LUMEN ET VIRTUS
ISSN: 2177-2789

ALOPECIA AREATA AND AUTOIMMUNE THYROID DISEASES: CLINICAL ASSOCIATION AND SCREENING STRATEGIES

ALOPECIA AREATA E DOENÇAS AUTOIMUNES DA TIREOIDE: ASSOCIAÇÃO CLÍNICA E ESTRATÉGIAS DE RASTREAMENTO

ALOPECIA AREATA Y ENFERMEDADES AUTOINMUNES DE LA TIROIDES: ASOCIACIÓN CLÍNICA Y ESTRATEGIAS DE CRIBADO

https://doi.org/10.56238/levv17n63-020

Submission date: 07/10/2026
Publication date: 08/10/2026

Luciano Custódio dos Santos Lima, Anne Katlen Damasceno Uchoa Thome[1], Isabela Rizzieri Zaque de Jesus[2], Maria Cecília Rodrigues Henckel[3]

ABSTRACT

Introduction: Alopecia areata (AA) is an immune-mediated, nonscarring hair-loss disorder that frequently coexists with other autoimmune conditions. Autoimmune thyroid diseases (AITD), particularly Hashimoto thyroiditis and Graves disease, are among its most commonly reported endocrine associations. Nevertheless, uncertainty remains regarding which patients should undergo thyroid screening and which laboratory tests provide meaningful clinical information.

Objective: To examine the association between AA and AITD, summarize the shared immunological and genetic mechanisms, identify clinical factors associated with thyroid abnormalities, and discuss practical strategies for selective thyroid screening.

Methods: A narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and LILACS. Observational cohorts, case-control studies, systematic reviews, meta-analyses, consensus statements, and genetic analyses evaluating thyroid dysfunction or thyroid autoantibodies in children and adults with AA were considered. Eight recent publications were analyzed in greater detail, with additional studies used to support the clinical and pathophysiological discussion.

Results and Discussion: The available evidence consistently showed a higher prevalence of AITD among patients with AA, with Hashimoto thyroiditis being more frequent than Graves disease. Thyroid autoantibody positivity may occur despite normal thyroid-stimulating hormone (TSH) and free thyroxine (FT4) concentrations, indicating autoimmune susceptibility without established hormonal dysfunction. Extensive or persistent AA, early onset, nail involvement, Down syndrome, family history of thyroid disease, and the coexistence of other autoimmune disorders may increase clinical suspicion, although no isolated feature reliably predicts thyroid abnormalities. TSH is the most appropriate initial laboratory test when risk

[1] E-mail: annethome3@gmail.com
[2] E-mail: isarzj@hotmail.com
[3] E-mail: cissahenckel@gmail.com

factors, symptoms, or suggestive physical findings are present. Anti-thyroid peroxidase antibodies may be useful when autoimmune thyroiditis is suspected, whereas broader antibody panels and thyroid ultrasonography should be reserved for selected cases. Current evidence supports targeted rather than universal screening.

Conclusion: AA is clinically associated with AITD, but the absolute risk varies across populations and clinical phenotypes. Thyroid investigation should be individualized according to symptoms, examination findings, family history, age, associated autoimmune disorders, and disease course. This approach may improve detection of clinically relevant thyroid dysfunction while reducing unnecessary testing and overdiagnosis.

Keywords: Alopecia Areata. Autoimmune Thyroid Diseases. Hashimoto Disease. Thyroid Function Tests.

RESUMO

Introdução: A alopecia areata (AA) é uma doença imunomediada, não cicatricial, caracterizada pela perda de cabelos e pelos, frequentemente associada a outras doenças autoimunes. As doenças autoimunes da tireoide (DAT), especialmente a tireoidite de Hashimoto e a doença de Graves, estão entre suas associações endócrinas mais frequentemente descritas. Entretanto, ainda existem incertezas quanto aos pacientes que devem ser submetidos ao rastreamento da função tireoidiana e aos exames laboratoriais que fornecem informações clinicamente relevantes.

Objetivo: Examinar a associação entre alopecia areata e doenças autoimunes da tireoide, sintetizar os mecanismos imunológicos e genéticos compartilhados, identificar fatores clínicos associados às alterações tireoidianas e discutir estratégias práticas para o rastreamento seletivo da função tireoidiana.

