

ORIGINAL ARTICLE

Prognostic factors that affect the response to topical treatment in patchy alopecia areata

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Abstract

Background Alopecia areata (AA) is an organ-specific, T-cell-mediated autoimmune disease that is characterized by non-scarring hair loss.

Objective We aimed to find the factors that may affect the response to topical therapy in AA.

Methods The study included a total of 60 patients with AA and 30 healthy control patients. The AA patients were randomized into two groups. 40 patients used 0.05% clobetasol propionate cream, and 20 patients used petrolatum (placebo). Both groups applied topical treatments to their lesions twice daily for 12 weeks.

Results The mean extent of AA was $21.88 \pm 16.75\%$ in patients with autoantibodies and $12.16 \pm 13.55\%$ in those who were negative for autoantibodies ($P = 0.021$). Ophiasic pattern and nail involvement were observed more frequently in patients with atopy ($P < 0.05$). Relapse was more frequent in patients with atopy ($P = 0.002$) and nail involvement ($P = 0.02$).

Conclusions We observed that the presence of autoantibodies was associated with more extensive AA, and that ophiasic hair loss pattern and nail dystrophy were significantly associated with atopy. Topical clobetasol propionate treatment produced a modest advantage in hair regrowth as compared with placebo. Notably, atopic AA patients have a higher risk of relapse and redevelopment of AA after completing a course of topical corticosteroid treatment.

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Conflict of interest

None declared.

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Introduction

Alopecia areata (AA) is a common disease that is characterized by non-scarring hair loss. It is an organ-specific, T-cell-mediated autoimmune disease that targets hair follicles.^{1,2} Lifetime risk of developing AA is 1.7% for the general population in the USA.³ AA can be present anywhere on the body; however, the hair loss is usually seen on the scalp.⁴ It is usually characterized by sudden, patchy hair loss in oval-shaped areas. Sometimes, patchy AA can develop into severe forms named alopecia totalis (AT), which affects the whole scalp, and alopecia universalis (AU), which involves complete body hair loss.^{5,6}

The factors affecting onset and progression of AA include genetics,⁷ stress,⁸ hormones,⁹ diet,¹⁰ infectious agents¹¹ and vaccinations.¹² The frequency of a positive family history in AA cases varies from 3 to 42%.^{13,14} Other risk factors for AA include history of atopy and autoimmune diseases.^{14–18} Asthma, atopic dermatitis

and hay fever have been reported in 10–60% of AA patients.^{15,16} AA frequently occurs in association with other autoimmune disorders. Some studies have shown that 12% of patients go on to develop an autoimmune disease.³ Autoimmune thyroid disorders, pernicious anaemia, psoriasis and vitiligo are frequent autoimmune diseases in AA patients.^{16–18}

Materials and methods

A total of 60 patients with alopecic plaques affecting the scalp, eyebrows and/or beard and 30 healthy control patients, all of whom had been referred to the Department of Dermatology of Elazig Education and Research Hospital between October 2010 and December 2011, were enrolled in this study.

Exclusion criteria:

- 1 AT and AU.
- 2 Use of systemic drugs to treat AA.
- 3 Immunosuppression.

- 4 Use of immunosuppressive treatments.
- 5 Pregnancy.

Study plan

The enrolled patients were questioned about the age at onset, family history and presence of atopy and other autoimmune diseases (vitiligo, pernicious anaemia and Hashimoto's thyroiditis) that influence the response to therapy. In the dermatological examination, clinical pattern (patchy, reticular, ophiatic), presence of activity and nail involvement were evaluated.^{19–21} Activity was defined by exclamation point hair. Patients were questioned about their previous treatments and responses to these treatments. Telogen effluvium, androgenic alopecia, trichotillomania and tinea capitis were excluded in differential diagnosis. To exclude tinea infection, native preparations were examined.^{19–21}

The scoring system of the American National Alopecia Areata Foundation was used to calculate extent of disease involvement (%), involvement score and disease duration score. The activity of the disease and the response to the treatment were compared between the groups using these values.²²

Disease percentage (SALT score) was calculated as follows:²²

Percentage hair loss in any one of the four areas of the scalp = % hair loss/% surface area of the scalp in that area. The SALT score is the sum of the scalp hair losses for individual areas.

