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STUDY PROTOCOL

Evaluation of SALT score severity in correlation with trichoscopic findings in alopecia areata: a study of 303 patients

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Abstract

Background Alopecia areata (AA) is a common autoimmune non-scarring type of hair loss characterized by round patches on the scalp or body. AA is the second most common form of hair loss after male-pattern baldness in men, and female-pattern baldness in women.

Aim of the study This study aims to examine the correlation between trichoscopic patterns and AA severity, evaluated by the Severity of Alopecia Tool (SALT) score, and to analyze the impact of demographic and clinical factors on treatment outcomes.

Patients and methods A prospective cross-sectional single-center study was conducted involving 303 patients with AA, assessing the relationships between trichoscopic findings, clinical features, treatment responses, and SALT scores.

Results Our cohort comprised 50.83% females and 49.17% males, ranging in age from 6 months to 84 years, with a mean age of 23.67 ± 14.79 years and an average disease onset at 21.68 (± 14.30) years. Localized and Multiple patchy AA were the most observed subtypes, with ophiasis, alopecia universalis, and totalis being less frequent. Small vellus hairs, exclamation mark hair, and coudability hair emerged as prevalent trichoscopic findings. A significant prevalence of comorbidities (82.18%) was observed, particularly micronutrient deficiencies, autoimmune and endocrine disorders, and atopic diseases. Key predictors of more severe AA, as indicated by higher SALT scores, include prolonged disease duration, nail involvement, and neuropsychiatric comorbidities. Shorter disease duration was correlated with active disease markers such as broken hairs, black dots, yellow dots, and exclamation mark hair. Markers like cumulus-like white dots and v-sign correlated with severe disease stages, whereas pigtail hairs, small vellus hairs, and upright regrowing hairs indicated new hair growth or positive treatment responses.

Conclusion Trichoscopy proves crucial for assessing AA severity and treatment efficacy, facilitating personalized treatment approaches and improving patient management outcomes.

Keywords Alopecia areata · Trichoscopy · SALT score · Hair loss · Comorbid conditions · Epidemiological data · Nail involvement · Disease severity

Abbreviations

AA	Alopecia Areata
ACE	Alopecia Areata Consensus of Experts
AGA	Androgenic Alopecia
AO	Alopecia Ophiasis
ANA	Antinuclear Antibodies
AT	Alopecia Totalis
AU	Alopecia Universalis
BD	Black Dots
BH	Broken Hair
CH	Coudability Hair
CI	Confidence Intervals
CLWD	Cumulus Like White Dots
DPCP	Diphenylcyclopropenone
EMH	Exclamation Mark Hair
FOV	Field of vision
HHG	Hypopigmented Hair Growth
HT	Hypertension
IFN-γ	Interferon-gamma
NAAF	National Alopecia Areata Foundation
OR	Odds Ratio

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PH	Pigtail Hairs
PPC	Pohl Pinkus Constrictions
PS	Perifollicular Scaling
SALT	Severity of Alopecia Tool
SVH	Small Vellus Hairs
T2DM	Type 2 Diabetes Mellitus
TC	Tinea Capitis
TE	Telogen Effluvium
TGAb	Thyroglobulin Antibody
TH	Tulip Hairs
TPOAb	Thyroid Peroxidase Antibody
TTM	Trichotillomania
URH	Upright Regrowing Hair
YD	Yellow Dots

Introduction

Alopecia areata (AA) is a multifactorial, organ-specific autoimmune disorder that targets anagen hair follicles, leading to non-scarring, patchy hair loss on the scalp and other areas. It results from an immune attack that disrupts the hair cycle and impairs the immune privilege of hair follicles, causing them to transition prematurely into the catagen or telogen phase, leading to the miniaturization of the hair follicles [1]. AA affects approximately 1–2% of the population, with a slight female predominance, and typically occurs before the age of 30 years, although it can occur at any age [2]. The global prevalence of AA increased from 1990 to 2019, likely due to enhanced diagnostic techniques and increased awareness of conditions [3, 4]. Furthermore, recent studies have suggested that AA incidence may have increased following COVID-19, with both the severity of the infection and vaccination linked to the onset or exacerbation of the disorder [5]. Analyses of social media's impact emphasize its significant role in shaping public perceptions of AA, highlighting the need for healthcare providers to actively engage on these platforms to improve patient guidance and support [6].

Socioeconomic factors such as education, income, and health insurance play a significant role in AA diagnosis rates, underscoring the need for targeted interventions to improve access to economically disadvantaged groups [7]. Advances in the understanding of AA etiology emphasize the interaction between genetic susceptibilities, especially immune-related genes such as HLA, and environmental factors such as viral infections (e.g., Epstein-Barr Virus, Cytomegalovirus), stress, oxidative stress, and allergies [8]. AA prognosis varies based on factors such as disease duration, systemic involvement, and specific clinical features. Indicators of poorer prognosis include ophiasis patterns, eyebrow and eyelash involvement, disease lasting over five years, and nail abnormalities. Additionally, a family history of AA, other autoimmune diseases, or atopy increases the risk of more severe forms, such as alopecia totalis (AT) or universalis (AU), and may limit treatment efficacy [9].

AA manifests in various clinical forms, ranging from discrete, patchy baldness on the scalp (most common) to more extensive presentations like AT and AU. Less common patterns include ophiasis (AO), where hair loss appears as a band-like distribution along the scalp periphery [10]. This variability suggests that AA may encompass a spectrum of related disorders, each driven by distinct mechanisms. Localized immune responses often underlie patchy AA, while systemic immune dysfunction is thought to drive more severe forms like AU [11].

Diagnosis primarily relies on clinical evaluation, with trichoscopy enhancing diagnostic accuracy by revealing characteristic features, such as yellow dots and exclamation mark hair. In atypical cases, skin biopsy may confirm the diagnosis [12]. These diagnostic methods, along with tools such as the SALT score, provide a comprehensive framework for assessing disease severity and guiding treatment [13]. Tools like the SALT score help assess disease severity and guide treatment, although misdiagnosis remains common, especially in women and individuals with darker skin tones [14]. This highlights the need for improved diagnostic protocols that combine clinical assessments with patient-reported outcomes for more accurate and inclusive AA management [15].

AA frequently coexists with other autoimmune conditions such as thyroid disease, vitiligo, and psychological issues, including anxiety and depression. This comorbidity pattern underscores the importance of integrated care that addresses both medical and psychological needs [16]. Early onset and extensive initial scalp involvement are significant predictors of relapse, with over two-thirds of patients experiencing recurrence within a year of remission [17]. Studies suggest that AA severity and relapse frequency tend to decrease with later disease onset, emphasizing the need to adapt treatment strategies for different age groups [18, 19]. Given the chronic, recurrent nature of AA, regular follow-up is critical for monitoring treatment and providing psychological support. Innovations like tele-trichoscopy facilitate more consistent monitoring, while global and trichoscopic photography are vital for tracking disease progression. However, standardized protocols are required to ensure the quality and consistency of clinical assessments [20, 21].

Despite numerous treatment alternatives, patients often encounter challenges in making informed decisions due to limited access to specialized care, inadequate information, and the psychological impact of the disease, potentially leading them to forego treatment [22]. Management is tailored to disease severity, with corticosteroids often prescribed for milder cases, while immunotherapy and JAK inhibitors are reserved for more extensive cases. The long-term safety of these advanced therapies remains to be monitored [23]. The

financial burden of AA treatment remains substantial, leading to significant out-of-pocket costs and economic stress in patients [24, 25]. Recent bibliometric analyses have shown a shift in AA research towards understanding treatment strategies, molecular mechanisms, and comorbidities [26].

Building on these insights, the present study aims to evaluate the correlation between trichoscopic findings and AA severity, as measured by the SALT score. By identifying specific trichoscopic features indicative of more severe disease, this research seeks to enhance diagnostic precision and inform treatment approaches.

Materials and methods

Study design and study population

This prospective, single-center, cross-sectional study was conducted at the Department of Dermatology, Ministry of Health, Gaziantep Nizip State Hospital, from July 2023 through November 2024. This study included all consecutive patients diagnosed with various subtypes of AA, including localized and multiple forms of patchy AA, AO, AT, and AU, who presented to the clinic during this period.

The diagnostic approach followed a structured flowchart, beginning with a comprehensive medical history, clinical examination, trichoscopic assessment, and hair pull test. In cases where standard diagnostic methods were inconclusive or potentially confounded with other types of alopecia, trichoscopy-guided 4 mm scalp biopsy was performed for pathological analysis to confirm or rule out the diagnosis.

Inclusion criteria for the study were all patients of both genders and all age groups, diagnosed with AA. Exclusion criteria included individuals with other scarring or non-scarring scalp disorders, congenital causes of hair loss, those who were pregnant, or who declined to participate. The study specifically focused on patients diagnosed with AA affecting the scalp; those with AA limited to eyebrows, eyelashes, or beard were excluded. To accurately assess the impact of treatment on trichoscopic findings, participants were categorized into two groups: treatment-naive patients and those who received topical, systemic corticosteroids, immunotherapy, or biological therapies in the last three months. This classification enhances our ability to compare the natural progression of the disease with the effects of specific therapeutic interventions, providing valuable insights into the efficacy of different treatments for AA.

Efforts to minimize selection bias involved including all eligible patients from a consecutive sample of clinic attendees, while diagnostic procedures were standardized to ensure consistency and reduce diagnostic bias. This manuscript adheres to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies.

Assessment of comorbidities

The inclusion of metabolic, autoimmune and endocrine, atopic, psychiatric and neurological, dermatological, and genetic comorbidities was based on their documented association with AA, underscoring the multifaceted systemic implications of the disease. Comorbidities were assessed using a standardized protocol, which integrated laboratory data, each patient's medical history, clinical examinations, and radiological imaging to identify systemic disease indicators. Data was gathered from the hospital's electronic medical records and a national health database, employing specific ICD codes and advanced filtering techniques to ensure relevance and accuracy. This comprehensive review provides a holistic view of each patient's health status, shedding light on the complex interactions between trichoscopic biomarkers and disease severity without specifically tracking therapeutic responses.

Laboratory evaluations included essential tests such as complete blood count, biochemical screening, thyroid function tests, and autoantibody screening for Antinuclear Antibodies (ANA), Thyroid Peroxidase Antibodies (TPOAb), and Anti-Thyroglobulin Antibodies (TGAb), crucial for evaluating thyroid health and autoimmune status. Trace element and micronutrient levels were measured to identify deficiencies that could influence disease progression. Clinically, nail changes such as pitting and trachyonychia have been documented as potential indicators of comorbidities.

Comorbidities were categorized on the basis of their nature and systemic involvement. Conditions like discoid lupus erythematosus (DLE), vitiligo, and psoriasis vulgaris were classified into both dermatological and autoimmune categories due to their overlapping features, while autoimmune thyroiditis was included in both endocrine and autoimmune categories reflecting its dual nature. This systematic classification facilitated a nuanced understanding of the comorbid landscape and aided the comprehensive clinical management of AA.

Ethical approval statement

Ethical approval for this study was granted by the Bezmialem Vakıf University Non-Interventional Clinical Research Ethics Committee, under protocol number 2023/275 (Reference: E-54022451-050.05.04–122609). Following this approval, further authorization was secured from the Provincial Health Directorate of Gaziantep (Reference: E-87825162-663.08-231669191) to ensure compliance with local regulatory standards. Informed consent was obtained from all participants before their inclusion in the study, affirming that all

procedures were conducted in accordance with the ethical standards set forth in the Declaration of Helsinki.

Trichoscopic examination

During trichoscopic examinations, a DermLite® DL5 (DermLite, Carlsbad, CA, USA) in conjunction with an iPhone 15 Pro Max® (Apple Inc., Cupertino, CA, USA) was used to capture high-resolution images of the follicular ostia, hair shafts, and scalp surface. This handheld device is equipped with polarizing filters, which effectively reduce surface reflections and allow for noninvasive and precise diagnostics without the need for immersion oil. Photographs from four distinct scalp regions (frontal hairline, vertex, parietal, and occipital areas) were taken to assess AA involvement, with each patient having at least ten high-resolution images captured to maintain consistency in feature assessment. A consistent field of vision (FOV) was maintained in each image to ensure a standardized assessment of trichoscopic features. The live photo feature captured dynamic changes, providing detailed views of hair and scalp dynamics crucial for assessing AA activity and progression.

