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Alopecia Areata: Etiopathogenesis, Diagnostics and Treatment – A Literature Review

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Abstract

Background. Alopecia areata is an autoimmune disease affecting approximately 2% of the global population. It is characterized by non-scarring hair-loss and is closely linked to severe mental health issues and other allergic or autoimmune diseases.

Aim. The aim of this review is to summarize epidemiology, pathophysiology, diagnostics and treatment in Alopecia Areata.

Material and methods. This literature review includes articles from PubMed and publishers' websites. The selection criteria specifically targeted clinical and scientific data published from the year 1995 onward, focusing on the management and characteristics of patients diagnosed with alopecia areata.

Results. The review demonstrates that Alopecia areata is driven by inflammatory response. Diagnostic approaches must include screening for other medical conditions and use trichoscopy to assess disease activity..

Conclusions. Alopecia areata requires an early and individualized approach that addresses both physical hair loss and psychiatric comorbidities. Modern targeted therapies offer results for severe cases, though future long-term clinical trials are necessary to establish standardized dosing regimens and minimize relapse rates after treatment discontinuation.

Keywords: alopecia areata, hair loss, trichoscopy, autoimmune disease, pathogenesis, JAK inhibitors, platelet-rich plasma, systemic treatment

1. Introduction

Alopecia areata (AA) is a common autoimmune condition characterized by non-scarring hair loss that affects approximately 2% of the global population. [1] Non-white ethnic groups, particularly people of Asian ethnicity, are more likely to develop alopecia areata. [2] The first onset of disease most commonly occurs in the third or fourth decade of life, although symptoms can appear at any age. The younger the age of onset, the greater the risk of developing a widespread form of disease over the course of a lifetime. [3] The prevalence, incidence and the increase in cases of alopecia areata were found to be higher in women than in men, but an overall decline in the number of cases was observed in the global population for both women and men between 1990 and 2021. However, it is projected to increase by 2050. [4]

The etiopathogenesis of alopecia areata is highly complex, involving genetic predisposition, environmental factors, and immune dysregulation. Key lifestyle and nutritional triggers include smoking, obesity, emotional stress, and microelement deficiencies. At the molecular level, the disease is driven by the collapse of the hair follicle's immune privilege during the active growth phase. The resulting cascade of proinflammatory cytokines, particularly interferon-gamma and various interleukins, activates the JAK/STAT signaling pathway, which prematurely halts the hair growth cycle.

Clinically, alopecia areata presents with high heterogeneity. It ranges from isolated, well-demarcated patches to complete shedding of scalp hair or total body hair. The condition frequently co-occurs with nail abnormalities and severe comorbidities, including thyroid autoimmunity, atopic diseases, and metabolic imbalances. Furthermore, alopecia areata carries a heavy psychological burden, often causing profound emotional distress, anxiety, and sleep disturbances, particularly in younger patients.

Managing the condition effectively requires accurate clinical assessment using specialized tools, hair pull tests, and dermoscopic visualization. While mild, localized presentations respond well to topical treatments, such as localized corticosteroids, minoxidil, or contact immunotherapy, extensive and rapidly progressing cases require aggressive systemic interventions. Traditional therapies include oral glucocorticoids and classic immunosuppressants. However, these often lead to high recurrence rates and adverse side effects.

Targeted therapies, such as Janus kinase (JAK) inhibitors and cytokine-targeting biologics have transformed the treatment of severe cases. Nevertheless, long-term safety data and permanent efficacy profiles for these new interventions remain limited.

This review provides a comprehensive analysis of the pathogenesis, diagnosis, and current treatment options for alopecia areata, establishing a framework for standardized, personalized clinical management.

2. Research materials and methods

This study is a literature review focused on the causes, symptoms, and treatment options for alopecia areata. To find relevant scientific data, a comprehensive search was performed across major medical databases, including PubMed and various academic publishers' websites. The search used specific keywords related to the topic, such as "alopecia areata", "pathogenesis", "JAK inhibitors", "biologics", and "clinical management". The selection criteria targeted peer-reviewed articles and clinical studies published from the year 1995 onward. This timeframe was chosen to focus on modern treatments and up-to-date scientific findings. The collected data were then analyzed and compared to create a clear overview of the current knowledge about the disease.

