Clinical, Cosmetic and Investigational Dermatology

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ORIGINAL RESEARCH

Case Series and Literature Review on Tofacitinib for Treating Severe Alopecia Areata

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Background: Alopecia areata (AA) is an autoimmune-mediated, non-scarring hair loss disorder. With the advancement of alopecia treatment research, the efficacy of Janus kinase (JAK) inhibitors in the clinical management of AA has been increasingly evaluated.
Objective: This study reports the efficacy and safety of tofacitinib in the treatment of severe AA and presents a literature review on tofacitinib in AA.
Methods: Collect patient information on severe AA treated at a tertiary hospital from April 2023 to May 2024, and analyze their clinical treatment outcomes.
Results: All five collected patients met the criteria for refractory alopecia areata. Three of these patients achieved favorable therapeutic outcomes with mild adverse reactions, consistent with current research findings.
Conclusion: This study supports the efficacy and safety of tofacitinib in AA treatment, providing further clinical evidence for its application.
Keywords: alopecia areata, JAK-STAT pathway, tofacitinib, JAK inhibitor

Introduction

Alopecia areata (AA) is an autoimmune hair loss disorder classified as one of the three major non-scarring forms of hair loss. Clinically, AA is characterized by the sudden onset of well-defined, round or ovoid patches of alopecia while the surrounding skin remains normal. The condition can occur at any age, but is more common in young and middle-aged individuals, with no significant gender differences observed. According to the Severity of Alopecia Tool (SALT), the condition can be classified into five grades: none (0%), mild (1%-20%), moderate (21%-49%), severe (50%-94%), and very severe (95%-100%).[1]

The pathogenesis of AA is complex and is generally considered to involve an interplay of genetic, immune and environmental factors, with dysfunctional immunity as the central driver. Hair follicle, functioning as immune-privileged (IP) unit, maintains homeostasis regulated by major histocompatibility complex (MHC) class I and II molecules.[2] Nonspecific stimuli (eg, infection, stress) trigger substantial release of interferon-γ (IFN-γ) around hair follicles, activating the Janus kinase 1/2 (JAK1/JAK2) signaling pathway. This leads to overexpression of MHC I/II molecules and interleukin-15 (IL-15) in the follicular epithelium, recruiting CD8+ T cells. These cells activate intracellular JAK1/JAK3 signaling via IL-15 binding, upregulating the NKG2D receptor and amplifying IFN-γ release, thereby creating a vicious cycle of inflammatory factor accumulation that disrupts follicular immune homeostasis.[1,3] In addition, factors such as genetic susceptibility,[4–6] psychoemotional stress,[7] endocrine dysfunction, oxidative stress,[8] micronutrient deficiencies,[9] and poor lifestyle habits[10] have also been correlated with the development of AA.

Received: 29 December 2024
Accepted: 7 May 2025
Published: 15 November 2025

The goal of AA treatment is to control disease progression and promote hair regrowth, thereby improving the quality of life for affected individuals. For patients with mild to moderate forms of the disease, common treatments include the use of topical hormones and minoxidil, while extensive hair loss may require oral corticosteroids or contact immunotherapy. For refractory cases, immunosuppressive therapies should cautiously employ because of adverse reactions. Recent advancements in the therapeutic landscape for AA have highlighted the efficacy and safety of Janus kinase inhibitors. Baricitinib, ritlecitinib, and deuruxolitinib have all received FDA approval for the treatment of severe AA, offering new therapeutic options for patients.[11,12] In addition, tofacitinib, the first JAK inhibitor approved for rheumatoid arthritis and other autoimmune diseases, has shown promising efficacy in the clinical management of AA and is preferred by patients due to its cost-effectiveness. In this study, we reviewed five patients with severe and very severe AA (SALT >50%) treated with tofacitinib. We also conducted a review of the literature on the use of tofacitinib in the treatment of AA.

Method

Patients Selection

Data were collected from patients who attended the Second Affiliated Hospital of Anhui Medical University from April 2023 to May 2024. Inclusion criteria required: 1) diagnosis of severe AA; 2) documented therapeutic failure to ≥3 guideline-recommended systemic agents; and 3) adherence to tofacitinib with regular follow-up for 6 months. Prior to treatment, each patient was screened to exclude patients with contraindications, including routine blood test, liver function, renal function, micronutrients, immunoglobulin E, antinuclear antibody titer, vitamin D assay, and infection-related indications (including T-cell spot test for tuberculosis, Hepatitis B surface antigen, Hepatitis C virus antibody, syphilis antibody test, human immunodeficiency virus antibody test, and chest computed tomography scan). All patients received tofacitinib at a dose of 5 mg twice daily. Patients who continued to receive tofacitinib had regular follow-up laboratory tests (including blood counts, lipids, liver and kidney function).

