Chinese Guidelines for the Diagnosis and Treatment of Alopecia Areata (2026 Edition)

Jinran Lin[1,†], Xiangqian Li[2,†], Cheng Zhou[2,*], Wenyu Wu[1,3,*]; Working Group for Hair Diseases, Association of China Medicine Education in Dermatology and Health Professional Committee; Working Group for Hair Diseases, Combination of Traditional and Western Medicine Dermatology; Working Group for Hair Diseases, Chinese Association of Rehabilitation Dermatology; Working Group for Hair Diseases, Chinese Society of Dermatology

[1] Department of Dermatology, Huashan Hospital, Shanghai Institute of Dermatology, Fudan University, Shanghai, China
[2] Department of Dermatology, Peking University People's Hospital, Beijing, China
[3] Department of Dermatology, Jing'an District Central Hospital, Shanghai, China

*__Correspondence:__ Wenyu Wu, Department of Dermatology, Huashan Hospital, Shanghai 200040, China.
E-Mail: wenyu_wu@fudan.edu.cn
Cheng Zhou, Department of Dermatology, Peking University People's Hospital, Beijing 100044, China.
E-Mail: chengzhou@live.cn

Abstract

Alopecia areata (AA) is an autoimmune, non-scarring hair loss disorder caused by collapse of hair follicle immune privilege and T cell-mediated inflammation. It affects individuals of all ages and carries substantial psychological and social burden. The condition shows marked heterogeneity in presentation and prognosis, influenced by genetic, immune, and environmental factors. The 2026 Chinese Guidelines for AA provide updated, evidence-based recommendations spanning diagnosis, severity assessment, and management. It endorses a multidimensional evaluation that integrates scalp involvement, extra-scalp features, treatment response, and psychosocial impact to guide individualized care. Therapeutic pathways cover topical, intralesional, and systemic options, including corticosteroids, Janus kinase (JAK) inhibitors, and selected biologics, with adjunctive modalities such as light-based therapies, microneedling, platelet-rich plasma, and oral minoxidil. For children, topical corticosteroids remain first-line; JAK inhibitors or biologics may be considered in carefully selected severe cases with appropriate monitoring. The guideline emphasizes long-term follow-up, screening and management of comorbidities, and shared decision-making to improve quality of life (QoL). It further highlights the need for research into biomarkers and novel therapeutic targets to fuel continued iterations on treatments for AA.

Keywords: alopecia areata; guideline; diagnosis and treatment; JAK inhibitors; severity

[†] These authors have contributed equally to this work.

Received: 27 February 2026; **Accepted:** 14 April 2026
DOI: 10.2738/SKIN.2026.0002

Introduction

Alopecia areata (AA) is an autoimmune, non-scarring hair loss disorder that can affect individuals of all ages and both sexes. It typically presents with sudden onset and variable extent, ranging from localized patches to complete scalp (alopecia totalis [AT]) or body hair loss (alopecia universalis [AU]). In addition to classic patchy disease, variants such as diffuse and band-like forms complicate diagnosis and management. Pathogenesis centers on the collapse of hair follicle immune privilege (HF-IP) and T cell-mediated attack on anagen follicles. Beyond visible hair loss, AA is frequently associated with other autoimmune diseases and imposes substantial psychological and economic burdens, impairing quality of life (QoL) and mental health. Given its unpredictable course, recurrence, and psychosocial impact, AA warrants active treatment and long-term follow-up.

Despite being among the most common causes of non-scarring alopecia, management of AA remains challenging. Conventional therapies, such as corticosteroids and immunosuppressants, provide limited efficacy and pose risks of adverse effects, particularly in extensive or refractory disease. Advances in immunopathogenesis have facilitated the development of targeted therapies, notably Janus kinase (JAK) inhibitors, while also optimizing existing modalities. Meanwhile, evidence from randomized trials, real-world studies, and systematic reviews has accumulated, with growing emphasis on treatment safety, laboratory monitoring, and long-term disease control.

In this context, the Chinese Hair Research Society (CHRS) has developed the 2026 edition of the AA guideline. Based on comprehensive literature review, updated evidence grading, and two rounds of expert consensus, this guideline provides standardized, practical, and up-to-date recommendations to assist Chinese dermatologists in clinical practice and improve patient outcomes.

Epidemiology

The prevalence of AA has steadily increased over time. It can affect individuals of any race, sex, or age, with higher rates reported in Asian patients. The peak incidence occurs between ages 19 and 50[1,2]. Approximately 2% of the general population will experience an episode of AA during their lifetime[3]. In China, a 2009 epidemiological study estimated a prevalence of 0.27%[4]. According to the 2021 Global Burden of Disease (GBD) study, there were approximately 3.49 million AA patients in China, with the heaviest disease burden among women aged 25–39 years[5]. The pediatric population represents an important subgroup of AA, carrying a substantial disease burden and associated comorbidities[6]. Moreover, earlier-onset AA tends to follow a more severe and refractory course[7,8].

Beyond physical manifestations, AA imposes significant psychological distress, with symptoms of depression, anxiety, and social phobia severely affecting QoL. A recent multicenter cross-sectional study showed that half of both adult and pediatric patients experience clinically significant anxiety and/or depression[9]. The economic burden is also considerable: the mean annual out-of-pocket cost per patient in China was estimated at $2178.1, with higher expenditures among adults and females[10].

Pathogenesis

The etiology and pathogenesis of AA remain incompletely understood. It is currently recognized as a T cell-mediated inflammatory disorder targeting anagen hair follicles, arising from an interplay of genetic predisposition and environmental triggers.

Genetics

Epidemiological data confirm heritability: positive family history is observed in 8.4%–25% of cases, with monozygotic twin concordance of 42%–55%[11]. Early studies linked AA to human leukocyte antigen (HLA) class I alleles (e.g., *HLA-B12* and *HLA-B18*). Genome-wide association studies (GWAS) have since identified more than 100 susceptibility loci, including immune-related genes (e.g., *ULBP3/6*, *MICA*, *CTLA-4*, *IL2*, *HLA-DRB1*, and *C6orf10*), hair follicle structural genes (e.g., *SMAECA2* and *TCF7L2*), and pigmentation-related genes (e.g., *MCHR2* and *MCHR2AS1*)[12–16]. To date, however, no single causative gene has been identified.

Immunology

Collapse of HF-IP is central to AA. HF-IP is maintained

by low expression of MHC class I in anagen bulbs, secretion of immunosuppressive "IP guardians" (e.g., transforming growth factor-β1 (TGF-β1), interleukin-10 (IL-10), α-melanocyte-stimulating hormone (α-MSH), indoleamine 2,3-dioxygenase (IDO), and vasoactive intestinal peptide (VIP)), and regulation by regulatory T cells (Tregs) and other immune cells[7,17].

In AA, $CD8^+NKG2D^+$ T cells and interferon-γ (IFN-γ) disrupt IP by inducing IL-15 in follicular epithelial cells via JAK1/2. IL-15 subsequently activates $CD8^+NKG2D^+$ T cells through JAK1/3, amplifying IFN-γ production and establishing a pro-inflammatory feedback loop. This cascade promotes dystrophic anagen and premature catagen, leading to hair loss. Importantly, bulge stem cells remain intact, enabling reversibility[1,18–20].