3. Research results

3.1. Etiopathogenesis

Alopecia areata is an autoimmune condition, characterized by nonscarring hair-loss with multiple factors involved in its etiopathogenesis. The etiology is not entirely known, but evidence suggests environmental, immunological, and genetic factors are involved. A positive family history or coexisting medical conditions may increase the likelihood of developing alopecia areata. [6] Family history of alopecia areata increases the likelihood of developing the disease – approximately 48% of patients have relatives with this condition, compared to 2% of the general population. The inheritance pattern of alopecia areata is complex. A 55% genetic concordance rate has been observed in monozygotic twins and a tenfold increase in risk has been noted among first-degree relatives of affected individuals. Moreover, deficiencies of iron, folate, zinc, selenium and vitamin D are associated with the onset of alopecia areata, as well as changes in its course and severity. Smoking increases the risk of developing alopecia areata due to increased level of proinflammatory cytokines. Alcohol consumption may contribute to immune dysregulation associated with alopecia areata, but some studies suggest that moderate consumption may reduce psychological stress and have a positive effect on the course of the disease. Obesity, which exacerbates various inflammatory skin conditions such as psoriasis or atopic dermatitis, may increase the risk of alopecia areata, likely through IL-17-mediated inflammation. Furthermore, mental health is an important factor regarding alopecia areata - 23% of patients reported an emotional crisis or traumatic event prior to the onset of exacerbation of the disease. [5] About 70% of patients with alopecia areata also suffer from mental disorders - patients in different age groups exhibit different trends in the prevalence of mental disorders. In patients before the age of 14 with alopecia areata, a link has been demonstrated between the presence of alopecia areata and anxiety and other mental disorders, however, in patients in whom alopecia areata developed after the age of 14, no correlation with these conditions was found. [4] Moreover, patients with alopecia areata may have comorbid conditions, including autoimmune diseases and atopic disorders, such as hay fever, asthma, atopic dermatitis. The most common comorbidities included hyperlipidemia (22.4%), hypertension (21.8%), thyroid disorders (13.1%), contact dermatitis or eczema (10.8%), depression (9.5%), and anxiety (8.4%). Comorbid autoimmune diseases included atopic dermatitis (2.8%), psoriasis (2.1%), chronic urticaria (1.5%), and rheumatoid arthritis (1.1%). [9] Patients with concomitant atopic

dermatitis, bronchial asthma, or Hashimoto's thyroiditis were significantly more likely to present with early-onset, severe, or prolonged alopecia areata compared to patients with alopecia areata alone. Coexisting bronchial asthma was associated with a higher risk of early onset, severe, or prolonged alopecia areata than coexisting atopic dermatitis. The coexistence of skin diseases showed a stronger association with the duration of alopecia areata than with age at onset or severity. [7] Therefore, patients with alopecia areata should be screened for coexisting autoimmune diseases due to the potential for a more severe course of the disease and the possibility that systemic and other immunotherapies may be required. Early identification may improve overall treatment outcomes. [8]

There are three phases of hair growth cycle: anagen, catagen and telogen. The anagen phase is the growth phase, during which the hair follicle produces the hair shaft. This phase can be divided into the proanagen and metanagen phases. During the proanagen phase, hair progenitor cells proliferate and the differentiation process begins. The metanagen phase begins when a new hair shaft appears on the skin's surface. The entire anagen phase can last up to several years. The catagen phase begins at the end of the anagen phase. It is characterized by regression of the hair follicles, which lose about one-sixth of its standard diameter. It lasts several weeks. The telogen phase is the resting phase. About 10-15% of the body's hair is in this phase. It lasts from 1 to 6 months. When another hairs re-enter anagen phase, hairs in telogen phase are extruded from the follicle, making space for a new anagen hair. [10, 11]

The immune system changes due to various reasons, immune cells can cause the inflammation of the hair follicle, resulting in the development of alopecia areata.

