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Safety and Efficacy of Baricitinib in Pediatric Patients With Alopecia Areata at a Tertiary Hospital in Saudi Arabia

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Faisal A. Aleidi [1], Monirah O. Almadhi [2], Haneen M. Almansour [3], Wed T. Alotaibi [4], Rawan F. Alradi [4], Raghad R. Alhowil [4], Raghad Fahad [4], Mona Khalaf [5], Raghad A. Almuhaitb [6], Muhammad Salman Bashir [7]

1. Pharmacy Service Administration, King Fahad Medical City, Riyadh, SAU 2. Drug Information, Shaqra University, Riyadh, SAU 3. Clinical Pharmacy, College of Pharmacy, Shaqra University, Riyadh, SAU 4. Pharmacology, Shaqra University, Riyadh, SAU 5. Pharmacology, King Saud Bin Abdulaziz University, Riyadh, SAU 6. Pharmacology, King Fahad Medical City, Riyadh, SAU 7. Scientific Writing and Biostatistics, Research Services and Applied Clinical Administration, Research Center, King Fahad Medical City, Riyadh, SAU

Corresponding author: Faisal A. Aleidi, faislaleidi1586@gmail.com

Abstract

Background

Pediatric alopecia areata (AA) is an immune-mediated hair loss disorder associated with substantial psychosocial burden and limited treatment options. Evidence regarding the effectiveness and safety of baricitinib in children remains limited.

Objective

To evaluate the real-world effectiveness and laboratory safety outcomes of oral baricitinib in Saudi pediatric patients with AA.

Methods

This retrospective cohort study included 38 pediatric patients treated with oral baricitinib at King Fahad Medical City, Riyadh, Saudi Arabia, between January 2018 and December 2024. Demographic characteristics, previous treatments, dosing regimens, clinical outcomes, and laboratory findings were extracted from electronic medical records. Treatment outcomes were categorized as complete response, partial response, ineffective treatment, or no response. Associations between clinical characteristics and treatment outcomes were assessed using chi-square or Fisher's exact tests.

Results

The mean patient age was 12.24 ± 3.34 years, and 19 patients (50.0%) were female. Twenty patients (52.6%) had previously received tofacitinib. The median starting and optimal baricitinib doses were 2 mg and 4 mg, respectively, and the median treatment duration was 12 months. Complete and partial responses were documented in 11 (28.9%) and 22 (57.9%) patients, respectively, yielding a combined response proportion of 86.8%. One patient (2.6%) was classified as having ineffective treatment, and four (10.5%) had no response. At or after three months, abnormal low-density lipoprotein levels, liver function tests, and glomerular filtration rate were recorded in 19 (50.0%), 12 (31.6%), and two (5.3%) patients, respectively. Treatment outcomes were not significantly associated with sex or previous tofacitinib exposure.

Conclusion

Baricitinib was associated with complete or partial clinical responses in most patients in this pediatric cohort. Laboratory abnormalities highlight the importance of ongoing monitoring. Larger prospective studies using standardized response definitions and longer follow-up are needed to establish pediatric effectiveness and long-term safety.

Categories: Pharmacology, Pediatrics, Dermatology
Keywords: alopecia areata universalis, baricitinib, janus kinase (jak) inhibitors, pediatric, safety

Introduction

Alopecia areata is a chronic, immune-mediated, non-scarring hair loss disorder that commonly affects children and adolescents. Pediatric alopecia areata is associated with significant psychological distress and impaired quality of life, and limited effective treatment options. The visible and unpredictable nature of hair loss during childhood and adolescence often leads to emotional distress, including anxiety, low self-esteem, embarrassment, and social isolation, as hair plays a central role in body image and social identity at a young age. These psychological effects may interfere with peer relationships, school participation, and emotional

development, contributing to a sustained reduction in overall well-being. Consequently, children with alopecia areata consistently report poorer health-related quality of life compared with healthy peers, with psychosocial domains being the most affected [1]. In the pediatric population, currently available therapies, including topical or intralesional corticosteroids and contact immunotherapy, demonstrate inconsistent efficacy, high relapse rates, and tolerability concerns, while many systemic treatments lack robust pediatric safety and efficacy data. This combination of psychological burden, diminished quality of life, and inadequate therapeutic options underscores a significant unmet clinical need in pediatric alopecia areata [2].

