Original Article

Comparative Evaluation of Oral Baricitinib and Tofacitinib in Alopecia Areata: A Retrospective Cohort Study Based on SALT Scores

Merve Küçükşahin, Erhan Ayhan

Department of Dermatology, Dicle University Faculty of Medicine, Diyarbakır, Türkiye

Abstract

Aim: Alopecia areata (AA) is an autoimmune, non-scarring hair loss disorder in which interferon-γ/interleukin-15 signaling amplifies inflammation via the Janus kinase (JAK)/STAT pathway, providing a mechanistic rationale for oral JAK inhibition. To assess the real-world effectiveness and safety of oral baricitinib and tofacitinib in AA and to examine clinical and lifestyle predictors of treatment response.

Materials and Methods: We retrospectively reviewed 65 patients (age 7-55 years) with AA/AT/AU who received tofacitinib or baricitinib at a tertiary dermatology clinic (Dicle University, Türkiye) between 15 August 2021 and 29 March 2024. Disease severity was assessed by the Severity of Alopecia Tool (SALT). The primary outcome was the change in SALT from pre- to post-treatment. Responses were categorized into no (0-24.9%), partial (25-49.9%), good (50-74.9%), or excellent (75-100%) reduction. Analyses were used paired t-tests, one-way ANOVA, with Bonferroni post-hoc tests, Pearson correlations, and χ^2/Fisher's exact tests (two-tailed, $P \leq 0.05$). Ethics approval was obtained, and procedures conformed to the Declaration of Helsinki.

Results: Cohort characteristics included AU 63.1%, AT 10.8%, AA 23.1%; severe SALT (50-100) in 86.2%. Overall SALT decreased from 91.15±18.76 to 49.08±38.20 ($P = 0.001$). In subgroup analyses, SALT fell from 96.80±7.61 to 43.40±40.63 when using tofacitinib ($P = 0.020$) and from 86.09±23.04 to 45.71±35.55 when using baricitinib ($P < 0.001$). Response distributions were as follows: tofacitinib-50.0% no, 6.0% partial, 6.0% good, 38.0% excellent; baricitinib-23.1% no, 20.5% partial, 10.3% good, 46.1% excellent. Between-drug SALT differences were not significant (ANOVA F = 1.66, $P = 0.198$). Tofacitinib duration correlated with greater improvement (r = 0.415, $P = 0.001$) and was longer in excellent responders compared to non-responders (23.08±17.95 vs. 6.28±5.19 months, $P = 0.005$); no duration-response correlation was observed with baricitinib ($P = 0.671$).

Conclusion: In a severe, predominantly AU cohort, oral JAK inhibitors produced clinically meaningful SALT reductions with acceptable safety. Effectiveness appeared comparable between agents, while longer tofacitinib exposure was associated with greater benefit, whereas baricitinib achieved substantial responses over shorter intervals. Prospective studies should clarify the roles of treatment duration, clinical phenotype, and lifestyle/metabolic factors in optimizing outcomes.

Keywords: Alopecia areata, janus kinase inhibitors, treatment

Introduction

Alopecia areata (AA) is an autoimmune disease of the scalp and body hair that does not leave scars and exhibits polygenic and multifactorial characteristics.[1] The clinical presentation varies depending on the pattern of involvement and prevalence. The most common form presents as well-defined patches on the scalp, while more severe cases may progress to extensive forms such as alopecia totalis and alopecia universalis. Clinical subtypes of AA include patchy, ophiasis, sisaipho, reticular, diffuse, and incognita forms.[2-5]

The etiopathogenesis of AA is not fully understood, but it is thought to be related to genetic predisposition, environmental

Submission: 22-Sep-2025 **Epub:** 03-Nov-2025
Acceptance: 23-Oct-2025 **Web Publication:** 27-Nov-2025

Access this article online

Quick Response Code:

Website: www.turkjdermatol.com

DOI: 10.4274/tjd.galenos.2025.15046

Adress for correspondence: Erhan Ayhan, Assoc. Prof., MD, Department of Dermatology, Dicle University Faculty of Medicine, Diyarbakır, Türkiye
Email: magnetar21@hotmail.com
ORCID ID: 0000-0003-1416-2636