In healthy individuals, the absence of alopecia areata is ensured by gaining by hair follicles immune-privileged status during the anagen phase, which protects anagen hair follicles against hair loss. This includes physical factors such as rich extracellular matrix and the lack of lymphatic vessels in the hair follicle region, local immunoinhibitory environment created by immunosuppressive molecules, but also NK cells suppression and downregulation of MHC. [5]

In alopecia areata the disruption of the immune-privileged of the hair follicle is occurred. In predisposed individuals, increased expression of MHC class I and II molecules, adhesion molecules and NKG2D ligands intensifies the activation of the defence mechanisms. Consequently, T lymphocytes migrate to the lower part of the hair follicle. This leads to the development of an autoimmune response in which autoreactive T lymphocytes play a particular role – primarily cytotoxic CD8+ T cells, especially the NKG2D+ subpopulation, CD4+ helper

T cells such as Th1, Th2 and Th17, but also regulatory T cells, NK cells and plasmacytoid dendritic cells. These cells, upon infiltrating the hair follicle during the anagen phase, initiate an inflammatory reaction by releasing proinflammatory cytokines, such as IFN-γ, IL-2, which leads to the further recruitment of immune cells and an intensified inflammatory response, resulting in damage and collapse of the hair follicle and induce keratinocyte apoptosis. IFN-γ plays a particularly important role, as it inhibits the hair growth cycle and contributes to premature hair loss. As a result, the structure of the hair follicle is damaged, hair growth is inhibited, and dystrophic hair develops. Severe inflammation may also lead to premature transition of the follicle from the anagen to the telogen phase, resulting in increased hair loss. [5]

At the molecular level, the activation of the JAK/STAT signalling pathway by IL-15 and other pro-inflammatory cytokines plays a significant role. This mechanism is involved in both the initiation and maintenance of the disease, as confirmed by experimental studies that inhibition of JAK-STAT signalling using JAK inhibitors led to hair regrowth. Additionally, a positive feedback loop between IFN-γ and IL-15 has been demonstrated, which sustains the activation of autoreactive T lymphocytes. [5]

Increasing evidence indicates that both innate and adaptive immune mechanisms are involved in the pathogenesis of alopecia areata. Plasmacytoid dendritic cells regulate the activity of T lymphocytes, myeloid dendritic cells, mast cells, NK cells and B lymphocytes, promoting their activation, differentiation, and production of cytokines and chemokines, amplifying the inflammation. Furthermore, autoantigens directed against hair follicle components, such as hair-specific keratin and trichohyalin, have been detected in patients with alopecia areata, however, their exact role in disease pathogenesis remains not fully understood. [5]

3.2. Clinical presentation

Alopecia areata can be a single episode or have a recurrent course. Mostly the first symptom is a single, enlarging patch of non-scarring hair loss, followed by the appearance of additional patches, which may merge with one another. [12]

Types of Alopecia Areata:

Patchy Alopecia Areata

Alopecia Universalis

Alopecia Totalis

Ophiasis Alopecia

Ophiasis Inversus (Sisaipho)

Alopecia Incognita

Diffuse Alopecia Areata

Marie Antoinette Syndrome (Canities Subita)

Patchy Alopecia Areata is the most common type of alopecia areata - it is characterized by the presence of one or more coin-sized patches on the scalp or other parts of the body. Alopecia Universalis involves complete hair loss over the entire body, including the eyebrows and eyelashes. Alopecia Totalis involves complete hair loss on the scalp. Alopecia in an ophiasis pattern is characterized by band-like hair loss in the temporal and occipital regions. Ophiasis Inversus (Sisaipho) is a rare type, the inverse of the ophiasis pattern - hair loss occurs in the central part of the scalp, sparing the temporal and occipital areas. Alopecia Incognita is a diffuse hair loss with positive pull test, short, miniaturized regrowing hairs and yellow dots, without nail involvement. Diffuse Alopecia is characterized by sudden, extensive thinning of the hair, involving the entire scalp - typically, hair regrows within a few months. Moreover, Diffuse Alopecia Areata may present as an acute episode in which the immune system destroys melanocytes in the hair follicles within a matter of weeks, leaving the unpigmented, white hair - a condition known as Marie Antoinette Syndrome (Canities Subita). [13]

