



CLINICAL GUIDELINES OPEN ACCESS

Systemic Treatment of Moderate to Severe Alopecia Areata in Adults: Updated Australian Expert Consensus Statement

Daniella Kushnir-Grinbaum^{1,2} | Laita Bokhari³ | John Frewen¹ | Anthony Moussa¹ | Daranporn Triwongwanat^{1,4} | Ragini Ghiya¹ | Flavia Rodrigues Dias¹ | Shin Shen Yong¹ | Bevin Bhojrul¹ | Zeyad Dabbagh¹ | Ahmed Kazmi^{1,5,6} | Adam Daunton^{1,7} | Jane Li^{8,9,10,11} | Leona Yip¹² | Vivian Lai¹³ | Katherine York^{14,15} | William Cranwell¹⁶ | Dmitri Wall^{17,18,19,20} | Samantha Eisman¹ | Rodney Sinclair¹

¹Sinclair Dermatology, East Melbourne, Victoria, Australia | ²Department of Dermatology, Emek Medical Centre, Afula, Israel, affiliated with the Ruth and Bruce Rappaport Faculty of Medicine of the Technion Institute of Technology, Haifa, Israel | ³Sinclair Direct, East Melbourne, Victoria, Australia | ⁴Department of Dermatology, Siriraj Hospital, Mahidol University, Bangkok, Thailand | ⁵Fairfield Dermatology Clinic, Melbourne, Victoria, Australia | ⁶University of Western Australia, Perth, Western Australia, Australia | ⁷Clinical & Experimental Dermatology, Oxford, United Kingdom | ⁸Skin Health Institute, Melbourne, Victoria, Australia | ⁹Department of Medicine (Dermatology), St Vincent's Hospital Melbourne, The University of Melbourne, Melbourne, Victoria, Australia | ¹⁰Department of Dermatology, Eastern Health, Melbourne, Victoria, Australia | ¹¹Box Hill Dermatology, Melbourne, Victoria, Australia | ¹²Skin Partners, Brisbane, Queensland, Australia | ¹³Royal Children's Hospital, Melbourne, Victoria, Australia | ¹⁴Sunshine Coast University Hospital, Birtinya, Queensland, Australia | ¹⁵Buderim Skin Centre, Sunshine Coast, Queensland, Australia | ¹⁶Dermatology Clinics Australia, Newstead, Queensland, Australia | ¹⁷Hair Restoration Blackrock, Dublin, Ireland | ¹⁸Charles Institute of Dermatology, UCD School of Medicine, University College Dublin, Dublin, Ireland | ¹⁹Department of Dermatology, Mater Misericordiae University Hospital, Dublin, Ireland | ²⁰National and International Skin Registry Solutions, University College Dublin, Dublin, Ireland

Correspondence: Daniella Kushnir-Grinbaum (daniella.kushnir-grinbaum@sinclairdermatology.com.au)

Received: 26 July 2025 | **Revised:** 26 July 2025 | **Accepted:** 28 August 2025

Keywords: alopecia areata | alopecia totalis | alopecia universalis | baricitinib | hair loss | Janus kinase inhibitors | ritlecitinib

ABSTRACT

Over 5000 patients are newly diagnosed with Alopecia areata (AA) in Australia each year. AA severity varies from a single small patch to complete loss of scalp hair, body hair including eyelashes and eyebrows. Approximately 40% of affected individuals experience only a single patch and achieve spontaneous, complete and durable remission within 6 months (acute AA). A further 27% develop additional patches but still attain complete remission within 12 months (chronic AA). Chronic persistent AA (CPAA) is defined by an episode duration of > 12 months and occurs in approximately 33% of patients. Without systemic treatment, 55% of individuals with CPAA will have persistent multifocal relapsing and remitting disease, 30% will progress to alopecia totalis (AT) and 15% will ultimately develop alopecia universalis (AU). The physical disfigurement, unpredictable course, social isolation and rejection contribute to the psychological distress attributable to AA. A wide range of topical, intralesional and systemic agents used to treat AA were evaluated in the 2018 Australian expert consensus statement. In 2020, the international Alopecia Areata Consensus of Experts (ACE) publication stated that if reimbursed, Janus Kinase inhibitors (JAKi's) would be an ideal systemic treatment for adults with AA. TGA approval of baricitinib in 2023 and ritlecitinib in 2024 for severe AA is the first step on the pathway for these systemic medications to be reimbursed on the Australian Government Pharmaceutical Benefits Scheme (PBS). Reimbursement would significantly transform the Australian therapeutic landscape for AA. The purpose of this 2025 Update on the Australian Expert Consensus Statement on the treatment of chronic, moderate to severe AA is to augment the 2018 treatment algorithm to include these TGA-approved medications and to address indications for initiation, continuation and dose titration of systemic JAKi treatment, appropriate choice of agent, satisfactory outcome measures and to provide guidance on when to discontinue successful or unsuccessful treatment.

Abbreviations: AA, alopecia areata; AT, alopecia totalis; AU, alopecia universalis; CPAA, chronic persistent alopecia areata.

This is an open access article under the terms of the [Creative Commons Attribution-NonCommercial-NoDerivs](https://creativecommons.org/licenses/by-nc-nd/4.0/) License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

© 2025 The Author(s). *Australasian Journal of Dermatology* published by John Wiley & Sons Australia, Ltd on behalf of Australasian College of Dermatologists.

1 | Introduction

Alopecia areata (AA) is an organ-specific autoimmune disease with a characteristic histology. Acute, intense peribulbar lymphocytic inflammation of anagen follicles initiates apoptosis of terminal hair bulb epithelium leading to hair fibre breakage (anagen effluvium) and premature catagen. Chronic lesions show an increased ratio of catagen and telogen follicles and follicular miniaturisation. Premature catagen, prolonged telogen and repeated failure to re-establish anagen lead to prolonged baldness.

Until recently there had been no new Therapeutic Goods Administration (TGA) approved treatment for AA in over 50 years. In May 2023, Baricitinib, a Janus kinase (JAK)1/2 inhibitor, was approved under the trade name *Olumiant* for the treatment of severe AA. In July 2024, Ritlecitinib, a JAK3/TEC kinase inhibitor, was approved under the trade name *Litfulo* [1, 2]. Also in 2024, a third agent, deuruxolitinib, was approved by the U.S. Food and Drug Administration (FDA). Although deuruxolitinib is not yet approved by the TGA, its FDA approval underscores the growing global momentum toward systemic JAK inhibitor therapy for AA. Its potential future availability in Australia may further expand treatment options and inform updates to national treatment guidelines. Inclusion of ritlecitinib and baricitinib on the PBS would represent a critical step toward equitable access for Australians living with severe AA and pave the way for potential future reimbursement of emerging agents such as deuruxolitinib. Currently, several other treatments are in phase 2 or phase 3 development.

Treatment of chronic, severe AA with ritlecitinib and baricitinib results in meaningful hair regrowth in over two-thirds of patients [3, 4]. With ongoing treatment, approximately 90% of responders maintain their response out to 152 weeks [5]. Most patients who respond within the first 8–24 weeks go on to achieve a SALT score < 20 by 52 weeks, whereas fewer than 10% of late responders reach this threshold [6].

Response rates to JAKi are comparable in patients with baseline SALT scores ranging from 20 to 95; however, outcomes are significantly poorer in patients with AT and in those with an episode duration greater than 4 years [7, 8].

These approvals materially alter the therapeutic landscape for AA and necessitate an update of the 2018 Australian expert consensus guidelines [] to support dermatologists managing patients with AA, who must carefully weigh the safety and efficacy of long-term JAKi therapy against the negative impact of delayed treatment on long-term prognosis.

1.1 | Burden of Disease

The global prevalence of AA is 2% [9]. In Australia, the estimated incidence of AA is 0.28 per 1000 patient-years, with the peak age of onset occurring between 19 and 34 years. The point prevalence of AA in 2020 was estimated at 0.13% (1.26 per 1000 individuals) [10].

Amongst patients with newly diagnosed AA, 17% were prescribed antidepressant and 9% anxiolytic medication. This is more than double the national average for the prescription of these therapies

[10]. Individuals with AA frequent their primary care provider more than matched controls, particularly women, rural dwellers and those of lower socioeconomic status. The proportion of individuals with AA referred to secondary care has increased over time from 19.4% in 2009 to 27.9% in 2017 [11]. Significant anxiety, stress and a substantial negative impact on self-perception and quality of life is prevalent amongst these individuals. Psychosocial impact is greatest amongst individuals with SALT score 50%–94%, and stigma is linearly correlated with AA severity [12].

Patients' self-perception of disease severity and involvement of eyebrows/eyelash loss have been identified as the strongest predictors of diminished quality of life rather than physician-recorded SALT scores [13, 14]. Although quality of life impact assessment in AA can be established using the Dermatology Life Quality Index [15] and specific quality of life questionnaires for use in AA have been developed, there remains no standardised health-related quality of life (HRQoL) outcome measure for use in AA [15].

1.2 | Aetiology and Pathogenesis

AA pathogenesis involves interferon-gamma mediated collapse of hair follicle immune privilege. Immune privilege is characterised by down-regulation of classical human leukocyte antigen (HLA) expression and up-regulation of non-classical HLA expression on the lower portion of the follicle, from the bulb to the bulge [16].

Hair follicle immune privilege is a requirement for long hair. Ageing hair bulbs increasingly express cell surface antigens such as MICA and ULBP-3 as a marker of senescence. In non-immune privileged tissues, co-expression of these senescent antigens together with classical HLA antigens triggers immune-mediated apoptosis and/or necroptosis [16, 17]. In immune privileged anagen follicles, apoptosis is not triggered in senescent matrix cells and catagen is prevented. The lack of cell surface expression of HLA-C and B (combined with the up-regulation of HLA E, F and G) antigens masks senescent antigens and prevents them from triggering immune-mediated apoptosis and necroptosis. Collapse of immune privilege, with cell surface expression of classical HLA-A, B and C and loss of HLA-E, F and G antigens unmasks previously hidden senescent antigens to antigen presenting T cells (APC) [16, 18–19].

APCs recruit and activate CD8+ NKG2D+ effector T cells that induce apoptosis in follicle outer root sheath cells, resulting in anagen arrest and the initiation of catagen within the proband hair follicle [20, 21].

Local factors transmit immune privilege collapse and T cell mediated anagen arrest to neighbouring follicles, producing circular patches of alopecia. The subsequent appearance of secondary patches of AA suggests systemic as well as local factors are involved in patients with multifocal disease. The demonstration of an altered inflammasome in non-lesional hair bulbs using RT-PCR further supports the role of systemic factors in the pathogenesis of AA [22, 23].

Genetic aetiology is conferred as a complex polygenic trait. A genome-wide association study identified 139 single nucleotide

polymorphisms from eight genomic regions that are significantly associated with AA. Each of these genomic regions contains several genes controlling: the activation and proliferation of regulatory T cells (Treg cells); cytotoxic T-lymphocyte-associated antigen 4 (CTLA4); interleukin (IL)-2/IL-21, IL-2 receptor A (IL-2RA; CD25); Eos (also known as Ikaros family zinc finger 4; IKZF4); HLA; PRDX5 and STX17 genes expressed in the hair follicle itself; as well as ULBP (cytomegalovirus UL16-binding protein) gene cluster on chromosome 6q25 that encodes activating ligands of the natural killer cell receptor NKG2D [24].

Although AA was previously considered a Th1-driven disease, there is increasing recognition that Th2 cytokine activation has a role in the disease [25]. Highlighting that the underlying mechanisms of AA are not yet fully understood.

