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ORIGINAL ARTICLE

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Efficacy, Safety, and Real-World Aspects of Janus Kinase Inhibitors to Treat Patients With Alopecia Areata

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ABSTRACT

Alopecia areata (AA) is an autoimmune disease characterized by nonscarring hair loss on the scalp and body, leading to a reduced quality of life and psychosocial burden for patients. The only US Food and Drug Administration–approved treatments for severe AA are the Janus kinase inhibitors (JAKis) baricitinib, ritlecitinib, and deuruxolitinib. Herein, we discuss the efficacy, safety, and real-world aspects of JAKi treatment for patients with AA for an advanced practice provider audience. We review the clinical characteristics and diagnosis of AA and discuss treatment expectations for patients using JAKis.

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INTRODUCTION

Alopecia areata (AA) is an autoimmune disease characterized by nonscarring hair loss on the scalp and body, with periods of relapse and remission, or complete hair loss on the scalp (alopecia areata totalis [AT]) or whole body, including the eyebrows and eyelashes (alopecia areata universalis [AU]).[1,2] Adults with AA seek outpatient primary and dermatological care more frequently than adults without AA, for both AA and comorbid conditions, negatively impacting mental health, social interactions, and employment.[3] A model predicting demands for dermatological care in the US through the year 2030 suggests that nurse practitioners (NPs) and physician assistants (PAs) will become crucial providers in the dermatology workforce to avoid shortages in care; thus, continuing to educate NPs and PAs in dermatological conditions and treatments is critical.[4]

Alopecia areata affects 2% of the global population.[1] Prevalence is higher in females than in males, which may be influenced by a greater tendency for females to seek medical care.[5] Reported incidence is greatest in Black and Hispanic/Latino patients.[6] While AA can develop at any time, the average age of diagnosis is 38.9 to 40.6 years; in a subgroup analysis, incidence is highest in adults 18 to 44 and children and adolescents 12 to 17 years of age.[5] Occurrences of AA in identical twins and siblings support that AA is hereditary;[7] genes associated with AA are involved in immune functions and Janus kinase (JAK) signaling pathways.[7] Environmental factors, including traumatic events and acute stress, also influence the development and progression of AA.[8] Pathologically, AA develops due to the loss of protection of hair follicles from immune cells (collapse in immune privilege), leading to the release of proinflammatory cytokines that activate JAK signaling pathways (Figure 1A).[9,10]

Comorbid Conditions and Psychosocial Burden

Based on a meta-analysis (n = 102 studies), autoimmune and inflammatory disorders are common comorbidities in patients with AA, including thyroid (autoimmune thyroiditis [prevalence, 41.4%] and autoimmune hypothyroidism [4.5%]), atopic (allergic rhinitis [26.5%] and allergic conjunctivitis [21.4%]), dermatological (skin infection [13.2%] and acne vulgaris [12.8%]), and rheumatic (rheumatoid arthritis [1.0%] and systemic lupus erythematous [0.3%]) diseases.[11] Additional comorbidities include cardiovascular disease (13.4%), iron deficiency anemia (6.3%), and vitamin D deficiency (1.0%).[11] A systematic review of 4 studies indicated that, globally, 66% to 74% of patients with AA suffer from psychiatric disorders, including anxiety and depression; an additional study reported that over half suffer from impaired quality of life (QoL).[12]

Clinical Presentation and Diagnosis

Alopecia areata presents as patchy hair loss without scarring or inflammation; besides minor itching and tingling, most patients are asymptomatic prior to and during AA episodes.[1,2] Hair loss may visually differ between patients depending on the AA subtype, including patchy alopecia, ophiasis, AT, and AU.[2]

Alopecia areata is typically diagnosed through clinical examination and trichoscopy, although skin biopsy might be necessary when a diagnosis is uncertain.[2,8] Clinical features of AA by trichoscopy include yellow dots, short vellus hairs, black dots, tapered hairs, broken hairs, exclamation mark hairs, upright regrown hairs, and pigtail hairs.[2] As AA is hereditary and often comorbid with other conditions, family history and medical background should be considered when diagnosing AA.[7,8,11]