Trichoscopy can reveal features of alopecia areata, although some of them may also be present in other types of alopecia. These features include: exclamation mark hairs, yellow dots, black dots, broken hairs, short vellus hairs. Exclamation mark hairs are common trichoscopic finding of alopecia areata, which indicates the active phase of the disease - hairs are thinner at the base and wider at the tip. Yellow dots are one of the most sensitive trichoscopic features of alopecia areata, although they are not easy to observe on pigmented skin. These are empty hair follicle openings filled with keratotic material and sebum. They are not identified as markers of active disease. Furthermore, they may also occur in androgenic alopecia, trichotillomania and connective tissue inflammation. [15, 16, 17, 18] Black dots may be useful, but nonspecific feature of active alopecia areata. They are remnants of broken hairs inside the follicle, which appear as small, black dots. This sign is observed in people with dark hair and may also occur in diseases such as trichotillomania, tinea capitis, dissecting cellulitis. [16, 19] Broken hairs are

also common findings observed in trichoscopy. They are fragments of pigmented hair shafts. Their prevalence is higher during the active phase of the alopecia areata, which may serve as an indicator of disease activity. [16, 20] Clinically, their disappearance indicates good treatment response. [21] Broken hairs at equal lengths are characteristic of alopecia areata, while broken hairs at varying lengths are characteristic of trichotillomania. [22] Short vellus hairs are thin, regrowing, nonpigmented white hairs. Like yellow dots, they are also considered as one of the most sensitive alopecia areata features. They are not specific to alopecia areata and may occur in androgenetic alopecia, trichotillomania, tinea capitis, traction alopecia, traumatic alopecia, congenital triangular alopecia, chronic and acute telogen effluvium, and primary cicatricial alopecia. [16, 23]

In diagnostically unclear cases, a skin biopsy is recommended. The histopathology of alopecia areata is characterized by stage-dependent features. In the acute and subacute stages, a “swarm of bees” pattern is observed, composed of CD4+ and CD8+ T lymphocytes surrounding hair follicles in the anagen phase. Furthermore, there is a transition from the catagen phase to telogen phase, with miniaturization of the hair follicles. In the chronic stage, inflammation may or may not be present, but the number of hairs in the catagen or telogen phase increases and pigment loss occurs. In the recovery stage, minimal inflammation is present and the number of hairs in the anagen phase increases. [14]

Moreover, nail changes are observed in approximately 20% of patients with alopecia areata and are more commonly associated with severe forms, such as alopecia totalis and alopecia universalis. The most common nail changes include pitting, longitudinal ridges, trachyonychia, and leukonychia. A red-mottled lunula is rare but appears to be associated with a more severe course of the disease. The most common nail symptom of AA is pitting. Nail pitting refers to punctate erosion on the surface of the nail plate. Pits in alopecia areata, unlike in nail psoriasis, are very superficial and often go unnoticed. There is a wavy or undulating appearance of the nail plate surface rather than erosions and a geometric, grid-like pattern. [24]

The severity of the alopecia areata is assessed by Severity of Alopecia Tool (SALT) and Alopecia Areata Score (AAS). The SALT score is a standardized clinician-reported measure quantifying scalp hair loss percentage in alopecia areata, ranging from 0-100%. A SALT score 1-20 corresponds to limited disease, 21-49 to moderate disease, 50-94 to severe disease, and 95-100 to very severe disease. [12, 25]