- a – for the left profile: 95% of 0.18 = 17.1
- b – for the right profile: 90% of 0.18 = 16.2
- c – for the vertex: 95% of 0.40 = 38
- d – for the posterior aspect: 55% of 0.24 = 13.2
- a + b + c + d = 17.1 + 38 + 16.2 + 13.2 = 84.5% hair loss, or a SALT score of 84.5

Percentage disease involvement was calculated as follows:²²

- S0 = no hair loss
- S1 < 25% hair loss
- S2 = 25–49% hair loss
- S3 = 50–74% hair loss
- S4 = 75–99% hair loss
- a = 75–95% hair loss
- b = 96–99% hair loss
- S5 = 100% hair loss

Duration of current episode was scored as follows:²²

- Score 1 = <3 months
- Score 2 = 3–12 months
- Score 3 = 12–24 months
- Score 4 = 2–5 years
- Score 5 = >5 years

Treatment protocols

The patients were randomized into two groups. Forty patients were treated with 0.05% clobetasol propionate cream and 20 patients were treated with pure Vaseline (placebo). The treatments were applied twice daily for 12 weeks.

Evaluation of treatment efficacy

Patients were evaluated in terms of hair regrowth by the same physician at 4-week intervals. The evaluation of hair regrowth after treatment was performed in accordance with the scoring system of the American National Alopecia Areata Foundation:²²

- Score 1: no hair regrowth or worse
- Score 2: vellus-type hair growth
- Score 3: non-pigmented terminal hair growth
- Score 4: presence of pigmented terminal hair
- Score 5: mixed-type hair growth (association of vellus and terminal hair)

In evaluation of percentage recovery in alopecic plaques, existing percentage hair loss was used as the baseline value. For example, for a patient referred with a percentage hair loss of 75% at the beginning of treatment and for whom the size of the alopecic plaque was 50% at the end of treatment, then percentage recovery = $(75-50)/75 = 0.33 = 33\%$.⁸ When calculating percentage recovery, the presence of pigmented terminal hair only was used to determine the baseline value. Vellus-type hair growth was recorded, but not used in the calculation of percentage recovery. After percentage recovery had been calculated, scoring was performed as follows:²²

Percentage recovery score:

- Score 0 = no change or further loss
- Score 1 = <25% regrowth
- Score 2 = 25–49% regrowth
- Score 3 = 50–74% regrowth
- Score 4 = 75–99% regrowth
- Score 5 = 100% regrowth

During the examinations, the presence of alopecic areas was also evaluated and recorded.²⁰ Possible side-effects were taken into consideration and recorded. Side-effects such as folliculitis, telangiectasy, atopy and stria were evaluated for treatment with clobetasol propionate and folliculitis for the placebo group.

Thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4) and vitamin B₁₂ levels, antithyroglobulin (anti-Tg), antithyroid peroxidase (anti-Tpo), antigliadin immunoglobulin A (AGA IgA), antigliadin immunoglobulin G (AGA IgG), antiendomysial (EmA) autoantibody levels and antiparietal cell antibody (PCA) levels were evaluated and recorded. Serum samples were collected prior to the start of the treatment.

Statistical analysis

The Statistical Package for Social Sciences (SPSS) for Windows (version 13.0) was used for the data analysis. Descriptive statistics were expressed as means $\pm$ standard deviations and percentage. The Student's *t*-test was used to compare parametric values and the χ^2 test was used to evaluate non-parametric values. Spearman's tests were employed in the correlation analysis.