1.3 | Natural History

Non-scarring hair loss most commonly involves the scalp and face but can affect any terminal hair follicle. Episode duration is the dominant predictor of the natural history of AA [26] with longer episodes being associated with a lower likelihood of spontaneous regrowth. Approximately two-thirds of affected individuals experience acute, self-limiting AA, whereas the remaining one-third develop CPAA. About 40% of episodes present as a single patch that undergoes spontaneous, complete and durable remission within 6 months. A further 27% of episodes involve secondary patches that appear sequentially but still achieve complete and durable remission within 12 months. Fewer than 5% of patients develop acute multifocal and/or diffuse AA that progresses rapidly over weeks to months to AT or AU. CPAA, defined by an episode duration >12 months, occurs in approximately 33% of patients. Consistent with the placebo response rate below 3% observed in phase 3 clinical trials, spontaneous remission in CPAA is rare, and nearly 50% of cases ultimately progress to AT or AU [27–29].

1.4 | Currently Available Treatments

Chronic persistent AA and moderate to severe AA generally require systemic therapy [30]. Systemic corticosteroids are only suitable for short-term use due to accumulated toxicity. A number of systemic non-steroidal anti-inflammatory medications are used off-label in combinations with systemic steroids as steroid-sparing agents. Their efficacy in this context is uncertain, and with the exception of ciclosporin, none have been investigated in a high-quality study as a monotherapy. In a placebo-controlled, randomised, double-blind trial investigating ciclosporin 4 mg/kg in patients with moderate to severe AA, 31.3% of patients achieved a 50% reduction in SALT score (SALT₅₀) and 6.3% of patients achieved 100% reduction in SALT score (SALT₁₀₀) at 12 weeks [26–29].

The cost per cure is a metric used to compare the price of different treatments. It takes into account the cost of that treatment and the number of patients who need to be treated for 1 patient to achieve a positive treatment outcome.

The PBS dispensed price for thirty ciclosporin 100 mg capsules is \$373.80 [31, 32]. According to the Australian Bureau of Statistics in 2017–2018, the average weight for Australian men was 87 kg and Australian women 72 kg [33]. For an 87 kg man, the estimated monthly cost of ciclosporin at 4 mg/kg is \$4329.15 per SALT50 response and \$31,674.41 per SALT100 response.

In comparison, the dispensed price for twenty-eight baricitinib 4 mg tablets is \$1046.01. The published SALT ≤20 response is 51.9% at 36 weeks. The cost per ‘cure’ (defined as SALT ≤20) with baricitinib for severe AA is \$2695.87. The published SALT ≤20 response rate to ritlecitinib 50 mg daily is 43% at 48 weeks [34]. The cost of ritlecitinib in Australia has not yet been established. The cost of 30 ritlecitinib capsules based on the United Kingdom National Health Service is £949.41, which is approximately \$1981.00 AUD [35]. The approximate cost per ‘cure’ for ritlecitinib 50 mg is \$2693.87 per month at current exchange rates. Although most ciclosporin responders eventually lose response, most responders to ritlecitinib sustain clinical response at 24 months with treatment [36, 37].

1.5 | Definition of Moderate to Severe Alopecia Areata

The Severity of Alopecia Tool (SALT) is the standard metric used to quantify the extent of hair loss in phase 2 and phase 3 clinical trials of AA [15]. A SALT score of 0 indicates no scalp hair loss, whereas a score of 100 represents complete loss of scalp hair [38, 39]. Subscript numerals are used to denote percentage improvement from baseline. For example, SALT₅₀ reflects a 50% reduction in hair loss compared to baseline.

The Alopecia Areata Investigator Global Assessment scale (AA-IGA) (Table 1) uses SALT as a singular measure to define disease severity. Specifically, a SALT score from 1 to 20 corresponds to limited disease; 21–49 to moderate disease; 50–94 to severe disease; and 95–100 to very severe disease (Table 1) [40]. These severity thresholds have been endorsed by a global expert consensus statement [26].

There is expert consensus that AA patients with moderate to very severe disease are candidates for systemic therapy, and that systemic corticosteroids reduce the risk of disease progression and disease relapse [30, 41–42].

In the pivotal phase 3 trials for patients with a baseline SALT >50 treated with either baricitinib or ritlecitinib, the primary outcome measure that was used to define a favourable treatment outcome was a SALT score of ≤20 [40].

Various visual aids are available to facilitate calculation of the SALT score. The original aid created by Olsen et al. allows clinicians to map the area of hair loss in each quadrant. It is most useful for patients with moderate to very severe disease. The Sinclair SALT seven score card (Figure 1) is a transparent business card with markings representing SALT scores of 0.125, 1, 2, 3 and 7, which can be placed on the scalp. It is particularly useful for rapid scoring of patients with limited or moderate disease [43].

TABLE 1 | The Alopecia Areata Investigator Global Assessment scale.

	None	Limited	Moderate	Severe	Very severe
	0	1	2	3	4
Extent of scalp hair loss (%) ^a	0	1–20	21–49	50–94	95–100

^aSALT is recommended to assess the extent of hair loss [40].

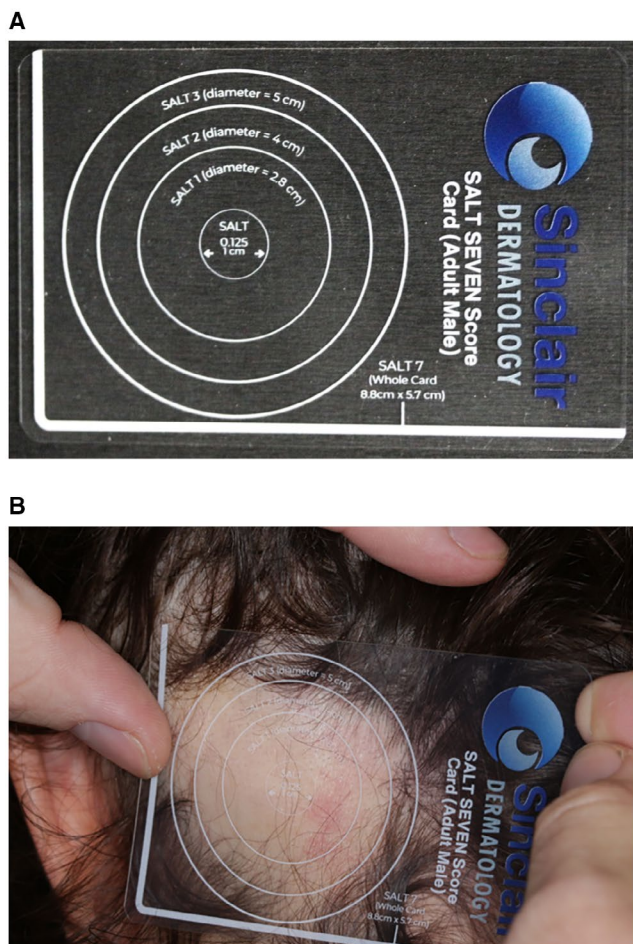


FIGURE 1 | (A) SALT Seven Score Card. (B) Demonstration of the SALT SEVEN score card being used in clinical practice (Score = 2).

2 | Methods

An initial exercise on reviewing current international and Australian guidelines for management of severe alopecia areata was conducted as well as a review of current literature. The grade of each therapy type was determined based on current data availability and assessed using the National Health and Medical Research Council (NHMRC) Body of Evidence Matrix (Table 2) [44]. Subsequently, a summary of treatment types was categorised according to the NHMRC grading (Table 3).

Following this exercise, discussions with local Australian dermatologists with specific expertise in treating AA were held, and key areas of need were identified.

An updated consensus statement for the management of chronic, moderate-to-severe AA was drafted from these discussions and

TABLE 2 | Definition of NHMRC grades of recommendations.

Grade of recommendation	Description
A	Body of evidence can be trusted to guide practice
B	Body of evidence can be trusted to guide practice in most situations
C	Body of evidence provides some support for recommendation(s) but care should be taken in its application
D	Body of evidence is weak and recommendation must be applied with caution

disseminated to Australian dermatologists with a subspecialty in hair and scalp disorders for input.

Treatment algorithms were developed to guide clinical decision-making in alopecia areata (AA), incorporating current evidence, clinical experience and key disease characteristics.

Two separate algorithms are presented: one for acute AA (disease duration <6 months) (Figure 2A) and one for chronic or chronic persistent AA (Figure 2B). Both provide a stepwise approach to treatment, considering disease severity, duration and prior treatment response.

3 | JAK Inhibitors (Level of Evidence A)

JAK inhibitors (JAKi), which block intracellular signalling of multiple pro-inflammatory cytokines including interferon- γ and interleukins involved in T-cell activation, have demonstrated promising therapeutic potential in the treatment of AA. In a 2023 systematic review and meta-analysis of seven randomised clinical trials with 1710 patients, JAK inhibitors were associated with patients achieving a 30%, 50% or 90% improvement in SALT scores from baseline compared with placebo. Results from notable large RCTs are outlined below.

3.1 | Baricitinib

Baricitinib is a selective inhibitor of JAK1 and 2 that is approved by the Therapeutic Goods Administration (TGA) for the treatment of severe and very severe alopecia areata in adults [45]. It

TABLE 3 | Summary of therapeutic type and grade of recommendation per NHMRC definition.

Therapy type	Grade of recommendation
Systemic therapy	
Baricitinib	Level A
Ritlecitinib	Level A
Prednisolone	Level B
Tofacitinib	Level C
Ciclosporin	Level C
Methotrexate	Level C
Contact immunotherapy	Level C
Phototherapy	Level D
Azathioprine	Level D
Sulfasalazine	Level D
Topical and adjunctive therapy	
Intralesional triamcinolone	Level B
Topical corticosteroids	Level B
Topical prostaglandin analogues	Level C
Oral/Sublingual minoxidil	Level C
Topical minoxidil	Level C
Topical JAKis	Level D
Topical calcineurin inhibitors	Level D
Platelet rich plasma (PRP)	Level D

is also TGA approved for the treatment of atopic dermatitis and rheumatoid arthritis [46].

In severe AA, Baricitinib is administered orally at a dosage of 2 mg once daily, which can be increased to 4 mg once daily in case of inadequate response. In very severe AA, therapy should be initiated at a dosage of 4 mg once daily, reduced to 2 mg once daily when an adequate response has been achieved [46].

3.1.1 | Efficacy

In the BRAVE-AA1 and AA2 Phase 3 randomised clinical trials, 36.8%–40.9% of participants with severe or very severe AA treated with baricitinib 4 mg and 21.2%–24.4% of those treated with baricitinib 2 mg achieved a SALT score of ≤ 20 at 52 weeks. With continued treatment, that response (SALT score of ≤ 20) was maintained through to 152 weeks in 89.1% of participants treated with 4 mg daily and 83.6% of those who received 2 mg daily. A SALT score ≤ 10 was achieved by 27.8%–29.9% of participants treated with 4 mg and 14.1%–16.7% of those treated with 2 mg at 52 weeks [3, 5, 47].

Outcome measures continued to improve after 18 and 26 months of treatment implying that long-term therapy could be especially important for patients with extensive disease at baseline [47]. A recent real-world study found 61.5% (59 out of 96 patients) achieved a SALT score of ≤ 20 at the end of a 52-week treatment period [48].

3.2 | Ritlecitinib

Ritlecitinib is a selective inhibitor of Janus kinase 3 (JAK3) and the tyrosine kinase expressed in hepatocellular carcinoma (TEC) family of kinases. It inhibits cytokine-induced phosphorylation of signal transducer and activator of transcription (STAT) mediated by JAK3-dependent cytokines, as well as blocking signalling of B and T-cell receptors dependent on TEC kinase family members [49].

Ritlecitinib was approved by the TGA for the treatment of severe AA in June 2024 in adults and adolescents 12 years and older. Ritlecitinib is administered orally at a dosage of 50 mg once daily [2, 50].