Evaluation of Efficacy

Patients were assessed with SALT at each clinical visit. Treatment response was assessed by calculating the percentage change in SALT score from baseline to six months of treatment. Treatment response was defined as: ineffective (≤5% change in SALT score), improvement (>5%), significant response (>50%), and complete regrowth (>95%).

Literature Review

A comprehensive literature search of Pubmed was conducted in May 2024. Search terms included “alopecia circumscripta”, “alopecia areata”, “alopecia universalis”, “tofacitinib”, “tasocitinib”, “tofacitinib citrate” and “Xeljanz”. Inclusion criteria were English-language studies in which: (1) patients had a diagnosis of severe or greater baldness and were treated with oral tofacitinib; and (2) the degree of hair regrowth was assessed using the Hair Loss Severity Instrument; and (3) data were available to assess treatment efficacy. Exclusion criteria were: (1) comorbidities with other diseases being treated; and (2) combinations of medications with clear efficacy in the treatment of AA; and (3) single case reports and letter articles.

Results

Patient Characteristics

Based on the predefined inclusion criteria, five patients who met the eligibility criteria and accepted longitudinal follow-up were subsequently enrolled in the study. Detailed descriptions of the five cases are shown in Table 1. All enrolled patients exhibited a disease duration of ≥8 years and had undergone multiple therapeutic interventions prior to initiating tofacitinib therapy. All patients had no family history.

Table 1 Characteristics of Patients with Alopecia Areata Treated with Oral Tofacitinib

Patient No.	Sex/Age, Years	Duration of Disease, Years	Lifestyle	Areas of Hair Loss	Comorbidity	Prior Treatment	SALT Score Before and After Treatment	Percentage of SALT Improvement	Adverse Reaction	Recurrence
1	F/24	9	Regular	Scalp	None	Minoxidil, TCS, OCS, ILC, TCM therapy	85 to 5	94.12	None	Yes at the ninth month of treatment
2	F/23	8	Poor sleep, academic pressure	Scalp	None	TCM therapy, minoxidil	60 to 5	100	None	Yes after 2 months
3	F/27	9	Regular	Scalp, eyebrow	None	TCM therapy, OCS, ILC	100 to 100	0	Scalp folliculitis	–
4	M/46	16	Poor sleep, smoking, alcohol	Scalp, body fur	None	Minoxidil, TCS, ILC	90 to 45	50	None	Yes after 5 months
5	M/15	8	Regular	Scalp, eyebrow, eyelash, body fur	Atopic dermatitis	Minoxidil, TCS, OCS	100 to 100	0	Folliculitis Of the scalp and face	–

Abbreviations: SALT, Severity of Alopecia Tool; TCS, topical corticosteroids; OCS, oral corticosteroids; ILC, intralesional corticosteroids; TCM therapy, traditional Chinese medicine therapy; F, female; M, male.

https://doi.org/10.2147/CCID.S512037

Treatment Outcome Evaluation

Patient 1, patient 2, patient 4 achieved significant response after treatment. Patient 3 and patient 5 had no hair regrowth at the end of treatment. For all patients, the median change in SALT score was 50% (mean 50%, range 0–100%). Representative images of patients who experienced significant response and complete regrowth in response to treatment (Patient 1, Patient 2, Patient 4) are shown in Figure 1. Five patients experienced mild adverse events limited to scalp or facial folliculitis (patients 3 and 5), and no opportunistic infections, thrombosis, tuberculosis, tumors, or other adverse events were observed.