Results

A total of 60 patients with AA and 30 healthy controls were enrolled in this study. Of the 60 patients, 40 were randomized to the clobetasol propionate group and 20 to the placebo group; 35 patients were male and 25 were female, and the male-to-female ratio was 1.4 : 1. The ages of the patients ranged from 3 to 50 years and the mean age was 24.26 ± 10.7 years for all patients, 24.15 ± 11.5 years for the clobetasol propionate group and 24.50 ± 9.1 years for the placebo group. The ages of the control group ranged from 7 to 57 years and the mean age was 21.13 ± 9.5 years. Disease duration was 13.50 ± 38.06 months in the clobetasol propionate group and 8.65 ± 7.74 months in the placebo group. No statistically significant difference between the groups in terms of age, gender, disease duration and disease duration score was identified ($P > 0.05$). Table 1 shows changes in patient demographics. There were no statistically significant differences between the groups in pattern of involvement, percentage involvement and involvement score before treatment ($P > 0.05$). Table 1 also shows the pattern of involvement. No statistically significant differences between the groups were found when the patients with AA were evaluated prior to treatment for positivity of activity, presence of atopy, nail involvement, disease duration and clinical pattern, which are important factors in terms of resistance to treatment ($P > 0.05$) (Table 1). None of the patients obtained successful results with previous treatments. Table 2 shows previous treatments in patients and their distribution according to the groups.

The efficacy of treatments was evaluated in terms of hair regrowth scores at weeks 4, 8 and 12, and recovery score and percentage recovery at week 12. Hair regrowth score at week 4 was 2.57 ± 1.66 in the clobetasol propionate group and 1.60 ± 0.94 in the placebo group. There was a statistically significant difference in recovery between the groups ($P = 0.018$). There was no statistically significant difference in hair regrowth score, recovery score and recovery percentage between the clobetasol propionate and

Table 2 The distribution of previous treatments of patients with alopecia areata according to the groups

	Clobetasol propionate group (%)	Placebo group	P
Topical corticosteroid	8 (20.0)	3 (15.0)	>0.05
Topical anthralin	2 (5.0)	1 (5.0)	>0.05
Alternative treatment	3 (7.5)	2 (10.0)	>0.05
No treatment	21 (52.5)	11 (55.0)	>0.05
Multiple treatments (topical corticosteroid, intralesional corticosteroid, topical minoxidil, alternative treatment.)	6 (15.0)	3 (15.0)	>0.05
Total	40 (100)	20 (100)	>0.05

placebo groups at weeks 8 and 12 of treatment ($P > 0.05$) (Table 3).

Associations between percentage recovery at 4, 8 and 12 weeks and serum autoantibody levels, prognostic factors such as the presence of atopy, disease duration and positivity of activity were evaluated. There was a statistically significant difference in recovery between patients with atopy and those without concomitant atopy at weeks 4, 8 and 12 (Table 3). Ophiasic pattern, nail involvement and relapse were observed more frequently in atopic patients compared to non-atopic patients ($P < 0.001$, $P < 0.001$ and $P = 0.002$ respectively). There were no statistically significant differences in disease duration, presence of activity and serum T3, T4, TSH, vitamin B₁₂ and autoantibody levels between patients with atopy and those without atopy ($P > 0.05$). The recovery ratios at 4, 8 and 12 weeks were lower, presence of atopy more common and relapse ratio higher in patients with nail involvement compared with patients without nail involvement. However, there were too few patients with nail involvement to perform a statistical analysis.