Beyond $CD8^+$ T cells, other immune cells, including iNKT10 (invariant natural killer T cells type), Tregs, T helper 17 (Th17), plasmacytoid dendritic cells, mast cells, and eosinophils, contribute to disease pathogenesis[17].

Environmental triggers and additional contributing factors

AA onset and progression are strongly associated with psychosocial stress, including chronic distress and anxiety, which can act as triggers or exacerbating factors and perpetuate poor treatment response. Environmental triggers include infections, vaccinations, hormonal fluctuations, medications, and diet[17,21]. Epigenetic modifications such as DNA methylation, histone modification, and microRNA regulation further enhance susceptibility[22–25]. Moreover, AA is associated with an increased prevalence of atopic and autoimmune diseases[26], including atopic dermatitis, allergic rhinitis, autoimmune hypothyroidism, systemic lupus erythematosus, and vitiligo.

Clinical subtypes

AA is characterized by the sudden onset of well-defined patches of hair loss on smooth skin and typically without signs of inflammation. Most patients are asymptomatic, although some report mild scalp itching, pain, or tightness. While scalp hair is most frequently involved, AA may also affect eyebrows, eyelashes, beard, axillary hair, pubic hair, and body hair. Nail changes are observed in a subset of patients, particularly in severe or extensive disease.

Clinically, AA can be categorized into several subtypes, which vary in severity, distribution, and extent of hair loss:

1. Patchy AA: Single or multiple round or oval patches of hair loss with distinct boundaries. Small patches often resolve spontaneously, but in some cases progress rapidly.

2. Reticular AA: Multiple, densely distributed patches forming a reticulated pattern.

3. Ophiasis: Band-like hair loss along the parieto-temporo-occipital hairline, often refractory to treatment.

4. Sisaipho (ophiasis inversus): Hair loss predominantly affecting the frontoparietal scalp with relative preservation of the occipital and temporal margins, in contrast to ophiasis.

5. Diffuse AA: Diffuse hair loss across most of the scalp, sometimes accompanied by patches. Short regrowing hairs may appear before shedding is complete. Acute diffuse and total alopecia (ADTA) cases often show better recovery potential.

6. AT: Complete or near-complete (> 95%) loss of scalp hair.

7. AU: Complete loss of scalp, facial, and body hair.

8. AA incognita (AAI): Acute, diffuse shedding of telogen hairs without distinct patches, resembling acute telogen effluvium[27]. Hair density is preserved, but the hair pull test is typically strongly positive.

9. Marie Antoinette syndrome: An acute diffuse AA variant characterized by sudden whitening, due to preferential shedding of pigmented hair[28].

Disease phases

Based on the clinical course of AA, the disease is typically divided into four phases:

1. Progressive/active phase: Expanding hair loss, either by enlargement of existing patches or emergence of new ones. This phase is often accompanied by broken hairs at the lesion margins and a positive hair pull test[29].

2. Stable phase: Hair loss ceases, with a negative hair pull test. Most patients with localized AA transition to the recovery phase after 3–4 months.

3. Recovery phase: Regrowth occurs in the affected areas, with fine, lightly pigmented vellus hairs progressively maturing into darker terminal hairs.

4. Relapse phase: Recurrence of AA-related hair loss following complete clinical remission (The Severity of Alopecia Tool [SALT] = 0)[29].

Evaluation of severity

SALT is the most widely applied metric in clinical practice and clinical trials to quantify scalp involvement in AA[1]. Scalp hair loss is stratified by SALT score as mild (SALT < 20), moderate (20 ≤ SALT < 50), severe (50 ≤ SALT < 95), and very severe (95 ≤ SALT ≤ 100). However, SALT alone does not fully capture individual disease burden, particularly in cases with extra-scalp involvement, poor prognosis, or significant impact on QoL.

To address this, the Alopecia Areata Scale (AAS) integrates additional dimensions, including eyebrow or eyelash loss, inadequate treatment response after ≥ 6 months, diffuse/multifocal positive hair pull tests consistent with rapidly progressive disease, and psychosocial impairment[3]. The Clinician-Reported Outcome (ClinRO) and Patient-Reported Outcome (PRO) are recommended for assessing eyebrow and eyelash involvement[4], which often carry greater aesthetic and emotional consequences.

Multiple validated instruments are available for assessing QoL and psychological states in AA. Commonly used tools include the Dermatology Life Quality Index (DLQI), Alopecia Areata Symptom Impact Scale (AASIS), and Alopecia Areata Patient Priority Outcome (AAPPO)[5]. The Hospital Anxiety and Depression Scale (HADS) is widely used to screen for anxiety and depression. However, these scales may be impractical in routine practice due to their length or complexity. To improve feasibility, the Chinese expert consensus recommends a simplified five-point patient-reported scale ("never affected", "rarely affected", "sometimes affected", "frequently affected", and "constantly affected"), which achieved 80% consensus support.

A systematic, multidimensional severity tool tailored to the Chinese population was recently proposed. Patients initially graded as mild, moderate, or severe by SALT should be upgraded by one severity level if any of the following seven criteria are met[29]:

1. Diffusely positive in hair pull test;

2. Ophiasis AA or Diffuse AA;

3. Inadequate response to previous topical and/or systemic agents (< 50% hair regrowth after 6 months with ≥ 3 topical agents (or ≥ 2 topical corticosteroids), or ≥ 2 systemic agents);

4. Complete eyebrow or eyelash loss (unilateral or bilateral);

5. Involvement of beard or other body hair;

6. Self-reported significant symptom worsening after 3 months of treatment;

7. Frequent or constant psychosocial and daily life impairment.

This multidimensional framework that incorporates scalp extent, extra-scalp features, treatment response, and psychosocial burden provides a more accurate reflection of overall disease severity and supports individualized treatment planning (Table 1).

Comorbidities

AA is associated with diverse comorbidities across organ systems. Autoimmune disorders such as autoimmune thyroid disease (e.g., Hashimoto's thyroiditis and Graves' disease), psoriasis, vitiligo, systemic lupus erythematosus, rheumatoid arthritis, and type 1 diabetes mellitus are well documented, reflecting a shared T cell-mediated

Table 1: A suggested method for severity assessment of AA

SALT score	Classification
SALT < 20	Mild
20 ≤ SALT < 50	Moderate
50 ≤ SALT < 95	Severe
95 ≤ SALT ≤ 100	Very severe

Additional criteria

Patients initially graded as mild, moderate, or severe by SALT should be upgraded by one severity level if any of the following seven criteria are met:

1. Diffuse positivity on hair pull test;
2. Ophiasis AA or Diffuse AA;
3. Inadequate response to previous topical and/or systemic agents (< 50% hair regrowth after 6 months with ≥ 3 topical agents (or ≥ 2 topical corticosteroids), or ≥ 2 systemic agents);
4. Complete eyebrow or eyelash loss (unilateral or bilateral);
5. Involvement of beard or other body hair;
6. Self-reported significant symptom worsening after 3 months of treatment;
7. Frequent or constant psychosocial and daily life impairment.

Abbreviations: AA, Alopecia Areata; SALT, Severity of Alopecia Tool.

autoimmune mechanism[30]. Atopic comorbidities including atopic dermatitis, allergic rhinitis, and asthma are also common. Elevated serum immunoglobulin E (IgE) levels, together with genetic and cytokine signatures, indicate a Th2-skewed immune profile in some patients, which may contribute to more severe or persistent disease[31,32]. Additionally, AA is strongly linked to psychological disorders such as anxiety, depression, and social phobia. Recognition and management of comorbidities are essential for comprehensive care, influencing prognosis, therapeutic decisions, and long-term outcomes.

