

Comparative Efficacy of JAK Inhibitors in Acute vs Nonacute Alopecia Areata: A Retrospective Cohort Study

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Despite advancements in systemic treatments, the efficacy of Janus kinase inhibitors in acute versus nonacute alopecia areata remains poorly defined, necessitating comparative studies to optimize therapeutic strategies. This retrospective, single-centre cohort study included 158 alopecia areata patients treated with Janus kinase inhibitors (tofacitinib or ritlecitinib) between 2021 and 2025, aimed at evaluating clinical outcomes and identifying predictors of super-responder status. Patients were stratified into acute ($n=41$) and nonacute ($n=117$) groups based on disease duration and treatment history, with efficacy assessed using the Severity of Alopecia Tool (SALT) through week 24. Our findings demonstrate that patients with acute alopecia areata achieved significantly superior outcomes, with 65 % reaching SALT100 by week 24 compared to 39.5 % in the nonacute group ($p=0.041$). Binary logistic regression analysis further identified tofacitinib treatment ($OR=2.883$, $p=0.009$) and mild-to-moderate baseline severity ($OR=2.802$, $p=0.015$) as significant predictors of super-responder status, while acute alopecia areata status showed borderline predictive value ($OR=2.418$, $p=0.098$). Although limited by its single-centre design and sample size, this study underscores that early intervention with Janus kinase inhibitors in acute alopecia areata leads to enhanced hair regrowth, emphasizing the critical importance of timely clinical management.

Key words: alopecia areata; tofacitinib; ritlecitinib; JAK inhibitors; super-responders.

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Alopecia areata (AA) is a chronic autoimmune skin disease characterized by nonscarring hair loss

SIGNIFICANCE

Alopecia areata is a challenging condition where the body's immune system mistakenly attacks hair follicles, leading to sudden hair loss and significant emotional distress. Our study shows that using modern medications early in the disease's "acute" stage leads to much better hair regrowth compared to waiting. By proving that timely action is crucial, our work helps doctors provide clearer guidance and more effective treatment plans. Ultimately, this research benefits society by offering hope for faster recovery, reducing the long-term psychological burden on patients and helping them regain their confidence and quality of life.

due to immune dysregulation and compromised hair immune privilege (1). While topical therapies remain the cornerstone for mild AA, recent advancements in systemic treatments and insights from similar skin diseases like psoriasis and atopic dermatitis highlight the importance of early intervention (2). Evidence shows that patients with prolonged severe AA have poorer outcomes. Early systemic treatment targeting immune dysregulation may significantly improve response rates and overall patient outcomes (3–6). Furthermore, the rapid alleviation of symptoms can greatly improve patient adherence to treatment, fostering greater commitment to therapy and increasing the likelihood of sustained positive results while reducing the risk of treatment discontinuation.

Despite current guidelines recommending Janus kinase (JAK) inhibitors as the first-line systemic treatment for moderate to severe AA, systemic corticosteroids remain the preferred choice for acute cases (2). However, in clinical practice, we are inclined to prescribe JAK inhibitors for patients with acute AA. This shift is primarily driven by concerns over the potential adverse effects associated with long-term corticosteroid use, such as weight gain, which can impose significant psychological burdens, particularly on female patients (7, 8). It is important to note that JAK inhibitor treatment for AA in China is a self-funded expense and, therefore, off-label use is becoming increasingly common.

Inspired by the 2024 European Consensus on Alopecia Areata (2), we aim to conduct a comparative study within our patient cohort to evaluate the differences in treatment outcomes between JAK inhibitor therapy for acute versus nonacute AA. By elucidating the distinctions in therapeutic responses, we hope to provide valuable insights to inform clinical decision-making and enhance treatment strategies for patients with AA. This research may contribute to a better understanding of the role of JAK inhibitors in managing acute AA, ultimately guiding more effective and patient-centred approaches to treatment.

METHODS

Study design and patients

This retrospective, observational, single-centre cohort study included AA patients initiating JAK inhibitor treatment from February 2021 to January 2025 at the Department of Dermatology in the Xiangya Hospital of Central South University (Hunan, China). Inclusion criteria: 1) clinical and dermatoscopic AA diagnosis; 2) ≥ 24 week follow-up; 3) complete records with the Severity of Alopecia Tool (SALT) scores at ≥ 3 of 4 time points (weeks 4, 8, 12, 24). Exclusion criteria: diffuse alopecia (due to challenges in assessing objective hair loss severity scores), concurrent systemic corticosteroids/immunosuppressants or prior JAK inhibitor use. Notably, due to limited data availability, the number of patients treated with baricitinib and upadacitinib was insufficient for analysis.