Defining Alopecia Areata Disease Severity

Alopecia areata severity is evaluated by different tools in clinical trials and clinical practice. In clinical trials, AA severity and treatment efficacy are assessed using the Severity of Alopecia Tool (SALT), from which an overall AA severity score is calculated (0 [no hair loss] – 100 [100% hair loss]).[13] The Investigator's Global Assessment defines clinically meaningful scalp hair regrowth as a SALT score ≤20 (≤20% scalp hair loss).[14] Clinician-reported outcome measures assess degrees of eyebrow and eyelash hair loss and nail involvement.[15] The amount of eyebrow and eyelash hair, nail damage, QoL, and psychological symptoms are also assessed using patient-reported outcome (PRO) questionnaires.[15-17]

A different AA disease severity scale, the Alopecia Areata Scale, was developed by dermatologists in 2022 for use in clinical practice.[18] With this tool, the extent of scalp hair loss at the time of the assessment is the primary basis for determining disease severity (≤20% scalp hair loss = mild AA, 21% - 49% scalp hair loss = moderate AA, and ≥50% scalp hair loss = severe AA).[18] If AA severity is mild or moderate, a clinician can increase the AA severity rating by one level if ≥1 of the following are present: noticeable involvement of the eyebrows or eyelashes, inadequate response after ≥6 months of treatment, diffuse (multifocal) positive hair pull test consistent with rapidly progressive AA, or negative impact on psychosocial functioning resulting from AA.[18]

Treatment Options for Alopecia Areata

Treatments for AA include topical therapies (corticosteroids, calcineurin inhibitors, minoxidil, immunotherapies [dinitrochlorobenzene, squaric acid dibutylester]), intralesional corticosteroids, nonprescription therapies (aromatherapy, acupuncture), and systemic therapies (corticosteroids, oral minoxidil, dupilumab).[19] Treatment decisions depend on patient age, disease duration, and disease severity.[19] These treatments may be sufficient to treat mild or moderate AA;[19] however, in the US, the JAK inhibitors (JAKis) baricitinib, ritlecitinib, and deuruxolitinib are the only US Food and Drug Administration (FDA)-approved treatments for patients with severe AA. Baricitinib and deuruxolitinib are indicated to treat severe AA in adults;[20,21] ritlecitinib is indicated to treat severe AA in adults and adolescents ≥12 years of age.[22] The most recent Alopecia Areata Investigational Assessment Guidelines for the US from 2004[13] and expert guidance from an international group on treatment options for AA that mentions JAKis both predate the first approval of JAKis for severe AA in the US in 2022.[19,20] Thus, updated guidelines on the use of JAKis for severe AA in the US are needed. This review discusses the efficacy, safety, and real-world aspects of using JAKis for the management of patients with severe AA by NPs and PAs.

Mechanisms of Action of JAK Inhibitors in Alopecia Areata

Baricitinib and deuruxolitinib have similar mechanisms of action (MOAs) and are both oral, reversible, selective JAK1 and JAK2 inhibitors;[23,24] ritlecitinib has a distinct MOA and is an oral selective inhibitor of JAK3 and 5 members of the tyrosine kinase expressed in the hepatocellular carcinoma (TEC) kinase family (Figure 1B).[25]

Clinical Trials, Efficacy, and Safety of JAK Inhibitors for the Treatment of Severe Alopecia Areata

Baricitinib

The efficacy and safety of baricitinib was demonstrated in the double-blind, parallel-group, randomized, placebo-controlled BRAVE-AA1 (NCT03570749; Phase 2/3) and BRAVE-AA2 (NCT03899259; Phase 3) studies of adult patients with severe AA who received 2 mg or 4 mg of once-daily (QD) oral baricitinib (approved doses for severe AA) or placebo for 36 weeks (additional trial details in Figure 2).[20,23] In both trials, a significantly greater percentage of patients treated with baricitinib vs placebo achieved a SALT score ≤20 at week 36 (P<0.001 for baricitinib 2 mg and 4 mg vs placebo; Figure 3).[23]