How to cite this article: Küçükşahin M, Ayhan E. Comparative evaluation of oral baricitinib and tofacitinib in alopecia areata: a retrospective cohort study based on SALT scores. Turk J Dermatol. 2025;19(4):227-234.

triggers, and loss of tolerance of the immune system against the hair follicle.[6] The secretion of IFN-γ by CD8+ NKG2D+ cytotoxic T-cells increases interleukin (IL)-15 production and creates a cycle that triggers inflammation via Janus kinase (JAK)/STAT pathways.[7-9] This process facilitates the transition to a dystrophic anagen phase in hair follicles, with peribulbar lymphocyte infiltration.[10]

While the severity and extent of AA are assessed using the SALT score, the patient's age and disease extent are decisive in treatment selection.[10,11] Although there are many topical and systemic treatment options, their efficacy is limited, especially in resistant cases, due to high relapse rates and side effects. Oral JAKis (JAKi), developed in recent years, are promising alternatives for patients requiring systemic treatment.[12]

This study aims to evaluate disease severity in AA patients treated with oral JAKi and to analyze prognostic and sociodemographic factors affecting treatment response.

Materials and Methods

Between August 15, 2021, and March 29, 2024, we retrospectively reviewed 65 patients aged 7-55 years who presented to the outpatient clinic of Dicle University Faculty of Medicine with diagnoses of AA, alopecia totalis, or alopecia universalis, and were treated with oral JAKis. The study protocol was approved unanimously by the Dicle University Faculty of Medicine Ethics Committee (approval number: 300, date: 17.04.2024), and all procedures complied with the principles of the World Medical Association Declaration of Helsinki.

We collected patients' sociodemographic data, clinical characteristics, prognostic factors of AA, previous treatments, SALT scores, the JAKis used, and adverse events. Treatment response to JAKis was assessed by changes in SALT score. Based on the percentage reduction in SALT from pre-treatment to post-treatment, responses were categorized into four groups: no response (0-24.9%), partial response (25-49.9%), good response (50-74.9%), and excellent response (75-100%).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics v21. Continuous variables are presented as mean ± standard deviation, and categorical variables as frequency and percentage (%). Normality of distributions was evaluated. For normally distributed paired measurements (pre- vs. post-treatment), the paired t-test was used. Differences in continuous variables among groups were analyzed with one-way ANOVA followed by Bonferroni post-hoc tests. The Pearson correlation coefficient was used to assess the direction and strength of associations between normally distributed continuous variables. For categorical comparisons, the chi-square (χ^2) test and Fisher's exact test were applied. All hypotheses were two-tailed, and $P \leq 0.05$ was considered statistically significant.

Results

The majority of participants (61.5%) were male, with an average age of 26.12±10.59 years. The age distribution ranged from 7 to 56 years, with a median age of 24. The majority of the sample was in the adult age group (81.5%), and in terms of marital status, the proportion of single individuals was prominent (64.6%). When body mass index levels were examined, more than half of the participants were classified as normal (46.2%) or overweight (33.8%). A high response rate was achieved in the group of non-smokers and non-drinkers using tofacitinib and baricitinib. A high response rate was observed in the group of normal weight individuals using tofacitinib and baricitinib. Tables 1 and 2 show the factors affecting the treatment of tofacitinib and baricitinib.

The demographic characteristics of the patients are shown in Table 3. However, no significant difference was observed in these relationships among all groups.

In this study, the most common comorbidity accompanying AA was thyroid disorders (5 patients). Thyroid disorders were followed by diabetes mellitus, depression, atopic dermatitis, Hepatitis-B carrier status, or chronicity. Cardiac pathologies observed in patients included MVP and patent foramen ovale. In the gastrointestinal system, ulcerative colitis, GER, and gastric ulcer were observed. There was diversity in dermatological conditions, such as oral lichen planus, urticaria, vitiligo, and atopic dermatitis, observed. Anxiety and depression were identified as psychosocial disorders in patients.