3.2.1 | Efficacy

An integrated analysis of the ALLEGRO Phase 2b/3 and long-term Phase 3 studies found 45.1% of participants who received ritlecitinib 50 mg and 45.9% of those receiving 200 mg/50 mg (200 mg daily loading dose for 4 weeks followed by 50 mg daily dose) achieved a SALT score of ≤ 20 at Year 1. Additionally, a SALT score of ≤ 10 was achieved by 34.2% of participants on 50 mg and 39.4% on the 200 mg/50 mg regimen in the first year [4]. A higher proportion of participants achieved a SALT score of ≤ 20 by Year 2.

Participants with AT or AU or with longer episodes of severe disease, showed lower response rates. These findings suggest that earlier initiation of treatment may result in better outcomes [34, 51].

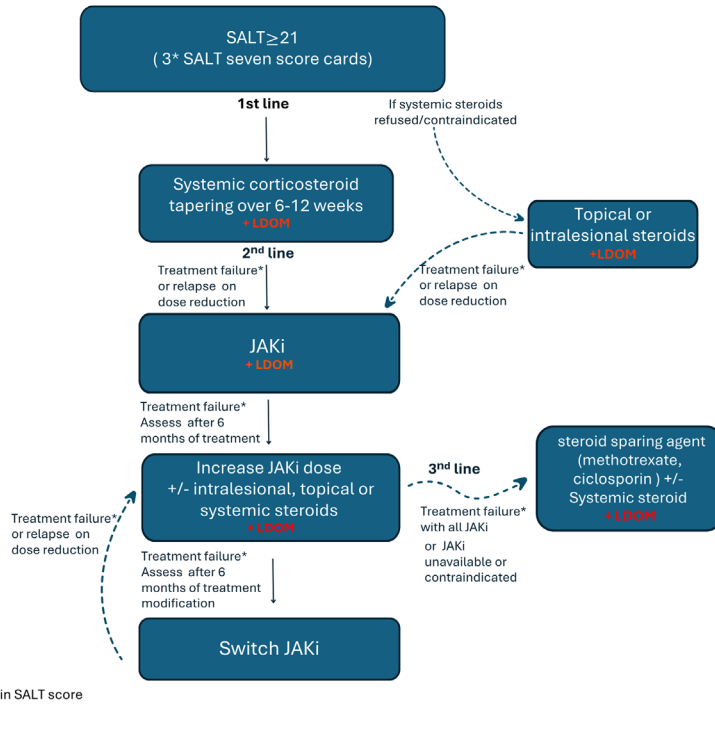
A recent systematic review evaluated the efficacy and safety of ritlecitinib versus baricitinib, showing comparable efficacy of ritlecitinib 50 mg daily and baricitinib 4 mg [52]. A comparison of side effects and laboratory investigation alterations reported with baricitinib and ritlecitinib in AA patients is described in Table 4.

3.3 | Assessment of Response to JAKi Treatment

Response to treatment is defined in current studies as achieving a SALT ≤ 20 with a secondary objective of achieving SALT ≤ 10 . Response to treatment can also be evaluated by percentage of change from baseline. AT and AU patients might perceive achieving eyebrow, eyelash and beard regrowth as a reasonable treatment response despite the SALT score remaining unchanged. The area of scalp involvement, whether it be easy to conceal by parting hair a certain way or through adopting a different hairstyle, can significantly influence patient satisfaction. This cosmetic impact may determine whether a patient feels content or distressed, regardless of their SALT score.

A

Treatment algorithm Moderate, severe and very severe acute alopecia areata



B

Treatment algorithm Moderate, severe and very severe chronic and chronic persistent alopecia areata

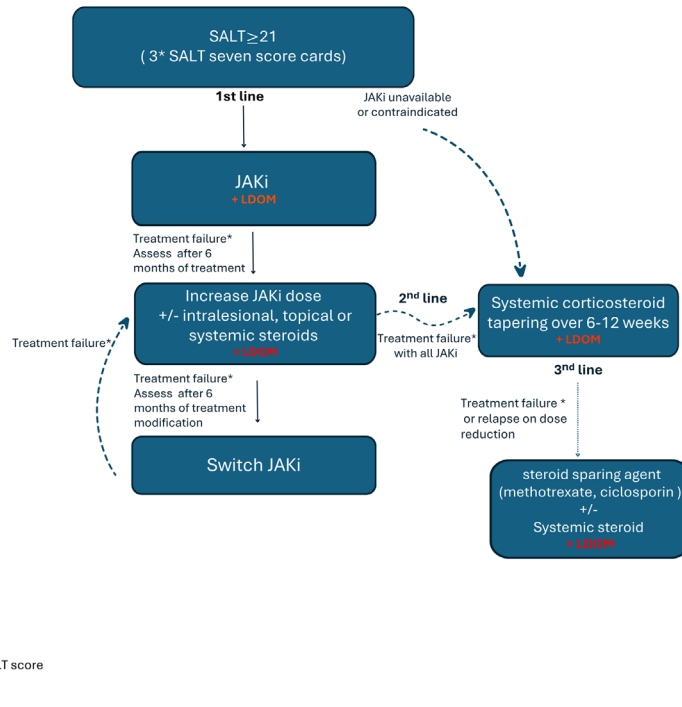


FIGURE 2 | (A) Treatment algorithm for moderate, severe and very severe acute alopecia areata. Solid arrows indicate the preferred treatment sequence. Dashed arrows indicate alternative treatment pathways or modifications based on clinical response or contraindications. (B) Treatment algorithm for moderate, severe and very severe chronic and chronic persistent alopecia areata. Solid arrows indicate the preferred treatment sequence. Dashed arrows indicate alternative treatment pathways or modifications based on clinical response or contraindications.

TABLE 4 | Side effects reported with baricitinib and ritlecitinib in alopecia areata patients [47].

Reported percentage of side effects (%)	Baricitinib (N= 1303)	Ritlecitinib (N= 1294)
Adverse events occurring in $\geq 5\%$ of patients ^a		
Nasopharyngitis	6.8%	12.4%
Acne	6.7%	10.4%
Headache	8.2%	17.7%
Upper respiratory tract infection	10.9%	10.2%
COVID-19	11%	15.5%
Urticaria	NR	6.8%
Urinary tract infection	6.1%	NR
Pyrexia	NR	7.6%
Cough	NR	7.4%
Fatigue	NR	7%
Adverse events of special interest		
Herpes Zoster	3.4%	1.5%
GI perforation	0.1% ^d	NR
Sensorineural hearing loss	NR	1.1%
MACE	0.1%	0.2%
Malignancy	0.3% ^e	0.5% ^b
Opportunistic infection	0.1%	<0.1%
Death	NR	0.2% ^c
DVT/PE	0.2%	<0.1%
Laboratory abnormalities		
Platelets		
> 600 $\times 10^9/L$	0.8%	NR
Anaemia		
Hg < 8 g/dL	0.2%	<0.1%
Neutropenia		
1000 to < 1500/mm ³	NR	5.1%
< 1000/mm ³	1.6%	0.8%
Lymphopenia		
< 500/mm ³	0.3%	2.2%
Blood creatine phosphokinase increase		
> 5*ULN	6.6%	5.9%
HDL cholesterol	53.3% (< 1.55 mmol/L)	0.6% (< 0.8*LLN)
LDL cholesterol	36.8% (> 3.36 mmol/L)	1.6% (> 1.2*ULN)
Triglycerides	1% (> 5.7 mmol/L)	8.2% (> 2.2 mmol/L)
ALT		
> 3*ULN	3.7%	2.5%
AST		
> 3*ULN	2.9%	2.3%

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LLN, lower limit of normal; MACE, major adverse cardiovascular events; NR, not reported; ULN, upper limit of normal.

^aWith one or both treatments.

^bIn the ritlecitinib study, four cases of breast cancer, one testicular cancer, one papillary thyroid cancer and one adjudicated malignant melanoma were reported. Time to onset of the event ranged from 68 to 299 days (median 156 days) after starting ritlecitinib.

^cTwo reported deaths during the study period that were determined by the investigator to be unrelated to the study treatment (breast cancer and acute respiratory failure/cardiopulmonary arrest).

^dOne patient diagnosed with perforated appendicitis, later categorised as a gastrointestinal perforation. The patient underwent appendectomy, recovered and remained in the trial.

^eOne case of chronic lymphocytic leukaemia, one case of B-cell lymphoma, one case of breast cancer and one case of melanoma in situ were reported.

3.4 | Effect of Episode Duration on Response to Treatment

Nearly 50% of patients with patchy alopecia areata persisting for over a year (CPAA) progress to develop AT or AU and have an estimated 0% chance of spontaneous recovery [29]. Longer disease duration confers a reduced response to treatment with JAKi, though the response to treatment is seen even in patients with a disease episode of over 4 years. The duration of the AA episode does not appear to affect the speed of response to treatment [6].

3.5 | When to Stop or Titrate Down Treatment

A study evaluating relapse rates following treatment withdrawal in patients who achieved a SALT score of ≤ 20 with baricitinib ($n = 40$) and maintained their response for 1 year found that 20% of patients sustained their response 3 years after discontinuation. Upon retreatment, most patients regained clinical response. Notably, patients who remained in remission for 3 years had a shorter current episode duration and shorter disease duration [5].

Similarly, for patients who were successfully treated with ritlecitinib for 6 months, defined as a 30% improvement in SALT score ($n = 22$), 18% (4/22) of patients maintained response 28 weeks post-discontinuation [53].

As approximately one-fifth of patients remain in remission after treatment withdrawal, discontinuation of JAK inhibitor therapy may be considered after 6–12 months of treatment in patients who have achieved their target response. Alternatively, gradual dose tapering can be considered where feasible. The decision should be made in discussion with the patient, ensuring they are fully informed of the potential consequences of continuing, stopping or reducing the dose.

3.5.1 | Interclass Switching

Treatment failure with one JAKi does not necessarily predict treatment failure with another. Emerging evidence has shown that patients that failed to respond to the pan JAKi, tofacitinib, may respond to treatment with baricitinib or ruxolitinib. These findings support the feasibility of interclass switching and suggest that trialling a different JAKi may be a reasonable strategy in non-responders [51, 52, 54].

4 | Guidelines for Treatment Initiation, Monitoring and Management With JAK Inhibitors

Prior to initiating JAK inhibitor (JAKi) therapy, patients should be assessed for risk factors that may increase the likelihood of morbidity or mortality with JAKi therapy (see Table 5). Although the only absolute contraindications in current drug prescribing information to JAKis are hypersensitivity to the drug or its excipients and active serious or opportunistic infections, other clinical conditions may warrant caution or even avoidance of

TABLE 5 | Recommended pretreatment evaluation [55, 56].

Patients at increased risk for morbidity or mortality
Age ≥ 50 with at least one cardiovascular risk factor ^a
History of PE, VTE, retinal artery occlusion, stroke
Current or past long-time smoking
Patient undergoing surgery or immobilisation
History of malignancy ^b —consider type, stage, duration and prior treatment

^aCurrent smoker, high-density lipoprotein level < 40 mg/dL, history of hypertension, diabetes, myocardial infarction or coronary heart disease.

^bExcluding resolved non-melanoma skin cancer.

TABLE 6 | Contraindications [46, 57].

Contraindications
Serious active infection ^a
Hypersensitivity to therapeutic agent ^a
Active malignancy (not including resolved non-melanoma skin cancer)
Low platelet count $< 100,000$ cells/mm ³
Haemoglobin < 8 g/dL
Absolute neutrophil count < 1000 cells/mm ³
Lymphocyte count < 500 cells/mm ³
Therapy with interacting medication
History of thromboembolism or pulmonary embolism
Pregnancy or intent to become pregnant ^{a,b}
Lactating women ^a
eGFR < 30 mL/min/1.73m ^{2a} ($30 < \text{eGFR} < 60$ mL/min/1.73 m ² requires dose adjustment) (baricitinib)
Severe hepatic impairment (Child-Pugh C, ritlecitinib)
Immunosuppressed patient

Note: Some contraindications listed above are relative, some absolute.