Diagnosis

Typical AA is diagnosed clinically through history taking, physical examination, and dermoscopy. In atypical cases, differential diagnosis should be guided by clinical and dermoscopic features; histopathology or laboratory tests may be used when necessary.

Hair pull test

The hair pull test is a simple yet reliable method to assess disease activity. After refraining from shampooing for 3–5 days, approximately 50–60 hairs are grasped between the thumb and index finger near the scalp and gently pulled. Extraction of > 6 hairs is considered positive, indicating active disease. Microscopy typically shows dystrophic anagen hairs with club-shaped or tapered roots.

Trichoscopy

Trichoscopy (hair and scalp dermoscopy) is an indispensable, non-invasive diagnostic and monitoring tool. Typical features include preserved follicular ostia, black dots, yellow dots, broken hairs, exclamation mark hairs, tapered hairs, short vellus hairs, upright regrowing hairs, and pigtail hairs[33].

Features such as black dots, exclamation mark hairs, broken hairs, and tapered hairs are commonly seen in active disease, indicating ongoing follicular damage. In contrast, short vellus hairs, upright regrowing hairs, and pigtail hairs are typically observed during the recovery phase, reflecting hair regrowth. Among these, exclamation mark hairs remain the most specific diagnostic hallmark of AA. Notably, trichoscopic patterns may not directly correlate with clinical severity[34].

In AAI, trichoscopic features differ from those in classic AA. Typical findings include numerous diffuse yellow dots, marked variability in hair shaft diameter, and short regrowing hairs of normal thickness, whereas black dots are rarely seen. These features can help differentiate AAI from other diffuse alopecias, such as telogen effluvium.

Scalp biopsy (histopathology)

Most AA cases can be diagnosed clinically and with trichoscopy. However, a 4-mm punch biopsy is valuable in atypical cases[7], especially ophiasis patterns or suspected coexisting cicatricial alopecia. Acute AA shows peribulbar lymphocytic infiltrates ("swarm of bees") targeting anagen follicles, with pigment incontinence and a few eosinophils and mast cells. In addition, miniaturized follicles in anagen morphology or follicles arrested in telogen may also be observed. In chronic AA, inflammatory infiltrates around the follicles are often less prominent, and miniaturized follicles with anagen morphology or follicles arrested in the telogen phase are

frequently observed. Additional diagnostic clues include the presence of pigment casts, lymphocytes within fibrous tracts, and eosinophils[35]. Mixed acute/chronic features within the same sample are common.

Laboratory evaluation

Tests are primarily used to identify comorbidities rather than confirm diagnosis. Suggested tests include thyroid function (thyroid-stimulating hormone (TSH) and free thyroxine (free T4)), thyroid autoantibodies (anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG)), ANA (antinuclear antibody), serum IgE, ferritin, zinc, and vitamin D in selected patients (e.g., those with thyroid disease, anemia, atopy, or severe/progressive AA). If an infection is suspected, fungal microscopy or treponemal serology should be performed to exclude tinea capitis or syphilitic alopecia. Baseline assessments are required before initiating systemic therapies.

Others

Several complementary tools may assist in the diagnosis and monitoring of AA. Trichogram and wash test can provide information on hair root morphology and hair cycle status but are primarily used as adjunctive methods. Additionally, other non-invasive techniques, such as super high-frequency ultrasound and reflectance confocal microscopy, have shown potential in evaluating hair loss disorders, including AA, although their clinical value requires further validation.

Differential diagnosis

AA should be differentiated from the following conditions:

1. Trichotillomania: Patchy, irregular hair loss with broken hairs of varying lengths. Common in children with hair-pulling behavior. Dermoscopy shows black dots, broken hairs, and split ends.

2. Tinea capitis: Common in children, with patchy hair loss, erythema, scaling, and crusting. Dermoscopy may show fungal hair shaft changes such as comma hairs[36]. Additional supportive signs include corkscrew hairs, zigzag hairs, barcode-like hairs with segmental white bands[37,38]. Fragile hairs often contain fungal elements and the diagnosis can be confirmed by potassium hydroxide (KOH) microscopy and fungal culture when needed.

3. Primary cicatricial alopecia (PCA): Permanent hair loss resulting from conditions such as discoid lupus erythematosus (DLE) and lichen planopilaris (LPP). It is characterized by inflammation, scarring, and destruction of follicular openings. Special consideration should be given to distinguishing diffuse AA from frontal fibrosing alopecia (FFA), as well as patchy AA from LPP and DLE.

4. Syphilitic alopecia: Mimics AA with “moth-eaten” patchy hair loss. Positive syphilis serology and secondary syphilis signs assist diagnosis.

5. Congenital alopecia: Present at or shortly after birth, often with sparse or absent hair and possible structural abnormalities. Unlike AA, congenital forms lack regrowth.

6. Temporal triangular alopecia: Non-scarring alopecia in the temporal region, appearing in early childhood. Lesions are stable and lack typical AA dermoscopic findings.

7. Anagen effluvium: Diffuse hair loss due to chemotherapy or toxins, reversible after stopping the causative agent.

8. Female Pattern Hair Loss: Gradual thinning over the frontal/vertex scalp, typically with a negative pull test and no AA-specific dermoscopic signs.

9. Telogen effluvium: Diffuse shedding triggered by stress, illness, or nutritional deficiencies. Pull test may be positive, but broken hairs and exclamation mark hairs are absent.

Treatment

General management

Patients should be advised to minimize psychological stress and maintain a healthy lifestyle, including sufficient sleep, a balanced diet, and moderate exercise. Comorbid inflammatory or autoimmune diseases, if

present, should be identified and managed appropriately.

Local therapy

Topical corticosteroids

Topical corticosteroids are the first-line treatment for mild to moderate AA. Potent or super-potent agents (e.g., halometasone, mometasone furoate, and clobetasol propionate) are applied once or twice daily. Solutions are preferred; creams, gels, and foams are alternatives. For extensive lesions, occlusion therapy may be used. Clinicians should reassess treatment efficacy if no improvement is observed after 3–4 months. Adverse effects include skin atrophy, telangiectasia, folliculitis, and hypopigmentation-mostly reversible. In patients undergoing occlusion therapy for amblyopia, regular intraocular pressure monitoring is recommended in those with pre-existing risk factors for glaucoma, such as high myopia or family history.

Intralesional corticosteroid injections

Intralesional corticosteroid injections are indicated for adults with stable or active patches of AA. Use diluted compound betamethasone (2.33–3.50 g/L) or triamcinolone (2.5–10.0 g/L), injecting about 0.1 mL per site intradermally, spaced 1 cm apart. Max dose: 7 mg betamethasone or 40 mg triamcinolone per session. Injections should be repeated every 3–4 weeks and discontinued if no regrowth after 3 months. Lower doses should be used for eyebrow areas. Side effects include localized atrophy, hypopigmentation, and folliculitis.