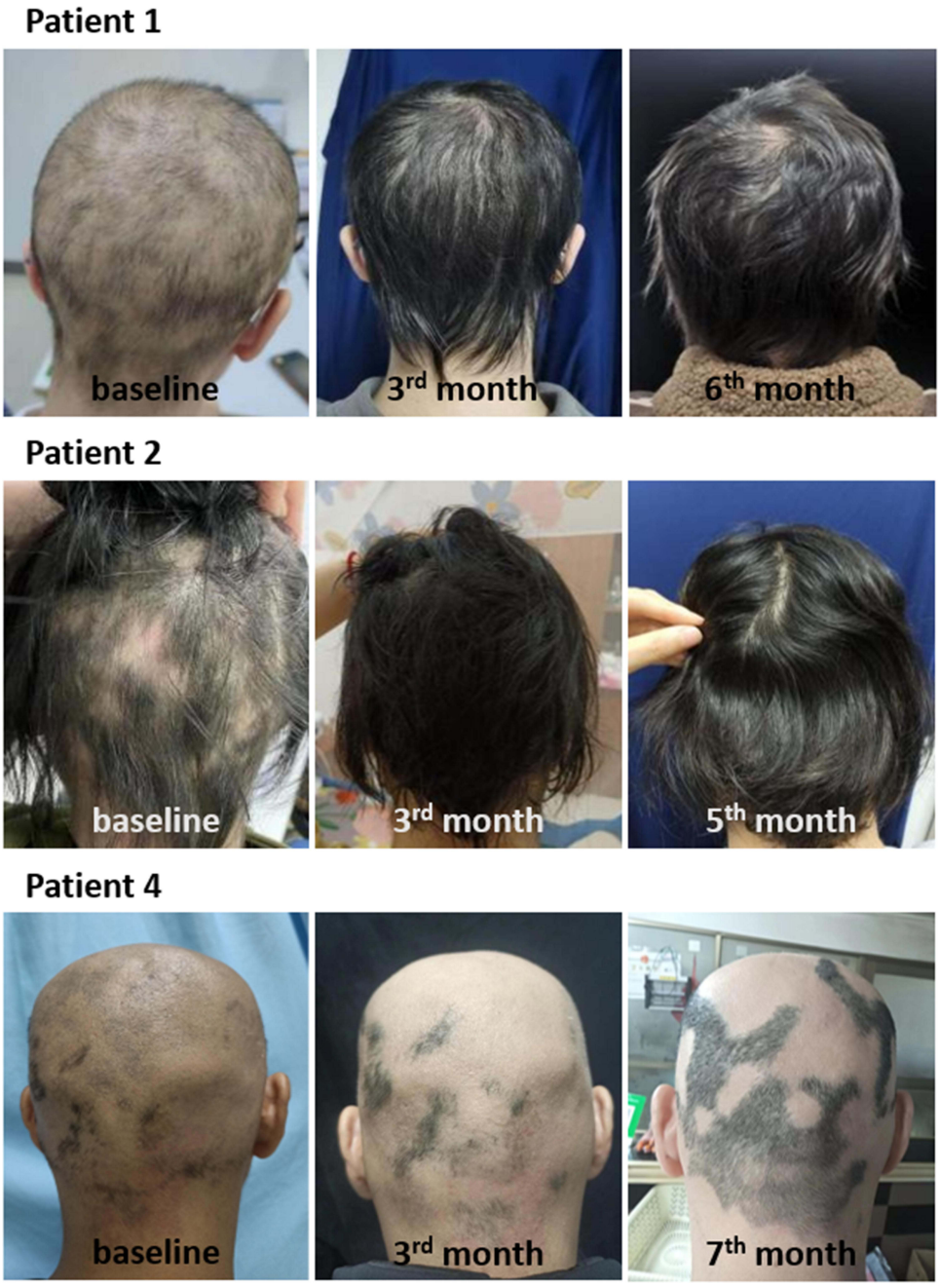

Figure 1 Tofacitinib treatment response in three patients. Patient 1 shows scalp hair at baseline, month 3, and month 6 visits. Patient 2 shows scalp hair at baseline, month 3, and month 5 visits in patient 2. Patient 4 shows scalp hair at baseline, month 3, and month 7 visits in patient 4.

Literature Review

A comprehensive search of PubMed identified 25 articles. After applying specific inclusion and exclusion criteria, 10 articles were selected for analysis. These articles included 9 case series and 1 prospective study. Patient characteristics, treatment details, and response data are summarized in Table 2. The total number of participants was 188, consisting of 83 males and 105 females, with an age range of 3 to 59 years. The literature review revealed that 120 patients experienced a greater than 50% improvement in SALT score, while 18 patients did not respond to treatment. Reported adverse events were generally mild and predictable and included primarily upper respiratory tract infections, elevated liver enzymes, acne-like lesions, and dyslipidemia.

Discussion

Alopecia areata is a chronic inflammatory disorder characterized by hair loss that significantly affects patients' quality of life and psychological well-being. While several treatment options are available for clinical AA, the efficacy and safety of conventional therapies—including corticosteroids, vasodilators, sensitizers and immunosuppressants—pose challenges for patients with extensive hair loss. Recently, the JAK-STAT pathway has received considerable attention in the context of dermatological diseases.[23–25] In particular, the presence of JAK1/JAK2 and JAK1/JAK3 in hair follicle epithelial cells and CD8+ NKG2D+ T cells, respectively, plays a critical role in the release of inflammatory factors. Inhibition of JAK in these cells may inhibit the release of downstream inflammatory mediators, reduce the accumulation of immune factors around hair follicles, and thereby control disease progression and facilitate the treatment of AA. Currently, both baricitinib and deuruxolitinib, which are approved for the treatment of AA, act as JAK1/JAK2 inhibitors. In addition, ritlecitinib, a JAK3 inhibitor, has also been approved for the treatment of AA based on its demonstrated efficacy in affected patients. All three selective JAK inhibitors have shown satisfactory results in clinical trials for AA.