Regarding the effect of relapse on patient recovery, there were statistically significant differences between relapse and percentage

Table 1 The distribution of demographic and clinical characteristics of patients with alopecia areata according to the groups

	Clobetasol propionate group	Placebo group	Control group	P
n	40	20	30	
Gender (M/F)	22/18	13/7	14/16	>0.05
Age (year)*	24.15 ± 11.5	24.50 ± 9.1	21.13 ± 9.5	>0.05
Disease duration* (month)	13.50 ± 38.06	8.65 ± 7.74		>0.05
Disease duration score*	1.90 ± 1.15	2.00 ± 0.95		>0.05
The percentage of involvement of lesions at the baseline*	12.82 ± 12.95	19.60 ± 18.31		>0.05
The involvement scores of lesions at the baseline*	1.22 ± 0.53	1.50 ± 0.60		>0.05
The positivity of activity at the baseline	8	5		>0.05
The presence of atopy	9	4	1	>0.05
Nail involvement	5	1		>0.05
Patchy type	25	15		>0.05
Ophiasic type	6	3		>0.05
Mixed type	9	2		>0.05

*(Mean $\pm$ SD).

Table 3 The recovery rates of patients with alopecia areata according to the groups and prognostic factors

	(n)	Hair regrowth score at week 4	Hair regrowth score at week 8	Hair regrowth score at week 12	The percentage recovery at week 12	The score recovery at week 12	P
	Clobetasol propionate group* (40)	2.57 ± 1.66 ^a	3.15 ± 1.61	3.10 ± 1.49	52.37 ± 43.58	3.60 ± 2.12	^a 0.018
	Placebo group* (20)	1.60 ± 0.94 ^a	2.89 ± 1.69	3.20 ± 1.60	39.00 ± 43.36	2.90 ± 2.02	
The presence of activity*	Present (13)	2.53 ± 1.76	3.23 ± 1.64	3.07 ± 1.44	61.92 ± 44.97	4.00 ± 2.16	>0.05
	Absent (47)	2.17 ± 1.46	3.00 ± 1.64	3.14 ± 1.56	44.04 ± 42.90	3.19 ± 2.07	
The presence of atopy*	Present (13)	1.61 ± 1.50	1.53 ± 1.12 ^a	1.69 ± 1.18 ^b	16.92 ± 37.27 ^c	1.84 ± 1.86 ^d	^a <0.001
	Absent (47)	2.42 ± 1.49	3.46 ± 1.50 ^a	3.53 ± 1.36 ^b	56.48 ± 41.59 ^c	3.78 ± 1.97 ^d	^b <0.001 ^c 0.003 ^d 0.002
Nail involvement*	Present (6)	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	
	Absent (54)	2.38 ± 1.54	3.57 ± 1.55	3.37 ± 1.41	53.24 ± 42.76	3.62 ± 2.04	
The duration of the disease*	< 3 months (26)	2.38 ± 1.52	3.26 ± 1.61	3.26 ± 1.45	61.53 ± 43.88	4.00 ± 2.13	>0.05
	3–12 months (21)	2.52 ± 1.66	3.42 ± 1.56	3.61 ± 1.49	47.85 ± 40.23	3.38 ± 1.85	
	12–24 months (5)	1.40 ± 0.54	2.00 ± 1.73	2.00 ± 1.22	10.00 ± 22.36	1.60 ± 1.34	
	2–5 years (7)	1.71 ± 1.49	2.14 ± 1.34	2.28 ± 1.38	31.42 ± 47.40	2.57 ± 2.37	
Pattern of hair loss*	Patchy type (40)	2.35 ± 1.51	3.27 ± 1.60	3.30 ± 1.48	53.37 ± 43.40	3.65 ± 2.10	>0.05
	Ophiasic type (9)	1.77 ± 1.30	2.55 ± 1.74	2.88 ± 1.83	37.77 ± 46.51	2.88 ± 2.20	
	Mixed type (11)	2.27 ± 1.79	2.63 ± 1.62	2.72 ± 1.42	36.36 ± 42.25	2.72 ± 1.95	
Development of side-effects*	Positive (13)	4.07 ± 1.44 ^a	4.38 ± 0.65 ^b	4.00 ± 0.40 ^c	84.23 ± 20.80 ^d	5.07 ± 0.95 ^e	^{a,d} <0.001
	Negative (47)	1.74 ± 1.11 ^a	2.68 ± 1.62 ^b	2.89 ± 1.63 ^c	37.87 ± 43.02 ^d	2.89 ± 2.08 ^e	^{b,e} 0.001 ^c 0.019
Relapse*	Present (32)	1.48 ± 1.00 ^a	2.09 ± 1.54 ^b	2.45 ± 1.76 ^c	13.78 ± 25.46 ^d	1.75 ± 1.32 ^e	^{a,b,c,d,e} <0.001
	Absent (27)	3.18 ± 1.54 ^a	4.22 ± 0.69 ^b	3.96 ± 0.33 ^c	89.62 ± 15.24 ^d	5.33 ± 0.73 ^e	
The presence of autoantibody*	Positive (18)	1.88 ± 1.23	2.88 ± 1.60	3.33 ± 1.57	41.94 ± 42.25	3.16 ± 2.03	>0.05
	Negative (42)	2.40 ± 1.62	3.11 ± 1.65	3.04 ± 1.51	50.47 ± 44.43	3.45 ± 2.14	
Thyroid function abnormalities*	Positive (10)	2.10 ± 1.59	2.80 ± 1.75	3.00 ± 1.76	46.00 ± 45.26	3.30 ± 2.16	>0.05
	Negative (50)	2.28 ± 1.52	3.10 ± 1.61	3.16 ± 1.48	48.30 ± 43.73	3.38 ± 2.10	