Topical immunotherapy

Topical immunotherapy is used for severe, long-standing, or treatment-resistant AA (e.g., multifocal, AT, AU). Agents include diphenylcyclopropenone (DPCP) and squaric acid dibutylester (SADBE), though not Food and Drug Administration (FDA)/National Medical Products Administration (NMPA) approved. This therapy requires institutional ethics approval and informed consent from patients. Treatment duration: ≥ 3–6 months. Response rates: 30%–50%. Adverse effects: contact dermatitis, lymphadenopathy, pigmentation changes, fever, and vitiligo.

Topical minoxidil

Topical minoxidil is recommended as adjunctive therapy in stable AA but is not recommended as monotherapy in active disease. The commonly used concentrations are 2% and 5%. The 5% concentration may offer better efficacy[39], and the foam formulation is given priority recommendation. Other side effects include hypertrichosis (reversible) and rare allergic reactions.

Systemic therapy

Systemic corticosteroids

Systemic corticosteroids are recommended for severe active AA (including multifocal, AT, and AU). Oral prednisone ≤ 0.5 mg/(kg·day) is commonly used, with effects seen within 1–2 months. Continue the initial dose for 2–4 weeks after regrowth, then taper gradually. Intramuscular long-acting corticosteroids (e.g., 1 mL compound betamethasone) may be given every 3–4 weeks for up to 3–4 months.

Relapse is common with rapid tapering. Some patients may require long-term low-dose maintenance (< 7.5 mg/day), and all patients should be closely monitored for systemic adverse effects. Systemic corticosteroids should be discontinued if no efficacy is observed after 3–6 months. In pediatric patients, systemic corticosteroids should be used with caution because of potential effects on linear growth and hypothalamic-pituitary-adrenal axis suppression[40,41].

Immunosuppressants

Agents such as cyclosporine (≤ 3 mg/(kg·day)) may be considered in patients unresponsive to or intolerant of corticosteroids. These immunosuppressive agents are often used in combination with low-dose corticosteroids, and patients should be closely monitored for toxicity and drug levels. Due to cost, relapse risk, and side effects, immunosuppressants are not recommended as first-line therapy.

JAK inhibitors

JAK inhibitors are recommended as first-line systemic therapy for patients aged ≥ 12 years with severe and

very severe chronic AA (disease duration > 6 months). JAK inhibitors may also be considered for patients with rapidly progressive AA or acute diffuse AA when systemic corticosteroids are ineffective, contraindicated, or declined.

Currently, JAK inhibitors approved for the treatment of AA in China include Baricitinib (for adults), Ritlecitinib (for adolescents aged ≥ 12 and adults), and Ivarmacitinib (for adults). These have shown significant efficacy and overall good safety[42–51].

Before initiating JAK inhibitors, baseline evaluation should include complete blood count, liver and renal function tests, lipid profile, and screening for tuberculosis, hepatitis B/C, and HIV when indicated[52]. Chest imaging may be considered if latent tuberculosis (TB) is suspected. Routine vaccinations should be updated before treatment. Live attenuated vaccines are generally not recommended during JAK inhibitor therapy, and inactivated vaccines can be administered without interrupting treatment[53]. Monitoring labs (complete blood count [CBC], liver/renal function, and lipids) should be repeated at 4 weeks and then every 3 months. Overall, JAK inhibitors have a favorable safety profile. Common mild adverse events including folliculitis, elevated liver enzymes, and hyperuricemia. In addition, JAK inhibitors should be avoided in patients with active infections, malignancy, or significant cardiovascular risk factors[52].

Baricitinib

Baricitinib was approved by NMPA for severe adult AA on March 27, 2023. It is a selective JAK1 and JAK2 inhibitor. It has demonstrated effectiveness across diverse populations in real-world settings, including refractory cases, children, and the elderly. In BRAVE-AA, more than one-third of patients receiving 4 mg baricitinib achieved the primary endpoint of SALT ≤ 20 by week 36[54]. By week 52, approximately 40% reached the primary endpoint, and 90.7% of these responders maintained benefit through week 104[45].

The recommended dose for adults is 4 mg/day[45]. The overall tolerability of baricitinib was favorable, with adverse events reported as mostly mild (e.g., laboratory abnormalities and mild infections) and no serious adverse events were observed, including in vulnerable groups.

Ritlecitinib

Ritlecitinib was approved by NMPA for severe AA in patients aged ≥ 12 years on October 19, 2023. It is the first oral selective JAK3 and TEC family kinase inhibitor. In the ALLEGRO-2b/3 trial, 23% of patients receiving ritlecitinib 50 mg once daily achieved SALT ≤ 20 at week 24; this proportion increased to 43% at week 48, and further rose to 73.5% at 24 months[46,47]. Better response was associated with lower baseline SALT, shorter disease duration, and younger age[55,56].

The standard dose is 50 mg once daily[46,48]. Ritlecitinib was generally well tolerated. Common adverse events include nasopharyngitis, folliculitis, headache, upper respiratory tract infection (URTI), and urticaria.

Ivarmacitinib

Ivarmacitinib was approved by NMPA for the treatment of adult patients with severe AA on June 30, 2025. It is a selective JAK1 inhibitor[51]. In a phase 3 study, over one-third of patients treated by 4 mg ivarmacitinib achieved SALT ≤ 20 at week 24[57]. The recommended dose is 4 mg/day.

Upadacitinib

Upadacitinib is currently being investigated for AA in the UP-AA clinical trial. It is a selective JAK1 inhibitor with demonstrated efficacy in AA, particularly in refractory and Th2-dominant cases[58,59]. Recent data from the UP-AA trial showed that at week 24, 44.6% and 54.3% of patients receiving upadacitinib of 15 mg and 30 mg, respectively, achieved SALT ≤ 20. The standard dose is 15 mg/day.

Other JAK inhibitors

Several other JAK inhibitors have also been used off-label for AA, including abrocitinib, tofacitinib, and gecacitinib, with real-world studies reporting encouraging effectiveness and an acceptable safety profile[60–64]. When clinically appropriate, these agents may be considered on a case-by-case basis, and dosing regimens are

typically extrapolated from their approved indications in other diseases.

Biologics

Biologics used in AA mainly include IL-4 receptor α-targeting agents such as dupilumab and stapokibart. They are recommended for patients with severe or greater AA with coexisting atopic disease or markedly elevated total IgE. For children aged 2–12 years with the same conditions, they are recommended if any of the following apply: (1) significant psychological impairment; (2) eyebrow or eyelash involvement; (3) no response to prior treatment within 6 months.

Dupilumab is a monoclonal antibody that targets the IL-4 receptor α subunit, inhibiting both IL-4 and IL-13 signaling. As an off-label therapy in AA, it is recommended for patients with IgE > 200[65,66]. Dosing generally follows atopic dermatitis (AD) regimens: adolescents/adults, 300 mg every 2 weeks.

Other therapies

Other treatments including Platelet-Rich Plasma (PRP)[67], 308-nm Excimer Light Therapy[68,69], and microneedling[70], have demonstrated effectiveness and good tolerability in AA. Antihistamines (e.g., ebastine and fexofenadine), compound glycyrrhizin, topical prostaglandin analogues, topical calcineurin inhibitors (TCI), psoralen plus ultraviolet A (PUVA), narrowband ultraviolet B (UVB), low-level laser therapy, localized cryotherapy, and Traditional Chinese Therapy and Medicine (including acupuncture, fire needle, total glucosides of White Paeony, etc) may also be considered as adjunctive treatment options.