Data collection utilized medical records or telephone interviews with suggestive cases undergoing dermatological examination. All patients provided written informed consent. Ethics approval was granted by the Medical Ethics Committee of Xiangya Hospital Central South University (2026030631). The analysis was conducted in accordance with the STROBE reporting guidelines for observational studies.

Treatment regimen

Patients received ritlecitinib 50 mg or tofacitinib 10 mg daily. Follow-ups occurred every 4 weeks (first 3 months), then every 12 weeks. Patients were stimulated to continue using medicated topical therapy, including topical corticosteroids and/or topical minoxidil. The decision to use combination topical therapy was based on patient preference; however, due to the retrospective nature of the study, this information was not collected as it was difficult to quantify objectively.

Data collection

We collected basic information, including age, sex, time since onset of AA, duration of the current episode,

involvement of eyebrows, eyelashes and nails, access to previous systemic treatments, atopic background, family history of AA, treatment choices, SALT grading according to the Alopecia Areata Investigator Global Assessment (AA-IGA) (9) and baseline SALT scores. The atopic background is defined as medical history or current conditions of atopic dermatitis, allergic rhinitis, allergic conjunctivitis, or allergic asthma. According to the AA-IGA, S1, S2, S3 and S4 categories represent 1–20%, 21–49%, 50–94% and 95–100% of the hair loss area, respectively (9).

Definition of acute alopecia areata and non-acute alopecia areata

In 2012, a study defined rapidly progressive AA as patients with positive hair-pull test results across the entire scalp during their first visit or those with a history of onset or rapid worsening of hair loss over the preceding six months. The 2024 European Consensus on Alopecia Areata on systemic treatment defined active AA as hair loss of less than 6-month duration in treatment-naïve patients. These definitions reflect the disease's progression and acute nature. However, there is no consensus on the definition of acute AA. To explore the relationship between acute/nonacute disease courses and subsequent treatment efficacy, we defined acute AA based on the duration of the current episode and the absence of systemic treatments during this period: 1) duration of the current episode ≤ 24 weeks; 2) no use of systemic treatments, including corticosteroids or JAK inhibitors, during this episode. Nonacute AA is defined as any patient who does not meet these criteria.

Definition of super-responders and non-super-responders

Achieving rapid hair regrowth represents an important therapeutic objective in AA management. Patients exhibiting marked clinical improvement with treatment are categorized as super responders (SRs). Building upon our previous work that systematically compared SRs with nonsuper responders (NSRs), we have established a conceptual framework for defining exceptional treatment responses in AA (10). Consequently, in the present study, we maintain the operational definitions established in our prior work, wherein SRs are characterized by achieving either ≥ 80 % reduction in SALT score by week 12 (SALT80) or complete hair regrowth (SALT100) by week 24, while NSRs fail to meet these predetermined response thresholds.

Outcome measures

The primary outcomes were the proportions of patients achieving 30%/50%/80%/100 % SALT score

improvement (SALT30/50/80/100) at Week 24 for acute AA and nonacute AA. Other secondary effectiveness outcomes included changes in SALT from baseline to week 4, 8 and 12. Safety assessments were based on reported adverse events.

Clinical efficacy was assessed using standardized scalp photography (front, back, left, right and top views) at each follow-up visit. SALT scores were independently evaluated by 2 trained dermatologists to ensure consistency; in cases of disagreement, a 3rd senior dermatologist was consulted to reach a consensus, thereby ensuring robust inter-rater reliability.

Statistical analysis

Categorical variables were expressed as numbers or proportions, whereas categorical variables were reported as mean±standard deviation (SD), median or range. The chi-square and Fisher's exact tests were used to analyse categorical variables, while continuous data were compared using Student's t-test and Mann-Whitney U-tests, when appropriate. Missing SALT scores ($n=22$) were imputed using linear interpolation between adjacent time points. This method was selected because SALT score is a longitudinal clinical measure that generally evolves gradually over time, and interpolation between closely spaced observations is considered a reasonable approximation. In addition, most missing values occurred between 2 observed time points, which further supports the use of this approach. Binary logistic regression analysis was used to explore factors predicting SRs, as well as to compare the differences between SRs and NSRs. For analytical purposes, AA-IGA classifications (S1-S4) were dichotomized into mild-moderate (S1-S2) vs severe (S3-S4) in logistic regression analysis according to Severity of Alopecia Areata Tool (11). Different variables were assessed in univariate analyses, and those with p -value <0.1 were further included in multivariate binary regression. Analyses utilized SPSS 27.0 (IBM) with graphical representations created in Prism 10 (GraphPad).