Patients treated with baricitinib vs placebo also experienced significantly greater improvements in the Scalp Hair Assessment PRO (BRAVE-AA1: 2 mg, P=0.001; 4 mg, P<0.001; BRAVE-AA2: 2 mg, P=0.002; 4 mg, P<0.001) and eyelash and eyebrow hair regrowth (P<0.001), and a greater percentage achieved a SALT score ≤10 at week 36 (2 mg, P=0.002; 4 mg, P<0.001).[23] Response rates for both baricitinib doses increased from baseline through week 52 of treatment, and patients who achieved a SALT score ≤20 maintained this response long-term, through week 104.[26,27] Additionally, a greater percentage of patients using baricitinib vs placebo experienced improvements in health-related QoL (HRQoL), anxiety, and depression after 36 and 52 weeks of treatment.[16,17]

Over 6 months of treatment, baricitinib outperformed traditional AA treatments (topical immunomodulators, corticosteroid injections, and immunosuppressants), with a greater percentage of patients achieving SALT scores ≤20 and ≤10.[28] At week 36, the frequency and severity of adverse events (AEs) were similar between baricitinib 2-mg, baricitinib 4-mg, and placebo treatment arms, and most AEs were mild or moderate in severity (Figure 4).[23,26,27]

Ritlecitinib

The double-blind, randomized, multicenter, Phase 2b/3 ALLEGRO (NCT03732807) trial demonstrated the efficacy and safety of ritlecitinib in patients ≥12 years of age (approved dose for the treatment of severe AA is 50 mg QD; additional trial details in Figure 2).[22,25] A significantly greater percentage of patients treated with ritlecitinib 50 mg vs placebo achieved a SALT score

FIGURE 1. (A) Molecular pathology of alopecia areata and (B) mechanisms of action of JAK inhibitors. (A) In alopecia areata, the hair follicle is exposed to increased expression of major histocompatibility complex I and II and NKG2D (not shown), which leads to recruitment of CD8+NKG2D+ T cells. These T cells produce inflammatory cytokines including IFNγ and IL-15, which use JAKs for intracellular signaling. (B) Baricitinib, ritlecitinib, and deuruxolitinib inhibit JAKs to block the production of cytokines and downstream pathways in alopecia areata.

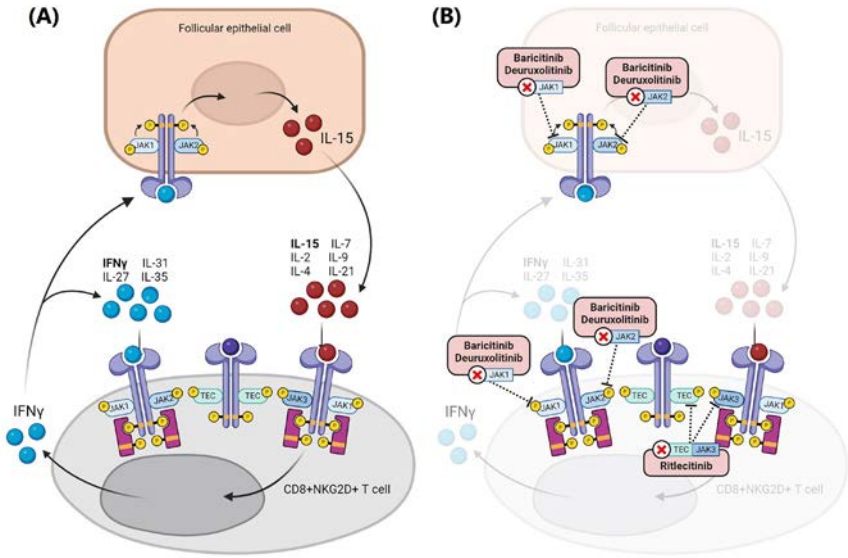


Figure created with BioRender.com.
CD, cluster of differentiation; IFNγ, interferon gamma; IL, interleukin; JAK, Janus kinase; NKG2D, natural killer group 2D; P, phosphorylation; TEC, tyrosine kinase expressed in hepatocellular carcinoma.

FIGURE 2. Study design details from key clinical trials of baricitinib, ritlecitinib, and deuruxolitinib for the treatment of severe alopecia areata.