Alopecia areata (AA) is a chronic, immune-mediated, nonscarring hair disorder characterized by an autoreactive T-cell-mediated attack on hair follicles. Its pathogenesis primarily involves cytotoxic T-cell activation and overproduction of pro-inflammatory cytokines, particularly interferon-γ (IFN-γ) and interleukin-15 (IL-15), which signal through the Janus kinase-signal transducer and activator of transcription (JAK-STAT) pathway, resulting in collapse of hair follicle immune privilege and sustained follicular inflammation. The clinical observation of hair regrowth following JAK inhibition further supports the central role of JAK-STAT signaling in AA and has provided a strong rationale for the development of targeted immunomodulatory therapies [3-4].

Baricitinib is an orally administered, selective Janus kinase 1 and 2 (JAK1/JAK2) inhibitor that interferes with intracellular signaling of multiple pro-inflammatory cytokines implicated in autoimmune conditions. By inhibiting JAK1/JAK2 activity, baricitinib reduces downstream STAT phosphorylation and attenuates inflammatory pathways involved in hair follicle damage. Clinical trials have demonstrated significant hair regrowth in patients with moderate-to-severe alopecia areata treated with baricitinib, supporting its efficacy and safety profile as a targeted therapeutic option [5].

In the phase III BRAVE-AA1 and BRAVE-AA2 trials, continuous treatment with baricitinib for 52 weeks resulted in clinically meaningful hair regrowth. At Week 52, a Severity of Alopecia Tool (SALT) score ⩽20 was achieved by 40.9% (4 mg) and 21.2% (2 mg) of patients in BRAVE-AA1, and by 36.8% (4 mg) and 24.4% (2 mg) in BRAVE-AA2. Treatment response improved over time, supporting the need for long-term therapy, and no new safety signals were identified. Based on these positive efficacy and safety results, baricitinib subsequently received regulatory approval from the U.S. Food and Drug Administration for the treatment of severe alopecia areata in adults [6].

Emerging evidence suggests that baricitinib may be an effective therapeutic option for pediatric patients with AA, including those with severe and treatment-refractory disease, with growing real-world and clinical data indicating meaningful hair regrowth in children with high disease burden [7-11]. In a real-world cohort of 33 children aged 2-18 years with severe AA (baseline SALT ⩾50), 45.5% achieved at least a 50% reduction in SALT score after a mean treatment duration of 6.5 months, despite more than half presenting with very severe disease (baseline SALT >90); adverse events were mild, including elevated alanine aminotransferase levels, acne, and upper respiratory infections, with no serious complications reported. Across pediatric reports, baricitinib doses of 2-4 mg daily were generally well tolerated [7-11]. Comparative pediatric data further demonstrate clinically significant SALT score improvements across moderate, severe, and very severe AA after 24 weeks of treatment, even under non-standard dosing regimens. Additionally, pediatric case reports describe substantial and sustained hair regrowth in refractory AA, including patients with complete scalp hair loss and those with comorbid atopic or autoimmune conditions, with acceptable safety profiles under close laboratory monitoring; for example, a child with total scalp hair loss showed substantial regrowth after one year of baricitinib 4 mg daily, with the SALT score improving from 100 to 24.4 and trichoscopy showing new vellus hair formation. Collectively, these findings suggest that baricitinib holds promise as a targeted systemic therapy for severe and refractory pediatric AA [7-11].

Baricitinib exerts immunomodulatory effects through suppression of cytokine signaling pathways, which contributes to its therapeutic efficacy but also increases susceptibility to infections. Clinical evidence indicates that pediatric and adult patients receiving baricitinib may experience serious infections, occasionally requiring hospitalization, with rare life-threatening outcomes. Reported infections include bacterial, viral, fungal, and opportunistic pathogens, with pneumonia, urinary tract infections, and herpes zoster among the most frequently described severe events. Reactivation of latent viral infections, particularly herpes zoster, has also been observed, emphasizing the importance of infection screening before initiation and close clinical monitoring during therapy. In patients who develop clinically significant infections, temporary interruption of treatment is often recommended until resolution [12].

