

Review Article Trichology

Clinical evidence for JAK inhibition in alopecia areata: A comprehensive review

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INTRODUCTION

JAK inhibitors are small-molecule agents that block the signalling of several cytokines involved in the pathogenesis of alopecia areata (AA), notably interferon- γ and interleukin-15, which activate and perpetuate CD8⁺ cytotoxic T cells, responsible for autoimmune inflammation against the hair follicle.^[1] Both topical and systemic formulations of JAK inhibitors have been utilized in the management of alopecia areata.^[2] A 2014 case report was the first to demonstrate that the JAK inhibitor tofacitinib (Xeljanz®) could successfully treat alopecia areata. Since then, multiple studies have confirmed the efficacy of JAK inhibitors in promoting hair regrowth in this condition.^[3] The FDA-approved oral JAK inhibitors for severe alopecia areata include baricitinib and deурuxolitinib, for adults >18 years, ritlecitinib inhibitor for adults and adolescents ≥ 12 years.^[4] We hereby discuss in detail the mechanism of action of individual JAK inhibitors and the efficacy data of the same.

PATHOGENESIS OF ALOPECIA AREATA

The hair follicle possesses a relative state of immune privilege, and alopecia areata (AA) is characterized by a breakdown of this privilege [Figure 1]. In particular, anagen hair follicles, especially those involved in pigment production, become targets of inflammatory cell infiltration.^[5] Cytotoxic CD8⁺ NKG2D⁺ T cells have been identified as key mediators in the pathogenesis of AA. Upregulation of interleukin-15 (IL-15) within the hair follicle microenvironment contributes to the loss of immune privilege by promoting the recruitment and activation of CD8⁺

NKG2D⁺ T cells, which subsequently produce interferon-gamma (IFN- γ).^[6] Genome-wide association studies have revealed associations between AA and multiple genes regulating regulatory T-cell activity and cytokine signaling, including IL-2/IL-21, cytotoxic T-lymphocyte-associated antigen 4 (CTLA4), human leukocyte antigen (HLA) region genes, and IL-2 receptor alpha (IL-2RA).^[7] While genetic predisposition is evident, the role of environmental factors in disease initiation remains uncertain.^[7]

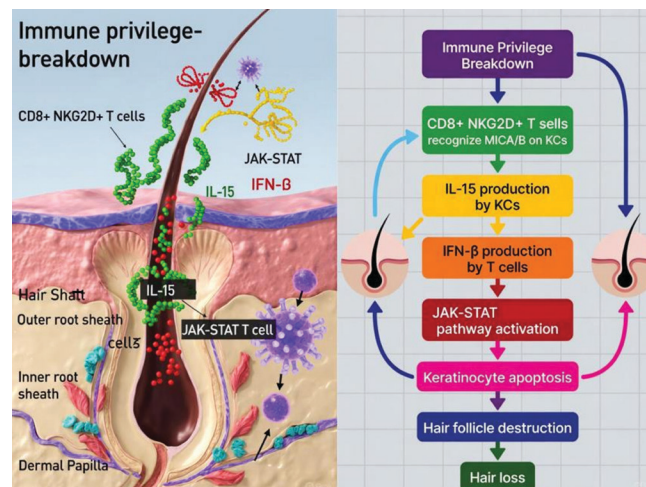


Figure 1: Depicts the immunopathogenesis of alopecia areata [created using Grok AI].

The Janus kinase (JAK) family comprises four intracellular tyrosine kinases: JAK1, JAK2, JAK3, and tyrosine kinase 2 (TYK2), which interact with type I and II cytokine receptors

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Received: 13 November 2025 Accepted: 19 December 2025 Published: 07 January 2026 DOI: 10.25259/JHRRM_17_2025

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to mediate downstream signal transduction. These kinases regulate fundamental cellular processes, including proliferation, migration, differentiation, and apoptosis.^[8] JAKs are integral to hematopoiesis, immune regulation, and host defense mechanisms. Cytokine signaling through IL-15, IFN- γ , and other cytokines implicated in AA (e.g., IL-2, IL-7, IL-13, and IL-21) occurs via the JAK–signal transducer and activator of transcription (STAT) pathway.^[9]

In AA, IFN- γ promotes IL-15 production in hair follicles through JAK1/2 signaling, while IL-15 in turn stimulates IFN- γ release by T cells via JAK1/3 signaling, establishing a positive feedback loop that amplifies perifollicular inflammation [Table 1].^[10] These insights have identified JAK inhibitors as promising therapeutic agents in the management of AA.