RESULTS

Baseline characteristics

The study cohort comprised 158 patients, including 41 (26.0%) with acute AA and 117 (74.0%) with nonacute AA (Table I). Baseline characteristics revealed that acute AA patients had later disease onset (28.2 vs 20.7 years, $p=0.002$) compared to nonacute AA patients. Extra-scalp involvement was more common in nonacute AA (50.4% vs 26.8%, $p=0.009$), while mild AA was more prevalent in acute AA (39.0% vs 19.7%, $p=0.043$). In terms of disease background, atopic history and

family history did not differ significantly between the two groups. Detailed baseline characteristics stratified by treatment type are presented in Table S1.

Clinical response to W24

Clinical response assessments at week 24 demonstrated similar efficacy trends for both tofacitinib and ritlecitinib (Figs 1–3). During the 4–24 weeks, both acute and nonacute AA patients showed comparable responses, with SALT30 being the most commonly achieved endpoint and SALT100 the least. By week 24, acute AA patients demonstrated superior outcomes: 65% of tofacitinib-treated acute AA patients achieved SALT100 compared to 39.5% in nonacute AA ($p=0.041$), while 76.2% of ritlecitinib-treated acute AA patients reached SALT80 versus 48.8% in nonacute AA ($p=0.038$). Similar patterns were observed in the overall cohort, with acute AA patients showing higher SALT100 rates as early as week 12 (26.8% vs 12.8%, $p=0.037$).

Sensitivity analyses were performed by excluding nonacute AA patients whose duration of the current episode was <6 months and who had systemic treatment during that time, to assess the robustness of our results. The sensitivity analyses consistently supported the main analysis, demonstrating that the SALT response observed at weeks 4, 8, 12 and 24 remained robust across all treatment groups, including tofacitinib-treated, ritlecitinib-treated, and the overall cohort (Figs S1–S3).

To further elucidate the impact of disease duration on treatment response, we performed additional subgroup analyses by stratifying nonacute AA cases into three categories: disease duration <1 year ($n=25$), 1–5 years ($n=65$) and ≥ 5 years ($n=27$). These subgroups were then compared with acute AA cases (disease duration <6 months, $n=41$) at various treatment timepoints. As illustrated in Figs S4–S6, this refined analysis yielded results largely consistent with our primary findings: acute AA patients consistently demonstrated superior treatment responses across all evaluated timepoints. Among nonacute AA subgroups, we observed a gradual decline in treatment efficacy with increasing disease duration, particularly evident in the 24 week SALT100 response rates (acute AA: 56.1 % vs <1 year: 32.0 % vs 1–5 years: 38.5 % vs ≥ 5 years: 25.9 %; $p<0.05$). This duration-dependent response pattern further supports our hypothesis that earlier therapeutic intervention may yield better outcomes in AA management.

Prediction of becoming super-responders

Super responder analysis revealed that 43.7 % of patients met SR criteria (Fig. S7). Although the difference between acute and nonacute AA did not reach statistical significance (56.1% vs 36.3%, $p=0.062$),