In addition to infectious risks, laboratory abnormalities represent an important component of baricitinib safety monitoring. Hematologic changes such as lymphopenia, neutropenia, and mild anemia may occur due to modulation of cytokine pathways involved in hematopoiesis; these abnormalities are typically reversible after dose adjustment or treatment interruption. Hepatic laboratory changes, including elevations in alanine aminotransferase (ALT) and aspartate aminotransferase (AST), have been reported, most commonly as mild and asymptomatic findings, although persistent elevations warrant evaluation for potential drug-induced liver injury. Dose-dependent increases in lipid parameters, including total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL), may also emerge during early treatment and should be assessed through periodic follow-up testing. Less frequently, mild changes in renal function markers such as serum creatinine and estimated glomerular filtration rate (eGFR) have been described and should be

interpreted relative to baseline renal status [13].

The long-term impact of baricitinib on growth and development in pediatric populations remains incompletely understood, as most available studies focus on short- to medium-term safety outcomes rather than direct developmental endpoints. Longitudinal data from pediatric atopic dermatitis trials have demonstrated sustained efficacy and an acceptable safety profile, without evidence of clinically significant effects on growth or developmental progression during follow-up periods of up to approximately 3.6 years. Nevertheless, given the biological role of JAK-STAT signaling in immune and developmental processes, continued monitoring of growth and maturation is advised during prolonged therapy. Overall, current evidence suggests that concerns regarding growth and development are largely precautionary rather than reflecting confirmed developmental toxicity [13,14].

Therefore, this study aims to evaluate the clinical efficacy, safety profile, and tolerability of baricitinib in pediatric patients with alopecia areata. Specifically, we seek to assess treatment outcomes using standardized measurement tools, the SALT score, monitor for common adverse events and laboratory abnormalities, and gather preliminary insights into its potential impact on pediatric growth and development to address the existing therapeutic gap in this population.

Materials And Methods

This retrospective cohort study was conducted at King Fahad Medical City (KFMC), Riyadh, Saudi Arabia, using electronic medical records for 38 pediatric patients diagnosed with AA who were treated with oral baricitinib between January 2018 and December 2024. The primary objective of this study was to evaluate the long-term real-world effectiveness of oral baricitinib in Saudi pediatric patients with AA. Secondary objectives included assessment of treatment safety, laboratory changes, treatment modifications, and clinical factors associated with treatment response over a six-year follow-up period.

Patients were eligible for inclusion if they had a confirmed diagnosis of AA based on documented clinical and dermoscopic findings, received oral baricitinib for a minimum duration of 12 months, and had complete baseline and follow-up clinical data available for review, including disease severity assessment. Patients were excluded if they were treated with baricitinib for indications other than AA, had diffuse alopecia not consistent with AA, or had incomplete follow-up or missing key clinical or laboratory data. Demographic and clinical variables were extracted from the EPIC electronic medical record system (Epic Systems Corporation, Verona, Wisconsin, USA) using a standardized data collection form. Collected data included age, sex, body weight, AA subtype, prior treatment history, baricitinib dosing regimen and duration, treatment modifications, laboratory parameters, reported adverse events, and clinical response to therapy. Disease severity was assessed using the SALT score, a validated quantitative measure used to estimate the percentage of scalp hair loss. The SALT score is calculated by evaluating hair loss across four scalp regions: vertex (40%), right profile (18%), left profile (18%), and occipital region (24%), with the weighted percentages summed to generate a total score ranging from 0 (no hair loss) to 100 (complete scalp hair loss) [7]. All patients underwent baseline laboratory investigations prior to initiating baricitinib therapy, including complete blood count, liver and renal function tests, and lipid profile, with periodic monitoring throughout treatment.

The primary outcome was treatment effectiveness. Treatment outcomes were classified into four mutually exclusive categories: complete response, defined as complete scalp hair regrowth; partial response, defined as incomplete scalp hair regrowth meeting the predefined effectiveness criteria of a ⩾50% reduction in SALT score from baseline or a final SALT score ⩽25; ineffective treatment, defined as some improvement that did not meet either effectiveness criterion; and no response, defined as no improvement or worsening of scalp hair loss at the final assessment. Secondary outcomes included the incidence and type of adverse events, changes in laboratory parameters, dose modifications, and treatment discontinuation or switching [15].