3.3. Treatment

Treatment of alopecia areata involves multiple topical and systemic options. The main indication for topical treatment is low severity alopecia areata (SALT $\leq$ 20) and slow progression. The indication for systemic treatment as first-line therapy is, in most cases, SALT 20 or higher. [31] Topical treatments include topical corticosteroids, intralesional corticosteroids, topical minoxidil, topical immunotherapy. Systemic treatments include oral corticosteroids, oral minoxidil, cyclosporine, JAK inhibitors (especially Baricitinib), cytokine-targeting biologics. [26, 27, 28] Treatment efficacy varies by disease severity, with topical corticosteroids dominating mild disease, topical immunotherapy leading moderate-to-severe disease, and systemic corticosteroids and JAK inhibitors showing superior outcomes in severe alopecia areata. For mild disease, intralesional corticosteroids achieved the highest efficacy, followed by topical corticosteroids. For moderate-to-severe disease, topical immunotherapy ranked highest, outperforming topical corticosteroids. For severe cases, tofacitinib, and possibly other JAK inhibitors, are very promising therapies. Minoxidil 5% solution was associated with better response to therapies in severe alopecia areata cases. Among cytokine-targeting biologics, dupilumab showed significant regrowth in 89% of cases and ustekinumab in 100% of patients. [28, 29, 30]

3.3.1. Topical treatments

Topical corticosteroids are used as a first-line treatment for mild to moderate alopecia areata. They may be used as monotherapy. Topical corticosteroids treat alopecia areata by reducing the immune attack on hair follicles, which is mediated by CD8+ T cells, due to their anti-inflammatory and immunosuppressive effects. A cream, lotion, or ointment containing 0.05% betamethasone dipropionate is typically used. If no improvement is seen after 3 months of use, clinicians usually discontinue this treatment. If improvement is noticeable, the patient continues using betamethasone, gradually reducing the dose. Side effects may include skin thinning, skin hypersensitivity, allergic reactions, hypopigmentation, and telangiectasia. [25, 26, 32, 33]

Intralesional corticosteroids such as triamcinolone acetonide are administered at concentrations ranging from 2.5-10 mg/mL for the scalp and 2.5-5 mg/mL for the face, applied to the scalp every 4 to 6 weeks for 6 months. It is a well-established treatment for limited-type alopecia areata, with response rates ranging from 60% to 95% across 12 studies. Side effects similar to those associated with topical corticosteroid therapy may occur. [25, 32]

Minoxidil supports hair regrowth by improving blood flow to hair follicles. Its effects are mediated through multiple mechanisms and includes stimulation of vascular endothelial growth factor (VEGF), extension of the anagen phase, and activation of potassium channels, all of which contribute to improved follicular perfusion and nutrient delivery. 5% topical minoxidil is characterized by significantly higher efficacy compared to minoxidil at a lower concentration. Side effects include hypertrichosis, dryness, postural hypotension, scalp irritation, dizziness, headache. Studies found no greater efficacy of oral minoxidil at a daily dose of 10 mg compared with 5% topical minoxidil in patients with alopecia areata. [26, 34, 35, 36]

The exact mechanism of action of topical immunotherapy in alopecia areata remains unknown, but it has been suggested that it involves the induction of allergic contact dermatitis at the application area through delayed-type hypersensitivity. The most commonly prescribed substances are dinitrochlorobenzene (DNCB), squaric acid dibutylester (SADBE), and diphenylcyclopropenone (DPCP). They are available as a powder or a solution in acetone. Initially, the patient is sensitized with a 2% solution for two weeks. Treatment then begins, starting with the lowest concentration. One side of the scalp is not treated with topical immunotherapy and remains as a control. The dose is typically increased weekly. Once hair regrowth is observed, the substance is applied to the entire scalp. If no hair regrowth is observed after 6 months, treatment should be discontinued. Adverse effects of topical immunotherapy include redness, severe itching, flu-like symptoms, lymphadenopathy, hyper or hypopigmentation. [26, 36]

3.3.2. Systemic treatments

Oral corticosteroids, such as prednisolone, are currently considered the first-line treatment for moderate to very severe alopecia areata. Treatment can be administered orally, intramuscularly, or intravenously pulse therapy. Treatment algorithms and efficacy vary across studies. Prednisolone was the preferred systemic corticosteroid, administered once daily, starting at a dose of 0.4–0.6 mg/kg/day, with gradual dose tapering over 6–12 weeks. Side effects of oral corticosteroids are weight gain, hypertension, swelling, fatigue, muscle weakness, thinning of skin. [25, 26]

