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Alopecia Areata Perspectives

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Alopecia Areata: The Clinician and Patient Voice

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Target Audience

This activity is intended for dermatologists, primary care physicians, nurse practitioners, physician assistants, and other healthcare professionals involved in the care of patients with alopecia areata (AA).

Goal Statement

The goal of this activity is for learners to be better able to diagnose and treat patients with AA.

Learning Objectives

Upon completion of this activity, participants will:

- Have increased knowledge regarding the evidence-based treatment options for AA
- Have increased competence with regard to counseling patients with AA

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Alopecia Areata: The Clinician and Patient Voice

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ABSTRACT

Alopecia areata (AA), an autoimmune disorder of hair follicles, results in varying degrees of scalp, facial, and body hair loss. In addition, it is associated with profound psychosocial and quality-of-life impairments, which can lead to anxiety and depression. The clinical course is unpredictable, with spontaneous remissions and relapses. There is no cure, and current treatments are limited by their efficacy, safety, and high relapse rates after discontinuation. This article reviews clinician and patient perspectives on AA, based on clinician and physician surveys, and discusses the unmet needs and gaps in care.

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INTRODUCTION

Alopecia areata (AA) was once considered a mere cosmetic condition. Now, we know that AA is a disease that profoundly impacts patients' lives.[1] Alopecia areata not only causes psychosocial distress but may also lead to depression and anxiety.[2] All too often, we hear patients with AA say, "*I am ashamed to go out in public; I have no eyebrows or eyelashes!*" or "*People stare at my bald patches; it is humiliating!*" How can we alleviate the distress and better care for our patients with AA?

This article began as two separate surveys: one survey for patients with AA and one survey for physicians developed by Antonella Tosti MD, a world-renowned dermatologist known for originating trichoscopy, which is a noninvasive method for diagnosing and assessing the severity of hair disorders.[3] Gathering responses from nearly 2500 physicians, primarily dermatologists and primary care physicians in community practices, the physician survey captured physicians' attitudes, knowledge, and beliefs around practice patterns for AA. Most respondents were actively managing patients with AA, 21% saw 16 or more patients with AA in a month, 37% saw 6 to 15 patients, and 35% saw 1 to 5 patients. The 40-patient survey, including responses from patients and caregivers, captured the impact of AA on patients' lives as well as patients' medical care and barriers to care. For patients surveyed, the extent of hair loss varied from mild (30%) to moderate (32%) to severe (38%), and two-thirds of patients reported hair loss often or all the time.

In this interview, Antonella Tosti MD reflects on the survey data, as well as her personal experiences, and highlights unmet needs in AA management and care.

DISCUSSION

There are several types of hair loss, but how do you define alopecia areata?

Alopecia areata is an autoimmune disorder of the hair follicles, characterized by sudden-onset, remitting-relapsing, nonscarring hair loss at any hair-bearing site (ie, scalp, eyebrows, eyelashes, beard, body hair).[4] Hair loss is variable from discrete bald patches to diffuse thinning to total hair loss involving the scalp (alopecia totalis) or total body (alopecia universalis) in severe cases. The patchy type localized to the scalp is the most common[4] and initially presents as a single patch in most cases.[5] Alopecia areata can also present with nail abnormalities, usually nail pitting. Nail involvement occurs in 10% to 15% of dermatology referral cases and is usually associated with severe forms of AA.[4]

As the second most common nonscarring alopecia,[4] AA can occur at any age, with the highest prevalence in patients between 30 and 49 years.[6] Men and women were thought to be equally affected, but recent data suggest that women have a 30% higher prevalence. Similarly, historical data suggested no ethnic differences in susceptibility, but newer data suggest a higher prevalence in Asian patients followed by Black and Hispanic patients compared with White patients.[6]

What makes a patient suspect that they may have alopecia?

Patients may suspect they have alopecia when they notice sudden patchy hair loss or more-than-usual hair loss. For example, it is very common to hear patients say, "*I got up one morning, and there were clumps of hair on my pillow,*" or "*When I was washing my hair, I saw clumps of hair on my bathroom floor*." Others may have been told by a family member, a friend, or their hairdresser that they have bald patches on their scalp. It is important to note here that the onset of hair loss is sudden, distinguishing AA from other types of hair loss with a slow onset.

How does alopecia areata impact patients?