Métodos: Foi realizada uma revisão narrativa utilizando as bases de dados PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library e LILACS. Foram considerados estudos observacionais de coorte, estudos caso-controle, revisões sistemáticas, metanálises, documentos de consenso e análises genéticas que avaliaram disfunção tireoidiana ou autoanticorpos tireoidianos em crianças e adultos com alopecia areata. Oito publicações recentes foram analisadas em maior profundidade, enquanto estudos adicionais foram utilizados para fundamentar a discussão clínica e fisiopatológica.

Resultados e Discussão: As evidências disponíveis demonstraram, de forma consistente, maior prevalência de doenças autoimunes da tireoide entre pacientes com alopecia areata, sendo a tireoidite de Hashimoto mais frequente do que a doença de Graves. A positividade para autoanticorpos tireoidianos pode ocorrer mesmo na presença de concentrações normais do hormônio estimulante da tireoide (TSH) e da tiroxina livre (T4 livre), indicando susceptibilidade autoimune sem disfunção hormonal estabelecida. Alopecia areata extensa ou persistente, início precoce da doença, acometimento ungueal, síndrome de Down, histórico familiar de doença tireoidiana e coexistência de outras doenças autoimunes podem aumentar a suspeita clínica, embora nenhuma característica isolada seja capaz de predizer de forma confiável alterações tireoidianas. O TSH constitui o exame laboratorial inicial mais apropriado quando estão presentes fatores de risco, sintomas ou achados sugestivos ao exame físico. Os anticorpos anti-peroxidase tireoidiana (anti-TPO) podem ser úteis quando

há suspeita de tireoidite autoimune, enquanto painéis ampliados de autoanticorpos e a ultrassonografia da tireoide devem ser reservados para casos selecionados. As evidências atuais apoiam uma estratégia de rastreamento direcionado, em vez de universal.

Conclusão: A alopecia areata está clinicamente associada às doenças autoimunes da tireoide, embora o risco absoluto varie entre diferentes populações e fenótipos clínicos. A investigação da função tireoidiana deve ser individualizada de acordo com sintomas, achados do exame físico, histórico familiar, idade, presença de outras doenças autoimunes e evolução clínica da alopecia. Essa abordagem pode melhorar a detecção de disfunções tireoidianas clinicamente relevantes, ao mesmo tempo em que reduz exames desnecessários e o sobrediagnóstico.

Palavras-chave: Alopecia Areata. Doenças Autoimunes da Tireoide. Doença de Hashimoto. Testes de Função Tireoidiana.

RESUMEN

Introducción: La alopecia areata (AA) es una enfermedad inmunomediada, no cicatricial, caracterizada por la pérdida de cabello y vello, que con frecuencia coexiste con otras enfermedades autoinmunes. Las enfermedades autoinmunes de la tiroides (EAT), especialmente la tiroiditis de Hashimoto y la enfermedad de Graves, se encuentran entre sus asociaciones endocrinas más frecuentes. Sin embargo, aún existen incertidumbres acerca de qué pacientes deben someterse a cribado tiroideo y cuáles son las pruebas de laboratorio que aportan información clínicamente relevante.

Objetivo: Examinar la asociación entre la alopecia areata y las enfermedades autoinmunes de la tiroides, resumir los mecanismos inmunológicos y genéticos compartidos, identificar los factores clínicos asociados con las alteraciones tiroideas y discutir estrategias prácticas para el cribado selectivo de la función tiroidea.

Métodos: Se realizó una revisión narrativa utilizando las bases de datos PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library y LILACS. Se consideraron estudios observacionales de cohortes, estudios de casos y controles, revisiones sistemáticas, metaanálisis, documentos de consenso y análisis genéticos que evaluaron la disfunción tiroidea o los autoanticuerpos tiroideos en niños y adultos con alopecia areata. Ocho publicaciones recientes fueron analizadas con mayor detalle, complementándose con estudios adicionales para respaldar la discusión clínica y fisiopatológica.