In severe, treatment-refractory AA with extensive hair loss and low likelihood of regrowth, discontinuation of therapy after counseling is acceptable. Camouflage options such as wigs, hairpieces, or cosmetic measures (e.g., eyebrow tattooing) can improve appearance and QoL.

Oral minoxidil (OM) has demonstrated good safety and efficacy in the treatment of AA. It can be used as an adjunct to JAK inhibitors or as a maintenance therapy[58–71].

Therapeutic strategies

The primary goals of AA treatment are to control disease progression, promote hair regrowth, prevent or reduce relapse, and improve QoL. Effective doctor–patient communication and psychological support are essential throughout treatment. Patients with limited, stable disease (e.g., solitary or few small patches) may be managed with observation or topical therapy alone. In contrast, patients with rapidly progressing or extensive hair loss should receive early, active intervention. For patients with long-standing AT, AU, or ophiasis-type AA unresponsive to treatment, therapy may be discontinued after thorough discussion. The use of wigs or hairpieces is also a reasonable supportive option.

Systemic therapy is indicated for patients with a SALT score ≥ 50%. For those with a SALT score of 20% to less than 50%, systemic treatment is recommended if any of the following Systemic Treatment Activation and Grading Evaluation for Alopecia Areata (STAGE-AA) Delphi criteria are met:

1. Diffusely positive hair pull test
2. Ophiasis or diffuse clinical pattern
3. Inadequate response to previous topical agents
4. Complete loss of eyebrow and/or eyelash hair (unilateral or bilateral)
5. Self-reported significant symptom worsening after 3 months of treatment
6. Frequent or constant psychosocial and daily life impairment

Figure 1 presents the recommended treatment pathway for adolescents ≥ 12 years and adults.

Children under 12 years

Topical corticosteroids remain the cornerstone of pediatric AA management, given their favorable safety profile. Intralesional steroids are effective but require careful use. Systemic steroids have limited durability and carry substantial side effects[72]. Topical minoxidil requires

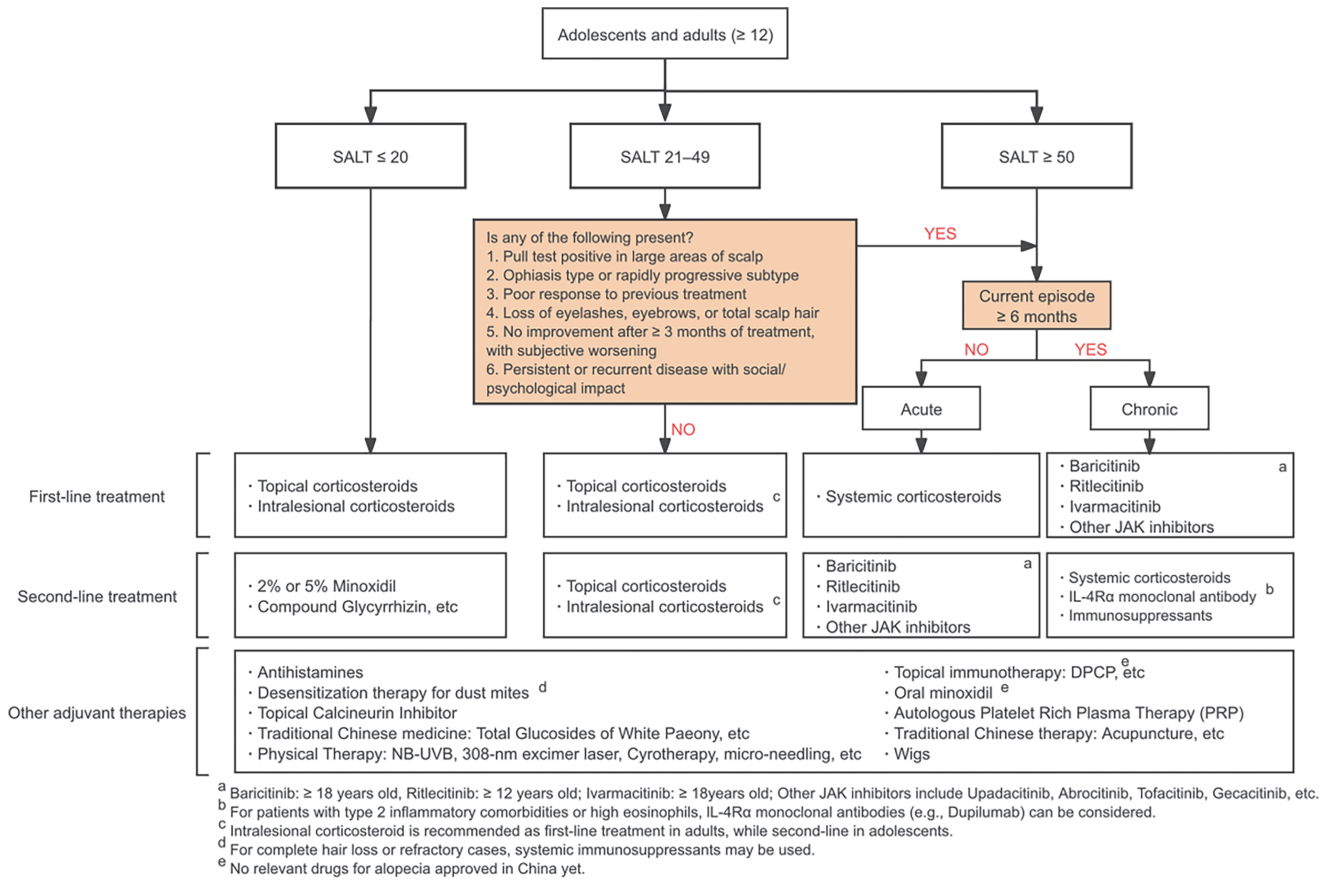

Figure 1: The recommended treatment pathway for adolescents ≥ 12 years and adults.

cautious use due to potential systemic absorption and irritation[73]. Contact immunotherapy offers a steroid-sparing alternative with manageable side effects[74]. Adverse drug reaction monitoring is essential due to off-label use. Routine labs and symptom checks are advised.

For pediatric patients aged 2–12 years with severe or very severe AA, treatment with JAK inhibitors may be considered if any of the following conditions are met: significant psychological distress, involvement of eyebrows and/or eyelashes, and no response to prior therapy of at least 6 months.

Baricitinib (2–4 mg/day) has shown the most promising results and good tolerability[75–77]. Ritlecitinib also shows potential in younger children[55,78]. Dupilumab may benefit children with comorbid atopic dermatitis, showing gradual regrowth and good safety in real-world practice[66,79]. Dosage should follow that recommended for AD in the same age group.

Prognosis

The clinical course and prognosis of AA vary widely among individuals. About 34%–50% of patients with mild disease achieve remission within one year, whereas spontaneous recovery in AT/AU is < 10%. Factors such as disease duration > 2 years, extensive scalp involvement, childhood onset, frequent relapses, ophiasis pattern, nail involvement, and comorbid atopic or autoimmune diseases are associated with poor prognosis. Relapse is common[80], and is defined as the reappearance of patchy hair loss after complete remission (SALT = 0).

Perspective

Further randomized controlled trials are needed to strengthen the evidence base for AA treatment. Research on predictive biomarkers is also an important priority.