Baricitinib

Baricitinib is a selective inhibitor of JAK1/2, demonstrating approximately 100-fold greater selectivity for these isoforms compared to JAK3. The reduced affinity for JAK3 may limit the immunosuppressive adverse effects typically associated with JAK3 inhibition. Baricitinib exhibits an oral bioavailability of about 80%, with metabolism primarily mediated by cytochrome P450 3A4 (CYP3A4). It has an elimination half-life of approximately 12 hours, with 75% of the administered dose excreted via urine and 20% via feces. Of the excreted fraction, 69% remains unchanged in urine and 15% in feces.^[11,12]

Baricitinib is an oral first-generation JAK1/JAK2–predominant inhibitor approved for the treatment of alopecia areata at doses of 1 mg, 2 mg, or 4 mg. The recommended starting dose is 2 mg orally once daily, which may be increased to 4 mg. In cases of severe alopecia areata or when eyelashes and eyebrows are affected, treatment may begin at 4 mg and then be reduced to 2 mg once an adequate response is achieved.^[13]

In adults from the BRAVE-AA1 (n = 465) and BRAVE-AA2 (n = 390) trials, treatment with baricitinib 4 mg and 2 mg resulted in SALT scores ≤ 20 at week 52 in 40.9% and 21.2% of patients in BRAVE-AA1, and 36.8% and 24.4% of patients in BRAVE-AA2, respectively.^[14] Since Baricitinib primarily targets JAK1 and JAK2, with limited activity against JAK3 and TYK2, lowers the likelihood of systemic side effects compared with broader JAK inhibitors.^[15]

Ritlecitinib

Ritlecitinib is an oral selective JAK3/TEC family kinase inhibitor, FDA-approved in June 2023 for severe alopecia areata in patients ≥ 12 years.^[16] In patients with alopecia areata, ritlecitinib treatment produced dose-dependent early decreases in absolute lymphocyte count (ALC), as well as reductions in CD3⁺, CD4⁺, CD8⁺ T cells, and natural killer

(NK) cells, while CD19⁺ B cells remained unaffected.^[16]

Ritlecitinib exhibits approximately dose-proportional pharmacokinetics up to 200 mg, with steady state achieved in about four days and peak plasma concentration (C_{max}) reached within one hour of oral dosing. The drug has an absolute oral bioavailability of ~64%, low plasma protein binding (14%), and a terminal half-life of 1.3–2.3 hours. It is metabolized primarily by glutathione S-transferases (GSTA1/3, GSTM1/3/5, GSTP1, GSTS1, GSTT2, GSTZ1, and microsomal GSTs 1–3) and cytochrome P450 enzymes (CYP1A2, CYP2C8, CYP2C9, CYP3A), with elimination via urine ($\approx 66\%$; 4% unchanged) and feces ($\approx 20\%$).^[17]

In the ALLEGRO-2a trial (n = 48), 50% of patients receiving ritlecitinib 200 mg once daily for 4 weeks followed by 50 mg once daily for 20 weeks achieved $\geq 30\%$ improvement in SALT score from baseline. In the larger ALLEGRO-2b/3 trial (n = 718), the proportion of patients achieving a SALT score ≤ 20 at week 24 was 31% in the 200 mg + 50 mg group, 22% in the 200 mg + 30 mg group, 23% in the 50 mg group, 14% in the 30 mg group, and 2% with placebo.^[18,19]

Deuruxolitinib

Deuruxolitinib is a selective JAK 1/2 inhibitor developed for the treatment of moderate-to-severe AA. Inhibiting JAK1/2 blocks the signaling of cytokines such as interferon- γ and interleukin-15, which are implicated in the autoimmune attack on hair follicles. It was approved for AA in the United States in July 2024, the third small molecule JAK inhibitor approved as therapy for alopecia areata, after baricitinib (in 2022) and ritlecitinib (in 2023).^[20]

Phase 3 trials (THRIVE-AA1 and THRIVE-AA2), 1209 patients were randomized to receive placebo (n=267), deuruxolitinib 8 mg twice daily (n=600), or 12 mg twice daily (n=342). Both deuruxolitinib doses achieved the primary efficacy endpoint of attaining a SALT score ≤ 20 at Week 24. Specifically, 31.0% of patients in the 8 mg BID group and 40.3% in the 12 mg BID group reached this target, compared with 0.8% in the placebo group (p<0.0001).