Resultados y Discusión: La evidencia disponible mostró de manera consistente una mayor prevalencia de enfermedades autoinmunes de la tiroides entre los pacientes con alopecia areata, siendo la tiroiditis de Hashimoto más frecuente que la enfermedad de Graves. La positividad de los autoanticuerpos tiroideos puede presentarse incluso con concentraciones normales de hormona estimulante de la tiroides (TSH) y tiroxina libre (T4 libre), lo que indica susceptibilidad autoinmune sin disfunción hormonal establecida. La alopecia areata extensa o persistente, el inicio precoz, la afectación ungueal, el síndrome de Down, los antecedentes familiares de enfermedad tiroidea y la coexistencia de otras enfermedades autoinmunes pueden aumentar la sospecha clínica, aunque ninguna característica aislada predice de forma fiable las alteraciones tiroideas. La TSH constituye la prueba de laboratorio inicial más adecuada cuando existen factores de riesgo, síntomas o hallazgos sugestivos en la

exploración física. Los anticuerpos antiperoxidasa tiroidea (anti-TPO) pueden ser útiles cuando se sospecha tiroiditis autoinmune, mientras que los paneles ampliados de autoanticuerpos y la ecografía tiroidea deben reservarse para casos seleccionados. La evidencia actual respalda un enfoque de cribado dirigido, en lugar de un cribado universal.

Conclusión: La alopecia areata se asocia clínicamente con las enfermedades autoinmunes de la tiroides, aunque el riesgo absoluto varía entre diferentes poblaciones y fenotipos clínicos. La evaluación de la función tiroidea debe individualizarse según los síntomas, los hallazgos de la exploración física, los antecedentes familiares, la edad, la presencia de otras enfermedades autoinmunes y la evolución clínica de la enfermedad. Este enfoque puede mejorar la detección de disfunciones tiroideas clínicamente relevantes, al tiempo que reduce la realización de pruebas innecesarias y el sobrediagnóstico.

Palabras clave: Alopecia Areata. Enfermedades Autoinmunes de la Tiroides. Enfermedad de Hashimoto. Pruebas de Función Tiroidea.

1 INTRODUCTION

Alopecia areata (AA) is an autoimmune inflammatory disorder characterized by nonscarring loss of scalp hair or body hair, typically presenting as sharply demarcated patches.[1] Its clinical spectrum ranges from localized involvement of the scalp to extensive forms, including alopecia totalis and alopecia universalis.[1] Although the hair follicle is not permanently destroyed, the clinical course is unpredictable, with spontaneous regrowth, recurrent episodes, or progression to more severe disease.[1] The pathophysiology involves collapse of the immune privilege of the anagen hair follicle and an immune attack primarily mediated by cytotoxic T lymphocytes.[2] Genetic susceptibility, environmental triggers, and immune dysregulation contribute to the initiation and persistence of the disease.[2] The frequent coexistence of AA with other autoimmune disorders supports its interpretation as a manifestation of a broader predisposition to autoreactivity.[2]

Among the autoimmune comorbidities associated with AA, autoimmune thyroid diseases (AITD) are particularly relevant because of their frequency and potential clinical consequences.[3] Hashimoto thyroiditis and Graves disease are the principal forms of this association, although thyroid autoantibodies may precede clinically apparent hormonal dysfunction by several years.[3] In many patients, thyroid antibodies are detected during the evaluation of apparently isolated hair loss.[3] This relationship has been reported across different age groups, including children, adolescents, and adults.[4] However, the magnitude of the association varies according to study design, population characteristics, alopecia severity, and the laboratory criteria used to define thyroid autoimmunity.[4] This lack of consistency makes it difficult to recommend a universal screening strategy for every patient with AA.[4]

The relationship between AA and AITD may be explained by shared immunogenetic mechanisms.[5] Both disorders involve dysregulated antigen presentation, activation of inflammatory pathways, and participation of helper and cytotoxic T lymphocytes.[5] Polymorphisms in genes involved in immune regulation may simultaneously increase susceptibility to follicular immune attack and thyroid autoimmunity.[5] Type 1 inflammatory cytokines, including interferon gamma, contribute to perifollicular inflammation and may also participate in the destruction of thyroid follicular cells.[6] Activation of the Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathway plays a central role in sustaining the inflammatory response observed in AA.[6] This biological overlap helps explain why thyroid abnormalities are detected more frequently in patients with autoimmune alopecia than in the general population.[6]