^aAbsolute.

^bWomen of childbearing potential should take appropriate precautions to avoid becoming pregnant during treatment with baricitinib and for at least 1 week after the final treatment and at least 1 month for ritlecitinib.

treatment (Table 6). Although not explicitly listed as absolute contraindications in the current prescribing information, their presence should prompt a careful individualised risk–benefit assessment. Recommended laboratory testing before and during JAKi therapy is outlined in Table 7.

4.1 | Vaccinations

Live vaccines are generally contraindicated immediately before and after treatment with JAKi, and if indicated should be received at least 1 month before starting immunosuppressive therapy [59].

TABLE 7 | Laboratory testing [46, 57–58].

Test	Prior to initiating treatment	During treatment
FBC	+	Within 1 month and then every 3 months
CMP	+	Within 1 month and then every 3 months
Hepatitis B and C virus serologic panel	+	
HIV serology	Consider	
Lipid profile	+	Within 1 month and then every 3 months (baricitinib)
PPD/QuantiFERON-TB Gold assay and (In immune-suppressed or history of tuberculosis) chest-X ray	+	Be alert for symptoms such as chronic cough, weight loss, fever or night sweats and consider further evaluation if these occur during treatment

Abbreviations: CMP, complete metabolic profile; FBC, full blood count; PPD, purified protein derivative (Mantoux test).

Completion of all age-appropriate immunisation is recommended prior to treatment initiation, as well as:

- Recombinant herpes zoster vaccine
- Influenza vaccine
- Pneumococcal vaccine
- COVID-19 vaccine

For vaccines given as a series (Shingrix), the first dose should ideally be given before starting therapy to ensure the best response, unless this would cause a significant delay in the start of a necessary treatment. All non-live vaccinations recommended prior to treatment can be given safely during therapy if necessary. Administration of adjuvanted recombinant zoster vaccine in rheumatoid arthritis patients treated with JAKi resulted in significant increases in anti-varicella-zoster virus immunoglobulin G titers [59–62].

4.2 | Indications for Interruption of Treatment

Treatment should be interrupted and restarted when values/status return to normal [46, 57].

- Absolute lymphocyte count $< 500/\text{mm}^3$
- Haemoglobin $< 8\text{g/dL}$
- Absolute neutrophil count $< 1000\text{ cells}/\text{mm}^3$
- Serious infection or opportunistic infection
- Liver function tests show elevations of ≥ 3 times the upper limit of normal
- In case of herpes zoster infection developing while on treatment, temporary interruption of treatment may be considered

Treatment should be discontinued:

- Platelet count $< 50,000/\text{mm}^3$

4.3 | AA Flare-Up During Treatment

During treatment, some patients may experience disease flare-up with new onset of discrete alopecic patches. Such cases can be managed by increasing the dose of JAKi if on baricitinib 2mg daily dose (increase to 4mg daily). Intralesional steroid injections if limited involvement, or in cases of extensive exacerbation, initiation of a short course of concomitant systemic corticosteroids, with gradual tapering.

4.4 | Adjuvant Minoxidil Therapy

A synergistic effect has been demonstrated when incorporating minoxidil with JAKi treatment [63, 64]. A recent systematic review, found increased efficacy when combining oral minoxidil with JAKi (85%) [65].

5 | Systemic Corticosteroids (Level of Evidence B)

Prednisolone or similar systemic corticosteroids are currently considered first-line treatment for acute moderate to very severe AA.

Treatment can be administered orally, intramuscularly or intravenously as pulse therapy [66–69]. A trial of systemic steroids can be considered for chronic moderate to very severe AA. Relapse on dose reduction or cessation of treatment is common; however, severe short-term and cumulative toxicity preclude their use for more than 3 months.

Therapeutic protocols and effectiveness vary across studies and there is no evidence to clearly rank the various systemic steroid agents or therapeutic protocols.

In the Alopecia Areata Consensus of Experts (ACE) study, prednisolone was the preferred systemic corticosteroid, with once daily mane administration [41] starting with a dose of 0.4–0.6mg/kg/day with gradual tapering off over 6–12 weeks.

Pre-screening investigations for untreated infections should be considered to minimise the steroid-related complications. Patients on treatment should be monitored regularly for sleep and mood disturbance, diabetes, hypertension, peptic ulcer disease, ocular toxicity and acute infections. Prophylaxis against osteopenia should also be considered.

6 | Ciclosporin (Level of Evidence C)

To date, there is only one RCT that has investigated the efficacy of ciclosporin (CSA) monotherapy in AA [31]. In this double-blind, randomised, placebo-controlled study, 32 patients (18–65 years) with moderate–severe AA were randomised to receive either CSA (4 mg/kg/day) or placebo for 3 months. Although the proportion of patients achieving a 50% reduction in the SALT score was higher in the ciclosporin group (31.3%) compared to the placebo group (6.3%), the results were not statistically significant ($p = 0.07$).

A 2021 meta-analysis assessing the therapeutic efficacy of CSA in AA incorporated seven studies evaluating CSA monotherapy and eight studies examining combination with systemic corticosteroids. The authors' assessment was that CSA exhibits a beneficial therapeutic impact on AA. The overall mean proportion of responders, defined as terminal hair growth exceeding 50%, was 73% (95% CI, 57%–85%), with a mean recurrence rate of 39% (95% CI, 10%–78%).

The mean response rate for CSA monotherapy was 66% (95% CI, 50%–79%), whereas combination therapy with corticosteroids showed a response rate of 78% (95% CI, 48%–93%). Recurrence rates were 55% (95% CI, 6%–96%) for monotherapy and 28% (95% CI, 6%–72%) for combination therapy [70].

In the ACE study, it was agreed that CSA is an effective monotherapy agent in AA, with a recommended dose of 3–5 mg/kg/day for a maximum treatment duration of 6–12 months [41]. CSA, however, is not TGA-approved for the treatment of AA and based on the current evidence, its use as a first-line therapy is not recommended by this group. Where other first-line therapies are not available, or contraindicated, it can be used as a second-line (off-label) treatment, either as a monotherapy or in combination with systemic steroids as a steroid-sparing agent in patients who respond initially to systemic steroids but relapse on dose reduction.

7 | Methotrexate (Level of Evidence C)

There have been no randomised, placebo-controlled studies investigating methotrexate (MTX) in AA. In view of the significant rate of spontaneous remission seen in patients with acute and chronic AA, it cannot be assumed that MTX is superior to placebo. Nevertheless, a 2019 meta-analysis of 16 studies concluded that MTX was an effective treatment for AA [71].

The overall rate of complete hair regrowth with MTX was 35.8% (95% CI, 25.0%–48.3%). Subgroup analysis revealed that the pooled complete response rate in adults was 44.7% (95% CI, 32.9%–57.1%), significantly higher than the 11.6% (95% CI,

5.1%–24.5%) observed in the paediatric population ($p = 0.001$). A recent 2023 double-blinded RCT of 89 patients with chronic and refractory AT and AU evaluated the efficacy and tolerance of MTX, both as monotherapy (25 mg/week) and in combination with low-dose prednisone (20 mg/day for 3 months, then 15 mg/day for 3 months) [72]. At the end of the 12-month period, the authors concluded that while MTX alone generally resulted in partial hair regrowth in patients with chronic AT or AU, whereas combining it with low-dose prednisone led to complete hair regrowth in up to 31% of patients.

In the ACE study, it was agreed that the recommended MTX dose for the treatment of AA is 15–20 mg once weekly [41].

8 | Azathioprine (Level of Evidence D)

An open-label RCT involving 50 patients with AA (SALT score ≥ 10) compared 300 mg weekly azathioprine (AZA) pulse therapy (WAP) to 5 mg betamethasone on 2 consecutive days every week (mini-pulse therapy) for 4 months [73]. The authors concluded both treatments to be effective for AA, with 44.52% of patients who received WAP therapy demonstrating hair regrowth.

In a Cochrane analysis, these results were of low certainty [74]. AZA has also been used with success in the paediatric AA population; however, its benefits must be considered against its potential risks [75].

9 | Topical Therapy

Topical therapy can be used as monotherapy or as an adjunctive treatment.

9.1 | Topical and Intralesional Steroid Therapy

Topical corticosteroids (TCS) are used in cases of limited area of involvement. They can be used as monotherapy or as an adjunctive treatment [76]. High-potency TCS have been shown to be more efficacious than low-potency formulations [77]. Intralesional triamcinolone acetonide (ILK) is a well-established treatment for limited-type AA, with response rates ranging from 60% to 95% across 12 studies [78]. A systematic review and meta-analysis identified that the optimal regimen for the administration of ILK is at 3–4 weekly intervals for up to 6 months, at concentrations of 2.5–10 mg/mL for the scalp and 2.5–5 mg/mL for the face [79].

Potential side effects of TCS and ILK include folliculitis, atrophy, striae, telangiectasia and acneiform eruptions [80].

9.2 | Contact Immunotherapy

Topical contact immunotherapy with diphenylcyclopropane or squaric acid dibutyl ester can promote hair regrowth in patients with AA. A meta-analysis of 45 studies ($n = 22,227$) found that 65% of patients showed some improvement, with

32.3% achieving complete regrowth with a 49% recurrence rate. However, most studies were not randomised, placebo-controlled trials, which limit the strength of the evidence [81].

As the mechanism of action relies on inducing allergic contact dermatitis, side effects are common. These include local erythema, eczematous reactions (e.g., blistering, scaling, exudation), pruritus, regional lymphadenopathy, generalised eczema, hyperpigmentation and flu-like symptoms [76, 81].

9.3 | Topical Calcineurin Inhibitors

Topical calcineurin inhibitors are not considered a first-line treatment for AA at present, as there is no evidence of clinical benefit [82–84].

9.4 | Topical JAK Inhibitors

Topical JAK inhibitors are a potential new treatment for AA with limited effectiveness [85]. A study conducted by Bokhari et al. involved applying 2% tofacitinib, 1% ruxolitinib, 0.005% topical clobetasol dipropionate and a placebo to the four scalp quadrants of 16 patients with AU for 28 weeks. Partial hair regrowth was observed in the tofacitinib (6/16), ruxolitinib (5/16) and clobetasol (10/16) groups, but not in the placebo group (0/16) [86].

In an open-label, single-arm pilot study, Liu et al. assessed the efficacy and safety of topical tofacitinib in 10 patients with AA. Regrowth occurred in 3 patients (significant regrowth in 1 patient with 61% regrowth and partial regrowth of 35% in 2 patients), whereas seven patients experienced no regrowth [87]. Topical ruxolitinib and delgocitinib were investigated with unsatisfactory results [88, 89]. We currently do not advocate using topical JAKi for scalp AA.

9.5 | Topical Minoxidil

Topical minoxidil can induce accelerated hair regrowth in alopecia patches and reduce disease activity in AA cases by lowering perifollicular lymphocytic infiltration [90]. The most common side effect is skin irritation [76, 91]. Topical minoxidil has inconclusive evidence to support its use as an adjunct therapy in AA [41].

9.6 | Topical Prostaglandin Analogues

Several studies have explored the efficacy of bimatoprost and latanoprost for scalp alopecia areata, with mixed results [92, 93]. Improved efficacy was seen when combined with topical betamethasone or clobetasol [94, 95].

10 | Emerging Treatments for AA

Several JAK inhibitors are in phase 1, 2 and 3 clinical trials for chronic severe and very severe AA. Notable agents under

investigation include tofacitinib, brepocitinib, deuruxolitinib, ruxolitinib, deucravacitinib, upadacitinib, jaktinib and ivarmacitinib.