Significant improvement over placebo was evident as early as Week 8 (p<0.0005). Furthermore, 22.5% of patients in the 8 mg BID group and 31.6% in the 12 mg BID group achieved a SALT score ≤ 10 at Week 24, while none in the placebo group did (p<0.0001). For relative change in SALT from baseline, both deuruxolitinib doses demonstrated significant differences from placebo as early as Week 4 (p<0.01).^[21]

OFF LABEL JAK STAT INHIBITORS

Other JAK inhibitors that have been used off-label for AA include the first-generation agents tofacitinib and ruxolitinib.

These two off-label molecules are discussed in detail. More recently, a range of selective second-generation JAK inhibitors has been developed, such as upadacitinib, brepocitinib, ritlicitinib, abrocitinib, jaktinib, deucravacitinib, ifidancitinib, and delgocitinib have been tried with less evidence.^[22] Table 1 provides an overview of JAK-STAT inhibitors reported in the literature for the treatment of AA.

Table 1: Depicts the role of various cytokines in the pathomechanism of alopecia areata

Cytokines	Receptor	Main JAKs used	Down stream STAT	Role in AA
IFN- γ	IFNGR	JAK1 + JAK2	STAT1	Induces IL-15 in HF epithelium
IL-15	IL-15R α / γ ^c	JAK1 + JAK3	STAT5	Activates & expands CD8 ⁺ cells
IL-2	IL-2R	JAK1 + JAK3	STAT5	T-cell survival/proliferation
IL-21	IL-21R	JAK1 + JAK3	STAT3	Amplifies Th17/Tfh responses

JAK: Janus kinase inhibitors, AA: Alopecia areata, IFN- γ : Interferon-gamma, IL: Interleukin, IFNGR: Interferon-gamma receptor, STAT: Signal transducer and activator of transcription.

Tofacitinib

Tofacitinib, a pan JAK1/3>2, TYK inhibitor, has been used in topical and oral formulations [Table 2] for alopecia areata. The recommended dose of tofacitinib ranges from 2.5 mg to 15 mg once daily for AA. The oral bioavailability of ~74%, and the drug is primarily metabolized in the liver via CYP3A4, with a minor contribution from CYP2C19, producing inactive metabolites. Elimination occurs mainly through the urine (~70%) and partially via feces (~30%), with a half-life of ~3 hours, supporting a twice-daily dosing regimen. Steady-state concentrations are reached within 24–48 hours, and pharmacokinetics are generally dose-proportional, though co-administration with CYP3A4 modulators can alter exposure. As a pan JAK inhibitor, it inhibits the inflammatory cascade linked with AA, such as interleukin (IL)-2, IL-4, IL-7, IL-9, IL-15, IL-21, and interferon (IFN)- γ [Figure 2].^[23]

Studies using tofacitinib 5 mg twice daily have shown encouraging results in alopecia areata. In an open-label study of 66 patients, 64% responded after 3 months, with similar outcomes across subtypes.^[23] A 2019 retrospective study of 63 patients (73% with alopecia universalis) reported complete regrowth in 40%, partial regrowth in 43%, and minimal regrowth in 12% after 6 months, with only 5% non-responders. In comparison, regimens escalating up to 10 mg twice daily (as in Jabbari *et al.*) achieved >50% regrowth in

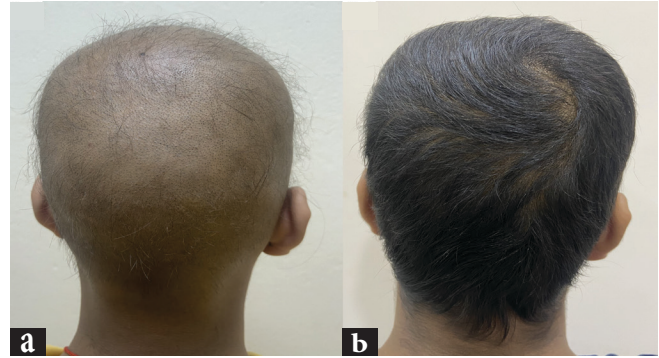


Figure 2: (a) Depicts the baseline, (b) Complete resolution of alopecia areata after 9 months of tofacitinib.