Special attention was paid to identifying key trichoscopic features indicative of AA activity, including abnormalities in hair shafts, follicle openings, and the overall condition of the scalp surface. The key features observed were yellow dots (YD), black dots (BD), broken hairs (BH), exclamation mark hair (EMH), tulip hair (TH), v-sign, hypopigmented hair regrowth (HHG), coudability hair (CH), upright regrowing hair (URH), small vellus hair (SVH), pigtail (circle) hairs (PH), Pohl pinkus constrictions (PPC), telangiectasia, cumulus-like white dots (CLWD), and perifollicular scaling (PS) [27]. The consistent observation of these features across multiple affected regions is critical for accurate diagnosis. All images and associated data were securely stored in a digital repository for detailed analysis and future research.

SALT score assessment

The Severity of Alopecia Tool (SALT) score, meticulously used to evaluate the extent of scalp hair loss in each study participant, classifies patients into five subgroups ranging from S1 to S5. Developed by the National Alopecia Areata Foundation (NAAF) and informed by a panel of experts, the SALT score provides a standardized and reliable method to quantify hair loss across different scalp regions, ensuring consistent application in clinical trials and routine assessments. The SALT scoring system categorizes the severity of alopecia as follows: S0, no hair loss, S1 = 1–24% hair loss, S2 = 25–49% hair loss, S3 = 50–74% hair loss, S4 = 75–99% hair loss; S5, 100% hair loss. This widely accepted scoring method has become a cornerstone in both research and clinical practice, allowing for consistent evaluation of scalp hair loss severity in patients with AA [28].

Statistical analysis

In our study of 303 individuals, the sample size was determined via power analysis, targeting 80% power at a 0.05 alpha level to detect significant clinical and trichoscopic differences. Statistical analyses were conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA), complemented by Python (version 3.8, Python Software Foundation, Wilmington, DE, USA) for advanced modeling and visualization. Python's capabilities enabled the deployment of machine learning algorithms and sophisticated data visualizations, enhancing the exploration of complex data relationships.

Descriptive statistics

Demographic and clinical characteristics, including age, gender, disease duration, SALT scores, and trichoscopic findings, were summarized using means, medians, ranges, standard deviations, and percentages. The Shapiro–Wilk test was used to assess the normality of continuous variables.

Comparative analyses

Non-parametric tests were employed for variables with non-normal distributions, while categorical variables were compared using Chi-Square or Fisher's Exact Test. For comparisons involving more than two groups, the Kruskal–Wallis test was used, followed by post-hoc pairwise analyses with Bonferroni corrections where applicable.

Correlation and regression analyses

Spearman's rank correlation coefficient assessed associations between continuous variables (e.g., disease duration, SALT scores) and trichoscopic findings. Ordinal logistic regression was utilized to identify predictors of disease severity, with SALT scores categorized into quartiles. Independent variables included demographic factors (e.g., gender), clinical characteristics (e.g., nail involvement), and comorbidities (e.g., neuropsychiatric and dermatologic conditions).

Model adjustments and interactions

Regression models were adjusted for potential confounders, and interaction terms were incorporated to evaluate moderating effects between comorbidities and disease severity. Odds ratios (ORs) and 95% confidence intervals (CIs) quantified the strength of associations, with statistical significance set at $p < 0.05$.

Sensitivity and specificity analyses

Sensitivity analyses confirmed the robustness of results across subgroups, while Bonferroni corrections were applied to control for Type I error in multiple comparisons.

This statistical framework facilitated the identification of key predictors of AA severity, elucidated relationships between trichoscopic findings and clinical outcomes, and evaluated the impact of treatment on disease progression.

Outcome measures

Primary outcome

The primary objective was to assess the prevalence and distribution of distinct trichoscopic features across AA subtypes including patchy AA, AO, AT, and AU. We hypothesized that specific trichoscopic characteristics would vary significantly across these subtypes, serving as reliable markers for differentiation. Additionally, the study aimed to correlate these trichoscopic findings with demographic variables including age, sex, and disease duration, enhancing our understanding of their relationship with the underlying disease process.

Secondary outcomes

Secondary objectives included exploring the association between trichoscopic findings and AA severity, quantified by the SALT score. We hypothesized certain patterns would correlate strongly with higher SALT scores, reflecting advanced disease stages. Comparative analysis between treatment-naive patients and those treated within the last three months evaluated treatment effects on trichoscopic markers. Regression analyses further assessed the influence of disease duration, nail involvement, and family history on severity, identifying specific features predictive of treatment response and overall disease dynamics.

Results

Demographic and clinical characteristics

This study included 303 patients diagnosed with AA, comprising 49.17% males and 50.83% females. The mean age of the patients was 23.67 ± 14.79 years, with a median age of 23 years, ranging from 0.5 months to 84 years. The average disease duration was 25.22 months, ranging from 0.25 to 732 months, and the average age at disease onset was 21.68 years. A positive family history of AA was reported in 22.44% of the patients, while nail changes were observed in 24.75% of the cohort. Among the AA subtypes, 42.98% of the patients had localized patchy AA, 32.29% had Multiple patchy AA, 18.75% had AO, and 5.99% had either AT/AU. The distribution of SALT scores revealed that 75.25% of the patients were classified as S1, 16.83% as S2, 2.64% as S3, 4.29% as S4, and 0.99% as S5. The median SALT score for the entire cohort was 6.4, with the median score for males being 4.8 and for females 8.1. Gender-based statistical analysis revealed that differences in onset age, disease duration, and SALT scores were not statistically significant ($p > 0.05$) (Table 1).

Prevalence of comorbidities

Associated comorbidities were present in 82.18% of patients with AA, with no significant differences in overall comorbidity prevalence observed between genders ($p = 0.376$). Comorbidities were categorized based on their pathophysiological relationships with AA into key groups: micronutrient deficiencies, metabolic and cardiovascular diseases, atopic conditions, thyroid disorders, autoimmune diseases, and others. In this cohort, micronutrient deficiencies, specifically vitamin D, vitamin B12, and iron, were the most common, affecting 47.85% of patients. Autoimmune and endocrine disorders were observed in 36.3% of patients. Atopic conditions, including asthma, allergic rhinitis, allergic conjunctivitis, and other atopic disorders, were present in 22.44% of patients. Additionally, 18.48% exhibited secondary dermatological conditions, and neuropsychiatric comorbidities affected 16.5% of the cohort. Metabolic complications such as type 2 diabetes mellitus (T2DM), hypertension (HT), and hyperlipidemia affected 9.24% of the patients. ANA positivity was observed in 9.6% of patients, TPOAb positivity in 7.92%, and elevated levels of TGAb in 8.25% (Table 2).

Significant gender differences were noted in the prevalence of certain comorbidities among AA patients. Micronutrient deficiencies were significantly more prevalent in females (56.49%) compared to males (38.93%, $p = 0.003$). Autoimmune and endocrine disorders were also more common in females (42.86% vs. 29.53%, $p = 0.022$), as were neuropsychiatric disorders (22.08% vs. 10.74%, $p = 0.012$). There were no significant gender differences noted in the prevalence of atopic diseases, dermatologic conditions, or metabolic complications (Fig. 1).

Characteristics and distribution of trichoscopic findings across AA subtypes

Trichoscopy plays a crucial role in diagnosing and monitoring AA, allowing rapid identification of hair shaft abnormalities without ex vivo sampling. In our evaluations, we categorized trichoscopic findings to identify patterns across AA subtypes and to explore their clinical relevance. Among the 15 evaluated features, trichoscopic findings in AA

Table 1 Demographic and clinical characteristics of patients with alopecia areata

Characteristic	Female	Male	Total	p-value
Total number of patients	154 (50.83%)	149 (49.17%)	303 (100%)	–
Mean age (years)	22.79 (± 14.03)	24.58 (± 15.54)	23.67 (± 14.79)	0.293
Median age (years)	23	22	23	–
Age range (years)	0.5–64.0	1.0–84.0	0.5–84.0	–
Age of onset (years)	20.93 (± 13.24)	22.46 (± 15.34)	21.68 (± 14.30)	0.353
Disease duration (months)	23.16 (± 65.40)	27.05 (± 56.94)	25.07 (± 61.32)	0.582
Family history present (%)	24.84%	20.13%	22.44%	0.345
Nail changes (%)	23.53%	26.17%	24.75%	0.574
SALT score distribution				
S1 (1–24% hair loss)	74.03%	76.51%	75.25%	0.713
S2 (25–49% hair loss)	20.13%	13.42%	16.83%	0.160
S3 (50–74% hair loss)	3.25%	2.01%	2.64%	0.756
S4 (75–99% hair loss)	1.30%	7.38%	4.29%	**0.020**
S5 (100% hair loss)	1.30%	0.67%	0.99%	1.000
Median SALT score	8.1	4.8	6.4	**0.008**
Alopecia subtypes				
Localized patchy	38.31%	47.65%	42.98%	0.127
Multiple patchy	35.71%	28.86%	32.29%	0.249
Ophiasis	22.73%	14.77%	18.75%	0.104
Totalis/Universalis	3.25%	8.72%	5.94%	0.076

Statistically significant p-values are in bold

The key demographic and clinical characteristics of a cohort of 303 patients diagnosed with AA, highlighting age, gender distribution, disease duration, and family history. The table also details the classification of SALT scores, prevalence of alopecia subtypes, and occurrence of nail changes, with p-values provided to note statistically significant gender differences

patients ranged from 1 to 13 per individual, averaging 5.89 overall, with no significant differences observed between genders ($p = 0.395$) or between treated and untreated patients ($p = 0.161$), illustrating the diverse presentation of the condition.

Follicular structures and dots

YD, BD, and CLWD were collectively analyzed as markers of inflammatory activity and structural changes within hair follicles. YD occurred in 51.82% of patients, BD in 41.25%, and CLWD in 19.14%, highlighting disruptions in the hair growth cycle and their potential role in predicting disease progression and guiding treatment decisions (Fig. 2).

Hair shaft abnormalities

Features such as BH, EMH, PPC, TH, and CH indicate mechanical and structural changes typical of AA. In our study, BH was observed in 50.83%, EMH in 58.75%, PPC in 35.64%, TH in 39.60%, and CH in 56.11% of patients, highlighting disease activity and its impact on hair shaft integrity (Fig. 3).

Regrowth features

Indicators of regrowth, such as SVH, HHG, PH, and URH, were prominent across the subtypes. SVH was the most frequent, seen in 68.98% of patients, followed by HHG at 30.69%, PH at 33.66%, and URH at 40.26%, illustrating the regenerative dynamics of hair follicles and aiding in evaluating treatment efficacy (Fig. 4).

Vascular and other dermoscopic findings

Vascular changes, such as telangiectasia and other distinctive patterns, including v-sign and PS, were categorized to highlight microvascular and other dermoscopic alterations. Telangiectasia was found in 28.38% of patients, while PS was seen in 18.15% and v-sign in 15.51%, respectively (Fig. 5).

Trichoscopic findings in AA provide critical insights into both superficial and histopathological changes, aiding the understanding of disease progression and treatment response. In the acute phase, BD and EMH indicate active inflammation and follicular damage linked to peribulbar lymphocyte infiltration and apoptosis in the hair matrix cells. The subacute phase is characterized by CH and TH, signaling a reduction in follicular activity as hair cycles from anagen to telogen, and continues to show structural damage.