	Baricitinib	Ritlecitinib	Deuruxolitinib
Phase 2/3 trial name and date[a]	BRAVE-AA1 (Sep 2018–Feb 2021) and BRAVE-AA2 (Jul 2019–Jan 2021)	ALLEGRO Phase 2b/3 (Dec 2018–Dec 2020)	THRIVE-AA1 (Nov 2020–Apr 2022) and THRIVE-AA2 (Jun 2021–Jun 2022)
Trial length, weeks	36; extension through 52 and 104; ongoing	24; extension for 24; ongoing, long-term, open-label efficacy and safety study	24; open-label extension for 108; ongoing, long-term, open-label efficacy and safety study
Patient age, years	Male, 18–60; female, 18–70	Adolescents, 12–17; adults, ≥18	18–65
Inclusion criteria	SALT score ≥50 Current AA episode lasting >6 months and <8 years without spontaneous improvement	SALT score ≥50 Current AA episode ≤10 years with no terminal hair regrowth within 6 moths of screening and baseline visits	SALT score ≥50 Current AA episode lasting 6 months to 10 years
Exclusion criteria	• "Diffuse" pattern AA or other forms of alopecia • Treatment with certain topical or systemic AA therapies • Inadequate response to oral JAKis after ≥12 weeks of treatment	• Other causes of alopecia • Clinically significant depression • Auditory conditions • History of disseminated herpes simplex/zoster or recurrent herpes zoster, or adolescent patients without documented varicella-zoster virus vaccination	• History of hormonally driven AA • Recent treatment with medications that could impact hair regrowth or immune response
Primary efficacy endpoint	Percentage of patients with a SALT score ≤20 at Week 36	Percentage of patients with a SALT score ≤20 at Week 24	Percentage of patients with a SALT score ≤20 at Week 24
Key secondary efficacy endpoints	• Percentage of patients with a SALT score ≤10 at Week 36 • Percentage of patients with a Scalp Hair Assessment PRO score of 0 or 1 with a ≥2-point decrease from baseline score at Week 36 • Percentage of patients with ClinRO Measure for Eyebrow and Eyelash Hair Loss score of 0 or 1 with a ≥2-point decrease from baseline score at Week 36	• Percentage of patients with a SALT score ≤10 at Week 24 • Percentage of patients with PGI-C of moderately or greatly improved at Week 24 • Percentage of patients achieving eyebrow and eyelash regrowth through Week 48	• Percentage of patients with a SALT score ≤10 at Week 24 • Percentage of patients who responded "very satisfied" or "satisfied" on the SPRO question at Week 24 • Percentage of patients with a SALT score ≤20 at Weeks 8, 12, 16, and 20
Safety assessments	AEs Clinical laboratory tests Vital signs TEAEs	AEs Audiological testing Neurological events reviewed	AEs Clinical laboratory tests Physical examinations Concomitant medications

[a]Date ranges indicate the study start dates and primary completion dates.
AA, alopecia areata; AE, adverse event; ClinRO, clinician-reported outcome; JAKi, Janus kinase inhibitor; PGI-C, Patient Global Impression of Change; PRO, patient-reported outcome; SALT, Severity of Alopecia Tool; SPRO, satisfaction of hair patient-reported outcome; TEAE, treatment-emergent adverse event.

≤20 at week 24 (*P*<0.0001 for 200-mg loading dose plus 50-mg maintenance dose [200 to 50 mg] and ritlecitinib 50 mg vs placebo; Figure 3), with response rates increasing through week 48.[25] Additionally, significantly greater percentages of patients treated with ritlecitinib 50 mg vs placebo achieved a SALT score ≤10 (200 to 50 mg, *P*<0.0001; 50 mg, *P*=0.0003); obtained a response of moderately or greatly improved from baseline in the Patient Global Impression of Change (*P*<0.0001) at week 24, with improvements through week 48; and experienced eyelash and eyebrow regrowth over 48 weeks of treatment.[25] Ritlecitinib had similar efficacy in adult vs adolescent patients and in patients with AT vs AU.[25] With up to 24 months of treatment, 45.5% of patients achieved a SALT score ≤20.[29] Through week 24, the frequency and severity of AEs and serious AEs were similar across treatment groups; most AEs were mild or moderate in severity (Figure 4).[30]