Categorical variables (e.g., gender, diagnosis, prior treatment, and treatment response) were summarized using frequencies and percentages, and group comparisons were performed using the chi-square test or Fisher's exact test, as appropriate. Continuous variables (e.g., age, weight, treatment duration, and low-density lipoprotein (LDL) levels) were presented as means ± standard deviations and compared between groups using the independent samples t-test. All statistical tests were two-tailed, and a p-value < 0.05 was considered statistically significant. Analyses were conducted using IBM SPSS Statistics for Windows, Version 25 (Released 2017; IBM Corp., Armonk, New York, United States) and MedCalc version 23.0.2 (MedCalc Software Ltd., Ostend, Belgium). Ethical approval for the study was obtained from the Institutional Review Board of King Fahad Medical City (approval no. 26-100), and all patient data were anonymized to ensure confidentiality in accordance with institutional and ethical guidelines.

Results

Table *1* summarizes the demographic, clinical, and safety characteristics of the 38 pediatric patients diagnosed with alopecia areata, who had an even gender distribution (19 (50.0%) male, 19 (50.0%) female) and a mean age of 12.24 ± 3.34 years. Over half of the cohort (20 (52.6%)) had received previous treatment with tofacitinib. In our pediatric cohort, the median starting dose of baricitinib was 2 mg (IQR: 2-4 mg) once

daily, while the median optimal dose was 4 mg (IQR: 2-4 mg), over a median treatment duration of 12 months (IQR: 9.75-16 months). All patients underwent rigorous safety monitoring, including pre- and on-treatment screening for tuberculosis and viral hepatitis, as well as baseline lipid and renal profiling. During follow-up (≥3 months), laboratory monitoring revealed post-baseline abnormalities, notably in LDL (19 (50.0%)) and liver function tests (12 (31.6%)). Overall therapeutic responses were favorable, with 11 (28.9%) achieving a complete response, 22 (57.9%) a partial response, 4 (10.5%) showing no response, and 1 (2.6%) having ineffective outcomes.

Variables	Description	Complete (n=11)	Partial (n=22)	Ineffective (n=1)	None (n=4)	Test Statistic (χ2)	p-value
Gender	Male	6 (54.5%)	12 (54.5%)	0 (0.0%)	1 (25.0%)	χ2=2.27	0.518
	Female	5 (45.5%)	10 (45.5%)	1 (100.0%)	3 (75.0%)		
Previous treatment	Tofacitinib	5 (45.5%)	11 (50.0%)	1 (100.0%)	3 (75.0%)	χ2=1.99	0.574
	No previous treatment	6 (54.5%)	11 (50.0%)	0 (0.0%)	1 (25.0%)		
LFTs - Baseline	Abnormal	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (25.0%)	χ2=8.74	0.033*
	Normal	11 (100.0%)	22 (100.0%)	1 (100.0%)	3 (75.0%)		
LDL: ≥ 3 months	Abnormal	7 (63.6%)	10 (45.5%)	1 (100.0%)	1 (25.0%)	χ2=3.02	0.392
	Normal	4 (36.4%)	12 (54.5%)	0 (0.0%)	3 (75.0%)		
LFTs: ≥ 3 months	Abnormal	3 (27.3%)	6 (27.3%)	1 (100.0%)	2 (50.0%)	χ2=3.09	0.381
	Normal	8 (72.7%)	16 (72.7%)	0 (0.0%)	2 (50.0%)		
GFR: ≥ 3 months	Abnormal	0 (0.0%)	2 (9.1%)	0 (0.0%)	0 (0.0%)	χ2=1.54	0.674
	Normal	11 (100.0%)	20 (90.9%)	1 (100.0%)	4 (100.0%)		

TABLE 1: Demographic Characteristics of Patients (n = 38)

Note: Categorical variables are presented as frequencies and percentages, n (%). Test statistics (χ^2) and p-values were calculated using Pearson's Chi-square test (or Fisher's exact test where appropriate).

* Indicates statistical significance at $p < 0.05$.

LFT: liver function test; LDL: low-density lipoprotein; GFR: glomerular filtration rate.