Table 2 Prevalence of comorbidities among patients with alopecia areata

Category and Subcategory	Count
Metabolic and cardiovascular diseases	
Hypertension (HT)	14
Insulin independent diabetes mellitus (Type 2 DM)	11
Dyslipidemia	11
Coronary artery disease	3
Hepatosteatosis	3
Atopic diseases	
Seasonal allergic rhinitis	39
Allergic conjunctivitis	20
Asthma	26
Atopic dermatitis	2
Food allergy	1
Atopic disease (unspecified subtype)	6
Thyroid diseases	
Hashimoto's thyroiditis	17
Hypothyroidism	2
Hyperthyroidism	1
Subclinical hyperthyroidism	11
Subclinical hypothyroidism	5
Toxic multinodular goiter	1
Other autoimmune diseases	
Vitiligo	7
Celiac disease	1
Insulin independent diabetes mellitus (Type 1 DM)	1
Discoid Lupus Erythematosus (DLE)	3
Addison's disease	2
Hypoparathyroidism	2
Antinuclear Antibody Positivity (ANA)	29
Anti Thyroid Peroxidase Positivity (TPOAb)	24
Thyroglobulin Antibody Positivity (TGAb)	25
Neuropsychiatric diseases	
Anxiety disorder	26
Depression/depressive episodes	9
Bipolar disorder	1
Migraine	4
Brain cavernous hemangioma	2
Attention deficit hyperactivity disorder	1
Severe mental retardation	1
Nutrition-related diseases	
Iron deficiency anemia	61
Nonanemic iron deficiency	27
B12 deficiency	69
Vitamin D deficiency	46
Dermatological diseases	
Acne vulgaris	13
Seborrheic dermatitis	5
Pityriasis alba	1
Xerosis cutis	3
Psoriasis vulgaris	4
Dermatophytosis	4
Lichen planus	3
Verruca vulgaris	5
Melasma	2
Urticaria	1
Nevus sebaceous	1
Congenital temporal triangular alopecia	1
Genetic disorders	
Turner syndrome	1
Hereditary macular dystrophy	1
Combined oxidative phosphorylation deficiency	1
Down syndrome	1
Hypohidrotic ectodermal dysplasia	1
Others	
Renal and urinary diseases	3
Respiratory diseases (other than asthma)	5
Gastrointestinal diseases	6
Gynecological diseases	3

A detailed overview of comorbidities among patients diagnosed with AA reveals that micronutrient deficiencies, autoimmune and endocrine disorders, and atopic conditions are most prevalent, underscoring their significance in the clinical landscape of this condition

The chronic phase is marked by PPC, reflecting sustained follicular damage and hair shaft thinning due to persistent histopathological alterations. During recovery, SVH and URH highlight follicular regrowth, diminished inflammation, and effective treatment response. Together, these markers chart the pathology of AA across stages and form the basis for personalized clinical interventions [30].

Trichoscopic analysis highlights significant variability in findings across AA subtypes, reflecting differences in disease severity. YD were notably more prevalent in Patchy AA and Multiple patchy AA, at 53.08% and 60.20% respectively, indicating significant differences ($p = 0.045$), which underscores their diagnostic significance in these subtypes. SVH was highly prevalent in less severe forms, with 76.53% in Multiple patchy AA and 75.44% in AO, compared to only 27.78% in AT/AU, highlighting its utility in distinguishing these conditions ($p < 0.001$). CH was prominently present in AO at 77.19%, significantly marking its presence in this subtype ($p = 0.003$). PH was absent in AT/AU but observed in 43.86% of AO cases ($p = 0.005$), suggesting its diagnostic value in excluding more severe forms like AT/AU. TH showed significant distribution, with 49.12% in AO and only 16.67% in AT/AU, emphasizing its utility in differentiating these forms ($p = 0.010$). HHG was significantly more prevalent in AO at 45.61%, compared to 29.23% in Patchy AA and 26.53% in Multiple patchy AA ($p = 0.036$), highlighting its importance in diagnosing AO. V-sign was elevated in AT/

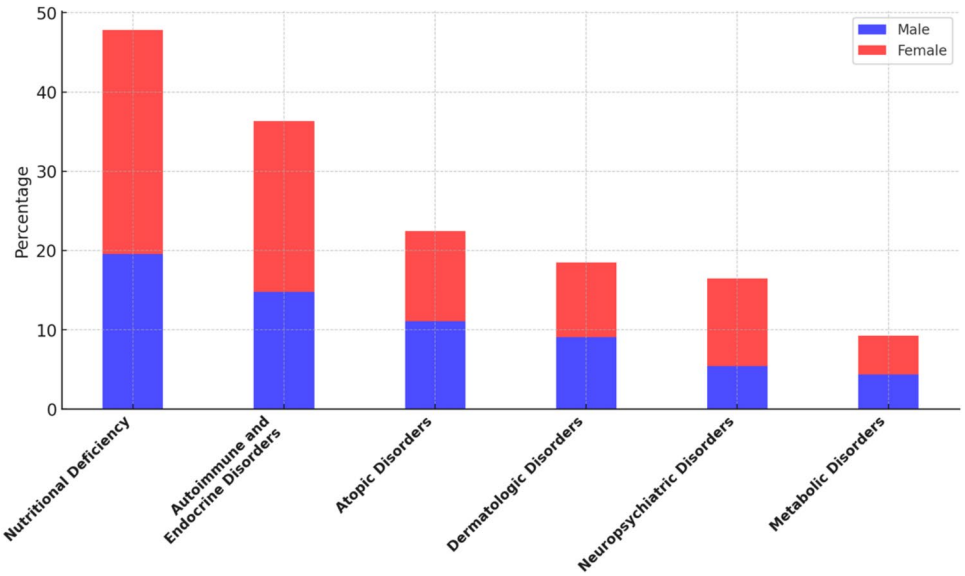

Fig. 1 Gender-based distribution of comorbidities among AA patients. This stacked percentage bar chart illustrates the distribution of comorbidities by gender, highlighting areas with notable gender-based differences. Significant gender differences were noted in the prevalence of micronutrient deficiencies ($p = 0.003$), autoimmune and endocrine disorders ($p = 0.022$), and neuropsychiatric conditions ($p = 0.012$). No significant differences were found in the prevalence of atopic diseases, dermatological conditions, or metabolic complications

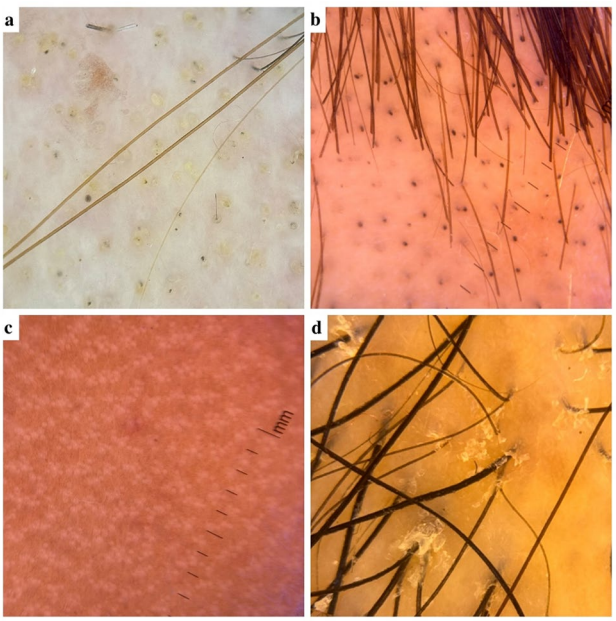

Fig. 2 Follicular structures and dots in alopecia areata. **a** Yellow dots: Trichoscopic image showing yellow dots, which are dilated follicular openings filled with keratotic material, commonly observed in AA. **b** Black dots: Presence of black dots, representing pigmented hair shaft residues broken at the scalp surface, a marker of active disease. **c** Cumulus like white dots: Appearance of cumulus-like white dots, indicative of clusters of tiny, white, globular structures around follicular openings, often observed in AA. **d** Perifollicular scaling: Visible perifollicular scaling, suggesting inflammation around hair follicles, frequently associated with AA

AU at 38.89% ($p = 0.006$), indicating its prevalence in more advanced stages of AA. CLWD were significantly prevalent in AT/AU at 77.78% ($p < 0.001$), marking them as indicative of advanced stages. (Table 3).

Analysis of trichoscopic findings: age range, disease duration, and correlation with SALT severity scores

Analysis of trichoscopic findings among patients with AA revealed significant differences across age groups (< 20, 20–40, and > 40 years). YD showed the highest prevalence in the 20–40 age range, with a statistically significant difference among the age groups ($p = 0.017$). Similarly, Telangiectasia ($p = 0.008$) and HHG ($p < 0.001$) were significantly more prevalent in the > 40 age group. HHG shows a notable rise in > 40 s at 58.33% ($p < 0.001$), indicative of progression or age-related symptom manifestation. URH is significantly more common in 20–40 s at 50.00% ($p = 0.013$), potentially reflecting a phase of active recovery or treatment response. V-sign prevalence escalates in > 40 s to 27.08% ($p = 0.038$), associated with advanced AA stages or cumulative disease impact. CLWD, while higher in > 40 s at 33.33%, shows a significant age-related increase ($p = 0.018$), suggesting more pronounced disease manifestations or chronicity in older age groups (Table 4).

Analysis of trichoscopic markers in AA patients revealed significant correlations with disease duration. Markers such as BH, BD, YD, and EMH were associated with shorter disease durations, with mean durations of 15.66, 18.26, 19.03, and 20.81 months, respectively ($p < 0.05$). These findings suggest that these markers are indicative of more acute or active disease phases. In contrast, CLWD was strongly associated with longer disease durations, with patients exhibiting this marker showing a mean duration of 62.30 months compared to 16.44 months without it ($p < 0.001$), underscoring its role as a marker of chronic disease stages. Markers such as SVH, CH, and Telangiectasia did not show significant differences in disease duration ($p > 0.05$), reflecting their limited direct association with AA progression. Other markers,

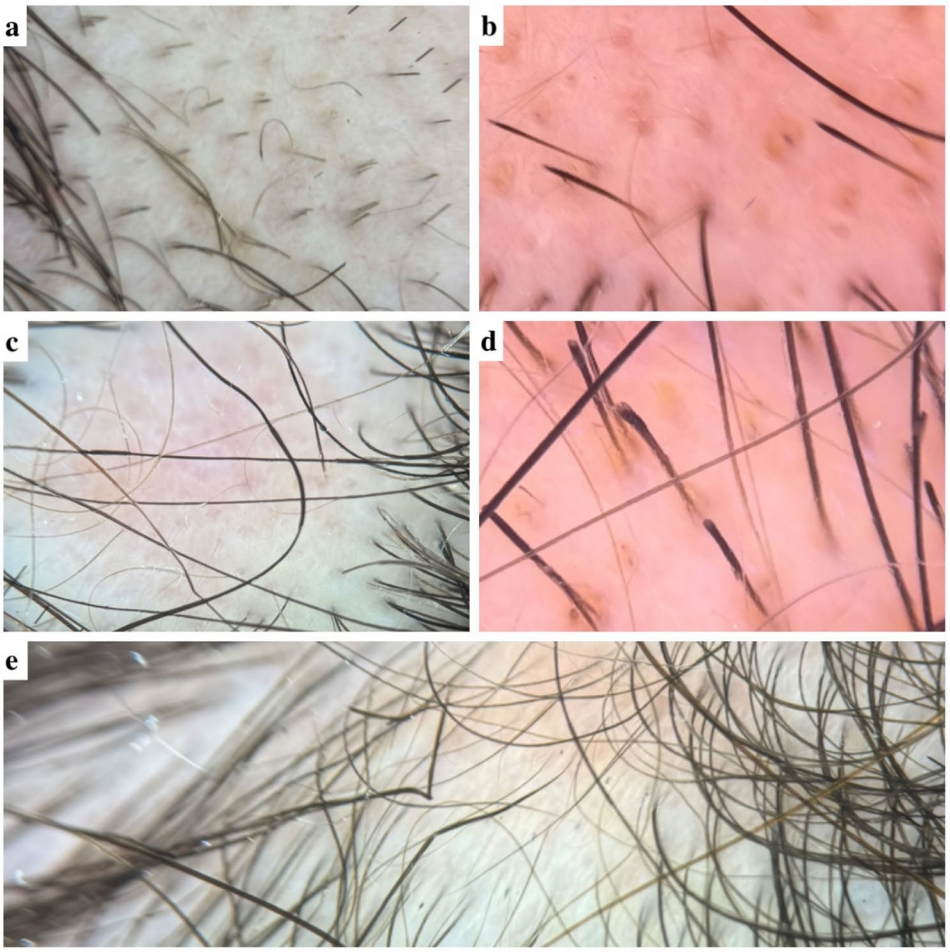

Fig. 3 Hair shaft abnormalities in alopecia areata. **a** Broken hairs: Trichoscopic image showing broken hairs, which are hair shafts that have fractured, often seen in areas of active hair loss in AA. **b** Exclamation Mark hair: Presence of exclamation mark hairs, characterized by a thicker distal end and a thinner proximal end near the scalp, commonly associated with AA. **c** Pohl pinkus constrictions: Visible Pohl-Pinkus constrictions, indicating areas where the hair shaft is constricted, resembling a string tied around the hair, suggesting mechanical stress or damage. **d** Tulip hair: Image displaying tulip hairs, where the distal end of the hair shaft tapers to a point, resembling the shape of a tulip, indicative of incomplete hair regrowth. **e** Coudability hair: Trichoscopic image of coudability hair, which bends easily upon mechanical stress, a sign of weakened hair structure often found in AA

such as TH and HHG, exhibited trends towards shorter durations but did not reach statistical significance, warranting further investigation (Table 5).