When evaluating the clinical characteristics of AA patients, alopecia universalis was observed in 63.1% of cases, AA in 23.1%, and alopecia totalis in 10.8%. Among the patterns of hair loss, patchy hair loss was the most common (84.6%), while ophiasis was detected in 32.3% of cases. Among the areas of involvement, hair loss was present in all patients (100%), followed by eyebrow and eyelash involvement (89.2%), body hair (70.8%), and nail involvement (43.1%). Among nail abnormalities, the most common involvement pattern was trachyonychia (29.2%), while longitudinal ridging and leukonychia were observed at similar rates. In terms of clinical severity, 86.2% of participants showed severe involvement-

Table 1. Sociodemographic comparison of tofacitinib treatment response

Variable	Non-response	Partial	Good	Very good	*P* -value
Age					
Adult	77.8%	50.0%	100%	75.0%	0.700
Child	22.2%	50.0%	0%	25.0%	
Gender					
Female	44.4%	50.0%	50.0%	50.0%	0.991
Male	55.6%	50.0%	50.0%	50.0%	
Education					
Primary	5.6%	50.0%	50.0%	25.0%	
Middle	11.1%	0%	0%	8.3%	0.522
High school	66.7%	50.0%	50.0%	33.3%	
University	16.7%	0%	0%	33.3%	
Marital status					
Single	77.8%	50.0%	50.0%	75.0%	0.725
Married	22.2%	50.0%	50.0%	25.0%	
Smoking					
No	83.3%	50.0%	50.0%	91.7%	0.307
Yes	16.7%	50.0%	50.0%	8.3%	
Alcohol					
No	94.4%	100.0%	100%	100.0%	0.822
Yes	5.6%	0%	0%	0%	
BMI					
Underweight	11.1%	50.0%	0%	8.3&	
Normal	55.6%	50.0%	0%	33.3%	0.469
Overweight	27.8%	0%	100%	50.0%	
Obese	5.6%	0%	0%	8.3%	

BMI: Body mass index

SALT score 50-100. Moderate involvement (SALT score 20-49.9) was observed in 12.3%, and mild involvement (SALT score 0-19.9) in 1.5%.

As shown in Table 2, the most common treatment method in AA patients, is topical corticosteroids, which all participants used. In addition, the most frequently preferred topical agents were anthralin (49.2%), minoxidil (43.1%), and calcineurin inhibitors (29.2%). Intralesional corticosteroid application was used in 64.6% of cases. Among systemic treatments, cyclosporine was prominent (75.4%), while other immunosuppressive agents were used less frequently (e.g., methotrexate 6.2%; azathioprine 1.5%). SADBE was used as immunotherapy in 18.5% of cases, while defensipron was used to a limited extent. Phototherapy preference was concentrated on the PUVA at 40%. The use of supplements was relatively low, with iron and zinc supplementation reported in 10.8% of cases. PRP applications were limited to 4.6% (Table 4). In this study, the JAKis most commonly used in patients diagnosed with AA were found to be tofacitinib (52.3%) and baricitinib (60%). Tofacitinib was generally administered at a dose of 5 mg twice daily (97%), while baricitinib was standardized at 4 mg once daily (100%). The average treatment duration for tofacitinib was 7.55±13.32 months, with a median of 3 months (a range 0-72 months). This duration was shorter for baricitinib, with an average of 3.68±4.43 months, a median of 3 months, and a range of 0-18 months. When treatment efficacy was assessed using the SALT score, the very good response rate (75-100% reduction) was 46.1% in the baricitinib group and 38% in the tofacitinib group. The non-response rate (less than 25% reduction) post-tofacitinib treatment was 50%, while this rate was 23.1% in the baricitinib group. The mean SALT score before JAK inhibitor treatment was 91.15±18.76 in all patients, while a decrease to 49.08±38.20 was observed after treatment (Table 5).

As shown in Table 6, no significant relationship was found between disease duration and treatment response in either JAK inhibitor group. In the tofacitinib group, disease duration ranged from 95 to 103 months depending on response levels ($P = 0.985$), while in the baricitinib group, this value ranged from 76 to 90 months, ($P = 0.988$). These findings indicate that disease duration has a limited effect in determining treatment response. However, a significant difference was observed in terms of treatment duration. A statistically significant relationship was found between treatment duration and response level in patients using tofacitinib ($P = 0.005$).