Alopecia areata has a profound negative impact on many aspects of patients' lives, including their social and family lives, intimate relationships and sex lives, and work or school performance; all of which exert a massive toll on their emotional and mental well-being.[1,7,8] Coping with AA is a daily challenge. In our patient survey, 70% of patients reported AA having a large or very large impact on their emotional and mental well-being. More worryingly, AA may lead to depression and anxiety. A recent study reported that one-third of patients have symptoms of depression and anxiety, while up to one-fifth of patients have depressive or anxiety disorders that require psychiatric care.[2] The significant psychological toll of AA does not appear to lessen over time.[1] Even during times of complete hair regrowth, patients live with the fear and anxiety of a relapse.

Coping with the disorder, specifically concealing the bald patches, takes a considerable time investment for patients, which may have been previously underrecognized.[1] On average, patients spend about 10 hours per week concealing hair loss. The time investment and the disease burden extend beyond the patient to include caregivers as well, as exemplified in this narrative from one patient's mother:

"*Helping my girl to survive the disease has been extremely difficult because as she got worse, we had to go shopping for weeks to try and find things to cover the patches, eyebrows, and eyelashes, and find somebody to tattoo the eyebrows.*"

Aside from hair growth, "not using or having to consider using a head covering or wig" was what the patients we surveyed most wanted from treatment, followed by "feeling more confident."

Is there an underappreciation of the burden of alopecia areata on patients?

Based on responses to our clinician survey, dermatologists and primary care physicians appear to underestimate—to some degree—the impact of AA on patients. This is reflected in patient anecdotes about interactions with clinicians where they hear statements such as:

"*This is not cancer. So, don't get so nervous. It's going to go away*" or "*It's just stress. Try to relax*," which is terrible for patients because they feel like, "*Oh, it's my fault. It's because I'm stressed that I'm getting this disease that's not going away*."

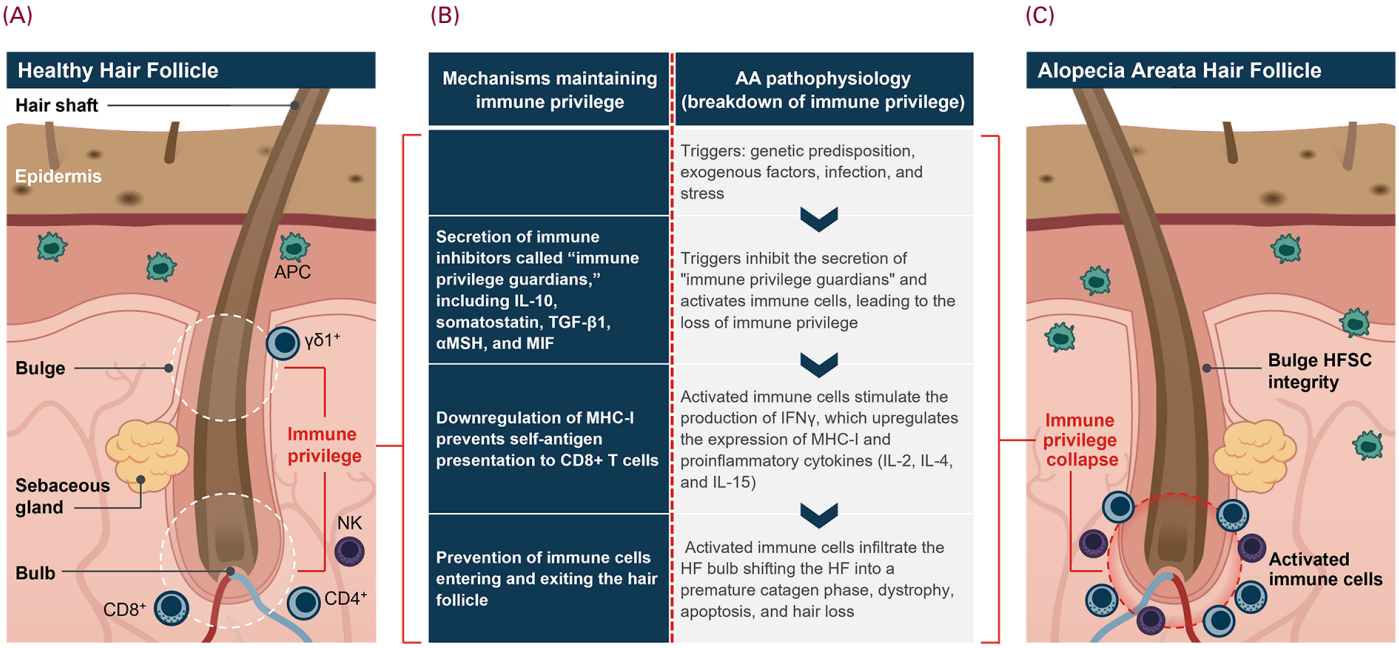
Patients with AA often feel that they are to blame for their condition. And historically, there has been poor education about disease burden in medical school curricula and residencies, which may partly explain the underappreciation of the disease burden seen in our survey.