Cyclosporine is a calcineurin inhibitor, originally used to prevent rejection of allogeneic transplants, as well as to treat many autoimmune diseases. In alopecia areata, it can be used both as monotherapy and in combination with systemic corticosteroids. It is suggested that the

optimal dose of cyclosporine in monotherapy is approximately 5 mg/kg/day, while in combination therapy with steroids it is approximately 3 mg/kg/day. It has also been noted that a longer duration of treatment may reduce the risk of disease recurrence. Available data indicate that cyclosporine combined with corticosteroids is more effective than cyclosporine alone. Importantly, the combination of these drugs allows for the use of lower cyclosporine doses, while significantly reducing the risk of disease recurrence, particularly in severe forms of alopecia areata. It has also been demonstrated that while the addition of corticosteroids only slightly increases treatment efficacy, it significantly reduces the frequency of relapses. Side effects of cyclosporine include gastrointestinal problems, hypertrichosis, hypertension, arrhythmia, decreased GFR, anxiety, headache, fever, increased occurrence of malignancies and infection. [26, 37, 38]

Janus kinase (JAK) inhibitors can be divided into two generations. First-generation drugs include tofacitinib, baricitinib, and ruxolitinib, while the second generation is represented by ritlecitinib and abrocitinib. The indication for their use is severe alopecia areata with more than 50% hair loss. The mechanism of action of JAK inhibitors involves blocking the JAK/STAT signalling pathway, which leads to the inhibition of the T-cell-mediated immune response directed against hair follicles. They also promote the activation of hair follicle stem cells and hair regrowth. Oral baricitinib is a selective and reversible inhibitor of JAK1 and JAK2 that suppresses the activation of T lymphocytes. Treatment typically requires long-term use to maintain therapeutic effects. The initial dose is usually 2 mg daily, with the option to increase to 4 mg daily if the response is insufficient after 3 months. In patients with severe symptoms, initiating therapy at a dose of 4 mg daily may be considered. Once a satisfactory response is achieved, the dose is reduced to 2 mg. However, if there is a lack of improvement after 6 months, treatment should be discontinued. Adverse effects of JAK inhibitors includes infections, especially viral, fungal and mycobacterial infectious disorders, bone marrow suppression, headache, increased risk of lymphoma and other malignancies, pulmonary embolism. [26, 32, 39]

Cytokine-targeting biologics remain an important treatment option, particularly for patients who cannot tolerate JAK inhibitors or for whom their use is contraindicated. Cytokine-targeted biologics include: Dupilumab, Secukinumab, Tralokinumab, Etanercept, Ustekinumab, Infliximab, Adalimumab and Tildrakizumab.

Dupilumab is a human IgG4 monoclonal antibody that binds to the IL-4Rα subunit, blocking the binding of interleukins IL-4 and IL-13 to their receptors. As a result, there is a reduction in the Th2-type inflammatory response and inhibition of the release of mediators, such as chemokines and immunoglobulin E. Administration of Dupilumab every two weeks led to complete or nearly complete hair regrowth in many patients within 2 to 24 months. In a phase IIa trial study, adverse events occurred in 25-26.5% of patients and included injection site reactions, conjunctivitis, upper respiratory tract infections and urinary tract infections. In case reports, dupilumab was generally well tolerated, and the observed adverse effects included Schamberg disease, dry eye, and conjunctivitis. Although dupilumab usually demonstrates high efficacy in the treatment of alopecia areata, isolated cases of hair loss developing during therapy with this drug have been reported. These changes usually appear several months after the start of treatment. It is unclear whether they represent a classic form of alopecia areata or a distinct form of drug-induced alopecia. It is hypothesized that dupilumab's mechanism of action shifts the immune response toward a Th1 profile and may promote hair loss. However, the exact cause of this phenomenon remains unknown. [28]

Ustekinumab is a human IgG1 monoclonal antibody targeting p40 subunit of interleukins IL-12 and IL-23. Its mechanism of action involves blocking the binding of these interleukins to the IL-12Rβ1 receptor, preventing the activation of the p35 (IL-12) and p19 (IL-23) and the subsequent transmission of pro-inflammatory signals. In case reports of patients treated with Ustekinumab 90 mg subcutaneously, significant hair regrowth was observed and improvement was typically achieved after approximately 3-4 months of therapy. The treatment was well tolerated, and no adverse effects were reported. [28]