For patients unresponsive to guideline-recommended

therapies, options include adding oral minoxidil[81,82]. Another option is switching to a different JAK inhibitor[83]. Novel biologics, such as the OX40L antibody amlitelimab, have shown favorable efficacy in phase II trials and may offer a new treatment option[84].

In relapsed patients, low-dose maintenance or retreatment with JAK inhibitors may be considered[80]. For patients who lose response after retreatment, switching treatment should be considered. Additional adjunctive options include allergen immunotherapy (e.g., dust mite desensitization)[85]. The Treg agonist rezpegaldesleukin and therapies targeting the skin–gut axis are also potential options[86,87].

Author contributions

Jinran Lin and Xiangqian Li take responsibility for literature search, manuscript preparation, and manuscript editing. Wenyu Wu and Cheng Zhou take responsibility for the integrity of the work.

Acknowledgements

Members of the Expert Steering Group (sorted by last name): Xufeng Du, Siqi Fu, Yiqun Jiang, Mingxing Lei, Ji Li, Xiangqian Li, Jinran Lin, Mao Lin, Guoyan Liu, Ling Liu, Xiaoyan Liu, Yuanjun Liu, Ying Luo, Zhongfa Lv, Xianfu Meng, Longquan Pi, Yongjun Piao, Xiuzu Song, Weiling Sun, Hao Wang, Peiguang Wang, Yanyun Wang, Aihua Wei, Sijian Wen, Wenyu Wu, Jun Xie, Nan Xu, Xuegang Xu, Yangchun Xu, Yadong Xue, Dingquan Yang, Shuxia Yang, Yanting Ye, Chenghui Zhang, Fan Zhang, Yong Zhang, Hengguang Zhao, Cheng Zhou, Jing Zhou, Qiang Zhou, Mingji Zhu, Fenglin Zhuo

This paper is funded by Key Specialty Research Centre of Shanghai Health Commission (2023ZZ02018), National Key Clinical Specialty, National Key Research and Development Program of China (2023YFC2509001), National Natural Science Foundation of China (82373504), Clinical Research Plan of SHDC (SHDC22025306), Jing'an District Discipline Development Education Program (2024PY01), and Shanghai Engineering Research Center of Hair Medicine (19DZ2250500).

The authors acknowledge the assistance of Weixin Fan, Department of Dermatology, The First Affiliated Hospital of Nanjing Medical University; Hong Fang, Department of Dermatology, the first affiliated hospital of Zhejiang University; Qinping Yang, Department of Dermatology, Huashan Hospital, Fudan University; Xingqi Zhang, Department of Dermatology, The First Affiliated Hospital, Sun Yat-sen University; Jianzhong Zhang, Department of Dermatology, Peking University People's Hospital.

The manuscript has been read and approved by all the authors. The requirements for authorship as stated earlier in this document have been met. Each author believes that the manuscript represents honest work, if that information is not provided in another form.

Ethics statement

Not applicable.

Data availability statement

No datasets were generated or analyzed for this work.

AI statement

The authors declare that no generative artificial intelligence (AI) or AI-assisted technologies were used in the preparation, analysis, or writing of this manuscript.

Conflict of interest

Wenyu Wu serves as a member of the Editorial Board of this journal. They were not involved in the editorial review or decision-making process for this manuscript. All editorial decisions were made independently by other members of the Editorial Board who have no conflicts of interest.

References

1. Strazzulla LC, Wang EHC, Avila L, et al. Alopecia areata: disease characteristics, clinical evaluation, and new perspectives on pathogenesis. *J Am Acad Dermatol*. 2018;78(1):1-12.
2. Jeon JJ, Jung SW, Kim YH, et al. Global, regional and national epidemiology of alopecia areata: a systematic review and modelling study. *Br J Dermatol*. 2024;191(3):325-335.
3. Lee HH, Gwillim E, Patel KR, et al. Epidemiology of alopecia areata, ophiasis, totalis, and universalis: a systematic review and meta-analysis. *J Am Acad Dermatol*.

2020;82(3):675-682.
4. Wang TL, Shen YW, Zhou C, et al. Alopecia areata in China: a survey on prevalence in 6 cities. *Chin J Dermatol*. 2009;42(10):668-670.
5. Li X, Liu H, Ren W, et al. Burden of alopecia areata in China, 1990–2021: Global Burden of Disease Study 2021. *Chin Med J (Engl)*. 2025;138(3):318-324.
6. Adhanom R, Ansbro B, Castelo-Soccio L. Epidemiology of pediatric alopecia areata. *Pediatr Dermatol*. 2025; 42 Suppl 1:12-23.
7. Pratt CH, King LE Jr, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. *Nat Rev Dis Primers*. 2017;3:17011.
8. Sibbald C. Alopecia areata: an updated review for 2023. *J Cutan Med Surg*. 2023;27(3):241-259.
9. Li X, Du X, Zhao H, et al. Patient-reported psychosocial impact of alopecia areata: a multicenter cross-sectional study. *J Am Acad Dermatol*. 2025;93(5):1294-1296.
10. Li X, Du X, Zhao H, et al. Patient-reported economic burden of alopecia areata in China: a multicenter cross-sectional study. *J Am Acad Dermatol*. 2025;93(5):1317-1319.
11. Blaumeiser B, van der Goot I, Fimmers R, et al. Familial aggregation of alopecia areata. *J Am Acad Dermatol*. 2006;54(4):627-632.
12. Petukhova L, Duvic M, Hordinsky M, et al. Genome-wide association study in alopecia areata implicates both innate and adaptive immunity. *Nature*. 2010;466(7302):113-117.
13. Connell SJ, Jabbari A. The current state of knowledge of the immune ecosystem in alopecia areata. *Autoimmun Rev*. 2022;21(5):103061.
14. Petukhova L, Christiano AM. Functional interpretation of genome-wide association study evidence in alopecia areata. *J Invest Dermatol*. 2016;136(1):314-317.
15. Betz RC, Petukhova L, Ripke S, et al. Genome-wide meta-analysis in alopecia areata resolves HLA associations and reveals two new susceptibility loci. *Nat Commun*. 2015;6:5966.
16. Rajabi F, Abdollahimajd F, Jabalameli N, Nassiri Kashani M, Firooz A. The immunogenetics of alopecia areata. *Adv Exp Med Biol*. 2022;1367:19-59.
17. Zhou C, Li X, Wang C, Zhang J. Alopecia areata: an update on etiopathogenesis, diagnosis, and management. *Clin Rev Allergy Immunol*. 2021;61(3):403-423.
18. Bertolini M, McElwee K, Gilhar A, Bulfone-Paus S, Paus R. Hair follicle immune privilege and its collapse in alopecia areata. *Exp Dermatol*. 2020;29(8):703-725.
19. Gilhar A, Laufer-Britva R, Keren A, Paus R. Frontiers in alopecia areata pathobiology research. *J Allergy Clin Immunol*. 2019;144(6):1478-1489.
20. Gilhar A, Etzioni A, Paus R. Alopecia areata. *N Engl J Med*. 2012;366(16):1515-1525.
21. Ma T, Zhang T, Miao F, et al. Alopecia areata: pathogenesis, diagnosis, and therapies. *MedComm*. 2025;6(5):e70182.
22. Wang EHC, DeStefano GM, Patel AV, et al. Identification of differentially expressed miRNAs in alopecia areata that target immune-regulatory pathways. *Genes Immun*. 2017;18(2):100-104.
23. Zhao M, Liang G, Wu X, et al. Abnormal epigenetic modifications in peripheral blood mononuclear cells from patients with alopecia areata. *Br J Dermatol*. 2012;166(2):226-273.
24. Tafazzoli A, Forstner AJ, Broadley D, et al. Genome-wide microRNA analysis implicates miR-30b/d in the etiology of alopecia areata. *J Invest Dermatol*. 2018;138(3):549-556.
25. Sheng Y, Qi S, Hu R, et al. Identification of blood microRNA alterations in patients with severe active alopecia areata. *J Cell Biochem*. 2019;120(9):14421-14430.
26. Holmes S, Harries M, Macbeth AE, et al. Alopecia areata and risk of atopic and autoimmune conditions: population-based cohort study. *Clin Exp Dermatol*. 2023;48(4):325-331.
27. Rodríguez-Tamez G, Maskan-Bermudez N, Tosti A. Alopecia areata incognita: current evidence. *Dermatol Ther (Heidelb)*. 2025;15(3):635-645.
28. Asz-Sigall D, Ortega-Springall MF, Smith-Pliego M, et al. White hair in alopecia areata: clinical forms and proposed physiopathologic mechanisms. *J Am Acad Dermatol*. 2023;89(4):758-763.
29. Zhou C, Du X, Fan W, et al. Expert consensus on systemic treatment activation and grading evaluation for alopecia areata. *Chin Med J (Engl)*. 2025. [Epub ahead of print]
30. Mostaghimi A, Soliman AM, Li C, Barqawi YK, Grada A. Immune-mediated and psychiatric comorbidities among patients newly diagnosed with alopecia areata. *JAMA Dermatol*. 2024;160(9):945-952.
31. Zhang XQ. T helper type 2 immune response in alopecia areata. *Chin J Dermatol*. 2022;55(11):1013-1018.
32. Xu W, Zhang H, Wan S, Xie B, Song X. Genetic links between atopy, allergy, and alopecia areata: insights from a Mendelian randomization study. *Allergy Asthma Clin Immunol*. 2024;20(1):32.
33. Waśkiel A, Rakowska A, Sikora M, Olszewska M, Rudnicka L. Trichoscopy of alopecia areata: an update. *J Dermatol*. 2018;45(6):692-700.
34. Mane M, Nath AK, Thappa DM. Utility of dermoscopy in alopecia areata. *Indian J Dermatol*. 2011;56(4):407-411.
35. Peckham SJ, Sloan SB, Elston DM. Histologic features of alopecia areata other than peribulbar lymphocytic infiltrates. *J Am Acad Dermatol*. 2011;65(3):615-620.
36. Miteva M, Tosti A. Hair and scalp dermatoscopy. *J Am Acad Dermatol*. 2012;67(5):1040-1048.
37. Waśkiel-Burnat A, Rakowska A, Sikora M, Ciechanowicz P, Olszewska M, Rudnicka L. Trichoscopy of tinea capitis: a systematic review. *Dermatol Ther (Heidelb)*. 2020;10(1):43-52.