Why is it important to understand the pathogenesis of alopecia areata?

Most clinicians know that AA is an autoimmune disorder but may be less familiar with its pathogenesis. Knowledge of AA pathogenesis is important to understand the targets and mechanism of action of immunotherapies for AA. However, there is a level of complexity surrounding AA's pathogenesis that can be off-putting. Explaining the pathogenesis in a simplistic way may be helpful.

Central to the pathogenesis of AA is loss of immune privilege of the hair follicle (HF).[9,10] Immune privilege is the suppression of an immune response to antigens in a localized area. In a healthy HF, immune privilege is localized to the hair bulge and anagen phase of the hair bulb, where it protects the HF from inflammatory processes and promotes immune tolerance (Figure 1a). Several mechanisms are in place to maintain the integrity of immune privilege: (1) secretion of immune inhibitors by the HF epithelium, such as interleukin (IL)-10, somatostatin, transforming growth factor beta-1 (TGF-β1), melanocyte-stimulating hormone alpha (αMSH), and macrophage migration inhibitory factor (MIF), which are referred to as "immune privilege guardians"; (2) downregulation of major histocompatibility complex I (MHC-I), preventing self-antigen presentation to CD8+ T cells; and (3) prevention of immune cells entering and exiting the HF through the HF extracellular matrix that serves as a barrier[10] (Figure 1b).

FIGURE 1. Schematic illustration of the breakdown of immune privilege in AA pathophysiology.[9,10]



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αMSH, melanocyte-stimulating hormone alpha; AA, alopecia areata; APC, antigen-presenting cell; HF, hair follicle; HFSC, hair follicle stem cell; IFNγ, interferon gamma; IL, interleukin; MHC-I, major histocompatibility complex I; MIF, migration inhibitory factor; NK, natural killer; TGF-β1, transforming growth factor beta-1.

In AA, there is a breakdown of these protective mechanisms.[9,10] The cause of this breakdown is not fully understood but may involve genetic and environmental factors. These factors inhibit the secretion of immune privilege guardians and activate immune cells (mostly CD8+ T cells and CD4+ T cells, mast cells, natural killer cells, and dendritic cells), leading to the loss of immune privilege. The activated immune cells stimulate the production of interferon gamma (IFNγ), which upregulates the expression of MHC-I and proinflammatory cytokines (IL-2, IL-4, and IL-15). The IFNγ-driven inflammation is mediated by Janus kinase (JAK).[11] The activated immune cells infiltrate the HF bulb like a "swarm of bees," shifting the HF into a premature catagen phase, followed by dystrophy, apoptosis, and hair loss[9,10] (Figure 1c). The bulge area of the HF is not affected by this immunologic insult, thus allowing for hair regrowth.

How is alopecia areata diagnosed?

Alopecia areata is diagnosed clinically.[4] A history of patient-reported patchy hair loss and regrowth highly indicates AA. In most cases, further tests are not required, although dermoscopy (also known as trichoscopy) may be used to validate the diagnosis. Salient dermoscopy features of AA include the presence of yellow dots, black dots, broken hairs, exclamation point hairs, and short vellus hairs (a sign of early regrowth). Skin biopsy for histopathology evaluation, fungal culture, or serology for other autoimmune diseases or infectious diseases is rarely necessary for diagnosing AA, except when clinical findings are inconclusive and to rule in or out other causes of hair loss.[4]

What are some of the causes for delay in alopecia areata diagnosis and treatment?

Usually, these patients are diagnosed. They are not treated, which is different. A retrospective analysis of administrative claims data from more than 68000 patients showed that only 25% were prescribed treatment within 7 days of an AA diagnosis, and 44% were not prescribed any treatment in the year following diagnosis.[12] Our patient survey findings were consistent with these data; despite the majority of patients reporting hair loss often or all the time and significant psychosocial impact, 52% had never been treated, 30% were not aware that AA treatments exist, and 22% had not been referred to a specialist.

How is the severity of alopecia areata assessed?