The presence of AITD does not necessarily indicate abnormal thyroid function at the time AA is diagnosed.[7] Some patients have positive anti-thyroid peroxidase antibodies (anti-TPO) or anti-thyroglobulin antibodies (anti-Tg) despite normal thyroid-stimulating hormone (TSH) and free thyroxine (FT4) concentrations.[7] This euthyroid phase may remain stable or gradually progress to subclinical or overt hypothyroidism.[7] In other patients, fatigue, cold intolerance, weight changes, palpitations, tremor, or menstrual irregularities may already suggest functional thyroid involvement.[8] Because these symptoms are nonspecific, they may be attributed to the psychological burden of hair loss or to unrelated clinical conditions.[8] A targeted clinical history and physical examination are therefore essential for identifying patients who require broader laboratory investigation.[8]

The severity and clinical pattern of AA may influence the likelihood of associated thyroid autoimmunity.[9] Extensive disease, early onset, prolonged duration, nail abnormalities, and a family history of autoimmune disorders have been proposed as possible markers of increased risk.[9] Coexisting vitiligo, atopic dermatitis, systemic lupus erythematosus, type 1 diabetes mellitus, or other autoimmune diseases may further indicate a shared predisposition to immune dysregulation.[9] Nevertheless, findings remain inconsistent, and not all studies have demonstrated a direct relationship between the extent of hair loss and the prevalence of thyroid abnormalities.[10] Part of this disagreement may result from small study populations and the use of different methods for grading alopecia severity.[10] The decision to investigate thyroid disease should therefore not be based solely on the proportion of scalp involvement.[10]

Laboratory screening may include measurement of TSH, FT4, anti-TPO, and anti-Tg.[11] In selected cases, thyroid-stimulating hormone receptor antibodies and thyroid ultrasonography may provide additional diagnostic information.[11] TSH remains the most useful initial test for identifying thyroid dysfunction, although a normal result does not exclude preclinical thyroid autoimmunity.[11] Conversely, indiscriminate use of extensive laboratory panels may increase costs and identify abnormalities that do not immediately change clinical management.[12] Isolated antibody positivity may also cause anxiety, unnecessary repetition of laboratory testing, and referrals without a clear clinical indication.[12] The central challenge is to balance early identification of clinically relevant disease against excessive investigation in asymptomatic patients with a low probability of thyroid dysfunction.[12]

A rational screening strategy should consider age, symptoms, personal and family history, alopecia extent, and the presence of other autoimmune disorders.[13] Children with Down syndrome, a family history of thyroid disease, or clinical findings suggestive of thyroid dysfunction may require earlier testing and periodic follow-up.[13] In adults, thyroid-related symptoms, goiter, menstrual abnormalities, infertility, concomitant vitiligo, or extensive AA

may increase the clinical value of screening.[13] Identification of AITD allows appropriate endocrinological follow-up and treatment of thyroid dysfunction when indicated, although normalization of thyroid hormone levels does not necessarily result in hair regrowth.[14] AA and thyroid disease should therefore be managed as associated but clinically independent conditions with distinct courses and therapeutic responses.[14] This narrative review aims to examine the association between AA and AITD, identify patient groups with a higher probability of thyroid abnormalities, and propose a practical approach to clinical and laboratory screening.[14]

2 OBJECTIVES

The primary objective of this narrative review was to examine the clinical association between alopecia areata (AA) and autoimmune thyroid diseases (AITD). Secondary objectives were to summarize the shared immunological mechanisms between these conditions; assess the reported prevalence of thyroid dysfunction and thyroid autoantibodies in patients with AA; identify clinical characteristics associated with a higher probability of thyroid abnormalities; evaluate the usefulness and limitations of thyroid-stimulating hormone, free thyroxine, anti-thyroid peroxidase antibodies, and anti-thyroglobulin antibodies; and propose a practical, risk-based strategy for thyroid screening and follow-up in children and adults with AA.

3 METHODOLOGY

A narrative review of the literature was conducted to identify evidence concerning the association between AA and AITD. Searches were planned in PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and LILACS. Reference lists from relevant articles were also examined to identify additional publications addressing thyroid autoimmunity, thyroid dysfunction, and clinical screening in patients with AA.