Correlation analysis between trichoscopic markers and SALT scores revealed that CLWD exhibited a strong positive correlation (Spearman's $r = 0.29$, $p < 0.001$), indicating a significant association with higher disease severity. Similarly, v-sign demonstrated a notably positive correlation (Spearman's $r = 0.19$, $p = 0.001$), reinforcing its role as an indicator of advanced AA. PH also showed a positive correlation (Spearman's $r = 0.11$, $p = 0.050$), suggesting its potential relevance in disease progression. Other markers, such as PS and PPC, showed trends towards significance but did not meet the statistical threshold, underscoring the specificity of CLWD and v-sign in assessing disease severity and progression (Table 6).

Distribution of trichoscopic findings among treated and naive patients

In this study, two groups of AA patients were compared: treatment-naive patients and those under treatment or in follow-up after various therapies such as topical corticosteroids, intralesional corticosteroids, immunotherapy, and biological therapy. Significant differences were observed in the prevalence of specific trichoscopic markers between groups. PH and URH showed a markedly higher prevalence in treated patients, at 68.00% and 76.00% respectively, compared to 22.37% and 28.51% in naive patients, suggesting these markers as reliable indicators of treatment response ($p < 0.001$ for both). In contrast, BH, BD, and EMH showed higher incidences in naive patients than in treated patients, indicating their association with greater disease activity or severity. This reduction in marker frequencies following treatment (BD: 29.33% vs. 45.18%, $p = 0.022$; BH: 34.67% vs. 56.14%, $p = 0.002$; EMH: 46.67% vs. 62.72%, $p = 0.021$) suggests therapeutic effectiveness in mitigating signs of active disease. Meanwhile, an increase in telangiectasia from 24.56% to 40.00% in treated patients ($p = 0.015$) may reflect side effects or consequences of prolonged disease or therapy, emphasizing the complexity of treatment effects in AA (Table 7) (Fig. 6).

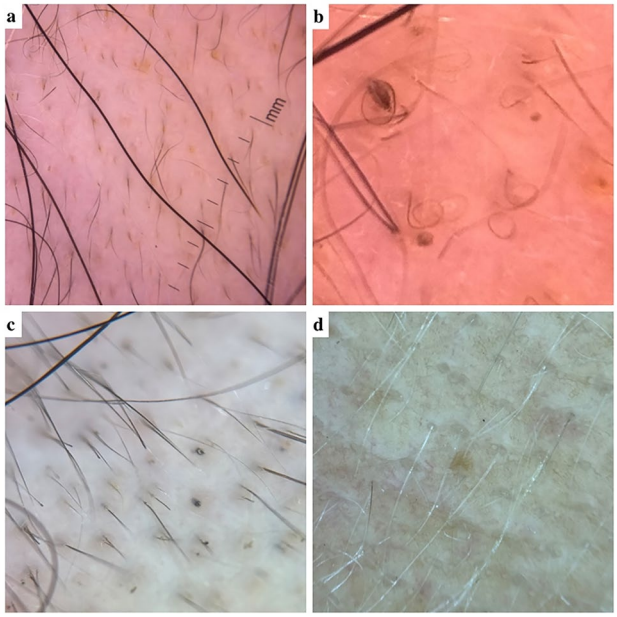

Fig. 4 Regrowing hair and minor hair findings in alopecia areata. **a** Small vellus hairs: Trichoscopic image displaying small vellus hairs, which are fine, short, and non-pigmented hairs, commonly observed during the early stages of hair regrowth in alopecia areata. **b** Pigtail hairs: Appearance of pigtail (circle) hairs, characterized by their looped structure, indicating initial regrowth and a positive sign of hair recovery. **c** Upright regrowing hair: Image showing upright regrowing hairs, which are newly emerging, straight, and often pigmented hairs growing vertically from the scalp. **d** Hypopigmented hair regrowth: Hypopigmented hair regrowth, reflecting the re-emergence of hair with reduced pigmentation, often seen in the regrowth phase after an episode of hair loss

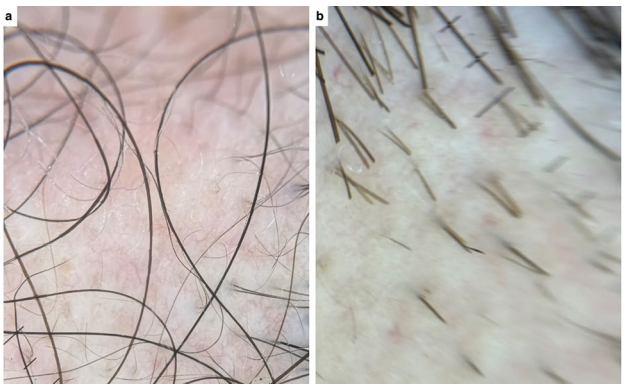

Fig. 5 Vascular and other dermoscopic findings in alopecia areata. **a** Telangiectasia: Trichoscopic image showing telangiectasia characterized by visible dilated blood vessels on the scalp, which may indicate underlying inflammation associated with AA. **b** V-sign: The appearance of the v-sign, where two or more hairs emerge from a single follicular opening and form a "V" shape, suggesting early regrowth or changes in follicular dynamics in patients with AA

Ordinal regression analysis of factors associated with SALT score severity

Ordinal regression analysis investigating the factors influencing SALT score severity in patients with AA identified several critical variables that significantly impact disease severity. Key findings showed that each additional month of disease duration was associated with a statistically significant increase in severity, with an odds ratio (OR) of 1.034 (95% CI: [1.021, 1.048], $p < 0.001$). Nail changes also emerged as a significant exacerbator, drastically increasing severity with an OR of 2.31 (95% CI: [1.57, 3.40], $p = 0.001$). Moreover, the presence of neuropsychiatric conditions notably heightened disease severity, with an OR of 9.165 (95% CI: [1.751, 47.985], $p = 0.009$). Conversely, variables such as gender did not show a statistically significant impact, despite males having an OR of 3.91; this finding was not statistically significant ($p = 0.114$), indicating a lack of conclusive evidence to suggest that males have a decreased risk of severe AA compared to females. Other factors including family history of AA, micronutrient deficiencies, autoimmune and endocrine disorders, atopic diseases, and metabolic complications also did not exhibit significant associations with SALT score severity (Table 8) (Fig. 7).

Discussion

This prospective, single-center, cross-sectional study included 303 patients with AA ranging in age from 6 months to 84 years over a period of 1.5 years. The primary objective was to assess the clinical, demographic, and comorbid associations in these patients and explore the correlation between trichoscopic findings and disease severity. Research shows that AA prevalence varies significantly across ethnic groups in the U.S., with the highest rates in Asians and the lowest in Whites, indicating a disproportionate impact on people of color. Geographically, the incidence varies from 0.54% in North America to 4.92% in Europe and 1.92% in Asia, highlighting the influence of regional and ethnic factors [31, 32]. A retrospective study in our country reported a 3.02% prevalence of AA among dermatology outpatients, predominantly affecting individuals under 40 years of age, suggesting an increasing prevalence over time and regional variability [33]. Following the earthquake in our region in February 2023, it is plausible that the prevalence of AA may have increased, given that significant psychological stress can potentially exacerbate autoimmune conditions like AA. In our study, AA demonstrated a balanced gender distribution with a slight female predominance, aligning with a single-center study in our country that indicated a more balanced gender distribution [33], and contrasting with a multicenter study that reported a more pronounced male predominance [34].

Table 3 Distribution of trichoscopic findings across alopecia areata subtypes

Trichoscopic Finding	Patchy AA (%)	Multiple patchy AA (%)	Alopecia ophiasis (%)	Alopecia Totalis/ Universalis (%)	p-value
Yellow dots	53.08	60.20*	38.60	38.89	**0.045**
Black dots	39.23	39.80	50.88	33.33	0.399
Broken hairs	50.00	51.02	57.89	33.33	0.336
Exclamation mark hair	58.46	54.08	66.67	61.11	0.493
Small vellus hairs	66.15	76.53†	75.44†	27.78‡	**< 0.001**
Coudability hair	54.62	47.96	77.19†	44.44	**0.003**
Tulip hair	39.23	38.78	49.12	16.67	0.104
Pigtail hairs	30.77	37.76	43.86†	0.00‡	**0.004**
Telangiectasia	27.69	27.55	33.33	22.22	0.780
Perifollicular scaling	14.62	19.39	28.07	5.56	0.075
Pohl pinkus constrictions	39.23	27.55	45.61	22.22	0.060
Hypopigmented hair growth	29.23	26.53	45.61†	16.67	**0.036**
Upright regrowing hair	45.38	38.78	35.09	27.78	0.346
V-sign	10.77	13.27	22.81	38.89†	**0.006**
Cumulus like white dots	10.77	11.22	33.33	77.78‡	**< 0.001**

Statistically significant p-values are in bold

Trichoscopic analysis reveals distinctive patterns across AA subtypes: CLWD and V-sign are prominently elevated in AT/AU, at 77.78% and 38.89% respectively, signifying their association with more severe disease stages. Conversely, the absence of PH in AT/AU highlights its relevance to milder forms. SVH is notably prevalent in less severe subtypes, with 76.53% in Multiple patchy AA and 75.44% in AO, sharply contrasting with 27.78% in AT/AU ($p < 0.001$). Additionally, HHG's prevalence in AO at 45.61% versus lower rates in Patchy AA and Multiple patchy AA (29.23% and 26.53%) emphasizes its diagnostic utility ($p = 0.036$). Statistical significance is denoted by: * for $p < 0.05$, † for $p < 0.01$, and ‡ for $p < 0.001$, indicating significant differences across age groups in the distribution of trichoscopic findings in AA

Such variations may be attributable to regional differences or methodological disparities.

Our study reported an average patient age of 23.67 years and an average disease onset age of 21.68 years, with 43.2% of our patients under the age of 20 years. This contrasts with findings from a larger epidemiological study, where the mean age at diagnosis was 33.6 years, with fewer young patients and incidence rates peaking between 20 and 39 years of age [35]. Additionally, 11.55% of our cohort experienced disease onset at age 40 or older. These findings indicate significant demographic or genetic factors driving the earlier onset of AA in our cohort, reflecting variations in age-specific susceptibility or environmental influences. Our data showed that AA incidence peaks in the 20–30 year age group, yet the presence of cases across all ages highlights its persistent lifetime risk, emphasizing the importance of exploring age-related dynamics in its etiology and management. In our study, the youngest patient was only 6 months old, underscoring the rarity of AA in very young children, likely influenced by immune system immaturity. Among our pediatric patients, the average age at diagnosis was 9.1 years, with an onset age of approximately 8.1 years, which is later than the 5.9 years reported in the National Alopecia Areata Registry [36]. The relatively lower age of onset in our cohort indicates an earlier manifestation of the disease. The close proximity between average patient age and onset age suggests prompt healthcare seeking behaviors in our patients, likely a result of heightened health awareness or improved healthcare accessibility.

