

A latest Review on Alopecia Areata

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ABSTRACT

Alopecia areata is a prevalent autoimmune disorder characterized by temporary, non-scarring hair loss that occurs even though the hair follicles remain viable. The condition most frequently manifests as isolated bald patches on the scalp. Statistical research suggests that nearly 2% of the global population will encounter this condition at some point in their lives. This article provides a comprehensive evaluation of the latest clinical insights and therapeutic breakthroughs in the field of alopecia areata as of 2025. The precise cause of alopecia areata (AA), an autoimmune-mediated condition, remains largely elusive. Statistically, it ranks as the third most prevalent skin disorder among children, with a lifetime incidence rate estimated between 1% and 2%. Clinicians typically diagnose AA through a direct assessment, relying on a combination of specific patient history and distinctive markers identified during a physical examination.

In the clinical management of alopecia areata, corticosteroids continue to be the primary therapeutic choice. Beyond standard treatments, this review delves into the underlying causes and immune-mediated pathways of the disorder, while providing a detailed assessment of the various therapeutic options currently available.

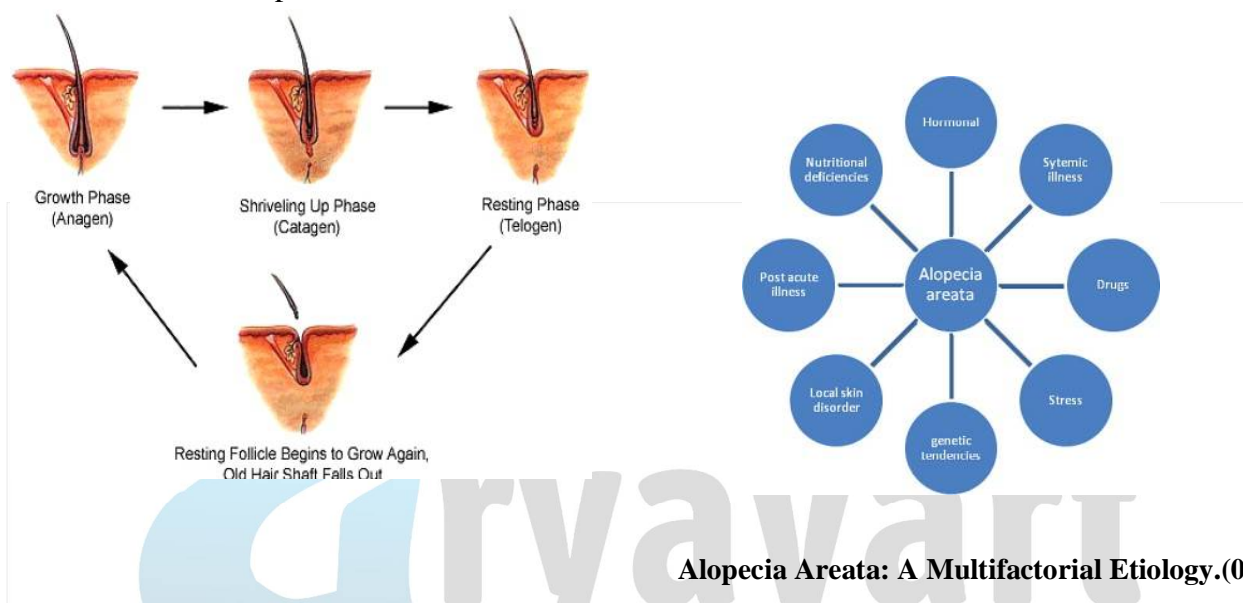
KEYWORDS

Alopecia areata, corticosteroids, Janus kinase, immunopathogenesis

INTRODUCTION

Alopecia areata (AA) is an immune-mediated condition defined by temporary, non-scarring alopecia that leaves the underlying follicle structure intact. The presentation of hair loss is highly diverse; it can manifest as isolated, circular patches, generalized thinning, or the total loss of body hair. Of these variations, the most frequent clinical observation is localized patchiness on the scalp. Current epidemiological data suggest a lifetime prevalence of roughly 2% across the global population.¹The clinical significance of alopecia areata is often undervalued or misidentified as a simple aesthetic issue. In reality, the disorder carries a heavy disease burden, frequently resulting in deep psychological distress, diminished self-regard, and a compromised quality of life for those affected².Progression to more severe forms occurs in roughly 1% to 2% of cases; this includes alopecia totalis, characterized by the complete denudation of the scalp, and alopecia universalis, which involves the total loss of all terminal body hair. Current data indicates an incidence rate between 0.1% and 0.2%, with a cumulative lifetime risk of approximately 1.7%. Furthermore, the disorder demonstrates no gender bias, impacting males