Table 2. Sociodemographic characteristics and baricitinib treatment response

Characteristic	Non-response	Partial response	Good response	Very good response	*P* -value
Age group					
Adult	100.0%	87.5%	75.0%	77.8%	0.497
Child	0.0%	12.5%	25.0%	22.2%	
Gender					
Female	37.5%	12.5%	50.0%	27.8%	0.530
Male	62.5%	87.5%	50.0%	72.2%	
Education level					
Primary	0.0%	12.5%	50.0%	5.6%	
Middle	12.5%	12.5%	25.0%	0.0%	0.067
High school	37.5%	37.5%	25.0%	72.2%	
University	50.0%	37.5%	0.0%	22.2%	
Marital status					
Single	75.0%	62.5%	75.0%	50.0%	0.593
Married	25.0%	37.5%	25.0%	50.0%	
Smoking history					
No	75.0%	25.0%	75.0%	61.1%	0.167
Yes	25.0%	75.0%	25.0%	28.9%	
Alcohol history					
No	100.0%	100.0%	100.0%	94.4%	0.767
Yes	0.0%	0.0%	0.0%	5.6%	
BMI category					
Underweight	0.0%	12.5%	25.0%	5.6%	
Normal	62.5%	50.0%	50.0%	50.0%	0.436
Overweight	37.5%	12.5%	0.0%	28.9%	
Obese	0.0%	25.0%	25.0%	5.6%	

BMI: Body mass index

Tablo 3. Demographic characteristics of participants (n = 65)

Variable	Statistic/category	n (%)
Gender	Female/male	25 (38.5%)/40 (61.5%)
Age	Mean ± SD/median (min.-max.)	26.12±10.59/24 (7-56)
Age group	Adult/child	53 (81.5%)/12 (18.5%)
Marital status	Single/married	42 (64.6%)/23 (35.4%)
Body mass index	Mean ± SD/median (min.-max.)	26.43±4.64/24.50 (11-37)
Smoking	Yes/no	20 (30.8%)/45 (69.2%)
Alcohol use	Yes/no	2 (3.1%)/63 (96.9%)

SD: Standard deviation, Min.: Minimum, Max.: Maximum

The duration of use was significantly longer in patients who responded very well (23 months), while it was only 6 months on average in those who did not respond. This suggests that tofacitinib treatment is more effective when used in the long-term.

In the baricitinib group, no significant difference was found between treatment duration and response (P = 0.671). Regardless of response levels, the duration of use ranged from approximately 4 to 6 months, while the "very good response" rate remained quite high (46.1%). This indicates baricitinib's potential to achieve maximum effect in a shorter time.

In this study, the relationship between the SALT score and the duration of JAK inhibitor use was evaluated using Pearson correlation analysis. A moderate positive and statistically significant relationship was found between the duration of tofacitinib use and the SALT score (r = 0.415; P = 0.001). This finding indicates that clinical efficacy increases with longer duration of tofacitinib use. No significant relationship was found between duration of baricitinib use and SALT score (r = 0.097; P = 0.443), suggesting that response to baricitinib treatment may be independent of duration.

Table 4. Treatments used in patients with alopecia areata

Treatment type	Treatment modality	n	%
Topical treatments	Topical corticosteroids	65	100.0
	Vitamin D analogs	4	6.2
	Topical retinoids	16	24.6
	Topical calcineurin inhibitors	19	29.2
	Topical minoxidil	28	43.1
	Topical anthralin	32	49.2
	Topical tar preparations	18	27.7
	Topical methoxsalen	6	9.2
	Topical prostaglandin analogs	11	16.9
	Intralesional corticosteroids	42	64.6
Systemic treatments	Methotrexate	4	6.2
	Cyclosporine	49	75.4
	Systemic corticosteroids	19	29.2
	Sulfasalazine	5	7.7
	Azathioprine	1	1.5
Immunotherapy	SADBE	12	18.5
	Diphencyprone	2	3.1
Phototherapy	PUVA therapy	26	40.0
Supplemental therapies	Iron	7	10.8
	Magnesium	2	3.1
	Zinc	7	10.8
	Biotin	3	4.6
	Vitamin D	2	3.1
Other procedures	PRP application	3	4.6

Additionally, a negative correlation was observed between the duration of use of tofacitinib and baricitinib (r = -0.384; P = 0.002), which may reflect differences in treatment preference and patient groups.