Tofacitinib, a pan-JAK inhibitor, is used in both oral and sublingual preparations for AA. A meta-analysis showed a 54% significant hair regrowth rate, but a high relapse rate after cessation [96]. Oral tofacitinib is given at a twice-daily dose. Sublingual tofacitinib offers pharmacokinetic benefits and a longer half-life, potentially allowing once-daily dosing [97].

Brepocitinib, a selective TYK2 and JAK1 inhibitor, was evaluated against ritlecitinib in a phase 2a RCT with 142 participants [98]. At 24 weeks, 60% of brepocitinib users and 50% of ritlecitinib users achieved a 30% improvement in SALT scores, versus 2% in the placebo group. In addition, 81% reported eyelash regrowth and 72% observed improvements in eyebrows.

Ruxolitinib, a selective reversible JAK1/2 inhibitor, has shown effectiveness in treating moderate-to-severe AA across various case series [99, 100]. In an open-label study of 75 patients, both ruxolitinib and tofacitinib achieved similar rates of complete remission (21%). However, 84.2% of patients on ruxolitinib reached SALT50 or better, versus 78.4% on tofacitinib [99].

Deuruxolitinib, a deuterated form of ruxolitinib, approved by the FDA since July 2024 for treatment of severe AA, demonstrated significant improvements in SALT scores in phase 3 studies. At 24 weeks, 42% of patients in the 12 mg group achieved a SALT score of <20 [101].

Upadacitinib, a JAK1 inhibitor, is being evaluated in a phase 3 trial in adults and adolescents [96]. Data from case reports and retrospective cohort studies suggest upadacitinib is effective in treating AA, particularly in patients with concurrent atopic dermatitis [102–104]. One study involving 25 patients demonstrated a median SALT score improving from 50 to 5 after 24 weeks of treatment [105]. Another study with 19 patients showed that 57.1% achieved complete hair regrowth at 28 weeks [106].

Jaktinib, a novel JAK1/2 inhibitor previously evaluated for myelofibrosis, is in a phase 3 RCT for severe AA [107].

Ivarmacitinib, a JAK1 inhibitor, has shown promising outcomes in a phase 2 RCT involving adults with moderate-to-severe AA [108]. After 24 weeks, the percentage reduction in SALT score from baseline was 19% in the placebo group compared to 56% in patients receiving 4 mg ivarmacitinib daily.

Filgotinib is a JAKi with high selectivity for JAK-1. It is approved by the FDA and EMA (European Medicines Agency) for the treatment of rheumatoid arthritis and has also been investigated for use in Crohn's disease. Complete scalp hair regrowth was reported in a patient with severe alopecia areata who received filgotinib as part of a clinical trial evaluating its efficacy in Crohn's disease [109].

Deucravacitinib, a TYK2 inhibitor, approved by the TGA for the treatment of psoriasis, is undergoing phase 2 clinical trials to assess its effectiveness in treating AA [110].

10.1 | Biologics

Dupilumab, an IL-4R α blocker approved for atopic dermatitis, may exert therapeutic effects in alopecia areata through modulating the Th2-pathway signalling [23]. In a phase 2a RCT involving 60 patients (23 patients with AD), 32.5% achieved SALT30, 22.5% SALT50 and 15% SALT75 by week 48. A significant correlation was observed between elevated baseline IgE levels and response to treatment [111]. In contrast, there are also case reports suggesting that dupilumab may paradoxically induce or exacerbate AA in certain individuals [112–114].

Tralokinumab, an IL-13 inhibitor, has shown effectiveness in a case study in mild to moderate AA patients with AD treatment [115].

Several immunomodulating agents are in phase 2 trials for AA, including daxdilimab (IL7 target), rosnilimab (PD-1 agonist), etrasimod (SJP receptor modulator), EQ101 (tri-specific IL-2, IL-9, IL-15 antagonist) and bempikibart (IL-7/TSLP receptor antagonist) [116–120].

Apremilast, a PDE-4 inhibitor that reduces IFN- γ activity, has been described in the treatment of AA; however, case series in which its use in AA has been described have revealed inconsistent effectiveness [121–125].

10.2 | Off Label Indications for Systemic Treatment—Facial Alopecia Areata

Baricitinib, ritlecitinib and brepocitinib have shown efficacy in promoting eyebrow and eyelash regrowth through phase 2–3 RCTs [34, 47, 126]. Tofacitinib has also shown effectiveness in a large case series [127]. Interestingly, the duration of eyebrow and eyelash loss did not significantly impact treatment outcomes, indicating these hair follicles may remain viable even after extended periods of inactivity. Topical tofacitinib and ruxolitinib have shown promise in smaller studies [128–131]. An eDelphi study found that systemic therapy is warranted for AA affecting eyebrows or eyelashes if it leads to functional or occupational impairment [26]. Beard alopecia areata (BAA) can have profound psychosocial effects [132]. Baricitinib, tofacitinib and ruxolitinib have been used successfully in case reports and small case series, though their efficacy in BAA has not been studied in RCTs [127, 132–133].

Prostaglandin analogues including bimatoprost and latanoprost have been shown to induce eyelash regrowth in several studies [93, 94, 134–138].

10.3 | Patient Registries

Given the need to understand the safety profile of emerging therapies and better describe the real-world effectiveness of existing and emerging mono and combination treatment regimens, the group strongly advocates for the registration of patients in high-quality patient registries. This concept has been supported internationally and has led to the development of the Global Registry of Alopecia Areata Disease Severity and Treatment

Safety (GRASS) [139, 140]. The pilot phase of this project has been led by the Australasian Hair and Wool Research Society and National and International Registry Solutions (NISR), an Irish charity, in Australia over the past 3 years, but has also established sites in Ireland and Italy, with multiple other international centres in the process of joining to provide comparable, harmonised data that will facilitate the development of valuable networks to help gather and interpret data from the use of therapies in AA.

11 | Conclusion

Guidelines for the management of moderate to very severe alopecia areata in paediatric and adolescent populations are needed; however, this was beyond the scope of the current work.

Given the accumulating evidence demonstrating the limited efficacy, higher cost and unfavourable side effect profiles of immunosuppressants such as MTX, CSA and AZA, we recommend reserving these treatments for cases in which systemic treatment is warranted but JAKi are contraindicated. Emerging data suggest that early initiation of treatment is associated with improved response rates and prognosis. Therefore, JAKi should be considered for patients with a SALT score of 21 and higher corresponding with moderate-to-severe severity. In cases of eyelash or eyebrow involvement, initiation of JAKi therapy may also be appropriate at lower SALT scores.

Conflicts of Interest

Dr. Kushnir-Grinbaum has served as an investigator for AbbVie, Eli Lilly, Merck, Pfizer, DermAbio, Sanofi, Novartis, Eli Lilly, Samson. She has received honoraria for professional services from AbbVie and Eli Lilly. Dr. Yip has been a consultant and/or advisory board member for L'oreal, Galderma, Eli Lilly, Pfizer, LEO Pharma, CryoMed. Dr. Wall reports receiving honoraria for consultancy from Bristol Myers Squibb, Eli Lilly, Pfizer, AbbVie and Sun Pharma; has received speaker fees from Almirall, Lilly and L'Oreal; and received support from AbbVie to attend conferences. He is a shareholder in Samson Clinical. He is an employee of National and International Skin Registry Solutions (NISR) and a director of Hair Restoration Blackrock. He is a steering committee member of the Global Registry of Alopecia Areata Disease Severity and Treatment Safety (GRASS) International and PI of GRASS Ireland.

Dr. Eisman is a member of the Australian Dermatology Advisory Board of Eli Lilly and is a principal investigator in clinical trials for Pfizer Inc., AbbVie, Arena Pharmaceuticals, Boston Pharmaceuticals, Bristol-Myers Squibb, Botanic, Dermira, Eli Lilly and Company, LEO Pharma, Novartis, Regeneron, Tigermed, Suzhou Connect Pharmaceuticals, Janssen, Kymab Ltd., Evelo Biosciences, KoBiolabs and Avance Clinical. Prof. Sinclair has been an investigator for and/or provided professional services to AbbVie, Aerotek Scientific, Akeso Biopharma, Amgen, Arcutis, Arena Pharmaceuticals, Ascend Laboratories, AstraZeneca, Bayer Pharmaceuticals, Boehringer Ingelheim, Bristol Myers Squibb, Celgene Co-herus BioSciences, Connect Biopharma, Cutanea, Dermira, Eli Lilly and Company, Galderma, GlaxoSmithKline, Janssen, LEO Pharma, MedImmune/AstraZeneca, Merck Sharp & Dohme, Novartis, On-cobiologics, Pfizer, Regeneron, Reistone Biopharma, Roche, Samson Medical Technologies, Sanofi, Sun Pharma and UCB Pharma.