Tofacitinib, a pan-JAK inhibitor that primarily targets JAK1 and JAK3 with weak inhibition of JAK2, has attracted considerable interest due to its remarkable efficacy in the treatment of AA and plaque psoriasis.[26,27] In an initial investigation into the treatment of AA, Kennedy et al reported that approximately 63% (42/66) of patients with severe AA experienced varying degrees of remission, as indicated by improvements in SALT scores greater than 5%, after three months of treatment with tofacitinib at a dosage of 5 mg twice daily. Notably, 50% (21/42) of these patients achieved a greater than 50% improvement in SALT scores.[28] Subsequently, Liu et al evaluated the efficacy of tofacitinib in a cohort of 90 patients with severe AA and found that up to 77% (50/65) experienced a 6% to 100% improvement in alopecia after 4 to 18 months of treatment; however, results were less favorable for patients with a disease duration greater than 10 years. Furthermore, this study showed that 52.2% (47/90) of patients had coexisting autoimmune disorders, suggesting a complex interplay between AA and autoimmunity.[29] Several case studies have further investigated and supported the efficacy of tofacitinib in the treatment of AA.[13–22] Tofacitinib was found to be more effective in the treatment of AA compared to alopecia totalis (AT) and alopecia universalis (AU). Patients who have been treated with tofacitinib at a dosage of 5 mg twice daily for 4 to 6 months without achieving satisfactory results may be considered for an increase in dosage. Meta-analyses and retrospective studies on the use of tofacitinib in adult alopecia suggest that the optimal time to evaluate treatment response is three months. In addition, the overall remission rate for tofacitinib at doses greater than 5 mg BID with continuous treatment longer than 6 months is higher than that for doses of 5 mg BID or less and treatment duration of 6 months or less. However, it should be noted that increasing the dosage may lead to a higher incidence of adverse events.[30] Notably, tofacitinib has regenerative effects not only on scalp hair but also on eyelashes and eyebrows.[31] Continuous treatment is necessary to maintain the therapeutic response, as discontinuation of the drug may result in recurrence of AA within three months. Some relapses may occur during the treatment period, possibly related to the long-term presence of resident memory cells in peripheral tissues.[32,33] In pediatric cases of AA, tofacitinib has also shown a favorable remission rate.[6]

In our study, all participants were rigorously diagnosed with refractory alopecia areata, defined by: (1) SALT score ≥50% indicating extensive hair loss; (2) disease duration exceeding 8 years with ≥3 documented recurrences; and (3) therapeutic resistance to ≥3 conventional systemic immunomodulatory regimens (including corticosteroid therapy). Among the five patients, three treatment responders exhibited the patchy subtype of alopecia areata, while the remaining two manifested severe

Table 2 Summary of Previous Studies on the Efficacy of Tofacitinib in Treating AA Patients