The change in SALT score was compared according to the JAK inhibitor used. According to the results of the one-way ANOVA test, no statistically significant difference was found among the groups using tofacitinib, baricitinib, or both drugs together (F = 1.66; P = 0.198). The highest mean SALT score was observed in the tofacitinib group (49.56±39.23), followed by baricitinib (35.37±32.28) and the combination therapy group (28.50±28.96). These findings indicate that, although both treatment options are effective, the differences in mean scores are not statistically significant).

In all patients, the SALT score decreased from 91.15±18.76 to 49.08±38.20 after JAK inhibitor treatment. In tofacitinib users, it decreased from 96.80±7.61 to 43.40±40.63 (P = 0.020), and in baricitinib users, it decreased from 86.09±23.04 to 45.71±35.55 (P < 0.001) (Table 7).

Discussion

JAK proteins regulate numerous physiological and immunological processes by transmitting signals from cell surface receptors to the nucleus.[13] The role of the JAK-STAT pathway in the pathogenesis of AA has been demonstrated through the release of interferon-gamma, which increases

Table 5. JAK inhibitor comparison table (tofacitinib vs. baricitinib)

Parameter	Tofacitinib	Baricitinib
Usage frequency	52.3% (n = 34)	60% (n = 39)
Average treatment duration (months)	7.55±13.32 (median: 3, range: 0-72)	3.68±4.43 (median: 3, range: 0-18)
Dosage	5 mg BID (97%) 2.5 mg BID (3%)	4 mg QD (100%)
Treatment response-SALT score reduction		
No response (< 25%)	50%	23.1%
Partial response (25-50%)	6%	20.5%
Good response (50-75%)	6%	10.3%
Excellent response (75-100%)	38%	46.1%

JAK: Janus kinase, SALT: Severity of Alopecia Tool

Table 6. Treatment response comparison by duration and drug use

Parameter	No response	Partial response	Good response	Excellent response	P -value (tofa)	P -value (bari)
Disease duration (months)	Tofa: 103.33±67.40	Tofa: 96±16.97	Tofa: 96±67.80	Tofa: 95±51.54	0.985	
	Bari: 87±53.57	Bari: 76.5±77.22	Bari: 90.75±63.98	Bari: 83.33±81.43		0.988
Drug use duration (months)	Tofa: 6.28±5.19	Tofa: 6±2.83	Tofa: 10±0.00	Tofa: 23.08±17.95	**0.005**	
	Bari: 4.25±1.48	Bari: 6.25±5.25	Bari: 6±2.44	Bari: 6.56±5.06		0.671

Table 7. Change in SALT score before and after JAK inhibitor use

Patient group	SALT score (before JAKi)	SALT score (after JAKi)	*P* -value
All patients	91.15±18.76	49.08±38.20	0.001
Tofacitinib users	96.80±7.61	43.40±40.63	0.020
Baricitinib users	86.09±23.04	45.71±35.55	< 0.001

JAK: Janus kinase, SALT: Severity of Alopecia Tool

IL-15 production in hair follicles. This mechanism contributes to the maintenance of the disease by triggering the inflammatory process. Therefore, JAKis are potential therapeutic agents that support the regrowth of hair follicles.[7-9]

Systematic reviews and meta-analyses on the efficacy of JAKis in the treatment of AA have generally reported positive results.[14,15] The high success rates of SALT50 with agents such as tofacitinib and ruxolitinib indicate that these treatments are promising in terms of clinical response.[16,17] Baricitinib, in particular, was the first JAK inhibitor to be approved for AA treatment after demonstrating superiority over placebo in phase 3 trials.[1] However, differences in study designs, heterogeneity of patient populations, and limited follow-up periods limit the generalizability of the results.