67% of 12 patients over 6–12 months, with median SALT score improvements of 70% in patch-type AA and 68% in ophiasis, but substantially lower in alopecia totalis (11.8%) and universalis (10.5%).^[24] In adults, as per a systematic review of 12 studies, out of 346 patients, 288 patients were on oral tofacitinib, and 58 were on oral ruxolitinib, and a 50% reduction in SALT score was seen in 66% of cases. Recurrence of AA was seen in 74% of the patients within 3 months after withdrawal of therapy.^[25]

As per the retrospective study by Huang *et al.* and Jerjen *et al.* in 11 preadolescent populations, 64% of the cases showed a 50% improvement in the SALT score earlier with oral tofacitinib alone, and 63.6% showed a 50% reduction in the SALT score in the latter study with oral tofacitinib and oral minoxidil.^[26,27] These data were in concordance with the results seen in the current series, but in previous studies, less than 50% of the cases of AT and AU were recruited. As per Indian data, a case report by Bhokare *et al.* showed that a young boy with therapy-resistant patch type AA responded to tofacitinib within 2 months and that complete hair growth was seen in 6 months.^[28]

Ruxolitinib

Ruxolitinib is a selective JAK1/e2 inhibitor that reaches peak plasma concentration within 1–2 hours and has a half-life of approximately 3–6 hours. It exhibits high oral bioavailability (~95%), with steady-state levels achieved within 4–5 days of twice-daily administration. The drug is primarily metabolized by CYP3A4. Clinical efficacy has been demonstrated at a dose of 20 mg twice daily in open-label studies.^[22]

In one trial involving 12 patients with moderate-to-severe AA (mean baseline SALT 66% \pm 28%), treatment with ruxolitinib 20 mg twice daily for 3–6 months resulted in a 41% reduction in SALT score at 3 months and 58% at 6 months.^[29] Another parallel open-label study compared ruxolitinib (20 mg BID) with tofacitinib (5 mg BID) in 75 patients with severe AA. The mean time to initial hair regrowth was significantly

Table 2: Summary of JAK inhibitors, dosing schedule and mechanism of action in AA

JAK inhibitors	Mechanism of Action	Route	Dosing Regimens	Efficacy
Baricitinib	JAK1/JAK2 inhibitor	Oral	4 mg or 2 mg once daily	Phase 3 BRAVE-AA trials show ~35–40% SALT \leq 20 at 36 weeks with 4 mg; ~20–25% with 2 mg. Mean SALT improvement ~45–50%.
Tofacitinib	JAK1/JAK2/JAK3/ TYK inhibitor	Oral	5 mg twice daily; 10 mg twice daily; 10–25 mg split twice daily	Real-world studies report 30–60% mean SALT improvement; up to 70% regrowth in responsive patients. Higher doses more effective but relapse common.
		Topical	2% twice daily	Limited evidence—10–20% SALT improvement; more effective for eyebrows/patchy AA.
Ruxolitinib	JAK1/JAK2 inhibitor	Oral	20 mg twice daily; 5–15 mg twice daily; 10–25 mg twice daily	Small cohorts show 50–75% mean SALT improvement at 3–6 months; efficacy similar to tofacitinib in some series.
		Topical	0.6% twice daily; 1% twice daily; 1.5% twice daily	Topical formulations show modest benefit, ~15–25% SALT improvement; best responses in eyebrow/limited AA.
Ritlecitinib	JAK3/TEC inhibitor	Oral	200 mg once daily for 4 weeks, then 50 mg once daily for 20 weeks; 50 mg, 30 mg, or 10 mg once daily	ALLEGRO trials: ~25–30% SALT \leq 20 at week 24 with 50 mg; mean SALT improvement ~40%. Stronger responses in severe AA with continuous use.
Brepocitinib	TYK2/JAK1 inhibitor	Oral	60 mg once daily for 4 weeks, then 30 mg once daily for 20 weeks	Phase 2 data show ~50% SALT improvement; ~25–30% achieve SALT \leq 20. Efficacy comparable to ritlecitinib.
Upadacitinib	JAK1 inhibitor	Oral	30 mg daily	Limited AA-specific data; small studies show 35–50% SALT improvement at 24 weeks, better responses at higher doses.
Abrocitinib	JAK1 inhibitor	Oral	200 mg daily; 100 mg or 200 mg once daily	Early reports/real-world data suggest 30–40% SALT improvement, but robust AA-specific trials are limited.
Ifidancitinib	JAK1/JAK3 inhibitor	Topical	0.5% or 0.1% twice daily	-
Delgocitinib	JAK1/JAK2/JAK3/TYK2 inhibitor	Topical	30 mg/g twice daily	-

BRAVE: Baricitinib Alopecia Areata Clinical Program; JAK: Janus kinase inhibitors, AA: Alopecia areata, TYK: Tyrosine kinase inhibitor, SALT: Severity of alopecia tool.