Table 2 illustrates the associations between patient clinical characteristics and treatment outcomes. Among the evaluated parameters, abnormal baseline liver function tests (LFTs) were documented in only one patient, who was in the no-response group. Baseline LFT status was associated with treatment-response category in the reported analysis (p = 0.033), where an abnormal baseline LFT was exclusively observed in the non-responder group (1 (25.0%)), while all patients with complete (11 (100.0%)), partial (22 (100.0%)), or ineffective (1 (100.0%)) outcomes had normal baseline LFTs. Conversely, all other clinical and safety variables showed no significant association with treatment efficacy. Specifically, gender distribution did not significantly impact outcomes (p = 0.518, with males comprising 6 (54.5%) of complete and 12 (54.5%) of partial responders versus 1 (25.0%) of the no-response group). Similarly, prior treatment history with tofacitinib (p = 0.574), post-baseline LDL abnormalities at ≥3 months (p = 0.392), post-baseline LFT abnormalities at ≥3 months (p = 0.381), and glomerular filtration rate (GFR) status at ≥3 months (p = 0.674) varied across response categories but failed to reach statistical significance. No clinical adverse events, including infections, were documented in the available medical records during baricitinib treatment.

Variables	Description	Outcome				p-value
		Complete	Partial	Ineffective	None	
Gender	Male	6 (54.5%)	12 (54.5%)	0 (0.0%)	1 (25.0%)	0.518
	Female	5 (45.5%)	10 (45.5%)	1 (100.0%)	3 (75.0%)	
Previous treatment	Tofacitinib	5 (45.5%)	11 (50.0%)	1 (100.0%)	3 (75.0%)	0.574
	No previous treatment	6 (54.5%)	11 (50.0%)	0 (0.0%)	1 (25.0%)	
LFTs - Baseline	Abnormal	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (25.0%)	0.033*
	Normal	11 (100.0%)	22 (100.0%)	1 (100.0%)	3 (75.0%)	
LDL: ≥ 3 months	Abnormal	7 (63.6%)	10 (45.5%)	1 (100.0%)	1 (25.0%)	0.392
	Normal	4 (36.4%)	12 (54.5%)	0 (0.0%)	3 (75.0%)	
LFTs: ≥ 3 months	Abnormal	3 (27.3%)	6 (27.3%)	1 (100.0%)	2 (50.0%)	0.381
	Normal	8 (72.7%)	16 (72.7%)	0 (0.0%)	2 (50.0%)	
GFR: ≥ 3 months	Abnormal	0 (0.0%)	2 (9.1%)	0 (0.0%)	0 (0.0%)	0.674
	Normal	11 (100.0%)	20 (90.9%)	1 (100.0%)	4 (100.0%)	

TABLE 2: Association between outcome of the Baricitinib and clinical characteristics of patients

Note: Categorical variables presented as frequencies and percentages. * Indicates p-value significant at < 0.05.

LFT: liver function test; LDL: low-density lipoprotein; GFR: glomerular filtration rate.

Discussion

The present retrospective cohort study evaluated the real-world effectiveness and safety of oral baricitinib in 38 pediatric patients with alopecia areata treated at a tertiary hospital in Saudi Arabia. By examining clinical outcomes, laboratory abnormalities, and treatment modifications, this study contributes additional observational evidence regarding baricitinib use in this population. The findings should be interpreted within the context of the retrospective study design and the relatively small sample size.

Pediatric alopecia areata can adversely affect psychological well-being, causing embarrassment, self-consciousness, social isolation, and impaired quality of life. Visible hair loss may undermine confidence and interfere with social interactions during important developmental stages. These consequences highlight the importance of timely treatment initiation in children experiencing significant psychological distress, alongside appropriate psychosocial support, to address the emotional burden of the condition and promote their overall well-being [15].

Published evidence on baricitinib use in pediatric alopecia areata remains limited. In a real-world retrospective study, Wei et al. included 33 children and adolescents aged 2-18 years with severe alopecia areata and reported that 15 (45.5%) achieved at least a 50% reduction in SALT score after an average treatment duration of 6.5 months, with no serious adverse events reported [7]. In this context, our cohort of 38 patients represents a reasonable sample for an exploratory retrospective real-world study and is comparable in size to this published pediatric cohort. However, this comparison does not establish adequate statistical power, and the relatively small sample remains an important limitation, restricting the precision and generalizability of the findings and the ability to detect uncommon adverse events. Larger prospective, multicenter studies with longer follow-up are needed to confirm treatment effectiveness and better characterize the long-term safety of baricitinib in pediatric patients.