38. Wang HH, Lin YT. Bar code-like hair: dermoscopic marker of tinea capitis and tinea of the eyebrow. *J Am Acad Dermatol*. 2015;72(Suppl 1):S41-S42.
39. Freire PCB, Riera R, Martimbianco ALC, Petri V, Atallah AN. Minoxidil for patchy alopecia areata: systematic review and meta-analysis. *J Eur Acad Dermatol Venereol*. 2019;33(9):1792-1799.
40. Beuschlein F, Else T, Bancos I, et al. European society of endocrinology and endocrine society joint clinical guideline: diagnosis and therapy of glucocorticoid-induced adrenal insufficiency. *J Clin Endocrinol Metab*. 2024;109(7):1657-1683.
41. Improda N, Chioma L, Capalbo D, Bizzarri C, Salerno M. Glucocorticoid treatment and adrenal suppression in children: current view and open issues. *J Endocrinol Invest*. 2025;48(1):37-52.
42. Piraccini BM, Pampaloni F, Cedirian S, et al. Real-life effectiveness and safety of baricitinib in patients with severe alopecia areata: a 24-week Italian study. *J Eur Acad Dermatol Venereol*. 2025;39(6):1143-1151.
43. Tang GT, Triwongwaranat D, Sinclair R, et al. Treatment of moderate-to-severe alopecia areata in patients over the age of 65 years with baricitinib. *Clin Exp Dermatol*. 2024;49(8):875-878.
44. Bi L, Wang C, Du Y, et al. Effectiveness and safety of baricitinib in patients with moderate-to-severe refractory alopecia areata in real world: an open-label, single-center study. *J Cosmet Dermatol*. 2024;23(4):1417-1421.
45. Senna M, Mostaghimi A, Ohyama M, et al. Long-term efficacy and safety of baricitinib in patients with severe alopecia areata: 104-week results from BRAVE-AA1 and BRAVE-AA2. *J Eur Acad Dermatol Venereol*. 2024;38(3):583-593.
46. Tziotzios C, Sinclair R, Lesiak A, et al. Long-term safety and efficacy of ritlecitinib in adults and adolescents with alopecia areata and at least 25% scalp hair loss: results from the ALLEGRO-LT phase 3, open-label study. *J Eur Acad Dermatol Venereol*. 2025;39(6):1152-1162.
47. Piliang M, Soung J, King B, 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. *Br J Dermatol*. 2025;192(2):215-227.
48. Zhang X, Ye Y, Sun W, et al. Efficacy and safety of ritlecitinib in Asian patients with alopecia areata: a subgroup analysis of the ALLEGRO phase 2b/3 trial. *J Dermatol*. 2025;52(4):603-614.
49. Sheng Y, Wang Q, Tao K, et al. Real-world efficacy and safety of ritlecitinib in alopecia areata: a single-centre experience. *J Eur Acad Dermatol Venereol*. 2025;39(12):e996-e998.
50. Huang J, Jian J, Li M, et al. Real-world efficacy of ritlecitinib in treating alopecia areata across various anatomical sites: potential rapid response predictors. *J Am Acad Dermatol*. 2025;93(5):1236-1242.
51. Zhou C, Yang X, Yang B, 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. *J Am Acad Dermatol*. 2023;89(5):911-919.
52. Huang J, Tan Z, Tang Y, Shi W. Screening for latent infectious disease in patients with alopecia areata before initiating JAK inhibitors therapy: a single-center real-world retrospective study. *Front Med (Lausanne)*. 2023;10:1287139.
53. National Clinical Research Center for Dermatologic and Immunologic Diseases (Peking Union Medical College Hospital), Chinese Association of Rheumatology and Immunology Physicians, Dermatology Professional Committee of Chinese Rehabilitation Medical Association. Chinese expert consensus on the treatment of immune-mediated inflammatory diseases with Janus kinase inhibitors. *Chin J Intern Med (Zhonghua Nei Ke Za Zhi)*. 2025;64(12):1187-1210. (in Chinese)
54. King B, Ohyama M, Kwon O, et al. Two phase 3 trials of baricitinib for alopecia areata. *N Engl J Med*. 2022;386(18):1687-1699.
55. Huang J, Jian J, Li M, Ji R, Li J, Shi W. Effectiveness and tolerability of ritlecitinib in children with alopecia areata: a case series. *J Am Acad Dermatol*. 2025;92(6):1395-1397.
56. Zhang W, Li X, Zhang Y, Luo Y, Li H, Zhou C. Ritlecitinib in alopecia areata: a multicenter retrospective real-world analysis. *J Am Acad Dermatol*. 2025;93(4):1136-1137.
57. Zhou C, Yang C, Fan W, et al. Ivarmacitinib for the treatment of adults with severe alopecia areata: results from a phase 3 trial. *J Am Acad Dermatol*. 2026;94(1):161-171.
58. Wang Q, Shen L, Sha Y, et al. Upadacitinib for refractory alopecia areata unresponsive to prior systemic therapies: a retrospective study. *J Am Acad Dermatol*. 2025;93(3):761-763.
59. Battilotti C, Azzella G, Dattola A, et al. Efficacy of upadacitinib in treating alopecia areata, atopic dermatitis, and Th1 comorbidities in pediatric patients: a comprehensive case series and literature review. *J Clin Med*. 2025;14(11):3881.
60. Lin J, Li Z, Shen L, et al. Successful treatment of severe diffuse alopecia areata with abrocitinib. *An Bras Dermatol*. 2025;100(3):580-582.
61. Zhang Y, Wang F, Li X, et al. Oral abrocitinib for treatment of refractory alopecia areata: a retrospective study. *J Am Acad Dermatol*. 2025;92(1):153-155.
62. Huang J, Qian P, Tang Y, Li J, Liu F, Shi W. Effectiveness and predictive factors of response to tofacitinib therapy in 125 patients with alopecia areata: a single-centre real-world retrospective study. *Acta Derm Venereol*. 2023;103:adv12425.