In our study, no significant gender differences were observed in mean age, onset age, disease duration, family history, ANA positivity, nail involvement, or overall comorbidity burden ($p > 0.05$). However, we noted a notably higher prevalence of micronutrient deficiencies, neuropsychiatric disorders, and autoimmune and endocrine comorbidities in female patients. This gender-specific disparity aligns with the findings of Lundin et al., who reported a higher incidence of thyroid disorders in females with AA. In contrast, unlike Lundin et al., we did not find significant gender differences in nail involvement [37]. Our findings suggest that gender does not play a primary role in determining the severity of AA. This highlights the multifactorial nature of the disease, where genetic predispositions and environmental influences emerge as the key drivers of pathogenesis, while gender-related differences appear to have a minimal and indirect impact on disease severity. Population-based studies typically reveal no significant gender differences in the epidemiology of AA, yet variations in hospital-based data suggest differing healthcare access, emphasizing the multifactorial nature of AA's etiology [2].

Table 4 Distribution of trichoscopic findings by age range in patients with alopecia areata

Trichoscopic finding	Age ranges (years)			
	< 20 (%)	20–40 (%)	> 40 (%)	p-value
Yellow dots	43.51*	61.29*	50.00*	**0.017**
Black dots	47.33	35.48	39.58	0.153
Broken hairs	57.25	45.16	47.92	0.141
Exclamation mark hair	58.02	59.68	58.33	0.962
Small vellus hairs	70.99	70.97	58.33	0.221
Coudability hair	50.38	58.59	64.58	0.171
Tulip hair	41.22	35.48	45.83	0.406
Pigtail hairs	30.53	35.48	37.50	0.584
Telangiectasia	19.85†	32.26†	41.67†	**0.008**
Perifollicular scaling	19.08	18.55	14.58	0.778
Pohl pinkus constrictions	32.06	37.90	39.58	0.513
Hypopigmented hair growth	22.90‡	28.23‡	58.33‡	**< 0.001**
Upright regrowing hair	32.06†	50.00†	37.50†	**0.013**
V-sign	11.45*	15.32*	27.08*	**0.038**
Cumulus like white dots	18.32*	14.52*	33.33*	**0.018**

Statistically significant p-values are in bold

Analysis across age groups in AA patients reveals distinct trichoscopic patterns. YD peaks in the 20–40 age range ($p = 0.017$). TG and HHG show significant increases in > 40 s ($p = 0.008$, $p < 0.001$, respectively), indicating age-related changes. URH is more prevalent in 20–40 s ($p = 0.013$), suggesting active or responsive disease phases. V-sign's prevalence increases in > 40 s ($p = 0.038$). CLWD, although higher in > 40 s, reaches statistical significance ($p = 0.018$), highlighting potential age-related differences in disease manifestation. Statistical significance is denoted by: * for $p < 0.05$, † for $p < 0.01$, and ‡ for $p < 0.001$, indicating significant differences across age groups in the distribution of trichoscopic findings in AA

In our study, 22.44% of patients reported a positive family history of AA, consistent with the literature suggesting a genetic predisposition linked to key immune regulatory genes, including PTPN22, CTLA4, IL-2, and various HLA variants [38]. Familial cases often show a correlation between the age of onset in index patients and their first-degree relatives, a trend also reflected in our findings, indicating earlier onset in patients with a family history [39]. Interestingly, previous studies have noted that mothers with AA are more likely to have offspring that develop autoimmune, inflammatory, atopic, thyroid, and psychiatric disorders [40].

Nail changes, observed in 24.75% of our cohort, are generally painless yet have a significant cosmetic impact, potentially diminishing quality of life. These alterations are notably more prevalent in patients with elevated disease severity scores, consistent with the literature, which indicates that up to 30% of AA patients experience nail changes, particularly in severe forms such as AT and AU. The primary manifestations, including pitting and trachyonychia, are attributed to abnormal keratinization in the nail matrix, likely resulting from a loss of immune privilege analogous to that in affected hair follicles [41].

Patients are generally asymptomatic, although some may experience tingling, itching, or dysesthesia before hair loss occurs [42]. In our study, AA primarily presented as localized patchy AA (42.98%), followed by Multiple patchy AA (32.29%), AO (18.75%), AU (4.29%), and AT (1.65%). This distribution, with more common patchy forms and less frequent extensive types, aligns with the prevalence patterns described in the literature, highlighting the typical variability of AA [43]. Differentiating AA from conditions like tinea capitis (TC), trichotillomania (TTM), telogen effluvium (TE), and temporal triangular alopecia often requires histopathology for a definitive diagnosis [44]. Scalp dermoscopy, or trichoscopy, is employed as a valuable, noninvasive tool that not only helps distinguish AA from other forms of hair loss but also correlates with disease severity and activity [45]. In this study, trichoscopic images were captured using a Dermlite DL5 device paired with an iPhone 15 Pro Max, providing high-quality images at magnifications ranging from 10× to 50×. Images were securely stored in a digital cloud to facilitate comprehensive and detailed trichoscopic evaluations conducted via computer analysis. This advanced imaging setup minimizes the need for unnecessary biopsies and assists in identifying optimal biopsy sites, thereby enhancing the diagnostic accuracy and patient care [46].

Our study indicated a high comorbidity rate of 82.18% in AA patients, reflecting its connection to broader systemic health issues due to potential shared genetic predispositions and immune dysregulation. Despite the cost-effectiveness concerns of comprehensive screening for autoimmune conditions in patients with AA, targeted testing based on age and specific risk factors is recommended [47]. Accordingly, our findings underscore the necessity for nuanced patient management strategies that integrate dermatological and systemic health considerations to optimize care and resource allocation.

In our study, 18.48% of AA patients had dermatological conditions, slightly higher than the 17.9% reported in previous studies [48]. This increase could be linked to prolonged disease duration and severity, which may increase the risk of secondary skin disorders. Additionally, treatments such as corticosteroids and immunosuppressants may exacerbate these risks by triggering or worsening other dermatological conditions.

In our study, 15.1% of patients exhibited vitamin D deficiency, which is noteworthy given its immunomodulatory effects, especially on lymphocytes involved in autoimmune responses. Meta-analyses have established that vitamin D deficiency occurs 3.55 times more frequently in AA patients than in controls [49]. Furthermore, a related study indicated

Table 5 Correlation between trichoscopic findings and disease duration in alopecia areata

Trichoscopic finding	Mean duration with feature (months)	Mean duration without feature (months)	P-value (Mann–Whitney U test)
Yellow dots	19.03	31.87	**0.007**
Black dots	18.26	30.11	**0.023**
Broken hairs	15.66	35.10	**0.003**
Exclamation mark hair	20.81	31.50	**0.028**
Small vellus hairs	19.79	37.29	0.848
Coudability hair	23.97	26.82	0.794
Tulip hair	16.92	30.66	0.112
Pigtail hairs	18.78	28.49	0.841
Telangiectasia	21.57	26.93	0.595
Perifollicular scaling	22.46	25.83	0.100
Pohl pinkus constrictions	20.00	28.11	0.966
Hypopigmented hair growth	17.55	28.62	0.086
Upright regrowing hair	24.30	25.84	0.184
V-sign	19.83	26.21	0.258
Cumulus like white dots	62.30	16.44	**< 0.001**

Statistically significant p-values are in bold

The correlations between trichoscopic markers and disease duration in patients with AA revealed distinct patterns. BH, BD, YD, and EMH were significantly associated with shorter disease duration, indicating their association with more acute or active phases. In contrast, CLWD is strongly correlated with a longer disease duration, highlighting its relevance as a marker of chronic disease stages. Other markers such as SVH, CH, and Telangiectasia did not show significant differences, reflecting their variable roles in disease progression

Table 6 Correlation between trichoscopic findings and SALT score in patients with alopecia areata

Trichoscopic finding	Frequency (n, %)	Median SALT score (IQR)	Spearman's correlation coefficient (r)	p-value
Yellow dots	157, (51.82%)	6.3 (14.00)	– 0.06	0.332
Black dots	125, (41.25%)	7.6 (23.30)	0.06	0.305
Broken hairs	154, (50.83%)	6.3 (20.50)	0.00	0.950
Exclamation mark hair	178, (58.75%)	6.8 (22.40)	0.05	0.364
Small vellus hairs	209, (68.98%)	7.2 (14.40)	0.03	0.587
Coudability hair	170, (56.11%)	7.2 (22.35)	0.09	0.104
Tulip hair	120, (39.60%)	6.3 (22.65)	0.00	0.998
Pigtail hairs	102, (33.66%)	8.0 (21.55)	0.11	**0.050**
Telangiectasia	86, (28.38%)	7.0 (11.58)	– 0.04	0.530
Perifollicular scaling	55, (18.15%)	7.6 (19.00)	0.04	0.539
Pohl pinkus constrictions	108, (35.64%)	7.8 (22.05)	0.07	0.255
Hypopigmented hair growth	93, (30.69%)	7.2 (20.70)	0.08	0.172
Upright regrowing hair	122, (40.26%)	6.0 (13.53)	– 0.03	0.572
V-sign	47, (15.51%)	16.0 (32.35)	0.19	**0.001**
Cumulus like white dots	58, (19.14%)	27.3 (57.45)	0.29	**< 0.001**

Statistically significant p-values are in bold

Correlation analysis highlighted the strong associations of CLWD and v-sign with higher SALT scores, emphasizing their importance in assessing disease severity ($p < 0.001$ for both). Pigtail hair also showed a positive correlation, suggesting its potential relevance to AA progression ($p = 0.050$). Other markers, such as PS and PPC, did not reach statistical significance, reinforcing the specificity of CLWD, v-sign, and PH in evaluating AA severity

Table 7 Distribution of trichoscopic findings among treated and naive patients with alopecia areata

Trichoscopic Finding	Naive patients	Treated patients	p-value
Yellow dots	121 (53.07%)	36 (48.00%)	0.529
Black dots	103 (45.18%)	22 (29.33%)	**0.022**
Broken hairs	128 (56.14%)	26 (34.67%)	**0.002**
Exclamation mark hair	143 (62.72%)	35 (46.67%)	**0.021**
Small vellus hairs	151 (66.23%)	58 (77.33%)	0.097
Coudability hair	127 (55.70%)	43 (57.33%)	0.910
Tulip hair	96 (42.11%)	24 (32.00%)	0.157
Pigtail hairs	51 (22.37%)	51 (68.00%)	**< 0.001**
Telangiectasia	56 (24.56%)	30 (40.00%)	**0.015**
Perifollicular scaling	36 (15.79%)	19 (25.33%)	0.092
Pohl pinkus constrictions	81 (35.53%)	27 (36.00%)	1.000
Hypopigmented hair growth	71 (31.14%)	22 (29.33%)	0.881
Upright regrowing hair	65 (28.51%)	57 (76.00%)	**< 0.001**
V-sign	39 (17.11%)	8 (10.67%)	0.249
Cumulus like white dots	48 (21.05%)	10 (13.33%)	0.192

Statistically significant p-values are in bold

Differences in trichoscopic markers between naive and treated patients reveal significant insights into the dynamics of AA. PH and URH showed an increased prevalence in treated individuals, suggesting that these markers are responsive to treatment (PH: 68.00%, URH: 76.00%). Conversely, higher frequencies of BD, BH, and EMH in naive patients compared to those treated (BD: 45.18% vs. 29.33%; BH: 56.14% vs. 34.67%; EMH: 62.72% vs. 46.67%) highlight their association with greater disease activity or severity, underscoring the potential effectiveness of therapies in reducing these markers indicative of active disease. Additionally, the increased prevalence of telangiectasia in treated patients (24.56–40.00%) suggests possible treatment-related changes or nuances of disease progression

that specific trichoscopic features such as BH and EMH were more prevalent in progressive cases of AA and were associated with lower vitamin D levels, suggesting that these markers could serve as indicators of both disease activity and nutrient status [50]. Additionally, 29.04% of our patients were iron deficient. Although iron deficiency is commonly associated with other forms of hair loss, such as TE and androgenic alopecia (AGA), its direct role in AA remains unclear. Some evidence suggests that iron deficiency may trigger the onset of AA, although it does not appear to affect long-term disease progression. Unlike AGA, where correcting iron levels is beneficial, there is currently no clinical trial evidence to support similar benefits for AA [51]. Furthermore, 22.77% of patients had vitamin B12 deficiency. Despite its crucial role in DNA synthesis and cellular turnover, important for rapidly dividing tissues like hair follicles, the link between B12 deficiency and AA remains speculative. Routine screening for B12 deficiency in AA patients is generally not recommended unless other autoimmune conditions or symptoms are present [52].