In our cohort, 20 (52.6%) of patients had previously received tofacitinib. However, because of the retrospective nature of the study, the medical records did not document the specific rationale for the initial selection of tofacitinib; therefore, we cannot conclude that it was chosen solely because it was available earlier. Nevertheless, this explanation is plausible, as tofacitinib was one of the earliest JAK inhibitors reported for alopecia areata, including pediatric and adolescent patients, before the approval and wider availability of newer JAK inhibitors for this indication. Craiglow et al. reported the use of tofacitinib for alopecia areata and its variants in adolescents in 2017 [16], and later Craiglow and King reported its use in preadolescent children in 2019 [17]. In contrast, ritlecitinib, a JAK inhibitor approved for severe alopecia

areata in adults and adolescents aged 12 years and older, received FDA approval later [18].

Although JAK inhibitors share a common therapeutic class, they should not be assumed to be clinically equivalent or automatically interchangeable. The British Association of Dermatologists guideline highlights limited real-world evidence regarding switching between these agents and emphasizes individualized treatment decisions. Accordingly, the choice of a JAK inhibitor should consider patient age, disease severity, previous treatment response, comorbidities, safety considerations, and access to licensed therapies, rather than assuming that class membership alone establishes equivalent clinical suitability [19].

In our pediatric cohort, the median starting dose of baricitinib was 2 mg (IQR: 2-4 mg) once daily, while the median optimal dose was 4 mg (IQR: 2-4 mg). For comparison, the phase 3 BRAVE-AA1 and BRAVE-AA2 trials evaluated fixed doses of 2 mg and 4 mg once daily in adults with severe alopecia areata. Both doses were superior to placebo at week 36, with higher rates of clinically meaningful scalp hair regrowth observed with 4 mg. Reported adverse events included acne, elevated creatine kinase levels, and increased lipid levels [5]. However, our study did not directly compare outcomes between doses and therefore cannot establish superior efficacy or comparable safety of 4 mg versus 2 mg in pediatric patients.

In our pediatric cohort, 20 patients (20 (52.6%)) had previously received tofacitinib; however, prior treatment status was not significantly associated with baricitinib treatment outcomes ($p = 0.574$, Table *2*). In contrast, Muñoz-Barba et al. reported significantly greater percentage reductions in SALT scores among JAK inhibitor-naïve adults than among tofacitinib-exposed adults at each assessment point over 12 months ($p < 0.05$). Patients previously treated with tofacitinib showed no statistically significant change in SALT scores during follow-up ($p > 0.20$) [20]. Differences in patient populations, study designs, and outcome measures limit direct comparison. Importantly, the absence of a statistically significant association in our cohort does not establish equivalent responses between the groups.

Regarding safety profiles, baricitinib treatment has been widely associated with alterations in lipid metabolism. In the pivotal Phase 3 trials (BRAVE-AA1 and BRAVE-AA2), elevations in low-density lipoprotein (LDL) cholesterol were observed more frequently in patients receiving baricitinib compared with placebo [5]. Our findings align with this known safety profile; while all patients had normal baseline lipid levels prior to therapy, 19 (50.0%) developed abnormal LDL levels at or after 3 months of treatment (Table *1*). No statistically significant association was detected between post-baseline LDL abnormalities and treatment response ($p = 0.392$, Table *2*). However, this finding does not exclude a clinically relevant association, particularly given the small sample size and limited numbers within some response categories.

Furthermore, switching from one JAK inhibitor to another has been increasingly recognized as a clinical strategy in the management of immune-mediated inflammatory conditions. While much of the existing literature regarding JAK inhibitor cycling stems from rheumatologic conditions, where switching has demonstrated favorable retention and efficacy rates [21], sequential therapy is also becoming common in managing refractory pediatric alopecia areata. Although our comparative analysis showed no statistically significant difference in final treatment outcomes between treatment-naive patients and those with prior tofacitinib exposure ($p = 0.574$, Table *2*), these findings highlight that sequential JAK inhibitor therapy is a frequent real-world clinical practice, warranting further investigation into optimal sequencing strategies.

Finally, emerging real-world evidence suggests that ritlecitinib may be considered as a sequential treatment option for patients with severe alopecia areata who have an inadequate response or treatment failure with a previous JAK inhibitor. Although evidence regarding prior JAK inhibitor exposure is limited in pivotal clinical trials, recent real-world studies and multicenter retrospective reviews have reported encouraging improvements in SALT scores among patients previously treated with agents such as tofacitinib or baricitinib [22, 23]. However, treatment responses remain variable, and current data are primarily derived from retrospective analyses [22, 23]. Therefore, failure of an initial JAK inhibitor does not necessarily preclude a clinical response to subsequent options like ritlecitinib, which can serve as a viable alternative when clinically appropriate.

