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Review Article

Alopecia Areata: Current and Emerging Treatment Options

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Alopecia areata is an autoimmune condition that can cause patchy hair loss anywhere on the body, though it most often affects the scalp. The medical name for baldness or hair loss is "alopecia," and "areata" denotes that it happens in small, sporadic patches. Loss of immunological privilege in hair follicles causes the condition, and CD8+NKG2D+ T cells release cytokines such as interleukin-15 (IL-15) and interferon-gamma (IFN-γ). They cause inflammation and disrupt normal hair growth by activating the JAK-STAT pathway. The severity of the illness determines the course of treatment. For localised AA, topical and intralesional corticosteroids—especially intralesional triamcinolone—remain widely utilised. Adjunctive treatments include topical minoxidil, bimatoprost, latanoprost, and anthralin. Although side effects and frequent clinic visits may restrict their usage, contact immunotherapy using diphenylcyclopropenone (DPCP) or squaric acid dibutyl ester (SADBE) may be considered for widespread disease. JAK inhibitors have emerged from our growing understanding of the JAK-STAT pathway and represent a significant development in the treatment of AA. The JAK1/JAK2 inhibitor baricitinib, which is licensed for severe AA in adults, showed notable scalp hair regeneration in phase 3 trials. The JAK3/TEC-family kinase inhibitor ritlecitinib has shown sustained long-term effects and is approved for use in adults and adolescents ≥12 years of age. SALT scores have also significantly improved with the specific JAK1/JAK2 inhibitor deuruxolitinib. Other medications, including upadacitinib, brepocitinib, and tofacitinib, are being studied or utilised in specific contexts. Upper respiratory infections, acne, headaches, and test abnormalities such as elevated cholesterol and creatine phosphokinase are common side effects of JAK inhibitors. It is advised to keep an eye on blood counts, liver enzymes, cholesterol, and infection status. Despite these developments, long-term safety, non-response, relapse upon therapy discontinuation, and cost continue to be significant obstacles. New immune-targeted therapies and customised maintenance therapy are the key topics of future research. All things considered, patients with severe AA now have a far wider range of treatment options due to targeted JAK inhibition.

Keywords: alopecia areata, Janus kinase inhibitors, Baricitinib, Ritlecitinib, Beuruxolitinib, Biologic therapy

INTRODUCTION:

The immune system targets hair follicles in alopecia areata (AA), a chronic immune- mediated illness that results in non-scarring hair loss [30], [36]. About 2% of people worldwide are impacted by it at some point in their lives[36]. The activation of cytotoxic T lymphocytes (CD8+NKG2D+) is the primary cause of the pathogenesis, which is the loss of immune privilege in the hair follicle[3][10]. These cells generate interleukin-15 (IL-15) and interferon-gamma (IFN-γ), which communicate via the JAK-STAT pathway (JAK1, JAK2, and JAK3), resulting in an inflammatory feedback loop that stops hair growth and forces follicles into the telogen (resting) phase[19],[38],[50]. AA's clinical manifestation varies greatly. Although acute isolated patches frequently go away on their own, the emergence of secondary patches or failure to go away within six to twelve months indicates a worse prognosis, which may include the development of alopecia totalis (AT) or alopecia universalis (AU) [30]. In addition to greatly lowering quality of life, chronic AA raises the risk of suicidality, anxiety, and depression. Corticosteroids and conventional immunomodulators, which frequently failed to generate long-lasting results in severe cases, were the only available treatments for decades[41]. A "new era" of treatment has begun with the discovery that Janus kinase (JAK) inhibitors are promising treatments due

to recent advances in understanding the immunological mechanisms[31],[32].

Conventional and Topical Treatment Methods

A tiered approach depending on disease severity was used to control AA before the widespread use of JAK inhibitors. Topical and intralesional treatments continue to be the leading choice for mild-to-moderate patients because of their localised effects and low systemic adverse effects[18].

Corticosteroids are used topically and intralesionally.

The most popular first-line treatment is corticosteroids. They work by inhibiting the inflammatory infiltrate that surrounds the hair follicle. Although treatment is less practical for considerable scalp involvement (>50%), intralesional triamcinolone acetonide is quite efficient for patchy AA [7], [37]. To improve results, topical corticosteroids such as mometasone furoate (0.1%) are commonly used, sometimes in conjunction with other medications like bimatoprost or minoxidil [42]. Both mometasone furoate and bimatoprost may be useful, according to a comparison study, although combination therapy frequently produces better outcomes[42].