Data Availability Statement

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

References

1. "Baricitinib Approved by the Therapeutic Goods Administration of Australia for the Treatment of Alopecia Areata [Internet]." (2023), <https://www.tga.gov.au/resources/prescription-medicines-registrations/olumiant-eli-lilly-australia-pty-ltd-1>.
2. "Ritlecinib Approved by the Therapeutic Goods Administration of Australia for the Treatment of Alopecia Areata [Internet]." (2024), <https://www.tga.gov.au/resources/auspmd/litfulo-ritlecitinib>.
3. O. Kwon, M. M. Senna, R. Sinclair, et al., "Efficacy and Safety of Baricitinib in Patients With Severe Alopecia Areata Over 52 Weeks of Continuous Therapy in Two Phase III Trials (BRAVE-AA1 and BRAVE-AA2)," *American Journal of Clinical Dermatology* 24, no. 3 (2023): 443–451.
4. M. Piliang, J. Soung, B. King, et al., "Efficacy and Safety of the Oral Janus Kinase 3/Tyrosine Kinase Expressed in Hepatocellular Carcinoma Family Kinase Inhibitor Ritlecitinib Over 24 Months: Integrated Analysis of the ALLEGRO Phase IIb/III and Long-Term Phase III Clinical Studies in Alopecia Areata," *British Journal of Dermatology* 192, no. 2 (2025): 215–227.
5. B. King, J. Ko, O. Kwon, et al., "Baricitinib Withdrawal and Retreatment in Patients With Severe Alopecia Areata: The BRAVE-AA1 Randomized Clinical Trial," *JAMA Dermatology* 160, no. 10 (2024): 1075–1081.
6. B. King, J. Shapiro, M. Ohya, et al., "When to Expect Scalp Hair Regrowth During Treatment of Severe Alopecia Areata With Baricitinib: Insights From Trajectories Analyses of Patients Enrolled in Two Phase III Trials," *British Journal of Dermatology* 189, no. 6 (2023): 666–673.
7. J. M. Ko, T. T. Mayo, W. F. Bergfeld, et al., "Clinical Outcomes for Uptitration of Baricitinib Therapy in Patients With Severe Alopecia Areata: A Pooled Analysis of the BRAVE-AA1 and BRAVE-AA2 Trials," *JAMA Dermatology* 159, no. 9 (2023): 970–976.
8. B. Burroway, J. Griggs, and A. Tosti, "Alopecia Totalis and Universalis Long-Term Outcomes: A Review," *Journal of the European Academy of Dermatology and Venereology* 34, no. 4 (2020): 709–715.
9. H. H. Lee, E. Gwillim, K. R. Patel, et al., "Epidemiology of Alopecia Areata, Ophiasis, Totalis, and Universalis: A Systematic Review and Meta-Analysis," *Journal of the American Academy of Dermatology* 82, no. 3 (2020): 675–682.
10. R. Sinclair, S. Eisman, W. Song, et al., "Incidence and Prevalence of Alopecia Areata in the Australian Primary Care Setting: A Retrospective Analysis of Electronic Health Record Data," *Australasian Journal of Dermatology* 64, no. 3 (2023): 330–338.
11. M. Harries, A. E. Macbeth, S. Holmes, et al., "The Epidemiology of Alopecia Areata: A Population-Based Cohort Study in UK Primary Care*," *British Journal of Dermatology* 186, no. 2 (2022): 257–265.
12. N. Mesinkovska, B. Craiglow, S. G. Ball, et al., "The Invisible Impact of a Visible Disease: Psychosocial Impact of Alopecia Areata," *Dermatology and Therapy* 13, no. 7 (2023): 1503–1515.
13. M. Senna, J. Ko, M. Glashofer, et al., "Predictors of QOL in Patients With Alopecia Areata," *Journal of Investigative Dermatology* 142, no. 10 (2022): 2646–2650.
14. D. Wall, H. Rees, L. Bokhari, N. Meah, K. York, and R. Sinclair, "Signposts to the Promised Land in Alopecia Areata," *Journal of Investigative Dermatology* 143, no. 1 (2023): 9–10.
15. N. Meah, D. Wall, K. York, et al., "The Alopecia Areata Consensus of Experts (ACE) Study Part II: Results of an International Expert Opinion on Diagnosis and Laboratory Evaluation for Alopecia Areata," *Journal of the American Academy of Dermatology* 84, no. 6 (2021): 1594–1601.
16. M. Bertolini, K. McElwee, A. Gilhar, S. Bulfone-Paus, and R. Paus, "Hair Follicle Immune Privilege and Its Collapse in Alopecia Areata," *Experimental Dermatology* 29, no. 8 (2020): 703–725.
17. R. Paus and M. Bertolini, "The Role of Hair Follicle Immune Privilege Collapse in Alopecia Areata: Status and Perspectives," *Journal of Investigative Dermatology. Symposium Proceedings* 16, no. 1 (2013): S25–S27.
18. T. Ito, N. Ito, A. Bettermann, Y. Tokura, M. Takigawa, and R. Paus, "Collapse and Restoration of MHC Class-I-Dependent Immune Privilege," *American Journal of Pathology* 164, no. 2 (2004): 623–634.
19. F. Morandi and V. Pistoia, "Interactions Between HLA-G and HLA-E in Physiological and Pathological Conditions," *Frontiers in Immunology* 5 (2014): 394.
20. A. G. Messenger, D. N. Slater, and S. S. Bleehen, "Alopecia Areata: Alterations in the Hair Growth Cycle and Correlation With the Follicular Pathology," *British Journal of Dermatology* 114, no. 3 (1986): 337–347.
21. A. Gilhar, A. Etzioni, and R. Paus, "Alopecia Areata," *New England Journal of Medicine* 366, no. 16 (2012): 1515–1525.
22. J. M. Shin, D. K. Choi, K. C. Sohn, et al., "Induction of Alopecia Areata in C3H/HeJ Mice Using Polyinosinic-Polycytidylic Acid (Poly[I:C]) and Interferon-Gamma," *Scientific Reports* 8, no. 1 (2018): 12518.
23. I. Chim, R. Ghiya, R. D. Sinclair, and S. Eisman, "Novel Investigational Drugs for Alopecia Areata and Future Perspectives," *Expert Opinion on Investigational Drugs* 33 (2024): 441–449.
24. L. Petukhova, M. Duvic, M. Hordinsky, et al., "Genome-Wide Association Study in Alopecia Areata Implicates Both Innate and Adaptive Immunity," *Nature* 466, no. 7302 (2010): 113–117.
25. M. Suárez-Fariñas, B. Ungar, S. Noda, et al., "Alopecia Areata Profiling Shows TH1, TH2, and IL-23 Cytokine Activation Without Parallel TH17/TH22 Skewing," *Journal of Allergy and Clinical Immunology* 136, no. 5 (2015): 1277–1287.
26. ASAMI Consensus Survey Study Group, A. Moussa, M. Bennett, et al., "The Alopecia Areata Severity and Morbidity Index (ASAMI) Study: Results From a Global Expert Consensus Exercise on Determinants of Alopecia Areata Severity," *JAMA Dermatology* 160, no. 3 (2024): 341.
27. D. M. Peterson, B. G. Craiglow, N. A. Mesinkovska, J. Ko, M. M. Senna, and B. A. King, "It Is All Alopecia Areata: It Is Time to Abandon the Terms Alopecia Totalis and Alopecia Universalis," *Journal of the American Academy of Dermatology* 87, no. 5 (2022): e149–e151.
28. W. C. Cranwell, V. W. Lai, L. Photiou, et al., "Treatment of Alopecia Areata: An Australian Expert Consensus Statement," *Australasian Journal of Dermatology* 60, no. 2 (2019): 163–170.
29. T. Ikeda, "A New Classification of Alopecia Areata," *Dermatologica* 131, no. 6 (1965): 421–445.
30. L. Rudnicka, M. Arenbergerova, R. Grimalt, et al., "European Expert Consensus Statement on the Systemic Treatment of Alopecia Areata," *Journal of the European Academy of Dermatology and Venereology* 38, no. 4 (2024): 687–694.
31. V. W. Y. Lai, G. Chen, D. Gin, and R. Sinclair, "Cyclosporine for Moderate-To-Severe Alopecia Areata: A Double-Blind, Randomized, Placebo-Controlled Clinical Trial of Efficacy and Safety," *Journal of the American Academy of Dermatology* 81, no. 3 (2019): 694–701.
32. "The Pharmaceutical Benefit Scheme—Ciclosporin," <https://www.pbs.gov.au/medicine/item/13911E-5636P-6354K-8660T>.
33. "National Health Survey 2017–18 Australian Department of Health," <https://www.health.vic.gov.au/your-health-report-of-the-chief-health-officer-victoria-2018/burden-of-disease/overweight-and>.
34. M. Piliang, J. Soung, B. King, et al., "Long-Term Efficacy of Ritlecitinib up to Month 24 From the ALLEGRO Phase 2b/3 and Long-Term Phase 3 Clinical Studies in Alopecia Areata," *Journal of Skin* 8, no. 2 (2024): s394.

35. "United Kingdom National Institute for Health and Care Excellence NICE—Ritlecitinib for Treating Severe Alopecia Areata in People 12 Years and Over," (2024), Efficacy and Safety of Ritlecitinib in Adults and Adolescents With Alopecia Areata.
36. M. Piliang, C. Lynde, B. King, et al., "Sustained Hair Regrowth With Continued Ritlecitinib Treatment Through Week 48 in Patients With Alopecia Areata With or Without Early Target Responses: Post Hoc Analysis of the ALLEGRO Phase 2b/3 Trial," *Journal of the American Academy of Dermatology* 92, no. 2 (2025): 276–284.
37. J. Nowaczyk, K. Makowska, A. Rakowska, M. Sikora, and L. Rudnicka, "Cyclosporine With and Without Systemic Corticosteroids in Treatment of Alopecia Areata: A Systematic Review," *Dermatologic Therapy* 10, no. 3 (2020): 387–399.
38. E. A. Olsen, M. K. Hordinsky, V. H. Price, et al., "Alopecia Areata Investigational Assessment Guidelines—Part II," *Journal of the American Academy of Dermatology* 51, no. 3 (2004): 440–447.
39. E. A. Olsen and D. Canfield, "SALT II: A New Take on the Severity of Alopecia Tool (SALT) for Determining Percentage Scalp Hair Loss," *Journal of the American Academy of Dermatology* 75, no. 6 (2016): 1268–1270.
40. K. W. Wyrwich, H. Kitchen, S. Knight, et al., "The Alopecia Areata Investigator Global Assessment Scale: A Measure for Evaluating Clinically Meaningful Success in Clinical Trials," *British Journal of Dermatology* 183, no. 4 (2020): 702–709.
41. N. Meah, D. Wall, K. York, et al., "The Alopecia Areata Consensus of Experts (ACE) Study: Results of an International Expert Opinion on Treatments for Alopecia Areata," *Journal of the American Academy of Dermatology* 83, no. 1 (2020): 123–130.
42. M. J. Harries, A. Ascott, L. Asfour, et al., "British Association of Dermatologists Living Guideline for Managing People With Alopecia Areata 2024," *British Journal of Dermatology* 192, no. 2 (2025): 190–205.
43. "Hair," *Australasian Journal of Dermatology* 65, no. S1 (2024): 53–59.
44. National Health and Medical Research Council (NHMRC), *NHMRC Additional Levels of Evidence and Grades for Recommendations for Developers of Guidelines* (NHMRC Commonwealth of Australia, 2009).
45. E. Freitas, E. Guttman-Yassky, and T. Torres, "Baricitinib for the Treatment of Alopecia Areata," *Drugs* 83, no. 9 (2023): 761–770.
46. "Baricitinib Product Information Approved by the Australian Public Assessment Reports," <https://www.tga.gov.au/sites/default/files/auspra-r-baricitinib-190321-pi.pdf>.
47. M. Senna, A. Mostaghimi, M. Ohyama, et al., "LONG-TERM Efficacy and Safety of Baricitinib in Patients With Severe Alopecia Areata: 104-WEEK Results From BRAVE-AA1 and BRAVE-AA2," *Journal of the European Academy of Dermatology and Venereology* 38, no. 3 (2024): 583–593.
48. C. A. Vignoli, L. Gargiulo, L. Ibba, et al., "Baricitinib for the Treatment of Severe Alopecia Areata: Results From a 52-Week Multicenter Retrospective Real-World Study," *Journal of Dermatological Treatment* 36, no. 1 (2025): 2444494.
49. J. B. Telliez, M. E. Dowty, L. Wang, et al., "Discovery of a JAK3-Selective Inhibitor: Functional Differentiation of JAK3-Selective Inhibition Over Pan-JAK or JAK1-Selective Inhibition," *ACS Chemical Biology* 11, no. 12 (2016): 3442–3451.
50. B. King, J. Soung, C. Tziotzios, et al., "Integrated Safety Analysis of Ritlecitinib, an Oral JAK3/TEC Family Kinase Inhibitor, for the Treatment of Alopecia Areata from the ALLEGRO Clinical Trial Program," *American Journal of Clinical Dermatology* 25, no. 2 (2024): 299–314, <https://doi.org/10.1007/s40257-024-00846-3>.
51. B. King, X. Zhang, W. G. Harcha, et al., "Efficacy and Safety of Ritlecitinib in Adults and Adolescents With Alopecia Areata: A Randomised, Double-Blind, Multicentre, Phase 2b–3 Trial," *Lancet* 401, no. 10387 (2023): 1518–1529.
52. D. Aceituno, C. G. Fawsitt, G. M. Power, E. Law, S. Vaghela, and H. Thom, "Systematic Review and Indirect Treatment Comparisons of Ritlecitinib Against Baricitinib in Alopecia Areata," *Acta Dermatovenereologica* 39 (2024): 1134–1142.
53. E. Peeva, E. Guttman-Yassky, A. Banerjee, et al., "Maintenance, Withdrawal, and Re-Treatment With Ritlecitinib and Breprocitinib in Patients With Alopecia Areata in a Single-Blind Extension of a Phase 2a Randomized Clinical Trial," *Journal of the American Academy of Dermatology* 87, no. 2 (2022): 390–393.
54. A. Kazmi, A. Moussa, L. Bokhari, et al., "Switching Between Tofacitinib and Baricitinib in Alopecia Areata: A Review of Clinical Response," *Journal of the American Academy of Dermatology* 89, no. 6 (2023): 1248–1250.
55. L. Hoisnard, B. Lebrun-Vignes, S. Maury, et al., "Adverse Events Associated With JAK Inhibitors in 126,815 Reports From the WHO Pharmacovigilance Database," *Scientific Reports* 12, no. 1 (2022): 7140.
56. "Janus Kinase (JAK) Inhibitors: New Measures to Reduce Risks of Major Cardiovascular Events, Malignancy, Venous Thromboembolism, Serious Infections and Increased Mortality," <https://www.gov.uk/drug-safety-update/janus-kinase-jak-inhibitors-new-measures-to-reduce-risks-of-major-cardiovascular-events-malignancy-venous-thromboembolism-serious-infections-and-increased-mortality>.
57. "Litfulo Australian Product Information," <https://www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2024-PI-02188-1&d=20240909172310101&d=20240910172310101>.
58. J. M. Jackson and J. P. Callen, "128—Systemic Immunomodulators," in *Dermatology*, 5th ed., (2024), 2272–2294.
59. Australian Government, Department of Health and Aged care, "Australian Immunization Handbook," <https://immunisationhandbook.health.gov.au/contents/vaccination-for-special-risk-groups/vaccination-for-people-who-are-immunocompromised#inactivated-vaccines-that-are-routinely-recommended-in-people-who-are-immunocompromised>.
60. A. J. Tan, J. L. Streicher, J. F. Merola, and M. H. Noe, "Vaccine Considerations for Adult Dermatology Patients on Immunosuppressive and Immunomodulatory Therapies: A Clinical Review," *Dermatology Online Journal* 27, no. 9 (2021): 10–5070.
61. R. Seror, M. Camus, J. H. Salmon, et al., "Do JAK Inhibitors Affect Immune Response to COVID-19 Vaccination? Data From the MAJIK-SFR Registry," *Lancet Rheumatology* 4, no. 1 (2022): e8–e11.
62. V. Venerito, P. Stefanizzi, L. Cantarini, et al., "Immunogenicity and Safety of Adjuvanted Recombinant Zoster Vaccine in Rheumatoid Arthritis Patients on Anti-Cellular Biologic Agents or JAK Inhibitors: A Prospective Observational Study," *International Journal of Molecular Sciences* 24, no. 8 (2023): 6967.
63. C. G. Wambier, B. G. Craiglow, and B. A. King, "Combination Tofacitinib and Oral Minoxidil Treatment for Severe Alopecia Areata," *Journal of the American Academy of Dermatology* 85, no. 3 (2021): 743–745.
64. D. Dincer, E. Tanacan, and C. Kose Ozkan, "Efficacy of Systemic Minoxidil and Tofacitinib Combination in Treatment-Resistant Alopecia Universalis," *Journal of Cosmetic Dermatology* 20, no. 6 (2021): 1807–1809.
65. R. S. Raval, A. Nohria, D. Desai, et al., "The Use of Minoxidil in the Treatment of Alopecia Areata: A Systematic Review," *Journal of the American Academy of Dermatology* 91 (2024): S0190962224007965.
66. G. A. Bin Saif, M. M. Al-Khawajah, H. M. Al-Otaibi, et al., "Efficacy and Safety of Oral Mega Pulse Methylprednisolone for Severe Therapy Resistant Alopecia Areata," *Saudi Medical Journal* 33, no. 3 (2012): 284–291.
67. V. K. Sharma and S. Gupta, "Twice Weekly 5 Mg Dexamethasone Oral Pulse in the Treatment of Extensive Alopecia Areata," *Journal of Dermatology* 26, no. 9 (1999): 562–565.