and females at an equivalent rate.³ The interplay between psychiatric health and alopecia areata is well-documented, with psychological stress serving as both a possible trigger for and a result of the disease. This dual relationship underscores the necessity for a comprehensive, holistic management strategy for all patients. While corticosteroid treatments have historically been the foundation of clinical care, the therapeutic landscape has expanded significantly with the emergence of new, evidence-based alternatives. This review provides a systematic examination of the progression of alopecia areata and offers a critical analysis of the benefits and constraints of modern treatment protocols.⁴



Alopecia Areata: A Multifactorial Etiology.(04)

CLINICAL FEATURES

The most frequent clinical presentation of alopecia areata is the sudden appearance of isolated, clearly defined areas of hair loss that may expand outward over time. Patients may present with a solitary lesion or several distinct patches; in some cases, these areas merge to create more extensive regions of baldness. While any hair-bearing site can be affected, the scalp is involved in roughly 90% of all diagnoses.

Clinically, the condition is categorized by the severity and distribution of hair loss. While the localized "patchy" form is the most prevalent, the disease can progress to more severe variants. Alopecia totalis (AT) involves the total loss of hair on the scalp and may extend to the eyebrows, lashes, and beard. The most extensive form, alopecia universalis (AU), is defined by the complete absence of hair across the entire body. Progression from patchy hair loss to AT or AU occurs in approximately 5–10% of cases. However, age of onset is a significant prognostic indicator; individuals who develop the condition before puberty face a 50% risk of total scalp hair loss, whereas the risk for those diagnosed later in life is roughly 25%.⁵



Figure 1: Localized patch of hair loss in alopecia areata.

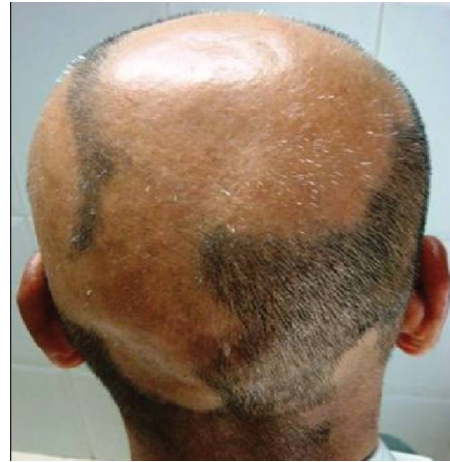


Figure 2: Small alopecia areata patches coalescing to form a larger affected area



Figure 3: Ophiasis



Figure 4: Sisaipho



Figure 5: Atypical presentation of alopecia areata exhibiting a linear pattern of hair loss

DIAGNOSIS

A thorough diagnosis requires a full review of medical history and a physical inspection of the scalp, body, and nails. Clinicians should routinely use the hair pull test and dermoscopy to identify characteristic markers. If the diagnosis is unclear, a scalp biopsy, fungal culture, or blood tests (to rule out syphilis or other autoimmune conditions) may be necessary⁶. To evaluate disease activity, clinicians perform a hair pull test by grasping roughly 20 hairs; extracting more than 10 indicates active hair loss and requires microscopic analysis. If the scalp shows signs of inflammation—such as scaling, pustules, or crusting—samples should be collected from the edge of the patch for bacterial and fungal cultures. Any fluid from pustular lesions should also be swabbed for sensitivity testing to rule out secondary infections.⁷

DIFFERENTIAL DIAGNOSIS

While various conditions can mimic the presentation of alopecia areata—specifically trichotillomania, tineacapitis, scarring alopecia, and telogen effluvium—a diagnosis is usually reachable through clinical observation alone. The distinct patterns of hair loss and the lack of scarring generally allow clinicians to distinguish it from these other disorders⁸.

TREATMENT

Studies indicate that baricitinib significantly improves both immediate and sustained hair regrowth when compared to a placebo. While data regarding the risk of severe side effects remains inconclusive, the low frequency of reported adverse events suggests that the clinical benefits of treatment generally outweigh the potential risks.¹⁴ To determine if a patient needs systemic treatment, clinicians use standardized tools like the SALT score or the Alopecia Areata Scale (AAS). Generally, a SALT score of 20 or higher—or a moderate-to-severe AAS rating—signals that it is time to consider more intensive therapy.

