing alternative with fewer adverse effects. Intralesional vitamin D3 in the treatment of alopecia areatacan be recommended either as monotherapy or combined with intralesional steroid to enhance the efficacy especially in refractory cases.

Competing interests

The authors declare that they have no competing interests.

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