Both Dupilumab and Ustekinumab demonstrate high therapeutic efficacy. Significant hair regrowth was observed in approximately 89% of patients with Dupilumab, while Ustekinumab was effective in all reported patients. However, further studies are necessary to optimize treatment algorithms. [28]

3.3.3. Other treatments

Platelet-rich plasma (PRP) is an autologous blood product with a baseline-above concentration of platelets, used therapeutically to deliver growth factors that promote tissue healing. It is a method widely utilized in wound healing, orthopaedics, gynaecology, as well as dermatology. PRP therapy is applied in various types of alopecia, such as androgenetic alopecia, alopecia

areata, and lichen planopilaris. The regenerative effects of PRP are primarily mediated through the release of multiple growth factors from activated platelets, which collectively enhance hair follicle proliferation, angiogenesis, and prolongation of the anagen phase of the hair cycle. Crucial to these processes is the complex composition of PRP, which contains both pro-angiogenic factors (VEGF, transforming growth factor-β and basic fibroblast growth factor) and anti-angiogenic mediators (platelet factor 4 and TSP-1). The net biological effects of these components depend on the platelet activation status and receptor-mediated signalling. Furthermore, in alopecia areata, transforming growth factor-β (TGF-β) modulates immune responses by suppressing monocyte chemoattractant protein-1 (MCP-1), thereby reducing immune cell infiltration into the follicular microenvironment, which may help reduce inflammation. [40] Evidence from multiple studies demonstrates PRP's consistent efficacy in treating alopecia areata. Systematic reviews and meta-analyses show that PRP is superior to placebo, comparable to triamcinolone acetonide, and more effective than 5% minoxidil. [40, 41, 42, 43] While individual response rates vary, a broad review of 32 studies confirms PRP's strong performance both as a monotherapy and alongside conventional treatments. Furthermore, side effects remain minimal and mostly minor, including burning sensation, pain during injection, erythema, edema, headache. [40]

Phototherapy, which includes psoralen and ultraviolet A (PUVA) therapy, represents a therapeutic approach for managing severe, hard-to-treat forms of alopecia areata, including alopecia totalis and alopecia universalis. However, there is still a lack of sufficient data regarding its long-term benefits and safety profile. PUVA works through localized immunomodulation. By combining a light-sensitizing medication (psoralen) with ultraviolet A radiation, the treatment penetrates the skin to clear away the inflammatory cells surrounding the hair follicles. This action halts the autoimmune attack and allows the hair follicles to re-enter their normal growth phase. PUVA shows variable efficacy for alopecia areata, reflecting conflicting evidence about its clinical utility. Additionally, the treatment carries notable side effects like skin redness, burning, and itching. Therefore, the increased risk of skin malignancies associated with PUVA therapy remains a significant concern to be investigated and addressed. [44]