In our study, treatment with JAKis resulted in a significant reduction in SALT scores. The mean SALT score before treatment was 91.15±18.76, while after treatment this value decreased to 49.08±38.20. Scores decreased from 96.8 to 43.4 in patients using tofacitinib and from 86.09 to 45.71 in those using baricitinib. The decrease in SALT scores after treatment with both agents was statistically significant ($P < 0.05$). These data clearly demonstrate the clinical efficacy of JAKis. However, no significant difference was found between the tofacitinib and baricitinib groups in terms of the change in SALT score ($P = 0.07$). This suggests that both drugs may have similar levels of efficacy.

In the literature, treatment duration and dose are highlighted as key factors affecting response tofacitinib therapy.[18] Meta-analyses in pediatric patients have also reported longer treatment duration in responders.[19] In our study, the mean duration of use in patients who responded well to tofacitinib was 23.08±17.95 months, which was significantly longer than in non-responders ($P = 0.005$). Furthermore, a positive and moderately significant correlation was found between the SALT score and the duration of tofacitinib treatment ($r = 0.415$, $P = 0.001$). In baricitinib treatment, no significant difference was found in the duration of use between response categories ($P = 0.671$), and no relationship was observed between the change in SALT score (SALT) and duration of use ($r = 0.097$, $P = 0.443$). This may be attributed to the use of baricitinib for shorter and more homogeneous periods. Our findings suggest that treatment duration may be a decisive factor in clinical response, particularly for tofacitinib.

In our study, treatment response was higher in individuals who did not smoke, did not consume alcohol, and had no positive family history. Several epidemiological studies have found that smoking increases the risk of AA. Current smokers showed a higher risk of AA incidence compared to non-smokers, with a risk ratio of 1.88. The duration and volume of tobacco use are also associated with AA risk. A history of smoking for more than 10 years constitutes an increased risk for an aortic aneurysm (AA).[20] Smoking activates various cytokines and inflammatory pathways by increasing CD4+ Th1, TH4, and Th17.[21-23] Alcohol consumption can activate immunological mechanisms.[20] Mild ethanol intoxication impairs the ACTH and cortisol secretion response to intravenous CRH administration. This suggests that alcohol consumption may impair AA development.[24] Furthermore, obesity increases the risk of AA (odds ratio: 1.15).[25] However, the detailed mechanism of obesity-related AA remains unclear.[26] In our study, although it was observed that alcohol and tobacco consumption increased the risk of AA and negatively affected the response to treatment, the results were not statistically significant.

Findings in the literature suggesting that JAKis may be more effective in common disease forms such as AU and AT, are supported by our study.[27] This may be because AU or AT patients have more inflammation and that JAKi treatment suppresses excessive inflammation better. In particular, the treatment response was found to be statistically significant in individuals with common diseases using tofacitinib ($P < 0.001$).

A meta-analysis found that the JAK treatment response was more effective in women.[27] When treatment response was evaluated by sex in this study, response rates in the tofacitinib group were equal in women and men, while in the baricitinib group, the very good response rate was higher in men. However, this difference was not clinically significant. Treatment response is higher in adults than in children, but these results should be interpreted with caution due to the small number of children.

No significant relationship was found between disease duration and treatment response. This can be explained by factors such as the active phase of the disease and the individual immune response profile. Similarly, the literature reports that AA duration does not determine JAKi treatment response.[7,28]

Study Limitations

The results of our study were mostly related to adults. The relapse rates in JAKi treatment could not be evaluated. In order to calculate the effective relapse rate, patients needed to have discontinued JAKi treatment for at least 3 months. A longer period was needed to observe serious JAKi side effects in patients. The duration of baricitinib use in our patients was shorter.

Conclusion

JAKis are effective, and safe agents in the treatment of AA, and the duration of treatment may be decisive for the clinical response, especially for tofacitinib. Baricitinib, on the other hand, can provide similar responses in a shorter period. Considering that the response to treatment is shaped by individual, immunological, and lifestyle factors, it is important to clarify these relationships with larger sample sizes and longer-term studies.