Alternative and Adjuvant Treatments

Although they do not treat the underlying autoimmune disease, topical minoxidil (5%) and prostaglandin analogues (such as latanoprost and bimatoprost) are frequently used as adjuvant therapy to promote hair growth by prolonging the anagen phase[42]. According to one study, 45% of individuals had thicker, longer lashes after using topical latanoprost[25]. Another topical irritant that is utilised is anthralin, which can seriously irritate and discolour the skin[7]. While more recent adjuvant strategies, like platelet-rich plasma (PRP) and photobiomodulation (low-level laser therapy), are being investigated, the evidence supporting their effectiveness in AA is still weaker than in androgenetic alopecia[48],[53].

Janus Kinase (JAK) Inhibitors' Ascent

Numerous specific oral and topical inhibitors have been developed as a result of the discovery that the JAK-STAT pathway is a key mediator in AA pathogenesis[10],[19]. These substances "turn off" the immunological attack on the hair follicle by blocking the signalling of important cytokines such as IFN-γ, IL-2, IL-7, and IL-15 [19],[50].

Baricitinib

For severe AA in adults, baricitinib, an oral, selective, and reversible JAK1/JAK2 inhibitor, was the first to be approved by the FDA and EMA[2],[49]. Baricitinib 2 mg and 4 mg daily

were shown to be effective in the crucial BRAVE-AAl and BRAVE-AA2 Phase 3

trials[27],[28]. Efficacy Results: At 36 weeks, 38.8% (4 mg) and 22.8% (2 mg) of patients in BRAVE-AA1 and 35.9% (4 mg) and 19.4% (2 mg) of patients in BRAVE-AA2 had a SALT score ≤ 20 [27].

Long-Term Maintenance: At week 104, around 90% of respondents had a SALT score ≤ 20 thanks to ongoing therapy.

Ritlecitinib

Ritlecitinib (PF-06651600) selectively inhibits both the TEC family kinases and JAK3. [8],[20]. Notably, it is authorised for both adults and teenagers (12 years of age and up) [8],[22].

ALLEGRO Trials: In the Phase 2b/3 trials, 23% of patients on Ritlecitinib 50 mg daily had a SALT score ≤ 20 at week 24 [13]. Response rates increased to 60.8%–63.1% at 24 months, according to long-term data [8],[47]. Adolescent Outcomes: In a long-term examination, 78.6% of patients reached the SALT 0–10 category at 24 months, indicating that the medication is particularly effective in adolescents [26].

Deuruxolitinib (CTP-543)

Deuruxolitinib, also known as deuterium-modified ruxolitinib, is a selective JAK1 and JAK2 inhibitor that inhibits the JAK-STAT signalling pathway [14], [40].Results of the Meta- Analysis: At week 24, an evaluation of 1,372 patients revealed substantial changes in SALT scores from baseline (MD = -47.26, $p<0.00001$) [14]. RRs of 93.66 and 65.26 were attained by patients who reduced their SALT by 75% and 90%, respectively [14]. Breakthrough Therapy: Because of their substantial clinical impact, the FDA designated both Ritlecitinib and Deuruxolitinib as breakthrough therapies [2].

Additional Targeted JAK Inhibitors

Tofacitinib: An oral JAK1/JAK3 inhibitor that is frequently used off-label, with response rates in some pediatric cohorts of about 66% [7],[9]. Brepocitinib: After 24 weeks, 64% of patients achieved SALT30, demonstrating the significant efficacy of this JAK1/ TYK2 inhibitor [7],[24].* Upadacitinib: In case studies, this selective JAK1 inhibitor has demonstrated significant or total regrowth in 1-4 months [23],[35].

Emerging Novel Therapies and Biologics

Particularly for patients who do not respond to JAK inhibition, research into alternative targeted pathways is still ongoing.

Biologics Aimed toward Particular Cytokines

There have been conflicting findings in the evaluation of biologics that target inflammatory. pathways common in other disorders for AA [2],[5],[15]. Dupilumab is often less effective than JAK inhibitors for primary hair regrowth, despite its success in case reports for individuals with concomitant atopic dermatitis [5],[43],[51].