68. A. Agarwal, J. Nath, and K. N. Barua, "Twice Weekly 5 Mg Betamethasone Oral Pulse Therapy in the Treatment of Alopecia Areata," *Journal of the European Academy of Dermatology and Venerology* 20, no. 10 (2006): 1375–1376.
69. A. Friedli, M. P. Labarthe, E. Engelhardt, R. Feldmann, D. Salomon, and J. H. Saurat, "Pulse Methylprednisolone Therapy for Severe Alopecia Areata: An Open Prospective Study of 45 Patients," *Journal of the American Academy of Dermatology* 39, no. 4 (1998): 597–602.
70. H. Husein-ElAhmed and M. Steinhoff, "Efficacy and Predictive Factors of Cyclosporine A in Alopecia Areata: A Systematic Review With Meta-Analysis," *Journal of Dermatological Treatment* 33, no. 3 (2022): 1643–1651.
71. K. Phan, V. Ramachandran, and D. F. Sebaratnam, "Methotrexate for Alopecia Areata: A Systematic Review and Meta-Analysis," *Journal of the American Academy of Dermatology* 80, no. 1 (2019): 120–127.
72. P. Joly, A. Lafon, E. Houivet, et al., "Efficacy of Methotrexate Alone vs Methotrexate Plus Low-Dose Prednisone in Patients With Alopecia Areata Totalis or Universalis: A 2-Step Double-Blind Randomized Clinical Trial," *JAMA Dermatology* 159, no. 4 (2023): 403–410.
73. P. Gupta, K. K. Verma, S. Khandpur, and N. Bhari, "Weekly Azathioprine Pulse Versus Betamethasone Oral Mini-Pulse in the Treatment of Moderate-To-Severe Alopecia Areata," *Indian Journal of Dermatology* 64, no. 4 (2019): 292–298.
74. M. Mateos-Haro, M. Novoa-Candia, G. Sánchez Vanegas, et al., "Treatments for Alopecia Areata: A Network Meta-Analysis," *Cochrane Database of Systematic Reviews* 10, no. 10 (2023): CD013719, <https://doi.org/10.1002/14651858.CD013719.pub2>.
75. V. Saoji, S. Kulkarni, and B. Madke, "Alopecia Areata Treated With Oral Azathioprine: A Case Series," *International Journal of Trichology* 11, no. 5 (2019): 219–222.
76. L. C. Strazzulla, E. H. C. Wang, L. Avila, et al., "Alopecia Areata," *Journal of the American Academy of Dermatology* 78, no. 1 (2018): 15–24.
77. P. Lenane, C. Macarthur, P. C. Parkin, et al., "Clobetasol Propionate, 0.05%, vs Hydrocortisone, 1%, for Alopecia Areata in Children: A Randomized Clinical Trial," *JAMA Dermatology* 150, no. 1 (2014): 47.
78. J. M. Kassim, A. R. Shipman, W. Szczecinska, et al., "How Effective Is Intralesional Injection of Triamcinolone Acetonide Compared With Topical Treatments in Inducing and Maintaining Hair Growth in Patients With Alopecia Areata? A Critically Appraised Topic," *British Journal of Dermatology* 170, no. 4 (2014): 766–771.
79. B. E. Yee, Y. Tong, A. Goldenberg, and T. Hata, "Efficacy of Different Concentrations of Intralesional Triamcinolone Acetonide for Alopecia Areata: A Systematic Review and Meta-Analysis," *Journal of the American Academy of Dermatology* 82, no. 4 (2020): 1018–1021.
80. A. Alkhalifah, A. Alsantali, E. Wang, K. J. McElwee, and J. Shapiro, "Alopecia Areata Update," *Journal of the American Academy of Dermatology* 62, no. 2 (2010): 191–202.
81. S. Lee, B. J. Kim, Y. B. Lee, and W. S. Lee, "Hair Regrowth Outcomes of Contact Immunotherapy for Patients With Alopecia Areata: A Systematic Review and Meta-Analysis," *JAMA Dermatology* 154, no. 10 (2018): 1145–1151.
82. V. H. Price, A. Willey, and B. K. Chen, "Topical Tacrolimus in Alopecia Areata," *Journal of the American Academy of Dermatology* 52, no. 1 (2005): 138–139.
83. C. Kuldeep, H. Singhal, A. Khare, A. Mittal, L. Gupta, and A. Garg, "Randomized Comparison of Topical Betamethasone Valerate Foam, Intralesional Triamcinolone Acetonide and Tacrolimus Ointment in Management of Localized Alopecia Areata," *International Journal of Trichology* 3, no. 1 (2011): 20–24.
84. K. A. Feldmann, C. Kunte, A. Wollenberg, and H. Wolff, "Is Topical Tacrolimus Effective in Alopecia Areata Universalis?," *British Journal of Dermatology* 147, no. 5 (2002): 1031–1032.
85. K. Phan and D. F. Sebaratnam, "JAK Inhibitors for Alopecia Areata: A Systematic Review and Meta-Analysis," *Journal of the European Academy of Dermatology and Venerology* 33, no. 5 (2019): 850–856.
86. L. Bokhari and R. Sinclair, "Treatment of Alopecia Universalis With Topical Janus Kinase Inhibitors—A Double Blind, Placebo, and Active Controlled Pilot Study," *International Journal of Dermatology* 57, no. 12 (2018): 1464–1470.
87. L. Y. Liu, B. G. Craiglow, and B. A. King, "Tofacitinib 2% Ointment, a Topical Janus Kinase Inhibitor, for the Treatment of Alopecia Areata: A Pilot Study of 10 Patients," *Journal of the American Academy of Dermatology* 78, no. 2 (2018): 403–404.
88. J. Seneschal, K. Boniface, and C. Jacquemin, "Alopecia Areata: Recent Advances and Emerging Therapies," *Annales de Dermatologie et de Vénérologie* 149, no. 4 (2022): 222–227.
89. E. A. Olsen, D. Kornacki, K. Sun, and M. K. Hordinsky, "Ruxolitinib Cream for the Treatment of Patients With Alopecia Areata: A 2-Part, Double-Blind, Randomized, Vehicle-Controlled Phase 2 Study," *Journal of the American Academy of Dermatology* 82, no. 2 (2020): 412–419.
90. V. C. Weiss and D. P. West, "Topical Minoxidil Therapy and Hair Regrowth," *Archives of Dermatology* 121, no. 2 (1985): 191–192.
91. C. T. Sung, M. L. W. Juhasz, F. D. Choi, and N. A. Mesinkovska, "The Efficacy of Topical Minoxidil for Non-Scarring Alopecia: A Systematic Review," *Journal of Drugs in Dermatology* 18, no. 2 (2019): 155–160.
92. H. Zaher, H. I. Gawdat, R. A. Hegazy, and M. Hassan, "Bimatoprost Versus Mometasone Furoate in the Treatment of Scalp Alopecia Areata: A Pilot Study," *Dermatology* 230, no. 4 (2015): 308–313.
93. S. Bhat, S. Handa, and D. De, "A Randomized Comparative Study of the Efficacy of Topical Latanoprost Versus Topical Betamethasone Dipropionate Lotion in the Treatment of Localized Alopecia Areata," *Indian Journal of Dermatology, Venereology and Leprology* 87 (2021): 42–48.
94. M. Ghassemi, N. Yazdani, E. Behrang, M. Jafari, and A. Goodarzi, "Comparison of Efficacy, Safety and Satisfaction of Latanoprost Versus Minoxidil, Betamethasone and in Combination in Patients With Alopecia Areata: A Blinded Multiple Group Randomized Controlled Trial," *Dermatologic Therapy* 35, no. 12 (2022): e15943.
95. M. Rafati, R. Mahmoudian, M. Golpour, A. Kazeminejad, M. Saeedi, and Z. Nekoukar, "The Effect of Latanoprost 0.005% Solution in the Management of Scalp Alopecia Areata, a Randomized Double-Blind Placebo-Controlled Trial," *Dermatologic Therapy* 35, no. 6 (2022): e15450.
96. "A Study to Evaluate the Safety and Effectiveness of Upadacitinib Tablets in Adult and Adolescent Participants With Severe Alopecia Areata (Up-AA)," <https://classic.clinicaltrials.gov/ct2/show/NCT06012240>.
97. V. W. Y. Lai, L. Bokhari, and R. Sinclair, "Sublingual Tofacitinib for Alopecia Areata: A Roll-Over Pilot Clinical Trial and Analysis of Pharmacokinetics," *International Journal of Dermatology* 60, no. 9 (2021): 1135–1139.
98. B. King, E. Guttman-Yassky, E. Peeva, et al., "A Phase 2a Randomized, Placebo-Controlled Study to Evaluate the Efficacy and Safety of the Oral Janus Kinase Inhibitors Ritlecitinib and Brepocitinib in Alopecia Areata: 24-Week Results," *Journal of the American Academy of Dermatology* 85, no. 2 (2021): 379–387.
99. N. Almutairi, T. M. Nour, and N. H. Hussain, "Janus Kinase Inhibitors for the Treatment of Severe Alopecia Areata: An Open-Label Comparative Study," *Dermatology* 235, no. 2 (2019): 130–136.
100. J. Mackay-Wiggan, A. Jabbari, N. Nguyen, et al., "Oral Ruxolitinib Induces Hair Regrowth in Patients With Moderate-To-Severe Alopecia Areata," *JCI Insight* 1, no. 15 (2016): e89790.
101. Sunpharma.com, *Late Breaking Phase 3 Data at AAD 2023 Show Oral Investigational Medicine Deuruxolitinib Significantly Improved*

Scalp Hair Regrowth in Alopecia Areata (Sun Pharmaceutical Industries Limited, 2023), <https://sunpharma.com/wp-content/uploads/2023/03/Press-Release-Late-Breaking-Phase-3-Data-at-AA-2023-Show-Oral-Investigational-Medicine-Deuruxolitinib.pdf>.