Table I. Baseline demographic and disease characteristics

Characteristics	Total patients (n=158)			p-value
	Acute AA (n=41)	Nonacute AA (n=117)	Total (n=158)	
Age, years, mean (SD)	29.0 (13.5)	25.8 (12.0)	26.6 (12.4)	0.159
Median (range)	28.0 (13.0–61.0)	25.0 (7.0–58.0)	25.0 (7.0–61.0)	
<18 years, n (%)	10 (24.4)	36 (30.8)	46 (29.1)	0.439
≥18 years, n (%)	31 (75.6)	81 (69.2)	112 (70.9)	
Gender, n (%)				0.386
Female	28 (68.3)	71 (60.7)	99 (62.7)	
Male	13 (31.7)	46 (39.3)	59 (37.3)	
Age of diagnosis of AA (year), mean(SD)	28.2(13.9)	20.7(13.0)	22.7(13.6)	0.002
Median (range)	25.0 (7.0–60.0)	17.5 (1.0–58.0)	20.0 (1.0–60.0)	
Duration of current episode, months, mean (SD)	4.0 (5.4)	37.1 (44.6)	28.5 (41.1)	<0.001
Median (range)	3.0 (1.0–36.0)	24.0 (1.0–324.0)	12.0 (1.0–324.0)	
Eyebrow/ Eyelash/Nail affected, n (%)	11.0 (26.8)	59.0 (50.4)	70.0 (44.3)	0.009
Atopic background**, n (%)	7 (17.1)	16 (13.7)	23 (14.6)	0.595
Previous systemic treatment, n (%)	3.0 (7.3)	73 (62.9)	76 (48.4)	<0.001
Family history of AA, n (%)	4 (9.8)	13 (11.1)	17 (10.8)	0.810
Treatment, n (%)				0.068
Tofacitinib	20 (48.8)	76 (65.0)	96 (60.8)	
Ritlecitinib	21 (51.2)	41 (35.0)	62 (39.2)	
AA-IGA, n (%)				0.043
S1 (1–20%)	16 (39.0)	23 (19.7)	39 (24.7)	
S2 (21–49%)	5 (12.2)	33 (28.2)	38 (24.1)	
S3 (50–94%)	6 (14.6)	22 (18.8)	28 (17.7)	
S4 (95–100%)	14 (34.1)	38 (32.5)	52 (32.9)	
SALT score, mean (SD)	52.2 (33.9)	57.8 (31.9)	56.3 (32.4)	0.341

*Atopic background is defined as medical history or current atopic dermatitis, allergic rhinitis, allergic conjunctivitis, or allergic asthma.

AA:alopecia Areata; AA-IGA:alopecia Areata investigator global assessment; SALT:severity of alopecia tool.

acute AA status was considered a potential predictor of long-term treatment response.

So, we built a binary logistic regression model to explore patient characteristics that predict SRs, as shown in **Table II**. Univariate analysis identified eight factors with a p -value<0.1: age, gender, time since onset of AA, duration of current episode,

eyebrow/eyelash/nail affected, acute AA, choice of treatment and severity of AA. A test for multicollinearity revealed no significant correlations among the independent variables. In addition to these factors, we believe that whether or not there has been previous systematic treatment may also affect the results to a greater extent, so this factor