*(Mean ± SD). The superscript letters as a–e show the statistically significant *P* values between groups.

ratio at 4, 8 and 12 weeks compared with patients without relapse (Table 3). Atopy and nail involvement were significantly more frequent in patients with relapse compared to patients without relapse ($P = 0.002$ and $P = 0.02$ respectively). There was no statistically significant difference in disease duration, presence of the activity and serum T3, T4, TSH, vitamin B₁₂ and autoantibody levels between patients with relapse and those without relapse ($P > 0.05$).

Abnormalities of thyroid function were detected in six patients in the clobetasol propionate group (15%). Of these, three (7.5%) had T3, four (10%) had T4 and three (7.5%) had TSH abnormalities. In the placebo group, abnormalities of thyroid function were detected in four patients (20%). Of these, three (15%) had T4 and one (5%) had TSH abnormalities. Abnormalities of thyroid function were detected in 10 patients overall (16.6%). In the control group, thyroid function abnormalities were detected in only two patients (6.66%) and in both cases were T4 abnormalities. Mean disease duration was 30.50 ± 74.06 months in patients with thyroid function abnormalities and 8.16 ± 9.49 months in the patients without thyroid function abnormalities. This difference was statistically significant ($P = 0.039$). There was no statistically

significant difference in age, recovery percentage, percentage disease involvement and serum autoantibody levels between the patients with and without thyroid function abnormalities ($P > 0.005$). Serum T3, T4, TSH and vitamin B₁₂ levels in the two groups are shown in Table 4.

Overall, autoantibodies were positive in 18 patients and negative in 42 patients. In the clobetasol propionate group, AGA IgA was detected in one patient, AGA IgG in four patients, PCA in two patients and Anti-Tpo in four patients. Autoantibodies were detected in 10 patients. Of these, one had two autoantibody types and nine had one autoantibody type. In the placebo group, AGA IgG was detected in four patients, PCA in five patients and EmA in one patient. Autoantibodies were detected in eight patients. Of these, two had two autoantibody types and six had one autoantibody type. In the control group, AGA IgA was detected in two patients, AGA IgG in four patients, PCA in one patient and EmA in one patient. Autoantibodies were detected in six patients. Of these, two had two autoantibody types and four had one autoantibody type. Mean autoantibody levels are shown in Table 4.