Currently, baricitinib (for adults) and ritlecitinib (for ages 12+) are the only EMA-approved JAK inhibitors for severe cases. Other options, such as cyclosporine, methotrexate, and oral steroids, are frequently used off-label. While oral minoxidil is often added to these regimens, its effectiveness as a standalone treatment is still being studied. This review provides a clear framework for using these systemic therapies, covering when to start, what to expect regarding safety, and how long to continue treatment¹⁰.

Janus kinase (JAK) inhibitors have emerged as a promising treatment option, offering a Modern treatment for alopecia areata now focuses on blocking the specific immune pathways that attack hair follicles. The FDA has recently approved three Janus kinase (JAK) inhibitors—baricitinib, ritlecitinib, and deuruxolitinib—following successful clinical trials. Data from these trials highlight their effectiveness in restoring hair:

Baricitinib: Approximately 35–40% of patients achieved a SALT score of 20 or less by week 36. **Ritlecitinib:** Roughly 23% of participants reached the same threshold by week 24. **Deuruxolitinib:** About 31% of patients saw significant regrowth within 24 weeks.

Medication	Mechanism	Success Rate (SALT \leq 20)	Key Timeline
Baricitinib	JAK 1/2 inhibitor	35–40%	36 Weeks
Ritlecitinib	JAK 3/TEC inhibitor	23%	24 Weeks
Deuruxolitinib	JAK 1/2 inhibitor	31%	24 Weeks

Beyond these approved drugs, clinicians often see success using other JAK inhibitors like tofacitinib and ruxolitinib off-label. This review synthesizes the latest trial data to help providers make informed, evidence-based decisions and improve patient outcomes.¹¹

CONCLUSION

Alopecia areata is a complex autoimmune disorder with a multifactorial etiology and diverse clinical manifestations ranging from patchy hair loss to alopecia universalis. Although the condition is non-scarring and potentially reversible, it exerts a profound psychological and social impact on affected individuals. Accurate diagnosis requires careful clinical evaluation, supplemented by dermatoscopy and, when necessary, histopathology or laboratory investigations to rule out mimicking conditions.

In terms of management, corticosteroids remain a cornerstone of therapy, but advances in immunomodulatory agents—particularly Janus kinase (JAK) inhibitors—have significantly transformed the treatment landscape. Baricitinib, ritlecitinib, and deuruxolitinib represent major milestones, showing meaningful hair regrowth in clinical trials and offering hope for patients with severe and refractory forms of the disease. Nevertheless, challenges remain in terms of long-term efficacy, relapse prevention, safety, and cost.

RESULTS

The review of published literature highlights that alopecia areata (AA) is a common autoimmune hair loss disorder affecting nearly 2% of the population worldwide, with equal prevalence among men and women. Clinical manifestations are heterogeneous, ranging from localized patchy hair loss to complete scalp (alopecia totalis) or body hair loss (alopecia universalis). Early onset, extensive involvement, and nail changes are associated with poorer prognosis.

Diagnostic evaluation relies primarily on clinical features, supported by dermatoscopy and hair pull test, with biopsy reserved for atypical cases. Differential diagnoses such as trichotillomania, tinea capitis, and scarring alopecias must be considered.

Therapeutically, corticosteroids remain the most widely used first-line agents, although their efficacy is often limited by relapse. Immunosuppressive agents such as methotrexate, cyclosporine, and azathioprine have been used off-label with variable success. Recent evidence

strongly supports the role of Janus kinase (JAK) inhibitors as promising targeted therapies, with baricitinib, ritlecitinib, and deuruxolitinib showing significant hair regrowth in clinical trials. However, challenges remain regarding long-term safety, accessibility, and cost-effectiveness.

SUMMARY

Alopecia areata (AA) is a chronic, autoimmune, non-scarring hair loss disorder with a lifetime risk of nearly 2% in the general population. It presents in various forms, most commonly as patchy hair loss on the scalp, but may progress to alopecia totalis or universalis. Diagnosis is primarily clinical, aided by dermatoscopy and hair pull test, with biopsy or laboratory tests used in uncertain cases.

The condition has a multifactorial etiology involving genetic, immunological, and environmental factors, and is often associated with significant psychosocial stress. Corticosteroids remain the mainstay of therapy, while other immunosuppressants such as methotrexate, cyclosporine, and azathioprine are used off-label. Recently, Janus kinase (JAK) inhibitors including baricitinib, ritlecitinib, and deuruxolitinib have shown promising results, representing a major advancement in treatment.

Overall, AA is a disease with a considerable quality-of-life impact, and newer targeted therapies provide hope for more effective and sustained management.

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