The significant psychosocial impact of chronic conditions, such as AA, highlights the critical role dermatologists play in addressing the psychological aspects to enhance patient outcomes [53]. Our study found an 11.2% prevalence of psychiatric comorbidities such as anxiety, depression, and bipolar disorder in patients with AA. A cohort study revealed a 4.7% cumulative incidence of psychiatric disorders over five years in AA patients versus 3.1% in controls, marking a significantly higher risk with an adjusted hazard ratio of 1.38 for conditions such as depression and anxiety [54]. Our findings also align with those of recent studies

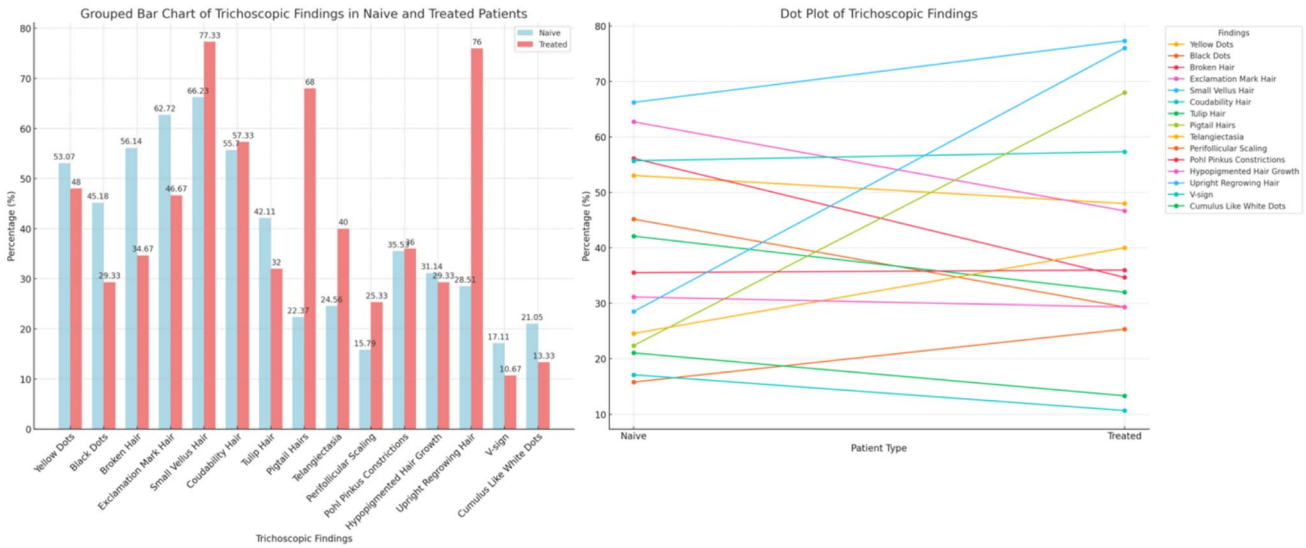

Fig. 6 Comparative distribution of trichoscopic findings in naive and treated patients with alopecia areata. The grouped bar chart and dot plot illustrate the distribution of trichoscopic findings across naive and treated patients, highlighting the effects of treatment on hair and scalp characteristics in AA

Table 8 Ordinal regression analysis of clinical and demographic factors influencing SALT score severity in patients with alopecia areata

Variable	Category	Odds ratio (OR)	95% CI	P-value
Gender	Male	3.91	[0.72, 21.22]	0.114
	Female (Ref.)	1.00	Reference	
Age	Per year increase	0.969	[0.905, 1.038]	0.373
Duration of disease	Per month increase	1.034	[1.021, 1.048]	**< 0.001**
Family history of AA	Yes	1.755	[0.367, 8.392]	0.481
	No (Ref.)	1.00	Reference	
Nail changes	Yes	8.361	[1.828, 38.255]	**0.006**
	No (Ref.)	1.00	Reference	
Micronutrient deficiencies	Present	1.315	[0.271, 6.391]	0.734
	Absent (Ref.)	1.00	Reference	
Autoimmune and endocrine diseases	Present	0.752	[0.153, 3.689]	0.726
	Absent (Ref.)	1.00	Reference	
Atopic diseases	Present	0.388	[0.049, 3.091]	0.372
	Absent (Ref.)	1.00	Reference	
Dermatologic comorbidity	Present	0.446	[0.056, 3.556]	0.446
	Absent (Ref.)	1.00	Reference	
Neuropsychiatric diseases	Present	9.165	[1.751, 47.985]	**0.009**
	Absent (Ref.)	1.00	Reference	
Metabolic complication	Present	0.097	[0.004, 2.568]	0.163
	Absent (Ref.)	1.00	Reference	

Statistically significant p-values are in bold

This analysis highlights key factors significantly influencing SALT score severity in AA patients. Notably, disease duration directly correlates with increased severity, demonstrated by an odds ratio (OR) of 1.034 for each additional month (95% CI: [1.021, 1.048], $p < 0.001$). Significant exacerbators also include nail changes (OR: 2.31, 95% CI: [1.57, 3.40], $p = 0.001$) and neuropsychiatric conditions (OR: 9.165, 95% CI: [1.751, 47.985], $p = 0.009$), underscoring their substantial roles in disease progression. Other factors such as gender and micronutrient deficiencies did not show significant impacts, suggesting more focused areas for potential clinical interventions. Odds ratios (ORs) quantify the strength and direction of associations, while the accompanying confidence intervals (CIs) provide a range of plausible values, helping to interpret the precision and reliability of the results. Reference categories provide baselines for these comparisons, with bold values denoting statistical significance ($p < 0.05$)

showing that women with AA in our cohort were notably more susceptible to psychiatric symptoms. This suggests a bidirectional relationship where each condition may exacerbate the other, potentially intensifying the challenges faced by these patients [55].

In our study, the prevalence rates of ANA positivity (9.6%), elevated TPOAb levels (7.92%), and TGAb (7.7%) were consistent with the general population averages, indicating no heightened autoimmune risk within our cohort. The balanced gender distribution and predominantly young demographics may account for the moderate autoimmune marker levels, typically higher in older or predominantly female groups. Despite the relatively short follow-up period potentially limiting the detection of new onset conditions, our findings concur with existing data on the prevalence of autoimmune and allergic conditions. For instance, a longitudinal UK study spanning ten years indicated a higher incidence of atopic and autoimmune conditions among patients with AA, including atopic dermatitis, allergic rhinitis, and autoimmune hypothyroidism, underscoring the need for extended monitoring to capture the full spectrum of associated conditions [56]. The interplay between Th1 and Th2 immune responses in AA adds complexity to the traditional Th1/Th2 paradigm, which is challenged by observations that both atopic and non-atopic AA patients exhibit significant systemic inflammation, indicating diverse immune profiles within the AA population [57]. Moreover, our observations of heightened AA activity during seasonal transitions highlight the potential role of environmental triggers in exacerbating AA, further supported by recent findings of seasonal exacerbation patterns, particularly in late autumn and early winter [58]. Allergen immunotherapy, especially targeting house dust mites, has shown promising results in reducing relapse severity and improving outcomes by modulating Th2 responses, particularly in patients without extensive disease and in pre-adolescents [59]. The cumulative prevalence of atopic conditions in our AA patients was 21.4%, with asthma, allergic conjunctivitis, and allergic rhinitis showing rates similar to those reported in the literature

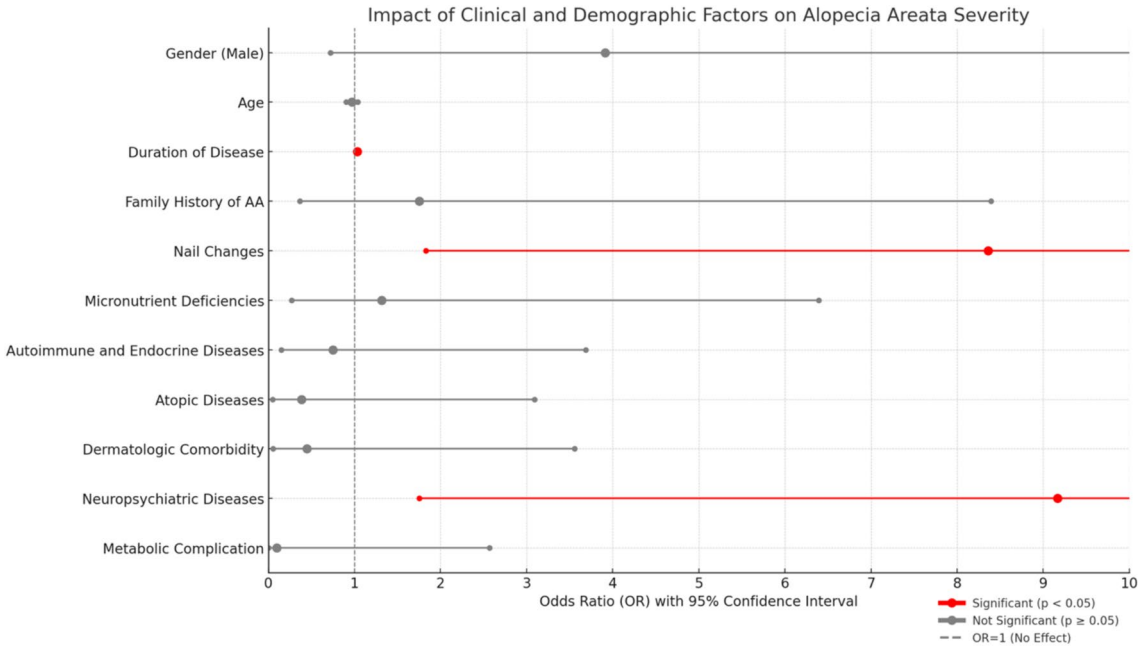

Fig. 7 Impact of clinical and demographic factors on alopecia areata severity. The diagram displays an ordinal regression analysis that evaluates the impact of various factors on SALT score severity in AA. Significant factors such as disease duration, nail changes, and neuropsychiatric conditions are highlighted in red, indicating their strong influence on disease severity. Conversely, factors like gender, age, family history of AA, and specific comorbidities—autoimmune and endocrine disorders, micronutrient deficiencies, atopic diseases, and metabolic complications—are shown in gray, underscoring their minimal impact. These findings help delineate the primary and secondary contributors to AA progression, emphasizing the differential impacts based on gender. Each variable's odds ratio is represented as a point on the horizontal axis, aligned with its confidence interval, providing a clear visual interpretation of its effect size and statistical significance

[60], underscoring the consistent susceptibility of this group to allergic conditions.