Study Number	Author	Year	Total Patients	Age, Years (Range)	Sex (M:F)	Comorbidity	Treatment Duration, Months (Range) [Daily Dosage]	Proportion of Improvement in SALT Scores After Treatment	Adverse Reaction	Recurrence
1	Jabbari, A[13]	2018	12	18-52	4:8	NA	6-18 [10–20 mg]	Significant remission (66.7%), improvement (25.0%), ineffctive (8.3%)	Respiratory infections (91.6%), urinary tract infections (33.3%)	6 patients relapsed 2 months after stopping the drug
2*	Cheng, MW[14]	2018	11	21-58	3:8	NA	4-27 [5–10 mg]	Significant remission (63.6%), ineffctive (36.4%)	Hyperlipidemia, multiple sclerosis	NA
3	Craiglow, BG[15]	2018	4	5-7	1:3	Atopic dermatitis	6-15 [10 mg]	Significant remission (25.0%), ineffctive (75.0%)	NA	NA
4	Akdogan, N[16]	2019	9	13-33	7:2	Allergic rhinitis, vitiligo, Hashimoto's thyroiditis	6 [10 mg]	Significant remission (11.1%), improvement (44.4%), ineffctive (44.4%)	Respiratory infections (22.2%), proteinuria (11.1%)	NA
5	Chen, YY[17]	2019	6	20-57	2:4	NA	1-9 [5–10 mg]	Significant remission (66.7%), improvement (16.7%), ineffctive (16.7%)	NA	NA
6	Kibbie, J[18]	2021	11	8-18	4:7	Hypothyroidism, atopic dermatitis	5-39 [10–20 mg]	Significant remission (72.7%), improvement (27.3%)	NA	NA
7	Dincer, RD[19]	2021	13	17-49	4:9	Vitiligo, Hashimoto's thyroiditis	3-15 [10 mg]	Significant remission (53.8%), improvement (7.7%), ineffctive (38.5%)	Acne-like lesion (61.5%), high transaminase (23.1%)	5 patients replased 2 months after stopping the drug, and 1 patient relapsed during treatment
8	Youssef, S[20]	2023	10	7-16	5:5	Hashimoto's thyroiditis, celiac disease	1-9 [10–20 mg]	Significant remission (100.0%)	Acne(10%)	NA
9	Huang, J[21]	2023	11	7-12	6:5	Atopic dermatitis	3-10 [5–10 mg]	Significant remission (63.6%), improvement (9.1%), ineffctive (27.3%)	High transaminase (9.1%), acne-like lesion (9.1%), low hemoglobin (9.1%)	1 patient relapsed 2 months after stopping the drug
10*	Nasimi, M[22]	2024	97	3-59	47:50	NA	6-29 [10 mg]	Significant remission (69.1%), improvement (10.3%), ineffctive (20.6%)	Dyslipidemia, respiratory infection, headache	NA

Notes: 2*: tofacitinib cream was used in 4 patients and data are not included in the table; 10*: Improvement was defined as a >25% change in SALT score.

https://doi.org/10.2147/CCID.S512037

presentations consistent with AT or AU. Of note, one patient demonstrated regrowth of both eyebrows and eyelashes, while another experienced a recurrence during the treatment period. These findings support the conclusion that tofacitinib has definite efficacy in the treatment of AA. The most common adverse reactions observed during the treatment included laboratory abnormalities such as dyslipidemia, elevated liver enzymes and leukopenia. Other reported adverse reactions included respiratory tract infections, urinary tract infections, herpes virus infections, acne and headaches. Some studies have suggested a potential risk of serious complications, including myocardial infarction, venous thromboembolism and malignant neoplasms.[34] In our study, folliculitis was observed in two cases with poor outcomes, and the underlying mechanisms warrant further investigation.

In this study, the timing of peak hair regrowth differed between male and female patients. Significant regrowth was observed in the first three months of treatment in female patients, whereas male patients experienced peak regrowth in the later stages of treatment. However, the sample size was small. Future studies should expand sample sizes and control for confounding factors to clarify sex-specific mechanisms.

This study has limitations: (1) the sample size was small and included only data from a single-center; thus, larger data sets are needed to validate the findings; (2) patients received multiple treatments prior to initiation of tofacitinib, which may have influenced the therapeutic effects and efficacy of the drug; and (3) the follow-up period was short, resulting in a lack of data on the extent of relapse after discontinuation of the drug.

Conclusion

This study further confirms the efficacy and safety of tofacitinib in patients with AA, offering additional data to support its clinical application in the management of AA.

Abbreviations

AA, alopecia areata; SALT, severity of alopecia tool; IP, immune-privileged; MHC I, major histocompatibility complex class I; MHC II, major histocompatibility complex class II; IFN-γ, interferon-γ; JAK, janus kinase; IL-15, interleukin-15; NKG2D, natural Killer group 2, member D; JAK-STAT, janus kinases-signal transducers and activators of transcription; AT, alopecia totalis; AU, alopecia universalis.

Ethics Approval and Informed Consent

This study is approved by the Ethics Committee of The Second Affiliated Hospital of Anhui Medical University (Hefei, China); approval number: SL-YW2022-005.

Consent for Publication

All patients, including parental consent for the patient under 18 years of age, provided informed consent to participate in this study, in accordance with the Declaration of Helsinki.

Acknowledgment

This work was supported by the "Clinical Research Cultivation Program" of the Second Affiliated Hospital of Anhui Medical University (2020LCYB12) and the Research Funds from Anhui Provincial Translational Medicine Research Fund Project (2021zhyx-C60). We are grateful to all persons who contributed their time and energy to developing this article.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported by the "Clinical Research Cultivation Program" of the Second Affiliated Hospital of Anhui Medical University (2020LCYB12) and the Research Funds from Anhui Provincial Translational Medicine Research

Fund Project (2021zhyx-C60). The team leader of the former is the corresponding author of this paper and the funding of the latter is also one of the authors of this article.

Disclosure

The author(s) report no conflicts of interest in this work.

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