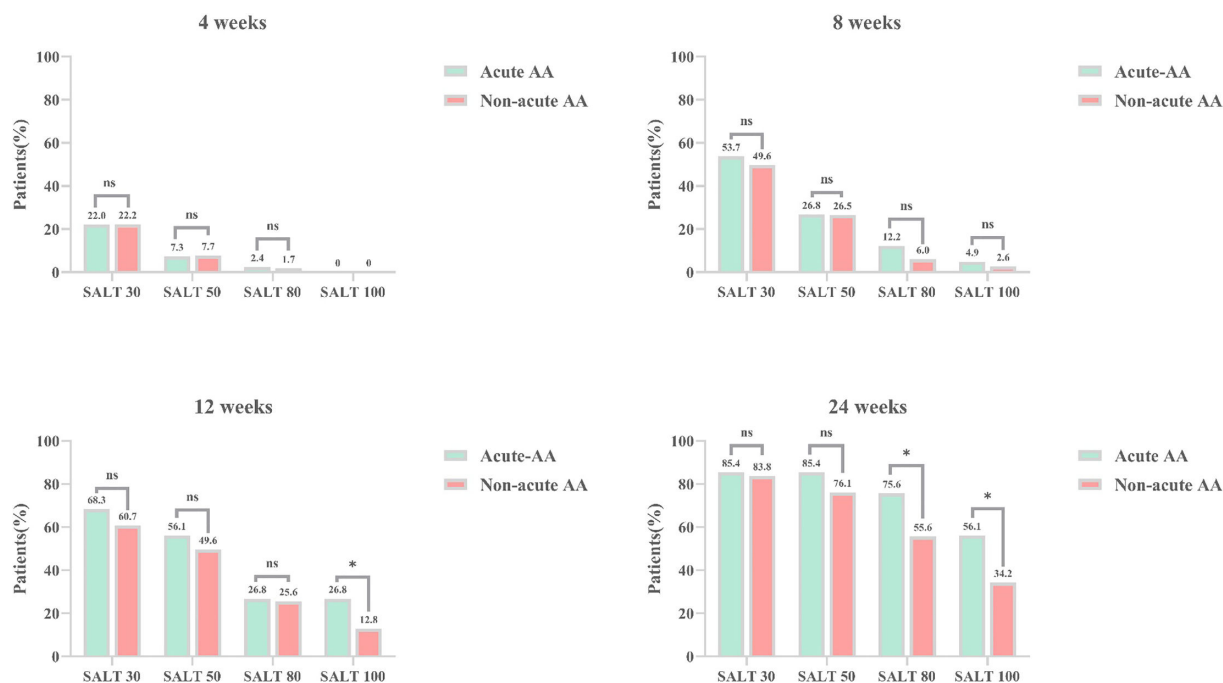


Fig. 1. Comparison of Severity of Alopecia Tool (SALT) response at week 4, 8, 12, 24 between acute alopecia areata (AA) and nonacute AA. ns: not significant; * p <0.05;

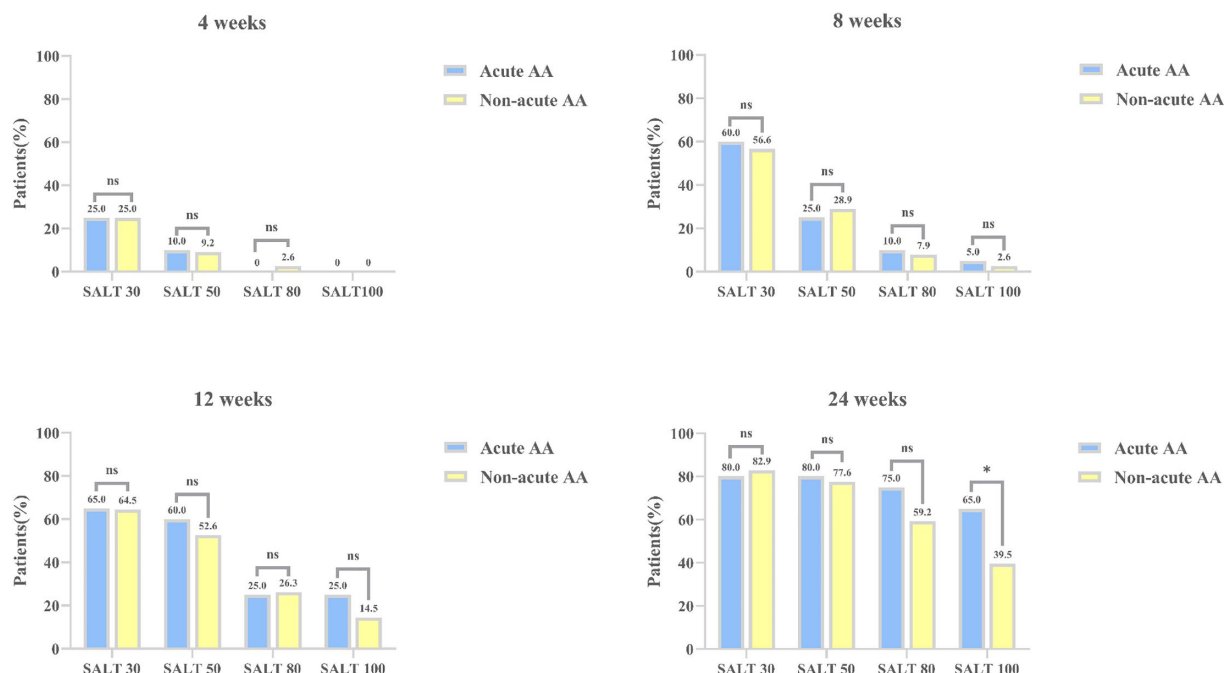


Fig. 2. Comparison of Severity of Alopecia Tool (SALT) response at week 4, 8, 12, 24 between acute alopecia areata (AA) and nonacute AA using tofacitinib. ns: not significant; * $p < 0.05$

was jointly included in the multifactorial analysis model for analysis. Multivariate logistic regression identified tofacitinib treatment (OR=2.883, 95% CI:1.299–6.400, $p=0.009$) and mild-moderate AA severity (OR=2.802, 95% CI:1.222–6.425, $p=0.015$) as significant predictors of SR status. Acute AA showed borderline predictive value (OR=2.418, 95% CI:0.850–6.883, $p=0.098$) but did not reach statistical significance in the final model.