Our study identified a 5.61% prevalence of autoimmune thyroid disease in patients with AA, closely mirroring the 4% prevalence noted in a multicenter European study [61]. The overall prevalence of thyroid disorders in our cohort was 12.54%, which was slightly lower than the 13.3% reported in a recent meta-analysis, reinforcing the established association between AA and autoimmune thyroid diseases [62]. Notably, contrary to similar studies, our findings did not indicate a higher prevalence of thyroid disease in severe forms of AA, nor were there significant variations in thyroid dysfunction across different clinical patterns of AA. Our analysis did reveal significant gender differences in the prevalence of autoimmune and endocrine disorders, with these conditions occurring more frequently in females ($p < 0.05$). A population-based study has highlighted a bidirectional relationship between AA and thyroid diseases, suggesting that each condition may elevate the risk of the other [63]. Given the presence of subclinical thyroid disorders and elevated thyroid autoantibodies in our patients, we emphasize the necessity for regular monitoring. This strategy is crucial for early detection and intervention to prevent the progression to overt thyroid disease, aligning with recommendations for biannual thyroid function tests in patients with documented antibodies or clinical signs of thyroid dysfunction.

Metabolic and cardiovascular diseases share pathogenic mechanisms with AA, notably through the disruption of immune privileges in the hair follicles. This disruption leads to increased expression of MHC I/II, ICAM-2, ECAM-1, and IFN-γ, which activate the JAK-STAT pathway and promote T cell infiltration [64]. In our study, the prevalence of T2DM was 3,63%, slightly higher than the 3.3% prevalence reported in a meta-analysis which suggests a lower association between AA and T2DM (OR = 0.64) [16]. Furthermore, we observed lower than expected rates of HT at 4.2% and dyslipidemia at 3.63%, which may be influenced by the younger mean age of our cohort or by specific immune system interactions. This contrasts with a cross-sectional study which reported higher rates of HT, obesity, hyperlipidemia, T2DM, and coronary artery disease among AA patients, though myocardial infarction rates were comparable to controls [65]. A systematic review found no significant association between AA and major cardiovascular diseases, including HT and T2DM, highlighting the intricate interplay of immune-mediated pathways in these conditions [66]. Notably, the use of JAK-STAT inhibitors, increasingly prescribed for AA, may offer additional benefits by managing systemic

inflammation, obesity, diabetes, and cardiovascular risk in these patients [67, 68].

Current data indicate that 34% to 50% of AA patients regrow hair within a year, while 15% to 25% may progress to total scalp or body hair loss, with less than 10% achieving full recovery in advanced cases [69]. Conversely, a long-term study showed that 67% of patients with less than 25% hair loss achieved full regrowth [18]. Our study confirms that disease duration significantly increases with age, with patients under 20 averaging approximately 14 months, those aged 20–40 averaging approximately 26 months, and those over 40 averaging approximately 65 months ($p < 0.001$). Additionally, we found a weak but statistically significant positive correlation between age and the SALT score ($r = 0.11$, $p = 0.03$), suggesting a slight increase in disease severity with age. These findings highlight the importance of early intervention in younger patients, as timely management may help prevent disease progression and increase disease severity over time. The Alopecia Areata Consensus of Experts (ACE) also notes that prognosis worsens after 6–12 months, and hair loss may become irreversible after 5–8 years [70].

In our study, the SALT score was used to classify the severity of scalp hair loss. However, recent perspectives have suggested a more comprehensive approach that combines clinical outcomes with patient-reported quality of life measures [71]. Our analysis demonstrated that the number of trichoscopic findings did not significantly correlate with the SALT score severity ($p = 0.809$), suggesting that the diversity of trichoscopic patterns does not directly reflect the clinical severity of AA. This highlights the complexity of the disease, indicating that comprehensive evaluation extending beyond trichoscopic examination is crucial for accurate assessment and management.

In our study, SVH was the most prevalent trichoscopic finding, observed in 68.98% of cases, indicating follicular miniaturization and partial hair regrowth. This aligns with the findings of Inui et al., who also identified SVH as a common feature in AA and noted its negative correlation with disease activity or severity, suggesting the potential for regrowth even when hair recovery is not yet visible [72]. However, a broader analysis of trichoscopic patterns revealed that while SVH is frequently seen across various types of alopecia, its presence is not specific to AA, as it also appears in TTM and TE [27, 73]. In our study, SVH are notably prevalent in less severe subtypes of AA, especially after treatment, indicating their potential as markers of positive therapeutic response and follicular recovery.

In our study, YD were observed in 51.82% of cases, highlighting their prominence among trichoscopic findings and underscoring their significant diagnostic value in identifying AA. While YD is a prevalent feature in AA across various studies, emphasizing its diagnostic significance [74], it is also seen in other scalp disorders, such as AGA, chronic cutaneous lupus erythematosus, and dissecting cellulitis [75]. Miteva and Tosti noted that YD is rare in prepubertal children because of underdeveloped sebaceous glands, consistent with our data showing a lower prevalence in individuals under 20 years of age [45]. In our study, YD prevalence peaked in individuals aged 20–40 ($p < 0.05$), a finding specific to our research, and a slight decline in individuals aged > 40 years. YD in frontal fibrosing alopecia has been associated with preserved sebaceous glands and has been linked to hair regrowth [76]. Our findings indicate that YD persists consistently in both treated and untreated groups in AA ($p = 0.529$), emphasizing its role as a stable marker of disease pathology and potentially reflecting chronic AA or a suboptimal treatment response [77].

In our study, BH was observed in 50.83% of cases and BD in 41.25%, both reflecting fragmented or damaged hair shafts, and commonly linked to AA disease activity. While these features are specific diagnostic markers for AA, their presence in conditions like TTM and TC requires careful differential diagnosis [78]. BD is generally associated with the early acute phase of AA and is less frequent in chronic cases or in patients showing regrowth after treatment [79]. Mane et al. demonstrated that BD, identified through dermoscopy, were significantly correlated with shorter disease duration, averaging 5.31 months compared to 16.33 months for patients without BD, underscoring their diagnostic value in the early stages of AA [80]. In our study, BH, BD, YD, and EMH were linked to shorter disease duration, following a sequential pattern from the most acute to transitional phases of AA, highlighting their significance as markers of disease activity.

EMH, characterized by a tapered base and broader tip, was observed in 58.75% of the cases in our study, making it the second most common trichoscopic finding. Although traditionally recognized as a marker of active AA stages, which is consistent with findings from our study, EMH has also been observed in stable, non-progressive phases according to some studies [81]. Research indicates that similar proximal tapering often correlates with more severe AA, reinforcing EMH as a valuable marker of disease activity [82]. Although EMH is observed in other hair loss disorders, its identification as one of the most frequently detected trichoscopic findings in our study emphasizes its pathognomonic significance in AA, highlighting its critical role in diagnosis.

CH, first identified by Shuster in 1984, is a key trichoscopic feature of AA, characterized by hair shafts that bend easily without breaking, reflecting structural weakness [83]. In our study, CH was observed in 56.11% of the cases, making it the third most common trichoscopic finding. Inui et al. showed that CH correlates with active disease markers, including positive hair-pull tests, short disease duration, BD, and EMH, indicating a rapid transition from the anagen

to catagen phase [84]. This suggests that, while CH reflects a structural weakness that allows follicles to continue producing compromised hair, it is particularly associated with the active phases of AA. Further studies by Vyshak, Nikam, and Mehta confirmed its association with active disease [85, 86], while Bains and Kaur proposed that it may serve as an indicator of more severe forms of AA [87].

TH is characterized by its light color and distinct darkened tip resembling a tulip, reflecting increased pigmentation and oblique hair rupture, as described by Rudnicka et al. [29]. In 39.60% of our AA cases, TH prevalence was notably higher than the 10% historically noted in AA, and closely aligned with the 48% found in TTM cases. This prevalence and the distinct morphology of TH suggest its utility not only in diagnosing AA and TTM, but also in understanding the timing and progression of these conditions, potentially indicating phases of active disease and hair fragility [78].

PPC was first described by Joseph Pohl-Pincus in 1885 as a region of reduced hair shaft thickness arising from disruptions in the metabolic and mitotic activity of hair follicles. These disruptions can result from various triggers, including chemotherapy, infections, malnutrition, or autoimmune conditions such as AA [88]. PPCs are typically observed during the active phase of AA and serve as indicators of heightened disease activity [89]. In our study, PPCs were present in 35.64% of the cases, a significantly higher prevalence than the 2–10% reported in previous studies [27].

In our study, PH, also known as circle hairs, was observed in 33.66% of cases, underscoring its role as a marker of therapeutic response and follicular recovery in AA. This finding is consistent with Waśkiel-Burnat et al., who identified PH as a predictor of regrowth during remission, highlighting its diagnostic importance in monitoring treatment progress through trichoscopy [90, 91]. Our findings reveal that PH in AA serves a dual role, predominantly indicating follicular recovery during treatment response and, to a lesser extent, reflecting the dynamics of disease progression in milder cases, thereby serving as a vital marker for evaluating both therapeutic efficacy and disease activity.

URH, characterized by distal tapering, indicates early stages of hair regrowth and is typically observed during the recovery phase in patients with AA [92]. In our study, URH was observed in 40.26% of cases, indicating active hair regrowth, particularly in the 20–40 age group, which suggests a higher treatment response or greater regenerative capacity in younger individuals. Similarly, Waśkiel-Burnat et al. identified URH as a reliable marker of regrowth and treatment response in AA, as it signals restored follicular activity with new hair shafts gradually thickening and regaining pigmentation [91].

In our study, HHG was observed in 30.69% of cases, indicating early signs of follicular recovery in AA. Notably, the prevalence of HHG increases in individuals over the age of 40 years, suggesting its association with chronic or longstanding cases of AA. These findings highlight that the immune-mediated destruction of pigmented hair follicles, potentially driven by melanogenesis-associated autoantigens, does not uniformly affect all age groups. Moreover, the alternating pigmentation observed during hair regrowth might reflect phases of melanocyte inactivity followed by recovery, which is indicative of dynamic changes in the hair follicle environment. These findings underscore the possibility of canities subita, where a sudden onset of white hair could signify an acute autoimmune response selectively targeting pigmented follicles, often resulting in permanent depigmentation. Conversely, the reversible nature of HHG suggests a variable impact on melanin-producing cells, with some patients experiencing transient depigmentation and others potentially regaining full pigment, in contrast to conditions such as poliosis, where depigmentation may indicate more prolonged or definitive melanin loss owing to enduring immune effects [93, 94].

CLWD, identified as clusters of white dots in trichoscopy linked to eccrine duct openings, suggests perifollicular fibrosis or sebaceous gland abnormalities, indicating follicular damage. Present in 19.14% of AA cases, CLWD is closely associated with more advanced, fibrotic stages of the disease, marked by statistically significant extended disease durations compared to cases without this feature ($p < 0.001$). Additionally, the prevalence of CLWD increases with age, demonstrating a significant age-related variation in this trichoscopic marker. Furthermore, a higher median SALT score in cases with CLWD ($p < 0.001$) confirms its critical role in indicating severe, chronic AA. These findings are consistent with Kibar et al.'s research, which also links CLWD to heightened AA severity and progression [81].

Telangiectasia, characterized by dilated small blood vessels visible on the surface of the skin, is a significant dermoscopic finding in AA and is often linked to underlying inflammation, chronic sun exposure, or other scalp conditions. It can also arise from intralesional steroid injections, which may indicate treatment-induced vascular changes, rather than disease severity. In our study, telangiectasia was observed in 28.38% of the cases, surpassing the 15.3% prevalence reported by Bains and Kaur [87]. Furthermore, our data indicate an age-related increase in telangiectasia, potentially exacerbated by factors such as sun exposure and chronic inflammation, and also suggest its utility as an indicator of response to treatments, such as intralesional corticosteroid injections. While thin arborizing vessels commonly appear in the temporal and occipital regions, the presence of thick arborizing vessels could signify underlying connective tissue diseases, such as DLE or dermatomyositis, or be a consequence of steroid-induced atrophy [95]. A study evaluating dermoscopic changes before and after immunotherapy in patients with AA

underscored the role of VEGF-mediated neoangiogenesis in treatment, emphasizing the potential of telangiectasia as a marker of therapeutic response and highlighting the critical role of microvascular formation in managing AA [96].

PS, observed in 18.15% of our AA patients, is a common but nonspecific dermoscopic feature, and is also found in conditions such as psoriasis, seborrheic dermatitis, and lichen planopilaris [88]. While PS may indicate active inflammation in AA, its presence necessitates careful differential diagnosis to consider other scalp disorders.