Alopecia areata severity can be assessed in daily clinical practice using the recently developed AA scale.[13] This scale classifies AA severity as mild, moderate, or severe, based on the extent of scalp hair loss of ≤ 20%, 21% to 49%, and 50% to 100%, respectively. In addition, the scale incorporates 4 secondary clinical features that contribute to disease severity—eyebrow and eyelash involvement, treatment-refractory disease, psychosocial impact of AA, and diffuse rapid hair loss—for a more comprehensive disease severity assessment (Table 1).[13] The presence of any of these secondary features increases the severity level. This AA scale is simple and easy to administer, allowing for an informative and consistent assessment of AA patients in clinical practice.

Alopecia areata severity is assessed in clinical trials using the Severity of Alopecia Tool (SALT) score.[14] This score exclusively measures the extent and density of scalp hair loss. The score is determined by combining the visually estimated percentage of hair loss in each of the 4 quadrants of the scalp (left side, right side, top, and back). Five subgroups of hair involvement are identified based on the score: S_0 = no hair loss, S_1 = < 25% hair loss, S_2 = 25% to 49% hair loss, S_3 = 50% to 74% hair loss, S_4 = 75% to 99% hair loss, S_5 = 100% hair loss. The SALT score is inherently limited by the omission of other clinical features of AA that contribute to disease severity. For daily clinical practice use, mapping out the scalp's surface area to determine the SALT score could be time-consuming. Clinicians, however, need to understand the SALT score to interpret clinical trial results that use this score.[14]

TABLE 1.

Alopecia Areata Scale[13]	
Scalp Hair Loss	
Severity	Extent of Scalp Hair Loss
Mild AA	20% or less scalp hair loss
Moderate AA	21% to 49% scalp hair loss
Severe AA	50% to 100% scalp hair loss
If mild or moderate, increase AA severity rating by 1 level if 1 or more of the following are present	
• Negative impact on psychosocial functioning resulting from AA	
• Noticeable involvement of eyebrows or eyelashes	
• Inadequate response after at least 6 months of treatment	
• Diffuse (multifocal) positive hair pull test consistent with rapidly progressive AA	

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What advice do you offer patients before initiating treatment?

Patients need to be informed upfront that there is no cure for AA.[10] In addition, currently available treatments do not prevent relapse and do not appear to alter the long-term clinical course of AA.[15] Spontaneous remission can occur, but it is highly variable and dependent on disease severity at presentation.[16] Small lesions of < 25% scalp have a greater probability of spontaneous resolution, seen in up to 68% of cases. Large lesions of > 50% scalp involvement have a poorer prognosis, with spontaneous resolution occurring in 8% of cases. Most patients, however, do worsen over time. The possibility of spontaneous remission may lead to watchful waiting and treatment delays.[12] Although watchful waiting may be reasonable in mild cases, treatment should be considered in moderate-to-severe cases where spontaneous resolution is less likely or when hair loss significantly impacts the patient's quality of life or causes undue anxiety.[10] Counseling patients on the likelihood of spontaneous remission is important as this would help patients to make informed treatment decisions. Further, patients should be informed that treatment response with current therapies takes time. Even with the most effective therapies, substantial hair regrowth may take months.

What are the current treatments for alopecia areata and their limitations?

Current evidence-based treatments for AA include intralesional corticosteroids, topical corticosteroids, oral corticosteroids, JAK inhibitors, minoxidil, and topical immunotherapy. Treatment choice is guided by the severity of hair loss and patient age.[10] Generally, mild-to-moderate disease is treated with topical agents, and moderate-to-severe disease is treated with systemic therapies. In our study, most patients were prescribed topical minoxidil (47%), followed by intralesional (32%), topical (32%), and oral corticosteroids (16%). The efficacy, safety, and place of current therapies are briefly summarized.

Intralesional corticosteroids are the standard of care in limited patchy AA and cosmetically sensitive areas, such as the eyebrows.[10,17] They can also be an adjunctive treatment in extensive disease. In limited patchy AA, monotherapy with intralesional corticosteroids has led to > 50% hair regrowth in more than 80% of patients after 3 months of treatment.[18]

In clinical practice, their use is limited by the number of injections a patient can tolerate. Usually, a SALT score of 30% is the upper limit for injections.[19] In our survey, one-third of dermatologists overestimated the AA severity where intralesional corticosteroids are effective. Since injections are placed 1 cm to 2 cm apart, alopecia affecting 30% of the scalp requires treating about 210 cm^2, which implies almost

100 injections.[20] Adverse effects of intralesional corticosteroids are limited to local skin changes at the injection site and may include hyper-/hypopigmentation, atrophy, telangiectasias, and striae with chronic treatment.These skin changes are usually transient and resolve (except striae) after treatment discontinuation.[10]