Tralokinumab and Secukinumab:

In clinical trials, biologics that target IL-17A and IL-13, respectively, have demonstrated only little efficacy [2],[5].

TNF Inhibitors: Research shows that TNF inhibitors are ineffective and, in certain situations, may even cause AA [18].

Innovative Regulatory T-Cell Treatments

Phase 2b clinical trials are underway for Rezpegaldesleukin (REZPEG), a new regulatory T-cell stimulating treatment [44]. It aims to reestablish immunological tolerance in the hair follicle in patients with severe-to-very-severe AA who are not on JAK inhibitors [44].

Traditional Systemic Immunosuppressants

In cases where JAK inhibitors are unavailable, traditional agents like methotrexate, cyclosporine, and azathioprine are used off- label [22],[46]. A comparative study found that methotrexate with steroid minipulse therapy showed slightly higher treatment efficacy (89.01%) than azathioprine (79.2%) after 6 months [45].

Efficacy and Comparative Analysis

Efficacy in AA trials is primarily measured using the Severity of Alopecia Tool (SALT) [47]. A SALT score ≤ 20 (80% or more scalp hair coverage) is widely considered a clinically. meaningful "success" 1)

Results of Network Meta-Analysis

A hierarchy of oral JAK inhibitor efficacy is provided by network meta-analyses. While Deucolitinib 12 mg and baricitinib 4 mg demonstrate top-tier performance for reaching. SALT75 and SALT90 in the severe population, Brepocitinib 30 mg is very beneficial for initial SALT score reduction [4],[15],[16]. Apremilast and biologics such as dupilumab, on the other hand, are rated far lower [2],[5]. Topical treatments are less effective than oral ways [12].

Regrowth of Eyebrows and Lashes

Baricitinib: From week 52 to week 104, rates of eyelash and eyebrow regrowth increased [11].

Ritlecitinib: At 24 months, eyelash response was 51.2%–62.7% and eyebrow response was 57.6%–61.0% [29].

Ruxolitinib: Up to 97% of scalp, eyebrow, and lash hair regeneration has been reported in case reports [19].

Monitoring, Safety, and Tolerability

In JAK inhibitor trials, the majority of adverse events (AEs) are mild to severe [9],[27].

Typical Adverse Events

Infections: Nasopharyngitis and upper respiratory tract infections (URTIs) affect 10% to 25% of individuals [6],[20].

Dermatologic: Acne is a common adverse effect, especially with baricitinib, Deucoruxolitinib, and Ritlecitinib.

Laboratory: Cholesterol and creatine phosphokinase (CPK) values are frequently elevated [41],[14],[21], [27].

Long-Term Risks and Serious Adverse Events

Serious adverse events are uncommon (0.77%–1.7%) [16]. Although JAK inhibitors have black-box warnings for cardiovascular events and cancers, these risks have not substantially increased in the AA trial groups over the course of one to two years [12].

Requirements for Monitoring

According to guidelines, full blood counts, liver enzymes, lipids, and CPK should be monitored at baseline and regularly [22],[52]. Additionally, screening for viral hepatitis and tuberculosis is crucial [52].

Management of Children and Adolescents

Youth Efficacy

Adolescents (ages 12-17) are permitted to use rilecitinib [22]. Research showed that at 24 months, 78.6% of teenage participants fell into the SALT 0–10 category [26]. In off-label pediatric groups, tofacitinib has demonstrated a 66% response rate [7].

Youth Safety

Adolescents' safety profile is similar to that of adults, with headaches and URTIs being the most frequent adverse events [11]. Serious side effects were uncommon and unrelated to the course of treatment [1].

Unfulfilled Clinical Requirements and Prospects

Relapse and Sustainability

Relapses of the disease are frequently caused by stopping JAK inhibitors [31]. Priorities include figuring out the best maintenance dosage and whether "drug holidays" are feasible [24],[33].

Non-Responders: Even after two years, over one-third of patients do not reach SALT ≤ 20 [29]. A new tactic is to switch between different JAK inhibitors [39].

Psychosocial Effects

To handle the emotional burden of AA, integrated care—including psychological support—is crucial [30],[32].

CONCLUSION

A new era in the treatment of alopecia areata has begun. Deuruxolitinib, Ritlecitinib, and baricitinib have raised the bar for efficacy. For patients with severe disease, targeted JAK inhibition is a game-changer, even though there are still issues with long-term safety, relapse, and access.

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