In our study, v-sign was observed in 15.51% of the AA cases and was characterized by two hairs breaking at the same length to form a v-shape. Although traditionally more common in TTM, Mani et al. reported that in 58% of cases, compared to only 8% in AA, v-sign is typically associated with mechanical trauma from hair pulling. In our AA cohort, however, v-sign appeared to be linked to higher disease severity and increased hair shaft fragility, possibly due to inflammatory processes compromising the hair structure. This fragility, likely exacerbated by follicular inflammation and altered hair cycle dynamics, suggests that the v-sign may serve as a marker of active disease or follicular stress in AA, indicating more than mechanical trauma alone [97].

EMH and CH are more commonly seen in AA, whereas trichoptilosis and flame hair are frequently associated with TTM, emphasizing the need to prioritize specific markers for accurate differentiation between these conditions [96]. AA is characterized by a higher terminal-to-vellus hair ratio compared to AGA and TE, which supports its diverse diagnostic trichoscopic features [98]. In our study, treatment-naive AA patients predominantly exhibited BH, BD, and EMH, markers of active disease and acute inflammation. Conversely, treated patients demonstrated increased prevalence of PH and URH, indicative of successful therapeutic responses and follicular recovery. Furthermore, although the trend in SVH prevalence among treated patients was not statistically significant, it suggests potential alignment with therapeutic response markers. The increased incidence of telangiectasia in treated individuals may reflect microvascular changes related to long-term treatment or chronic diseases. However, minimal changes in YD prevalence show its persistence and limited treatment responsiveness, aligning with literature findings [99]. Studies evaluating intralesional corticosteroids in AA noted rapid new SVH growth and a quick disappearance of AA markers like EMH, BH, and BD, while YD persisted [100, 101]. Similarly, another study on the effectiveness of DPCP showed significant reductions in BD and BH but slower and less pronounced responses in YD, SVH, and EMH [102]. In research investigating the efficacy of JAK inhibitors in AA, trichoscopic findings initially displayed a dense presence of YD and BH; however, as treatment progressed, particularly among responders, there was a notable decrease in BH and BD, accompanied by an increase in SVH and URH [103].

Our findings align with those of previous studies that highlight key trichoscopic features related to AA severity and treatment response (Table 9).

Our ordinal regression analysis identifies prolonged disease duration, nail changes, and neuropsychiatric comorbidities as primary determinants of increased severity in AA. These findings underscore the importance of addressing these factors in the clinical management and therapeutic strategies for AA, emphasizing a comprehensive approach that includes psychological assessment. Supporting these observations, You et al. highlighted a significant correlation between disease duration and AA severity, emphasizing the role of chronicity in disease management. They also noted nail changes as strong indicators of severity, underscoring their clinical relevance. Interestingly, our findings diverge in that early disease onset did not significantly affect disease severity, suggesting variability in the pathophysiological factors across different groups. Additionally, the link between severe AA and both thyroid and other autoimmune diseases suggests a systemic component of AA pathogenesis [109]. Jang et al. identified the extent of hair loss (extensive AA/AT/AU) as the most crucial factor influencing the progression of AA, alongside other significant factors such as early onset, severe nail abnormalities, atopy, and family history [110].

Conclusion

This study offers a nuanced examination of the correlations between trichoscopic findings and disease severity, duration, and comorbidities in AA, providing valuable insights into disease progression. Trichoscopic markers, such as CLWD and v-sign, robustly correlate with elevated SALT scores and extended disease durations, affirming their roles as indicators of chronicity. Conversely, markers like BH, BD, YD, and EMH are indicative of shorter disease durations and reflect active inflammatory phases of AA, highlighting their relevance in assessing disease activity. Additionally, findings such as PH, SVH, and URH suggest new hair growth or a positive treatment response, whereas telangiectasia may reflect prolonged disease or treatment-related changes. These insights emphasize the utility of trichoscopic markers not only in classifying AA severity and activity, but also in refining quantitative scoring systems for a more dynamic and nuanced assessment of the disease. Ordinal regression analysis in our study highlights prolonged disease duration, nail involvement, and neuropsychiatric comorbidities as significant predictors of disease severity in AA. The prevalence and pattern of comorbidities in this cohort align with those in conditions

Table 9 Insights from comparative research on trichoscopic patterns in alopecia areata

Study (Author, Year, Country)	Demographics (sample size, gender distribution, age)	AA subtypes (in order of frequency)	Most common trichoscopic findings (in order of frequency, with percentages)	Trichoscopic findings correlated with disease severity	Trichoscopic findings negatively correlated with disease severity
Inui et al., 2008, Japan [72]	N = 300, 33.7% male, 66.3% female Mean age: 33 years	1. Multiple patchy (38.3%) 2. Diffuse (19.0%) 3. Alopecia totalis (17.0%) 4. Universalis (12.3%) 5. Localized patchy (8.3%) 6. Alopecia ophiasis (5.0%)	1. Small vellus hairs (72.7%) 2. Yellow dots (63.7%) 3. Broken hair (45.7%) 4. Black dots (44.3%) 5. Exclamation mark hair (31.7%)	Yellow dots, black dots	Small vellus hairs
Hegde et al. 2013, India [104]	N = 75, 72% male [54], 28% female [21], Mean age: 26.94 years	1. Patchy (73.3%), 2. Alopecia ophiasis (12%), 3. Sisaphio (4%), 4. Diffuse (4%), 5. Alopecia totalis (1.3%), 6. Alopecia universalis (2.7%)	1. Black dots (84%), 2. Small vellus hairs (68%), 3. Yellow dots (57.33%), 4. Broken hair (37.33%), 5. Exclamation mark hair (18.67%)	Black dots, yellow dots	Small vellus hairs
Bapu et al., 2014, India [105]	N = 116, 53% male, 47% female Mean age: 25.3 years	1. Localized patchy (65.5%) 2. Multiple patchy (22.41%) 3. Alopecia ophiasis (4.31%) 4. Alopecia totalis (2.58%) 5. Alopecia universalis (2.58%)	1. Yellow dots (89.6%) 2. Small vellus hairs (78.4%) 3. Black dots (31.0%) 4. Exclamation mark hair (19.8%) 5. Broken hair (12.9%)	Yellow dots,	None explicitly mentioned
Jha et al., 2017, India [106]	N = 72, 56.94% male, 43.05% female Mean age: 24.43 years	1. Patchy AA (69.4%) 2. Non-progressive AA (22.22%) 3. Alopecia ophiasis (6.94%) 4. Alopecia universalis (6.94%) 5. Alopecia Totalis (5.55%)	1. Yellow dots (79.16%) 2. Black dots (70.8%) 3. Small vellus hairs (44.44%) 4. Broken hair (43.05%) 5. Exclamation mark hair (31.9%)	Yellow dots, black dots	Small vellus hairs, terminal hairs
Mahmoudi et al., 2018, Iran [107]	N = 126, 61.9% male, 38.1% female Mean age: 26.32 ± 11.92 years	1. Alopecia universalis (48.4%) 2. Multiple patchy (23%) 3. Alopecia totalis (12.7%) 4. Localized patchy (11.1%) 5. Alopecia ophiasis (4.8%)	1. Yellow dots (84.1%) 2. Small vellus hairs (62.6%) 3.black dots (48.4%) 4.exclamation mark hair (30.9%) 5.broken hair (9.5%)	Yellow dots	Vellus hairs, broken hair, exclamation mark hair
Waśkiel-Burnat et al., 2019, Poland [90]	N = 100, Children: 54% female, 46% male; Adults: 64% female, 36% male Mean age: Children: 7.6 years, Adults: 23.6 years	Not specified	1. Yellow dots (74%) 2. Empty follicular openings (56%) 3. Broken hair (54%) 4. Black dots (46%) 5. Exclamation mark hair (42%)	Yellow dots, black dots, broken hair, exclamation mark hair	Empty follicular openings, pigtail hairs

Table 9 (continued)

Study (Author, Year, Country)	Demographics (sample size, gender distribution, age)	AA subtypes (in order of frequency)	Most common trichoscopic findings (in order of frequency, with percentages)	Trichoscopic findings correlated with disease severity	Trichoscopic findings negatively correlated with disease severity
Bains & Kaur, 2020, India [87]	N = 52, 46.2% male, 53.8% female Mean age: 22.8 years	1. Localized patchy (17.3%) 2. Multiple patchy (53.9%) 3. Alopecia ophiasis (9.6%) 4. Alopecia totalis (7.7%) 5. Alopecia universalis (11.5%)	1. Black dots (82.7%) 2. Broken hair (69.2%) 3. Yellow dots (61.5%) 4. Small vellus hairs (57.7%) 5. Coudability hair (36.5%)	Yellow dots, broken hair	None explicitly mentioned
Vijay et al., 2020, India [108]	N = 260, 63% male, 37% female Mean age: 26.80 ± 9.816 years	1. Localized patchy (51.92%) 2. Multiple patchy (26.92%) 3. Diffuse (9.61%) 4. Alopecia ophiasis (4.61%) 5. Alopecia totalis (3.84%) 6. Alopecia universalis (3.08%)	1. Yellow dots (72.7%) 2. Small vellus hairs (61.53%) 3. Broken hair (59.23%) 4. Exclamation mark hair (38.46%) 5. Black dots (36.53%)	Broken hair, exclamation mark hair, trichoptilosis	Yellow dots, small vellus hairs
Kaya G., 2025, **Türkiye**	N = 303, 50.83% female, 49.17% male Mean age: 23.67 ± 14.79 years	1. Localized patchy (42.98%) 2. Multiple patchy (32.29%) 3. Alopecia ophiasis (18.75%) 4. Alopecia universalis (4.29%) 5. Alopecia totalis (1.65%)	1-small vellus hairs (%68.98) 2-exclamation mark hair (%58.75) 3- coudability hair (%56.11) 4- yellow dots (%51.82) 5-broken hair (%50.83) 6-upright regrowing hair (%40.26)	Cumulus like white dots, V-sign	Pigtail hairs*, upright regrowing hair, small vellus hairs, telangiectasia

Presented below is an analysis of seminal studies that explore trichoscopic patterns observed in patients with AA. It includes details on demographics, the prevalence of various subtypes of the disease, and common trichoscopic findings alongside their correlation with disease severity. This overview emphasizes the utility of trichoscopy in diagnosing and understanding AA progression, highlighting its role in tailoring patient-specific therapeutic approaches

*Pigtail hairs show a significant increase in treated patients compared to naive ones, indicating their involvement in both disease activity and recovery ($p < 0.001$). While their correlation with disease severity is modest, it reaches the threshold of statistical significance ($p = 0.050$), suggesting a complex role in disease progression

like psoriasis and hidradenitis suppurativa, positioning AA within the broader context of immune-mediated dermatologic disorders and underscoring immune dysregulation in its pathogenesis. Reflecting on real-world data and adhering to ACE guidelines, our findings emphasize the necessity for broader research across diverse populations to develop personalized treatment strategies that cater to the unique manifestations of AA. This approach not only enhances the personalization and effectiveness of AA management, but also addresses the inherent variability and heterogeneity of the disease, ensuring that clinical efforts are directly aligned with individual patient needs based on specific risk factors and demographic considerations.

Limitations

This cross-sectional design limits our ability to infer causality and track the temporal progression of AA. As a single-center study, the generalizability of our findings to broader and more diverse populations is restricted. The absence of long-term follow-up data further constrains our understanding of the chronicity of the disease and the long-term efficacy and safety of therapeutic interventions. Additionally, the inclusion of both treatment-naive and previously treated patients may have introduced variability in trichoscopic findings due to different stages of the hair cycle, potentially leading to underreporting of certain features in the treated group. To address these limitations, future research should focus on multicenter longitudinal studies with extended follow-up periods. Such studies would allow for a better evaluation of the stability of trichoscopic findings, the durability of treatment responses, and potential demographic or geographical variations in disease presentation and management.

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Declarations

Conflict of interest The authors declare no competing interests.

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