shorter with ruxolitinib (4 ± 3 weeks) compared to tofacitinib (7 ± 2 weeks; $p = 0.003$). However, by 6 months, both groups achieved comparable rates of hair regrowth ($\approx 95\%$ reduction in SALT score) and similar relapse rates at 3-month follow-up.^[30]

OFF LABEL TOPICAL JAK INHIBITORS IN AA

Topical formulations include tofacitinib, ruxolitinib, ifidancitinib, and deucravacitinib. Topical formulations have also been used for eyelash and facial hair regrowth, with a case series ($n = 119$) showing complete regrowth in 41% of eyebrows and 46% of eyelashes.^[31] In a review of 136 instances of topical JAK inhibitor (JAKi) therapy for alopecia areata, the vast majority were administered as monotherapy (135/136, 99.3%), with only one case involving combination therapy. Tofacitinib was the most frequently used agent (43.7%), followed by ruxolitinib (41.5%) and delgocitinib (14.8%). Treatment duration was reported in 107 cases, with a mean of 142.6 days (range: 28–330 days). SALT scores were available in 39% of cases, showing an average reduction of 24.9% from baseline, and 23.5% of cases

achieved $\geq 50\%$ improvement (SALT50). Follow-up data on disease recurrence were limited (11%), with 2 of 15 patients (13.3%) experiencing relapse at a mean of 2.3 months. Concurrent therapy was not documented. Adverse events were infrequent (3.7%), limited to scalp irritation (4 cases) and folliculitis (1 case), with no treatment discontinuations, indicating that topical JAKi therapy is generally well tolerated with modest efficacy.^[32]

ADVERSE EVENTS

The FDA has issued a black box warning for this class based on safety data from tofacitinib studies in rheumatoid arthritis. In dermatology, oral JAK1 inhibitors (abrocitinib, upadacitinib), the JAK1/2 inhibitor baricitinib, the TYK2 inhibitor deucravacitinib, and topical ruxolitinib are currently used, each with distinct safety profiles. Clinical trial data in dermatology show low rates of serious adverse events, including venous thromboembolism (0–0.5%), cardiovascular events (0–1.2%), serious infections (0.4–4.8%), nonmelanoma skin cancer (0–0.9%), and other malignancies

(0–0.7%), with most affected patients having pre-existing risk factors. Common side effects of oral JAK inhibitors include upper respiratory infections, nasopharyngitis, nausea, headache, and acne. Dermatologists are advised to assess baseline patient risk factors and implement appropriate screening and laboratory monitoring when initiating therapy with JAK inhibitors. JAK 2 inhibitors can also cause anemia, lymphopenia, neutropenia, thrombocytopenia, hyperlipidemia, and creatine kinase elevations.^[33]

WHEN TO CONSIDER JAKi TREATMENT OF AA, AND OPTIMIZING EFFICACY

Clinical trials of JAK inhibitors have primarily included adolescents and adults with 50–100% scalp hair loss of 6 months to 10 years' duration. In practice, treatment may also be considered for less severe scalp involvement, eyebrow/eyelash loss, refractory disease, significant psychosocial impact, or diffuse positive hair pull test, and even for episodes longer than 10 years. Trial analyses have identified two key factors influencing efficacy: duration of severe hair loss, with treatment within the first 3–4 years showing better outcomes, and presence of existing scalp hair, which predicts greater response compared to near-total hair loss.^[34]

TAPERING of JAK INHIBITORS (AUTHOR'S EXPERIENCE)

JAK inhibitor tapering is individualized, as no standardized guideline exists, and decisions are based on clinical stability, disease severity, and relapse risk. In general, tapering should be attempted only after sustained remission for at least 6–12 months, with close monitoring every 4–8 weeks. The most commonly used approach involves stepwise dose reduction, beginning with decreasing the dose from 5 mg twice daily to 5 mg once daily, or equivalently from 10 mg once daily to 5 mg once daily. If the disease remains controlled for 2–3 months, the dosing interval may be extended to alternate-day administration before eventual discontinuation. In patients with a history of severe disease, rapid progression, or frequent relapses, a slower or more conservative taper is preferred.^[35] This may include gradually reducing the number of weekly doses (e.g., from daily dosing to 3–4 days per week) over several months before complete withdrawal. Throughout tapering, routine laboratory monitoring (CBC, liver enzymes, lipid profile) every three months, along with disease-specific clinical scores, is essential. Tapering should be avoided in patients with recent disease activity, remission lasting less than six months, or those dependent on tofacitinib as the anchor drug. If a flare occurs, the prior effective dose should be reinstated for stabilization before considering a slower tapering strategy.