Deuruxolitinib

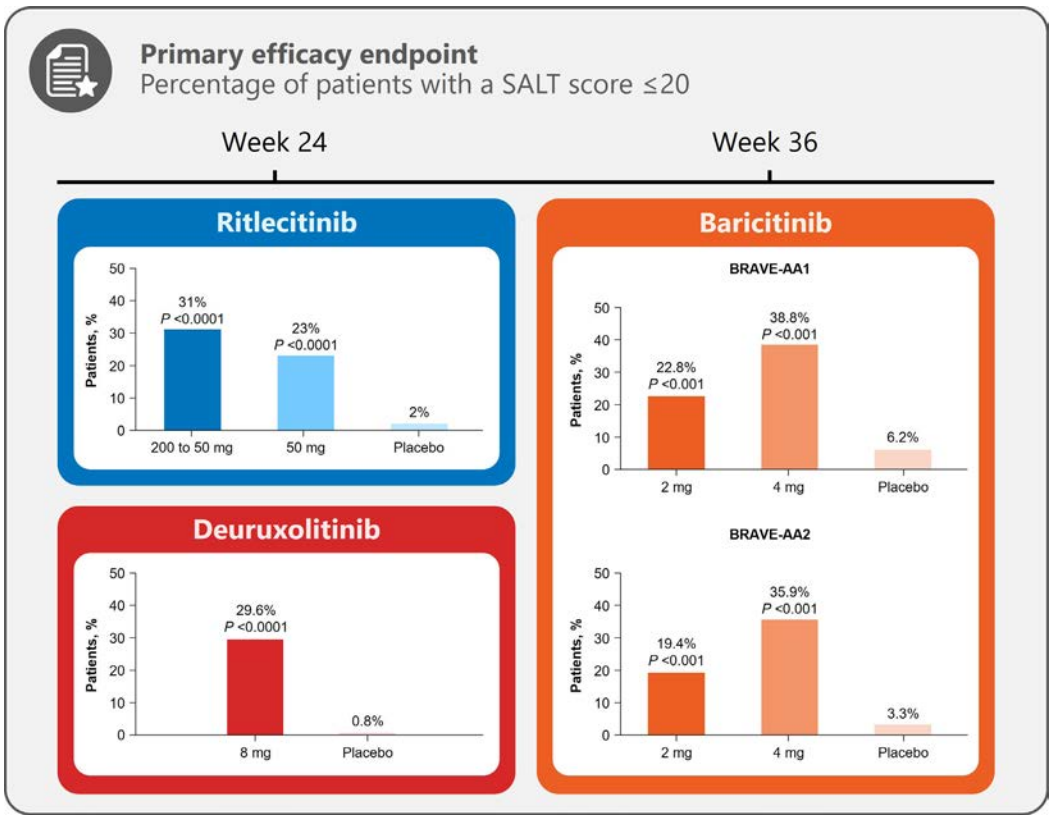
Deuruxolitinib efficacy and safety were evaluated in the double-blind, randomized, placebo-controlled THRIVE-AA1 (NCT04518995)[24] and THRIVE-AA2 (NCT04797650; submitted) studies of patients with severe AA who received 8 mg or 12 mg of twice-daily (BID) oral deuruxolitinib or placebo for 24 weeks (additional trial details in Figure 2). Deuruxolitinib 8 mg BID is approved for the treatment of adults with severe AA; deuruxolitinib 12 mg BID is not approved by the FDA.[21]

A significantly greater percentage of patients treated with deuruxolitinib 8 mg vs placebo achieved a SALT score ≤20 at week 24 (*P*<0.0001; Figure 3); a SALT score ≤20 at weeks 8 (*P*≤0.001), 12, 16, and 20 (*P*≤0.0001); and a SALT score ≤10 at week 24 (*P*≤0.0001).[24] A significantly greater percentage of patients receiving deuruxolitinib 8 mg vs placebo also reported being "very satisfied" or "satisfied" with their scalp hair in the satisfaction of hair PRO question at week 24, an improvement from baseline (*P*<0.001).[24] At week 24, the frequency and severity of AEs were similar across treatment arms, and most were mild or moderate in severity (Figure 4).[24]

Combination Therapies

In clinical settings, patients with severe AA may be treated with JAKis in combination with another AA therapy to obtain maximum therapeutic benefit (Figure 5). Though limited, evidence from smaller studies suggests combination therapies may reduce the time needed to achieve hair regrowth. In one case series, combination therapy of baricitinib 4 mg and low-dose prednisone resulted in complete hair regrowth by 3 months in 7 of 8 patients with very severe AA.[31] In a retrospective records review of 12 consecutive patients with severe AA using combined oral minoxidil with tofacitinib, 7 of 12 patients achieved a SALT score ≤20 by 9 months.[32]

FIGURE 3. Primary efficacy results from baricitinib, ritlecitinib, and deuruxolitinib clinical trials.