Ethics

Ethics Committee Approval: The study protocol was approved unanimously by the Dicle University Faculty of Medicine Ethics Committee (approval number: 300, date: 17.04.2024), and all procedures complied with the principles of the World Medical Association Declaration of Helsinki.

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.K., E.A., Concept: M.K., E.A., Design: M.K., E.A., Data Collection or Processing: M.K., E.A., Analysis or Interpretation: M.K., E.A., Literature Search: M.K., Writing: M.K., E.A.

Conflict of Interest: The authors declared that they have no conflict of interest.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. King B, Ohyama M, Kwon O, Zlotogorski A, Ko J, Mesinkovska NA, Hordinsky M, Dutronc Y, Wu WS, McCollam J, Chiasserini C, Yu G, Stanley S, Holzwarth K, DeLozier AM, Sinclair R; BRAVE-AA investigators. Two phase 3 trials of baricitinib for alopecia areata. N Engl J Med. 2022;386(18):1687-1699.
2. Alkhalifah A, Alsantali A, Wang E, McElwee KJ, Shapiro J. Alopecia areata update: part I. Clinical picture, histopathology, and pathogenesis. J Am Acad Dermatol. 2010;62(2):177-188, quiz 189-190.
3. Madani S, Shapiro J. Alopecia areata update. J Am Acad Dermatol. 2000;42(4):549-566; quiz 567-570.
4. Sato-Kawamura M, Aiba S, Tagami H. Acute diffuse and total alopecia of the female scalp. A new subtype of diffuse alopecia areata that has a favorable prognosis. Dermatology. 2002;205(4):367-373.
5. Ruiz-Arriaga LF, López-García L, Vega-Memije ME. Perinevoid alopecia: a case report. Skin Appendage Disord. 2019;5:94-96.
6. Fukuyama M, Ito T, Ohyama M. Alopecia areata: current understanding of the pathophysiology and update on therapeutic approaches, featuring the Japanese Dermatological Association guidelines. J Dermatol. 2022;49(1):19-36.
7. Phan K, Sebaratnam DF. JAK inhibitors for alopecia areata: a systematic review and meta-analysis. J Eur Acad Dermatol Venereol. 2019;33(5):850-856.
8. Gilhar A, Paus R, Kalish RS. Lymphocytes, neuropeptides, and genes involved in alopecia areata. J Clin Invest. 2007;117(8):2019-2027.
9. Xing L, Dai Z, Jabbari A, Cerise JE, Higgins CA, Gong W, de Jong A, Harel S, DeStefano GM, Rothman L, Singh P, Petukhova L, Mackay-Wiggan J, Christiano AM, Clynes R. Alopecia areata is driven by cytotoxic T lymphocytes and is reversed by JAK inhibition. Nat Med. 2014;20(9):1043-1049.
10. Strazzulla LC, Wang EHC, Avila L, Lo Sicco K, Brinster N, Christiano AM, Shapiro J. Alopecia areata: disease characteristics, clinical evaluation, and new perspectives on pathogenesis. J Am Acad Dermatol. 2018;78(1):1-12.
11. Olsen EA, Hordinsky MK, Price VH, Roberts JL, Shapiro J, Canfield D, Duvic M, King LE Jr, McMichael AJ, Randall VA, Turner ML, Sperling L, Whiting DA, Norris D; National alopecia areata foundation. Alopecia areata investigational assessment guidelines--Part II. National alopecia areata foundation. J Am Acad Dermatol. 2004;51:440-447.
12. Waśkiel-Burnat A, Kołodziejak M, Sikora M, Stochmal A, Rakowska A, Olszewska M, Rudnicka L. Therapeutic management in paediatric alopecia areata: a systematic review. J Eur Acad Dermatol Venereol. 2021;35:1299-1308.
13. Tanaka Y, Luo Y, O'Shea JJ, Nakayamada S. Janus kinase-targeting therapies in rheumatology: a mechanisms-based approach. Nat Rev Rheumatol. 2022;18(3):133-145.
14. Yan D, Fan H, Chen M, Xia L, Wang S, Dong W, Wang Q, Niu S, Rao H, Chen L, Nie X, Fang Y. The efficacy and safety of JAK inhibitors for alopecia areata: a systematic review and meta-analysis of prospective studies. Front Pharmacol. 2022;13:950450.
15. Liu LY, Craiglow BG, Dai F, King BA. Tofacitinib for the treatment of severe alopecia areata and variants: a study of 90 patients. J Am Acad Dermatol. 2017;76(1):22-28.
16. Yu DA, Kim YE, Kwon O, Park H. Treatment outcome of oral tofacitinib and ruxolitinib in patients with alopecia areata: a systematic review and meta-analysis. Indian J Dermatol Venereol Leprol. 2021;87(5):621-627.
17. Liu M, Gao Y, Yuan Y, Yang K, Shen C, Wang J, Tian J. Janus kinase inhibitors for alopecia areata: a systematic review and meta-analysis. JAMA Netw Open. 2023;6(6):e2320351.
18. Guo L, Feng S, Sun B, Jiang X, Liu Y. Benefit and risk profile of tofacitinib for the treatment of alopecia areata: a systemic review and meta-analysis. J Eur Acad Dermatol Venereol. 2020;34(1):192-201.
19. Chen Y, Zhu H, Shen Y, Zhu Y, Sun J, Dai Y, Song X. Efficacy and safety of JAK inhibitors in the treatment of alopecia areata in children: a systematic review and meta-analysis. J Dermatolog Treat. 2022;33(8):3143-3149.