LONG TERM SAFETY AND SUSTAINED MAINTENANCE OF EFFICACY

The risk of relapse after tapering or discontinuing tofacitinib

is significant, particularly in alopecia areata. Relapse is commonly reported in 50–80% of patients after dose reduction or discontinuation, reflecting the drug's short half-life and rapid loss of immune modulation.^[36] Preventing relapse and sustaining the therapeutic effect of tofacitinib in alopecia areata requires a combined strategy of prolonged remission, gradual tapering, adjunctive maintenance therapies, and close monitoring. Adjunctive maintenance options, including topical corticosteroids and intralesional corticosteroids for focal patches, and in multifocal and extensive cases, systemic therapy is preferred. Short-term mini-pulse oral steroids, azathioprine, and methotrexate may be considered in high-risk or extensive cases. If relapse occurs to the baseline, re-escalation to the last effective dose of tofacitinib and targeted local therapies often restores control. In individuals with severe phenotypes or repeated relapses, long-term low-dose maintenance therapy may be required to sustain regrowth and prevent disease recurrence; however, long-term safety in dermatological indications has not been explored.

A comprehensive safety analysis of JAK inhibitors in immune-mediated rheumatological diseases included 82 studies (selected from 973 screened), encompassing 66,159 patients exposed to JAK inhibitors, with approximately two-thirds of the studies being randomized controlled trials. The overall incidence of adverse events was 42.65 per 100 person-years, while serious adverse events occurred at a rate of 9.88 per 100 person-years. Specific safety outcomes included serious infections at 2.81 per 100 person-years, herpes zoster infection at 2.67 per 100 person-years, malignancy at 0.89 per 100 person-years, and major adverse cardiovascular events at 0.48 per 100 person-years. Importantly, all-cause mortality was not increased with JAK inhibitor therapy compared with placebo or active comparators (relative risk 0.72; 95% CI 0.40–1.28).^[37]

ROLE OF SWITCHING WITHIN JAK INHIBITORS

Switching JAK inhibitors (especially from tofacitinib → baricitinib or ritlecitinib → deuruxolitinib) represents a viable and often successful therapeutic strategy in refractory or relapsing alopecia areata, with real-world response rates of 60–80% in prior non-responders. This approach should be considered before abandoning the JAK inhibitor class entirely. Prospective studies are ongoing to confirm these findings.^[38]

CONCLUSION

JAK inhibitors represent a targeted, disease-modifying therapy for alopecia areata (AA) by disrupting the inflammatory cascade mediated by CD8⁺ NKG2D⁺ T cells, interferon- γ , and interleukin-15. Both oral and topical formulations have demonstrated efficacy in promoting hair regrowth across AA subtypes, including patchy, totalis, and universalis forms.

FDA-approved oral agents such as baricitinib, deuruxolitinib, and ritlecitinib, that have shown significant reductions in SALT scores, with the highest responses observed in patients with shorter disease duration and residual scalp hair at treatment initiation. Off-label therapies, including tofacitinib and ruxolitinib, also achieve meaningful regrowth, though relapse after discontinuation is common. Topical JAK inhibitors offer modest efficacy and a favorable safety profile, particularly for eyebrows and eyelashes.

Author contributions: SKG: Contributed towards study conceptualization, methodology design, funding acquisition, visualization and supervision, study resources and software work, performed formal analysis, validation, is also responsible for drafting, reviewing, and editing of the manuscript; SV: Performed formal analysis, study investigation, and is also responsible for study visualization, manuscript writing, reviewing, and editing; SG: Contributed towards methodology design, performed formal analysis, study investigation, and supervision; NT: Responsible for data curation, manuscript writing, methodology design, study visualization, supervision, resources, and software work, is also responsible for reviewing and editing the manuscript.

Ethical Approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship: Nil

Conflicts of interest: There are no conflicts of interest.

Use of Artificial Intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that they have used artificial intelligence (AI)-assisted technology for creating Figure 1 only. No AI assistance was employed in the generation of scientific content, data analysis or interpretation.

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How to cite this article: Gowda SK, Verma S, Gadegone S, Taneja N, Gupta S. Clinical evidence for JAK inhibition in Alopecia Areata: A comprehensive review. *J Hair Restor Regen Med.* 2026;1:17-23 doi: 10.25259/JHRRM_17_2025