SALT, Severity of Alopecia Tool.

Expectations for Patients

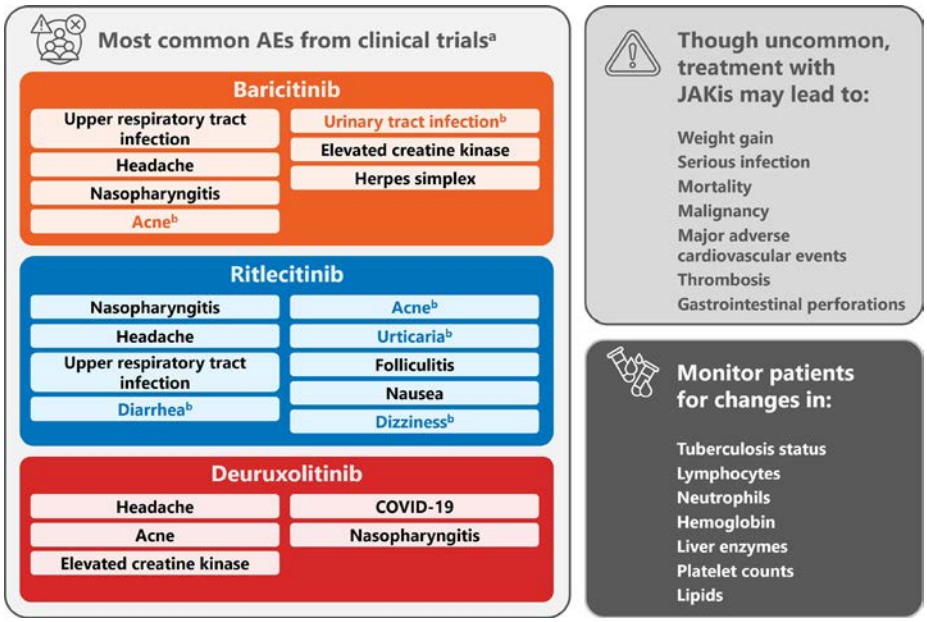
Efficacy for hair regrowth

Predictors of treatment success for patients with AA treated with JAKis include baseline disease severity and duration of current AA episode, with more severe disease and longer disease episodes associated with delayed or reduced efficacy (Figure 5).[29,33,34] Among patients receiving baricitinib up to 52 weeks, those with very severe AA had delayed and reduced response rates vs those with severe AA.[33,35] At week 24, among patients receiving ritlecitinib, those with AT or AU had lower response rates than those with partial hair loss.[25] Additionally, patients achieving a SALT score ≤20 at week 24 had less severe AA at baseline, less eyelash and eyebrow involvement, and shorter episode durations vs patients achieving a SALT score ≤20 by month 12 or 24 of treatment.[29] Similarly, for deuruxolitinib treatment, a greater percentage of patients with partial hair loss vs complete or near-complete hair loss achieved a SALT score ≤20 at week 24.[24] Notably, patients who did not achieve clinically meaningful scalp hair regrowth with baricitinib treatment still achieved eyebrow and eyelash regrowth.[17]

Impacts on HRQoL

Hair regrowth is associated with improvements in HRQoL and psychological symptoms (Figure 5). Patients with severe AA treated with ritlecitinib not only had decreased SALT scores after 24 weeks, but also experienced significant improvements in the Alopecia Areata Symptom Impact Scale.[36] Similarly, improvements in anxiety, depression, and QoL with 52-week baricitinib treatment were directly related to the amount of hair regrowth achieved.[17]

FIGURE 4. Safety considerations and monitoring of patients with alopecia areata using JAKis.



[a]AEs occurring in ≥5% of patients on approved doses of JAKis are presented from most to least common. Data from BRAVE-AA1 and BRAVE-AA2 (baricitinib), ALLEGRO (ritlecitinib), and THRIVE-AA1 (deuruxolitinib).
[b]Occurred more frequently in JAKi vs placebo treatment arms.
AE, adverse event; COVID-19, coronavirus disease 2019; JAKi, Janus kinase inhibitor.