Topical corticosteroids are the mainstay of treatment in patients younger than 10 years old or in patients unable to tolerate intralesional steroid injections.[10,17] Typically, they are less effective than intralesional corticosteroids and are used in conjunction with other treatments in less severe disease. As with intralesional corticosteroids, adverse effects involve skin changes. High-potency corticosteroids under occlusion can, however, be effective even in severe disease, even though they are not recommended in children due to the risk of systemic absorption.[21]

Oral corticosteroids can be used in the short-term treatment of AA,[17] with a 6-week course showing significant benefit in mild-to-extensive AA.[22] Pulse administration is recommended over continuous administration.[23] However, the benefit is not durable, with significant relapse rates after discontinuation.[17] Also, long-term use of oral corticosteroids is limited by their adverse effects, including suppression of the pituitary-adrenal axis, effects on bone growth or integrity leading to osteoporosis, ocular changes, and worsening of hypertension or diabetes. Because oral corticosteroids have a long history of use, the danger is that clinicians may become comfortable using them. Clinicians should always consider their long-term adverse effects and ensure that patient safety is not compromised.

Minoxidil, topical or oral, is often used as adjuvant therapy for AA to promote hair growth.[10,17] Topical and oral forms have been shown to be effective in stimulating hair regrowth; they are, however, less effective in severe AA. But in a recent case study of treatment-resistant alopecia universalis, co-administration of oral minoxidil with a JAK inhibitor (tofacitinib) appeared to improve the patient's SALT score from S_5 (100% hair loss) to S_2 (35% hair loss).[24] Adverse effects of topical minoxidil are usually mild, including scalp itching and dermatitis.[17] Hypertrichosis, lightheadedness, fluid retention, tachycardia, and headache are potential adverse effects of oral minoxidil.[10]

JAK inhibitors are the newest class of treatments for AA, with oral baricitinib being approved for use in adults with severe AA,[25] others in development (ie, ritlecitinib, deuruxolitinib), and several being used off-label or in clinical studies (ie, tofacitinib, ruxolitinib, and brepocitinib). The IFNγ- and IL-15-driven activation of cytotoxic T cells that contributes to AA pathophysiology is mediated in part by JAK, and JAK inhibition has been shown to reverse the effects of AA.[26] Oral, topical, and sublingual modes of delivery are being investigated. Current data suggest that oral formulations are the most effective, while the topical form lacks efficacy.[27,28] Compared to controls, oral formulations have a higher response rate (defined as 50% improvement in SALT score) (risk ratio of 6.86).[27]

With respect to baricitinib in phase 3 trials of patients with SALT scores ≥ 50, 36 weeks of treatment led to improved SALT scores of ≤ 20 in 36% to 39% of patients using 4 mg and in 19% to 23% of patients using 2-mg doses compared with 3.3% to 6.2% of patients using placebo.[29] Acne, elevated levels of creatine kinase, and increased levels of low- and high-density lipoprotein cholesterol were more commonly reported with baricitinib than with placebo. As a drug class, JAK inhibitors, including baricitinib, have a black box warning for serious infections, malignancy, major adverse cardiovascular events, and thromboembolic events.[10,25] These events are rare in patients with AA, and clinicians should discuss with their patients the efficacy and side effects of this new treatment option to reach a shared decision.[30]

Topical immunotherapy, diphenylcyclopropenone (DPCP) or squaric acid dibutyl ester (SADBE) may be used to induce contact dermatitis of the scalp and can modify the cytokine profile. This treatment is effective in approximately 30% of patients, including patients with severe AA.[31]

What are the unmet needs in alopecia areata?

Medical therapy: Do not delay treatment for AA. Spontaneous remission may occur, more so with localized disease involving < 25% of the scalp, but generally, mild-to-moderate disease should be treated with topical agents, while moderate-to-severe disease is treated with systemic therapies. Current treatments for AA are limited by their unsatisfactory efficacy and adverse event profiles. The newer JAK inhibitors have better efficacy and are generally safe, at least in the short term, but have a relapse rate of 54% with discontinuation.[27] Therefore, therapies with increased long-term efficacy and safety are needed.

Supportive therapy: Alopecia areata is associated with significant psychological burden requiring consideration of psychological interventions and access to support groups when developing treatment strategies. Further, treatment response with current therapies takes time. For example, while waiting for hair regrowth, patients require wigs and other camouflaging agents to conceal hair loss. Improved access to these agents, as well as counseling for coping strategies, is needed.

Education: Increased patient and public awareness and education on AA is needed to mitigate bullying and social stigma associated with hair loss and baldness and to improve the acceptance and inclusion of patients with AA.

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