Regarding the relationship between presence of autoantibodies and mean disease involvement percentage, the mean disease

Table 4 The mean serum autoantibody levels, thyroid functions, vitamin B12 levels between groups

	Clobetasol propionate group n = 40	Placebo group n = 20	Control group n = 20	P
T3	3.72 ± 1.24	3.36 ± 0.58	3.26 ± 0.67	
T4	1.52 ± 0.42 ^a	1.51 ± 0.18 ^b	1.32 ± 0.20 ^{a,b}	^a 0.02, ^b 0.002
TSH	1.66 ± 0.97	2.10 ± 1.21	1.57 ± 0.94	
Vitamin B12	269.70 ± 126.88	212.65 ± 105.01	256.10 ± 100.21	
AGA IgA	5.59 ± 12.56	2.36 ± 2.62	4.08 ± 6.07	
AGG IgG	11.14 ± 20.83	15.26 ± 29.80	4.95 ± 7.30	
Anti-Tg	7.67 ± 13.43 ^{a,b}	20.75 ± 32.21 ^{a,c}	1.47 ± 3.59 ^{b,c}	^a 0.03, ^b 0.01, ^c 0.002
PCA	7.53 ± 25.27	27.45 ± 66.73 ^a	2.41 ± 1.44 ^a	^a 0.04
Anti-Tpo	1.21 ± 3.62	0.16 ± 0.45	0.23 ± 0.36	

T3, triiodothyronine; T4, thyroxine; TSH, Thyroid-stimulating hormone; AGA IgA, antigliadin immunoglobulin A; anti-Tg, antithyroglobulin; anti-Tpo, antithyroid peroxidase. The superscript letters as a–e show the statistically significant *P* values between groups.

involvement percentage was 21.88 ± 16.75 in patients who were positive for autoantibodies and 12.16 ± 13.55 in those who were negative for autoantibodies. The relationship between presence of autoantibodies and mean disease involvement percentage was statistically significant ($P = 0.021$). There were no significant differences between the autoantibody-positive and -negative patients in terms of age, gender, disease pattern, presence of atopy, nail involvement, presence of activity, thyroid function abnormalities, disease duration and relapse ($P > 0.05$). The recovery ratio at 4, 8 and 12 weeks was higher in patients without autoantibodies than in patients with autoantibodies. However, the differences between the groups were not statistically significant ($P > 0.05$).

When the patients were evaluated with respect to side-effects due to the current treatment at 12 weeks, folliculitis had occurred in six patients (15%), telangiectasy in three (7.5%) and mixed side-effects in two (5%) in the clobetasol propionate group, while symptoms of folliculitis had occurred in two patients (10%) in the placebo group. The relationship between recovery ratio and side-effects was analysed by evaluating the efficiency of irritant therapies such as anthralin. The recovery percentages at 4, 8 and 12 weeks were significantly higher in patients with side-effects compared with those with no side-effects (Table 3). Also, interestingly, relapse was significantly more common in patients without side-effects compared with patients with side-effects ($P < 0.001$).

The patients were followed-up for 6 months after completion of treatment. When rates of relapse were investigated according to treatment group, relapse was detected in 20 patients in the clobetasol propionate group (50%) and 13 in the placebo group (65%). At the end of the treatments, there was no statistically significant difference between the groups with regard to relapse ($P > 0.05$).

Discussion

Patchy-type AA with scalp involvement is intrinsically limited in extent, and hair regrowth with or without treatment can occur.²³ Corticosteroids are the most commonly used agents in AA and

can be used in topical, intralesional and systemic forms. In several studies, topical corticosteroid treatment was not found to be superior to the placebo. In one study, Charuwichitrana²⁴ used 0.25% desoximetasone cream in 70 patchy AA patients and reported that desoximetasone was not superior to placebo in terms of full recovery at the end of a 12-week treatment period. Ucak *et al.*²⁵ used 0.05% clobetasol propionate in 30 patients as monotherapy and in 20 patients in comparison with 1% pimecrolimus. Recovery following the 12-week treatment period was acceptable (>50%) in 47% of patients who used 0.05% clobetasol propionate. This figure increased to 61% in the area treated with clobetasol propionate in the comparison group.