Safety outcomes

The safety analysis (Tables S2-S5) showed similar rates of treatment emergent adverse events between acute (26.8%) and nonacute AA patients (28.2%, $p=0.866$), and between super responders (29.0%) and nonsuper responders (27.0%, $p=0.779$). Acne/folliculitis was the most frequently reported adverse event (15.8%), followed by upper respiratory tract infections (2.5%). Severe adverse events occurred in 3.2 % of patients

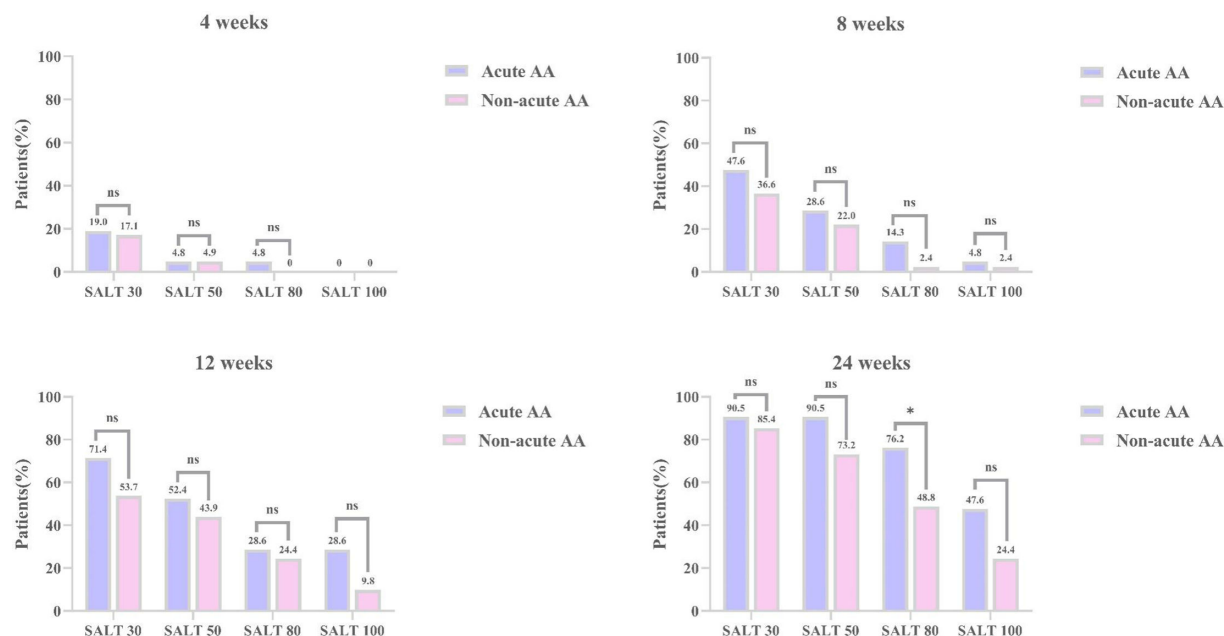


Fig. 3. Comparison of Severity of Alopecia Tool (SALT) response at week 4, 8, 12, 24 between acute alopecia areata (AA) and nonacute AA using ritlecitinib. ns: not significant; * $p < 0.05$

Table II. Univariate and multivariable binary logistic analyses for potential predictors of SRs

Variables	Univariate analyses			Multivariable analyses		
	OR	95% CI	p-value	OR	95% CI	p-value
Age						
<18 years						
≥18 years	2.936	1.380–6.249	0.005	1.772	0.529–5.928	0.353
Gender (M/F)	0.523	0.268–1.020	0.057	0.576	0.268–1.240	0.158
Time since onset of AA (year)						
<18 years						
≥18 years	3.224	1.644–6.321	<0.001	1.692	0.566–5.060	0.347
Duration of current episode (months)	0.988	0.977–0.998	0.026	0.988	0.974–1.002	0.086
Eyebrow/Eyelash/Nail affected	0.499	0.261–0.952	0.035	1.003	0.425–2.363	0.995
Atopic background	0.804	0.326–1.984	0.635			
Previous systemic treatment	1.309	0.696–2.462	0.403	2.372	0.987–5.700	0.053
Family history	1.166	0.4255–3.197	0.766			
Acute AA	1.972	0.960–4.051	0.064	2.418	0.850–6.883	0.098
Treatment						
Tofacitinib	1.744	0.905–3.362	0.097	2.883	1.299–6.400	0.009
Ritlecitinib						
Severity of AA						
Mild to moderate(1%–49%)	2.987	1.555–5.736	0.001	2.802	1.222–6.425	0.015
Severe (50%–100%)						