20. Dai YX, Yeh FY, Shen YJ, Tai YH, Chou YJ, Chang YT, Chen TJ, Li CP, Wu CY. Cigarette smoking, alcohol consumption, and risk of alopecia areata: a population-based cohort study in Taiwan. Am J Clin Dermatol. 2020;21(6):901-911.
21. Shan M, Yuan X, Song LZ, Roberts L, Zarinkamar N, Seryshev A, Zhang Y, Hilsenbeck S, Chang SH, Dong C, Corry DB, Kheradmand F. Cigarette smoke induction of osteopontin (SPP1) mediates T(H)17 inflammation in human and experimental emphysema. Sci Transl Med. 2012;4(117):117ra9.
22. Jiang R, Jiang Y, Xia P, Luo G, Huang W, Hu Z, Cheng G, Xiong Y, Wang Y, Cui T. Cigarette smoke extract promotes TIM4 expression in murine dendritic cells leading to Th2 polarization through ERK-dependent pathways. Int Arch Allergy Immunol. 2019;178(3):219-228.
23. Minokawa Y, Sawada Y, Nakamura M. Lifestyle factors ınvolved in the pathogenesis of alopecia areata. Int J Mol Sci. 2022;23(3):1038.
24. Waltman C, Blevins LS Jr, Boyd G, Wand GS. The effects of mild ethanol intoxication on the hypothalamic-pituitary-adrenal axis in nonalcoholic men. J Clin Endocrinol Metab. 1993;77(2):518-522.
25. Nakamizo S, Honda T, Adachi A, Nagatake T, Kunisawa J, Kitoh A, Otsuka A, Dainichi T, Nomura T, Ginhoux F, Ikuta K, Egawa G, Kabashima K. High fat diet exacerbates murine psoriatic dermatitis by increasing the number of IL-17-producing γδ T cells. Sci Rep. 2017;7(1):14076.
26. Hagino T, Okazaki S, Serizawa N, Suzuki K, Kaga M, Otsuka Y, Mikami E, Hoashi T, Saeki H, Matsuda H, Mitsui H, Kanda N. Dietary habits in Japanese patients with alopecia areata. Clin Cosmet Investig Dermatol. 2021;14:1579-1591.
27. Mao MQ, Ding YX, Jing J, Tang ZW, Miao YJ, Yang XS, Chen YH, Chen SZ, Wu XJ, Lu ZF. The evaluation of JAK inhibitors on effect and safety in alopecia areata: a systematic review and meta-analysis of 2018 patients. Front Immunol. 2023;14:1195858.
28. Hogan S, Wang S, Ibrahim O, Piliang M, Bergfeld W. Long-term treatment with tofacitinib in severe alopecıa areata: an update. J Clin Aesthet Dermatol. 2019;12(6):12-14.