In this study, none of the patients had obtained successful results with previous treatments. At week 4, the recovery ratios were significantly better in the clobetasol propionate group; however, there was no difference between the clobetasol propionate and placebo groups at weeks 8 and 12. This finding showed us that in the long term, improvement may occur without therapy.

We also assessed the effects of the prognostic factors serum autoantibodies, thyroid function abnormalities and serum vitamin B₁₂ level on the response to topical treatment in patients with patchy AA. The prognostic factors for AA are young age at onset, presence of atopy, presence of other autoimmune diseases, nail involvement, AT/AU, ophiasis and family history.^{26,27}

Some studies have reported a high frequency of atopy in AA. Goh *et al.*¹⁴ reported that 46% of AA patients had atopy, whereas Tan *et al.*²⁸ reported an atopy prevalence of 60.7% in AA patients. A study from the Netherlands showed a significant positive correlation between atopy and severe AA.²⁹ In our study, atopy was present in 21.6% of patients. We observed resistance to treatment in those patients. Recovery ratios differed significantly between patients with and without atopy. Also, we found significant associations between atopy and other prognostic factors, including nail involvement, ophiasic pattern and relapse. In contrast, there were no associations between atopy, presence of autoantibodies and thyroid function abnormalities.

Some reports have shown that 8–28% of AA patients have thyroid disease.^{26,27} In our study, abnormalities of thyroid function were detected in 16.6% of all patients. This finding was similar to those described in the literature. In the healthy control group, thyroid function abnormalities were detected in only 6.66% of patients. In addition, we found that mean disease duration was longer in patients with thyroid function abnormalities. Thus, thyroid function abnormalities influence the prognosis of AA by affecting the mean disease duration.

Associations between AA and autoimmune diseases have been demonstrated.³⁰ Also, an increased frequency of circulating serum autoantibodies has been reported.^{31,32} Some authors reported positive titres of the antithyroid antibodies anti-Tg (39.5%) and anti-TPO (33.3%) in AA patients. In addition, they reported autoimmune thyroid disease in 47.8% of patients.^{33,34} In this study, we found autoantibody positivity in 30% of patients. However, there were no statistically significant differences in serum autoantibody levels between patients with and without thyroid function abnormalities.

We further found an association between presence of autoantibodies and mean disease involvement percentage. We therefore hypothesized that autoantibodies could be used as a prognostic factor of AA. However, we found no associations between the presence of autoantibodies and the other prognostic factors. The recovery ratios at 4, 8 and 12 weeks were higher in patients without autoantibodies. However, there were no statistically significant differences between the groups.

The pattern of hair loss may be a prognostic factor for AA. Notably, ophiasic hair loss can affect the response to therapy.²⁷ In our study, an ophiasic pattern was present in 15% of patients, and recovery ratios were lower in ophiasic patients compared with patients with patchy AA. This showed that an ophiasic pattern affects the response to therapy. Nail involvement is another prognostic factor for AA.²⁷ We found nail involvement in 10% of the patients. The recovery ratios at 4, 8 and 12 weeks were lower, presence of atopy and relapse ratios higher in the patients with nail involvement compared with those without. We therefore conclude that nail involvement is a prognostic factor that affects the response to therapy. We believe that the high frequency of atopy in AA patients with an ophiasic pattern and nail involvement may contribute to the resistance to treatment.

Also, interestingly, the recovery percentages at 4, 8 and 12 weeks were significantly higher in patients with side-effects compared with those without. Also, relapse was significantly more frequent in patients without side-effects compared with those with side-effects. These findings suggest that irritative therapies are more effective in the treatment of AA.

In summary, we identified the ophiasic hair loss pattern, nail dystrophy and presence of atopy as important prognostic factors for the response to therapy in patchy AA. We further found an association between presence of autoantibodies and mean disease involvement percentage. So, we hypothesized that autoantibodies

could be used as a prognostic factor of AA. We also showed associations between the presence of atopy and nail involvement with relapse after therapy.

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