FIGURE 5. Expectations for patients with alopecia areata treated with JAKis.

AA, alopecia areata; JAKi, Janus kinase inhibitor.

Maintenance and risk of relapse

Continuous treatment with JAKis is likely required to maintain hair regrowth, as relapse is common (Figure 5). A withdrawal period from ritlecitinib and baricitinib was associated with loss of regrown hair and worsening SALT scores, and efficacy of ritlecitinib in the 24-week retreatment period was lower than the initial 24-week treatment period.[37,38] Relapse is in part dependent on disease duration, with longer episodes associated with greater likelihood of relapse.[38] However, with continuous baricitinib treatment, approximately 90% of patients maintained clinically meaningful hair regrowth through 152 weeks.[38]

Common and Rare Side Effects and Patient Monitoring

Across baricitinib, ritlecitinib, and deuruxolitinib clinical trials, most AEs were mild or moderate in severity; the most common AEs are presented in Figure 4.[23,24,30] No changes in vital signs, except increased weight gain, were observed.[24,28] However, reduced hemoglobin, neutrophils, and lymphocytes; elevated low- and high-density lipoprotein and creatine kinase; and increased and decreased platelet counts were observed; most changes were not clinically meaningful.[23-25] Thus, laboratory tests are required before and during JAKi treatment, including latent tuberculosis tests.[20-22]

While most AEs from clinical trials were mild or moderate, serious infections, mortality, malignancy, major adverse cardiovascular events, thrombosis, and gastrointestinal perforations are potential rare side effects of JAKi treatment listed as boxed warnings.[20-22] After 36 weeks of baricitinib treatment or 24 weeks of ritlecitinib or deuruxolitinib treatment, herpes zoster infection (baricitinib: 2 mg, 4/338 patients; 4 mg, 5/513; ritlecitinib: 200 to 50 mg, 1/131 patients; 50 mg, 5/130; deuruxolitinib: 8 mg, 1/350 patients), serious infections (baricitinib: 2 mg, 2/155 patients; 4 mg, 1/233; ritlecitinib: 200 to 50 mg, 2/131 patients; deuruxolitinib: 8 mg, 1/350 patients), cardiovascular events (baricitinib: 2 mg, 1/183 patients; ritlecitinib: 50 mg, 1/130 patients), and malignancies (baricitinib: 4 mg, 1/233 patients; ritlecitinib: 200 to 50 mg, 1/131 patients; 50 mg, 1/130) occurred infrequently.[23-25] Over 104 weeks of baricitinib and 24 months of ritlecitinib treatment, most AEs remained mild or moderate in severity; only 1 case of deep vein thrombosis (baricitinib) and 1 death (ritlecitinib; unrelated to study drug) occurred over the longer treatment durations.[27,39] However, since the chronic nature of AA suggests that patients could receive long-term JAKi treatment, continued long-term efficacy and safety studies are warranted.

CONCLUSION

In summary, JAKis are effective treatments for patients with severe AA, which causes patchy hair loss and a significant psychosocial burden for patients. Depending on the disease severity and AA episode duration, patients may experience variable responses to JAKis and will likely require long-term treatment to prevent relapse, which may increase the risk of experiencing a rare but serious AE. However, data from Phase 3 studies of baricitinib, ritlecitinib, and deuruxolitinib demonstrate that treatment for 24 or 36 weeks improves not only hair regrowth but also QoL; thus, JAKis are beneficial treatments for patients with severe AA.

DISCLOSURES

DD serves as a speaker and/or is an advisory board member for AbbVie, Amgen, Arcutis Biotherapeutics, Beiersdorf, Dermavant Sciences, Galderma, Johnson & Johnson, LEO Pharma, Lilly, Novartis, Sanofi/Regeneron, Takeda, and UCB. KK serves as a speaker and/or is an advisory board member for AbbVie, Amgen, Arcutis Biotherapeutics, Beiersdorf, Bristol Myers Squibb, Castle Biosciences, Ferndale, Galderma, Janssen, Johnson & Johnson, LEO Pharma, Novartis, Pfizer, Sun Pharma, Takeda, and UCB.

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