4. Discussion

Presented data show that alopecia areata (AA) is a complex disease, affecting about 2% of the world's population. Even though global cases slightly decreased between 1990 and 2021, numbers are expected to rise by the year 2050. AA is not just an aesthetic problem. About 70% of patients also suffer from mental disorders. Interestingly, there is a strong link between hair loss and anxiety or other mental issues in children who develop the disease before the age of 14, but this link is not found in patients who get the disease later in life. Also, patients with other health issues like asthma, atopic dermatitis, or Hashimoto's thyroiditis usually suffer from earlier, longer, and more severe hair loss. Because of this, doctors should always screen patients for other autoimmune diseases to start the right treatment as early as possible. At the biomolecular level, AA happens because of the collapse of the hair follicle's immune privilege during the active growth phase. Immune cells, especially CD8+ T cells, attack the lower part of the hair follicle. They release inflammatory proteins, such as interferon-gamma (IFN-gamma) and IL-2. This infiltration triggers a cascade of pro-inflammatory cytokines, where interferon-gamma plays a pivotal role in arresting the hair growth cycle and inducing premature catagen transition. The identification of the JAK/STAT signalling pathway, specifically sustained by a powerful feedback loop between interleukin-15 and interferon-gamma, provides a direct molecular target for advanced therapeutic modalities. The choice of treatment depends on how much hair the patient has lost, which is measured by the SALT score. For mild-to-moderate presentations (SALT≤20) topical treatments remain the first-line gold standard. Topical corticosteroids, such as 0.05% betamethasone dipropionate, and intralesional injections of triamcinolone acetonide manage the disease locally by suppressing T-cell-mediated follicular damage, though their clinical utility is frequently limited by long-term localized side effects like skin thinning and telangiectasia. To optimize hair regrowth, these anti-inflammatory agents are routinely combined with topical minoxidil (preferentially at a 5% concentration), which enhances follicular perfusion and extends the anagen phase through vascular endothelial growth factor (VEGF) stimulation and potassium channel activation. For patients with limited but resistant patches, topical immunotherapy using sensitizing agents like DPCP, SADBE, or DNCB offers an alternative approach by deliberately inducing a delayed-type hypersensitivity reaction to divert the autoimmune attack away from the hair follicle. For severe cases where patients lose more than 50% of their hair, systemic treatment is necessary. Oral corticosteroids like prednisolone have been used as a first-line treatment for severe AA to achieve quick

suppression of the disease. However, their long-term use is highly limited by significant adverse effects, including hypertension, weight gain, and muscle weakness. Cyclosporine works well in severe forms, especially when combined with systemic steroids, because this combination achieves higher efficacy and significantly reduces the frequency of disease relapses. Unfortunately, cyclosporine also carries heavy risks, such as decreased GFR and arterial hypertension. The Janus kinase inhibitors have completely revolutionized the prognosis for patients with more than 50% hair loss. First-generation drugs (such as baricitinib and tofacitinib) and second-generation drugs (such as ritlecitinib) directly block the JAK/STAT signalling pathway, which stops the T-cell-mediated immune response and activates hair follicle stem cells for regrowth. Oral baricitinib, for example, is highly effective but typically requires long-term use to maintain results, starting at 4 mg daily for severe cases and tapering down to 2 mg once hair regrows. Despite their superior outcomes, JAK inhibitors require strict medical monitoring due to serious potential side effects, including bone marrow suppression, viral or fungal infections, and pulmonary embolism. For severe patients who cannot tolerate or have contraindications to JAK inhibitors, monoclonal antibodies like dupilumab and ustekinumab have shown remarkable success, achieving significant hair regrowth in 89% and 100% of reported severe cases. Isolated cases have reported a paradoxical hair loss during dupilumab therapy, possibly due to an immune shift toward a Th1 profile, however, these biologics are generally very well tolerated. Platelet-Rich Plasma (PRP) therapy by releasing crucial growth factors, such as VEGF and TGF-beta, reduces local inflammation and enhances follicular cell proliferation, helping severe patients achieve better regrowth without any systemic toxicity. Phototherapies such as PUVA phototherapy are still used for severe alopecia, though they lack long-term safety data and carry a risk of skin malignancies.

5. Conclusions

Alopecia areata remains a highly challenging autoimmune disease, despite various available treatments. The findings are highly significant for clinical practice as they underscore that alopecia areata is not only a cosmetic issue, but a condition deeply linked with atopic, autoimmune, and psychiatric comorbidities, particularly in early-onset patients, which requires a personalized diagnostic approach. Treatment must be carefully tailored to the individual based on disease severity - while localized therapies like topical corticosteroids and minoxidil remain effective for mild cases, advanced systemic interventions including Janus kinase inhibitors,

cytokine-targeted biologics offer results for severe forms. However, the analysed data is lacking long-term evidence regarding the safety and permanent efficacy of these modern treatments. Future research should focus on conducting long-term clinical trials to establish standardized dosing routines for new medications. Additionally, researchers should explore the efficacy of combined therapies, such as mixing modern systemic drugs with regenerative methods, to achieve long-term remission and reduce side effects.

Disclossure

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