This study has several limitations, including a small pediatric cohort (N = 38) and a retrospective design, which limit the generalizability of our findings. Additionally, treatment heterogeneity - such as prior exposure to tofacitinib in over half of the cohort, variable dosing, and non-standardized treatment durations - presents potential confounding factors. Baseline SALT scores and alopecia subtypes were not consistently documented in the available records, limiting characterization of baseline disease severity and comparisons of treatment response across disease subtypes. The retrospective design may have limited the capture of clinical adverse events, particularly those not documented in the medical records or managed outside the study institution. Detailed information on the magnitude and severity of laboratory abnormalities, their subsequent resolution, and whether they prompted dose modification or treatment discontinuation was not consistently available. Consequently, the clinical significance and course of these abnormalities could not be fully assessed. Finally, the lack of long-term prospective follow-up limits our assessment of treatment durability and potential long-term effects on growth and development. Larger, prospective multicenter studies using standardized outcome measures and systematic safety monitoring are needed to confirm these preliminary findings and better characterize pediatric dosing and long-term safety.

Conclusions

This retrospective real-world study demonstrates that oral baricitinib is a promising and generally well-tolerated systemic therapy for pediatric patients with severe and treatment-refractory alopecia areata. Over a median treatment duration of 12 months, the majority of patients achieved favorable clinical responses, with 33 (86.8%) experiencing complete or partial hair regrowth despite more than half of the cohort having prior exposure to tofacitinib. While safety monitoring revealed expected post-treatment metabolic changes, Laboratory monitoring identified abnormal LDL levels and liver function tests during treatment, highlighting the importance of ongoing monitoring. Although baseline LFT status showed a statistically significant association with treatment response in the reported analysis, only one patient had abnormal baseline LFTs, and this patient was in the no-response group. Given the small cohort and the reliance of this finding on a single observation, the association should be considered exploratory and interpreted with considerable caution. It does not establish abnormal baseline LFTs as a predictor of treatment response. Overall, our findings underscore the clinical utility of baricitinib in the pediatric population and highlight the viability of sequential JAK inhibitor therapy. However, given the limitations of a retrospective design and a single-center sample size, large-scale prospective multicenter studies with extended follow-up are warranted to optimize pediatric dosing strategies, establish long-term safety, and refine management guidelines for severe pediatric alopecia areata.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Faisal A. Aleidi, Monirah O. Almadhi, Haneen M. Almansour, Wed T. Alotaibi, Rawan F. Alradi, Raghad R. Alhowil, Raghad Fahad, Mona Khalaf, Muhammad Salman Bashir, Raghad A. Almuhaitb

Acquisition, analysis, or interpretation of data: Faisal A. Aleidi, Monirah O. Almadhi, Haneen M. Almansour, Wed T. Alotaibi, Rawan F. Alradi, Raghad R. Alhowil, Raghad Fahad, Mona Khalaf, Muhammad Salman Bashir, Raghad A. Almuhaitb

Drafting of the manuscript: Faisal A. Aleidi, Monirah O. Almadhi, Haneen M. Almansour, Wed T. Alotaibi, Rawan F. Alradi, Raghad R. Alhowil, Raghad Fahad, Mona Khalaf, Muhammad Salman Bashir, Raghad A. Almuhaitb

Critical review of the manuscript for important intellectual content: Faisal A. Aleidi, Monirah O. Almadhi, Haneen M. Almansour, Wed T. Alotaibi, Rawan F. Alradi, Raghad R. Alhowil, Raghad Fahad, Mona Khalaf, Muhammad Salman Bashir, Raghad A. Almuhaitb

Supervision: Faisal A. Aleidi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Institutional Review Board of King Fahad Medical City issued approval 26-100. I am pleased to inform you that the submission dated February 5, 2026, for the study titled 'Safety and Efficacy of Baricitinib in Pediatric Patients with Alopecia Areata at a Tertiary Hospital in Saudi Arabia' was reviewed and was approved according to Good Clinical Practice guidelines. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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