Values in bold indicate $P<0.10$ in univariate analyses and $P<0.05$ in multivariable analyses.
AA:Alopecia Areata; AA-IGA:Alopecia Areata investigator global assessment; CI:Confidence interval; OR:Odds Ratio; SRs:super-responders.

overall, with higher incidence observed in tofacitinib-treated patients (5.2%) compared to ritlecitinib (0%). Herpes zoster cases were limited to nonacute AA patients (1.7%) and nonsuper responders (2.2%). These findings suggest disease duration does not significantly impact treatment safety.

DISCUSSION

The present study compares the efficacy of JAK inhibitors, tofacitinib and ritlecitinib, in treating acute and nonacute AA, providing insights into how disease chronicity influences treatment outcomes. By addressing a critical gap in the literature, our findings reinforce the key role of JAK inhibition in AA management and highlight the need for a deeper exploration of treatment optimization across different disease trajectories.

Current guidelines advocate early intervention, but the efficacy of systemic treatments in acute versus chronic cases remains poorly understood (2, 12–14). Our study addresses this gap by comparing outcomes between these subgroups, revealing that acute AA patients may experience better medium- and long-term responses, with higher rates of complete hair regrowth in acute AA patients. This indicates that the timing and duration of JAK inhibitor therapy are key factors in treatment outcomes, highlighting the need for future studies with extended follow-up to better understand the benefits of early and sustained intervention.

Another important finding is the comparable efficacy of tofacitinib and ritlecitinib in both acute and nonacute AA. Both agents demonstrated robust improvements in SALT scores, with acute AA patients achieving notably higher SALT100 rates by week 24. These results align

with the growing body of evidence highlighting JAK inhibitors' ability to alleviate AA symptoms and restore hair growth rapidly (15, 16). However, the borderline predictive value of acute AA status for SRs outcomes suggests a more complex interplay between disease chronicity and treatment efficacy. Additionally, our identification of tofacitinib treatment and mild-moderate AA severity as significant predictors of SR status adds another layer of intrigue. These findings are consistent with prior research indicating that less severe disease may be more likely to achieve robust, sustained responses (17). The broader inhibition of JAK pathways by tofacitinib may explain its superior efficacy, yet the comparable outcomes with ritlecitinib accentuate the need for individualized treatment selection (18).

Several limitations must be acknowledged. First, the retrospective design and single-centre setting may constrain the generalizability of our findings and reduce statistical power. Additionally, this limitation made it difficult for us to quantitatively capture all details, such as the usage of topical medications by patients. Second, the exclusion of other JAK inhibitors due to insufficient data restricts our ability to draw conclusions about the broader class of JAK inhibitors in AA treatment. Third, the 24-week follow-up period limits our ability to assess long-term outcomes, including treatment durability and relapse risk. Fourth, the retrospective nature of this study resulted in a lack of detailed clinical phenotyping (e.g. hair-pull tests and dermatoscopic signs) and systematic recording of adjunctive topical therapies, such as corticosteroids or minoxidil. These missing variables may serve as potential confounders influencing early treatment responses. Fifth, the safety findings should be interpreted with caution given the relatively small sample size and limited follow-up

duration, which may preclude the detection of rare or long-term adverse events. In addition, missing SALT scores were imputed using linear interpolation, which assumes a linear change between observations and may introduce some estimation bias; therefore, the results should be interpreted with caution. Finally, due to data limitations, we did not consider the therapeutic effects on hair loss in body parts.

In conclusion, our findings suggest that early and sustained JAK inhibitor therapy may be particularly beneficial for acute AA patients, leading to a more personalized approach to treatment. While our study provides important insights, it also raises critical questions that require further exploration. By addressing these unanswered questions, we can refine AA treatment strategies and ultimately improve clinical outcomes for patients with AA.

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Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

IRB approval status: Reviewed and approved by the institutional research ethics boards of Xiangya Hospital, Central South University (Changsha, China); approval number: 2026030631. All patients or their legal representatives provided written informed consent. This study was also conducted in adherence to the STROBE guidelines.

Conflict of interest: Ji Li and Wei Shi have received consultancy/speaker honoraria from Pfizer. Fangfen Liu and Wei Shi have participated as Principal Investigators in clinical trials sponsored by Pfizer.

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