63. Ji R, Huang J, Jian J, et al. Characteristics and predictive values of super-responders in alopecia areata under tofacitinib treatment: a single-centre retrospective study. *Clin Exp Dermatol*. 2025;50(8):1571-1577.
64. Huang J, Li T, Tan Z, et al. Effectiveness of tofacitinib in pre-adolescent alopecia areata: a retrospective case series and literature review. *Acta Derm Venereol*. 2023;103:adv13418.
65. Guttman-Yassky E, Renert-Yuval Y, Bares J, et al. Phase 2a randomized clinical trial of dupilumab (anti-IL-4Rα) for alopecia areata patients. *Allergy*. 2022;77(3):897-906.
66. David E, Shokrian N, Del Duca E, et al. Dupilumab induces hair regrowth in pediatric alopecia areata: a real-world, single-center observational study. *Arch Dermatol Res*. 2024;316(7):487.
67. Muhammad A, Saleem S, Khan S, et al. Comparison of efficacy of autologous platelet rich plasma therapy with 5% topical minoxidil spray in treating alopecia areata: a head-to-head assessment of novel approaches. *Cureus*. 2024;16(6):e61878.
68. Di Filippo Y, Montaudié H, Passeron T. Efficacy of 308-nm excimer therapy in alopecia areata: a retrospective study with long-term follow-up. *Photodermatol Photoimmunol Photomed*. 2022;38(5):507-510.
69. Kianfar N, Dasdar S, Mahmoudi H, et al. Comparison of the efficacy and safety of 308-nm excimer laser with intralesional corticosteroids for the treatment of alopecia areata: a randomized controlled study. *Lasers Surg Med*. 2022;54(4):502-510.
70. Qiao R, Zhu J, Fang J, et al. Microneedle transdermal delivery of compound betamethasone in alopecia areata-a randomized controlled trial. *J Am Acad Dermatol*. 2025;92(2):269-275.
71. Moussa A, Bokhari L, Sinclair RD. Systemic minoxidil as maintenance treatment in alopecia areata: a retrospective case series of 24 patients. *Clin Exp Dermatol*. 2022;47(4):753-755.
72. Fernando T, Goldman RD. Corticosteroids for alopecia areata in children. *Can Fam Physician*. 2020;66(7):499-501.
73. Rai AK. Minoxidil-induced hypertrichosis in a child with alopecia areata. *Indian Dermatol Online J*. 2017;8(2):147-148.
74. Esmaeili F, Vahabi SM, Abdoli M, et al. Topical immunotherapy with diphenylcyclopropenone in paediatric patients with alopecia areata-a retrospective study of 97 patients. *Skin Health Dis*. 2024;4(5):e441.
75. Zhao M, Wei Y, Cai L, Zhuo J, Zhang Z, Lin M. Baricitinib therapy for paediatric patients with severe alopecia areata. *J Eur Acad Dermatol Venereol*. 2025;39(1):e82-e83.
76. Asfour L, Bokhari L, Bhoyrul B, et al. Treatment of moderate-to-severe alopecia areata in pre-adolescent children with baricitinib. *Br J Dermatol*. 2023;189(2):248-250.
77. Zhan J, Cao J, Chen F, Jin Y, Huang C. Real-data on the use of baricitinib in adolescents with severe alopecia areata. *J Eur Acad Dermatol Venereol*. 2023. [Epub ahead of print]
78. Hordinsky M, Hebert AA, Gooderham M, et al. Efficacy and safety of ritlecitinib in adolescents with alopecia areata: results from the ALLEGRO phase 2b/3 randomized, double-blind, placebo-controlled trial. *Pediatr Dermatol*. 2023;40(6):1003-1009.
79. Huang J, Jian J, Li T, et al. Dupliumab therapy for alopecia areata: a case series and review of the literature. *J Dermatolog Treat*. 2024;35(1):2312245.
80. King B, Ko J, Kwon O, et al. Baricitinib withdrawal and retreatment in patients with severe alopecia areata: the BRAVE-AA1 randomized clinical trial. *JAMA Dermatol*. 2024;160(10):1075-1081.
81. Valido K, Craiglow BG. Baricitinib and oral minoxidil for the treatment of alopecia areata in pediatric patients. *J Am Acad Dermatol*. 2025;93(2):509-510.
82. Valido K, Kalil L, Peterson D, King B. Combination oral minoxidil and baricitinib treatment of alopecia areata. *J Am Acad Dermatol*. 2025;93(4):1071-1073.
83. Zhang X, Jiang Y. Predictors and management of inadequate response to JAK inhibitors in alopecia areata. *Am J Clin Dermatol*. 2024;25(6):975-986.
84. Kim M, Del Duca E, Dahabreh D, et al. Alopecia areata exhibits cutaneous and systemic OX40 activation across atopic backgrounds. *Allergy*. 2024;79(12):3401-3414.
85. Zeng Z, Li S, Ye Y, et al. Allergen desensitization reduces the severity of relapsed alopecia areata in dust-mite allergic patients. *Exp Dermatol*. 2023;32(7):1108-1119.
86. Silverberg JI, Rosmarin D, Chovatiya R, et al. The regulatory T cell-selective interleukin-2 receptor agonist rezpegaldesleukin in the treatment of inflammatory skin diseases: two randomized, double-blind, placebo-controlled phase 1b trials. *Nat Commun*. 2024;15(1):9230.
87. Burma NE, Ramien ML. Cutaneous and gut dysbiosis in alopecia areata: a review. *JID Innov*. 2025;5(4):100363.