102. A. G. Gambardella, G. Licata, G. Calabrese, A. De Rosa, R. Alfano, and G. Argenziano, "Dual Efficacy of Upadacitinib in 2 Patients With Concomitant Severe Atopic Dermatitis and Alopecia Areata," *Dermatitis* 32, no. 1S (2021): e85–e86.

103. M. Cantelli, F. Martora, C. Patruno, P. Nappa, G. Fabbrocini, and M. Napolitano, "Upadacitinib Improved Alopecia Areata in a Patient With Atopic Dermatitis: A Case Report," *Dermatologic Therapy* 35, no. 4 (2022): e15346.

104. L. Asfour, T. Getsos Colla, A. Moussa, and R. D. Sinclair, "Concurrent Chronic Alopecia Areata and Severe Atopic Dermatitis Successfully Treated With Upadacitinib," *International Journal of Dermatology* 61, no. 11 (2022): e416–e417.

105. A. Flora, E. Kozera, and J. W. Frew, "Treatment of Alopecia Areata With the Janus Kinase Inhibitor Upadacitinib: A Retrospective Cohort Study," *Journal of the American Academy of Dermatology* 89, no. 1 (2023): 137–138.

106. A. Chiricozzi, A. Balato, G. Fabbrocini, et al., "Beneficial Effects of Upadacitinib on Alopecia Areata Associated With Atopic Dermatitis: A Multicenter Retrospective Study," *Journal of the American Academy of Dermatology* 89, no. 6 (2023): 1251–1253.

107. clinicaltrials.gov, *Study to Evaluate the Safety and Efficacy of Jaktinib in Adults With Alopecia Areata (AA)* (National Library of Medicine, 2021).

108. C. Zhou, X. Yang, B. Yang, et al., "A Randomized, Double-Blind, Placebo-Controlled Phase II Study to Evaluate the Efficacy and Safety of Ivarmacitinib (SHR0302) in Adult Patients With Moderate-To-Severe Alopecia Areata," *Journal of the American Academy of Dermatology* 89, no. 5 (2023): 911–919.

109. N. Fagan, G. A. Doherty, N. Meah, R. Sinclair, and D. Wall, "Cross-Specialty Identification of the JAK1 Inhibitor Trial Agent Filgotinib as a Potential Therapy for Alopecia Areata," *British Journal of Dermatology* 188, no. 3 (2023): 442–443.

110. clinicaltrials.gov, *A Study to Evaluate Efficacy and Safety of Deucravacitinib in Participants With Alopecia Areata* (National Library of Medicine, 2022), <https://clinicaltrials.gov/study/NCT05556265?intr=Deucravacitinib&cond=Alopecia%20Areata&rank=1>.

111. E. Guttman-Yassky, Y. Renert-Yuval, J. Bares, et al., "Phase 2a Randomized Clinical Trial of Dupilumab (Anti-IL-4R α) for Alopecia Areata Patients," *Allergy* 77, no. 3 (2022): 897–906.

112. J. M. Carnicle, A. J. Hendricks, and V. Y. Shi, "Reactivation of Alopecia Areata After Dupilumab Therapy for Atopic Dermatitis," *Dermatitis* 32, no. 1S (2021): e80–e82.

113. K. Flanagan, L. Sperling, and J. Lin, "Drug-Induced Alopecia After Dupilumab Therapy," *JAAD Case Reports* 5, no. 1 (2019): 54–56.

114. J. Chung, C. L. Slaughter, and E. L. Simpson, "Alopecia Areata in 2 Patients Treated With Dupilumab: New Onset and Worsening," *JAAD Case Reports* 5, no. 8 (2019): 643–645.

115. G. Tavoletti, A. Chiei-Gallo, F. Barei, A. V. Marzano, and S. M. Ferrucci, "Tralokinumab as a Therapeutic Option for Patients With Concurrent Atopic Dermatitis and Alopecia Areata," *International Journal of Dermatology* 63, no. 3 (2024): 374–375.

116. clinicaltrials.gov, *Study of Daxdilimab for the Treatment of Moderate-to-Severe Alopecia Areata* (National Library of Medicine, 2022), <https://clinicaltrials.gov/study/NCT05368103?intr=daxdilimab&rank=2>.

117. clinicaltrials.gov, *A Study to Evaluate the Efficacy and Safety of Rosnilimab (ANB030) in Treatment of Subjects With Moderate-to-severe Alopecia Areata (AZURE)* (National Library of Medicine, 2022), <https://>

clinicaltrials.gov/study/NCT05205070?cond=Alopecia%20Areata&intr=Rosnilimab&rank=1.

118. clinicaltrials.gov., *Safety and Efficacy of Oral Etrasimod in Adult Participants With Moderate-To-Severe Alopecia Areata* (National Library of Medicine, 2020), <https://clinicaltrials.gov/study/NCT04556734?cond=Alopecia%20Areata&intr=etrasimod&rank=1>.

119. clinicaltrials.gov., *Study of EQ101 in Adult Subjects With Moderate to Severe Alopecia Areata* (National Library of Medicine, 2022), <https://clinicaltrials.gov/study/NCT05589610?cond=Alopecia%20Areata&intr=EQ101&rank=1>.

120. clinicaltrials.gov, *Randomized, Double-Blind, Placebo-Controlled Phase 2a, Proof-of-Concept Trial of ADX-914 Phase 2a Trial for the Treatment of Severe Alopecia Areata (SIGNAL-AA)* (National Library of Medicine, 2023), <https://clinicaltrials.gov/study/NCT06018428?cond=Alopecia%20Areata&intr=ADX-914&rank=1>.

121. A. Estébanez, N. Estébanez, J. Martín, and E. Montesinos, "Apremilast in Refractory Alopecia Areata," *International Journal of Trichology* 11, no. 5 (2019): 213–215.

122. B. Weber, S. Radakovic, and A. Tanew, "Apremilast for Extensive and Treatment-Resistant Alopecia Areata: A Retrospective Analysis of Five Patients," *European Journal of Dermatology* 30, no. 2 (2020): 165–168.

123. L. Y. Liu and B. A. King, "Lack of Efficacy of Apremilast in 9 Patients With Severe Alopecia Areata," *Journal of the American Academy of Dermatology* 77, no. 4 (2017): 773–774.

124. D. Mikhaylov, A. Pavel, C. Yao, et al., "A Randomized Placebo-Controlled Single-Center Pilot Study of the Safety and Efficacy of Apremilast in Subjects With Moderate-To-Severe Alopecia Areata," *Archives of Dermatological Research* 311, no. 1 (2019): 29–36.

125. N. Taneja and S. Gupta, "Apremilast Is Efficacious in Refractory Alopecia Areata," *Journal of Dermatological Treatment* 31, no. 7 (2020): 727–729.

126. B. King, E. Guttman-Yassky, E. Peeva, et al., "Safety and Efficacy of Ritlecitinib and Breprocitinib in Alopecia Areata: Results From the Crossover Open-Label Extension of the ALLEGRO Phase 2a Trial," *JID Innovations* 2, no. 6 (2022): 100156.

127. K. L. S. Kerkemeyer, J. M. John, R. Sinclair, and B. Bhojru, "Response of Alopecia Areata of the Beard to Oral Tofacitinib," *Journal of the American Academy of Dermatology* 82, no. 5 (2020): 1228–1230.

128. B. G. Craiglow, "Topical Tofacitinib Solution for the Treatment of Alopecia Areata Affecting Eyelashes," *JAAD Case Reports* 4, no. 10 (2018): 988–989.

129. K. L. S. Kerkemeyer, R. D. Sinclair, and B. Bhojru, "Topical Tofacitinib for the Treatment of Alopecia Areata Affecting Facial Hair," *British Journal of Dermatology* 185, no. 3 (2021): 677–679.

130. J. Song, A. Song, T. Palmares, M. Song, and H. Song, "Ruxolitinib Found to Cause Eyelash Growth: A Case Report," *Journal of Medical Case Reports* 11, no. 1 (2017): 189.

131. B. G. Craiglow, D. Tavares, and B. A. King, "Topical Ruxolitinib for the Treatment of Alopecia Universalis," *JAMA Dermatology* 152, no. 4 (2016): 490–491.

132. Y. Ramot and A. Zlotogorski, "Complete Regrowth of Beard Hair With Ruxolitinib in an Alopecia Universalis Patient," *Skin Appendage Disorders* 4, no. 2 (2018): 122–124.

133. A. Moussa, S. Eisman, R. D. Sinclair, and B. Bhojru, "Treatment of Alopecia Areata of the Beard With Baricitinib," *Journal of the American Academy of Dermatology* 88, no. 4 (2023): 948–950.

134. I. Coronel-Pérez, E. Rodríguez-Rey, and F. Camacho-Martínez, "Latanoprost in the Treatment of Eyelash Alopecia in Alopecia Areata Universalis," *Journal of the European Academy of Dermatology and Venereology* 24, no. 4 (2010): 481–485.

135. C. Sibbald, "Alopecia Areata: An Updated Review for 2023," *Journal of Cutaneous Medicine and Surgery* 27, no. 3 (2023): 241–259.
136. C. Mao, S. Bruce, D. Wirta, et al., "An Evaluation of the Safety and Efficacy of Bimatoprost for Eyelash Growth in Pediatric Subjects," *Clinical Ophthalmology* 10 (2016): 419–429.
137. T. Vila and F. Camacho Martinez, "Bimatoprost in the Treatment of Eyelash Universalis Alopecia Areata," *International Journal of Trichology* 2, no. 2 (2010): 86.
138. T. Chiba, K. Kashiwagi, K. Ishijima, et al., "A Prospective Study of Iridial Pigmentation and Eyelash Changes due to Ophthalmic Treatment With Latanoprost," *Japanese Journal of Ophthalmology* 48, no. 2 (2004): 141–147.
139. M. Harries, Y. Al-Nuaimi, D. Wall, A. Chandizura, S. Ahmed, and N. Meah, "The Global Register of Alopecia Areata Disease Severity and Treatment Safety—United Kingdom (GRASS-UK): Importance of Real-World Data in Alopecia Areata," *Clinical and Experimental Dermatology* 50 (2025): 1250–1252.
140. D. Wall, N. Meah, K. York, et al., "A Global eDelphi Exercise to Identify Core Domains and Domain Items for the Development of a Global Registry of Alopecia Areata Disease Severity and Treatment Safety (GRASS)," *JAMA Dermatology* 157, no. 4 (2021): 439–448.