The search strategy combined terms related to alopecia areata, Hashimoto thyroiditis, Graves disease, autoimmune thyroid disease, thyroid dysfunction, hypothyroidism, hyperthyroidism, thyroid autoantibodies, anti-thyroid peroxidase antibodies, anti-thyroglobulin antibodies, screening, and comorbid autoimmune disease. Boolean operators were adapted to each database. Recent publications were prioritized, while older studies were retained when they provided clinically important prevalence data, established diagnostic concepts, or information regarding the evolution of screening recommendations.

Eligible publications included observational cohorts, cross-sectional and case-control studies, systematic reviews, meta-analyses, clinical guidelines, consensus statements, and

studies evaluating thyroid function or thyroid autoantibodies in children or adults with AA. Studies were considered when they reported the prevalence of AITD, laboratory abnormalities, clinical predictors, disease severity, family history, associated autoimmune conditions, or recommendations for thyroid investigation.

Case reports, studies without clear diagnostic criteria, publications focused exclusively on other forms of hair loss, and experimental investigations without direct clinical applicability were excluded from the central synthesis. Because of differences in age groups, ethnicity, laboratory thresholds, definitions of thyroid autoimmunity, and severity classifications for AA, the findings were organized through qualitative comparison rather than quantitative pooling. Particular attention was given to the distinction between clinically manifest thyroid dysfunction and isolated antibody positivity without hormonal abnormalities.

4 RESULTS

The narrative synthesis prioritized eight recent publications that directly evaluated the relationship between alopecia areata (AA), autoimmune thyroid diseases (AITD), thyroid dysfunction, or thyroid autoantibodies. The evidence included nationwide population-based studies, observational cohorts, systematic reviews, meta-analyses, and Mendelian randomization analyses. The principal outcomes were Hashimoto thyroiditis, Graves disease, hypothyroidism, hyperthyroidism, thyroid-stimulating hormone (TSH) abnormalities, anti-thyroid peroxidase antibodies (anti-TPO), and anti-thyroglobulin antibodies (anti-Tg).

The prevalence of thyroid abnormalities varied substantially across studies. Differences were related to age, ethnicity, patient selection in specialized clinics, severity of hair loss, previous diagnosis of thyroid disease, and the laboratory criteria used to define autoimmunity. Studies that considered positive thyroid antibodies generally reported higher rates of abnormalities than those restricted to overt hormonal dysfunction.

Table 1

Main studies evaluating alopecia areata and autoimmune thyroid diseases

Reference	Population / assessment	Outcomes	Main conclusions
Dai et al., 2021	Nationwide population-based cohort using the Taiwanese health insurance database	Subsequent diagnoses of thyroid dysfunction, thyroiditis, Hashimoto thyroiditis, Graves disease, and other thyroid disorders	The association was bidirectional. Patients with AA had a greater probability of subsequently receiving a thyroid diagnosis, while individuals with thyroid disease also had an increased probability of developing AA.
Ly et al., 2023	Systematic review and meta-analysis of 102 studies, including 680,823 patients with AA and more	Medical and autoimmune comorbidities associated with AA	Hashimoto thyroiditis was strongly associated with AA, with an odds ratio of 4.31. The authors emphasized the need for better definition of clinically

Reference	Population / assessment	Outcomes	Main conclusions
	than 72 million controls		appropriate screening criteria.
Celorio et al., 2023	Latin American cohort of patients undergoing thyroid evaluation during the diagnostic investigation of AA	TSH, thyroxine, anti-TPO, anti-Tg, and clinical pattern of AA	Thyroid function and antibody abnormalities were identified across different clinical patterns. Alopecia distribution alone was not a reliable criterion for deciding who should undergo thyroid testing.
Aboalola et al., 2023	Scoping review of clinical studies published between 2011 and 2022	Association between different forms of alopecia and autoimmune thyroid disease	The review supported a clinically relevant association between AA and thyroid autoimmunity but highlighted substantial heterogeneity in prevalence estimates and screening practices.
Gao et al., 2024	Bidirectional two-sample Mendelian randomization study	Genetic associations between AA, Hashimoto thyroiditis, hypothyroidism, Graves disease, TSH, and other thyroid outcomes	Genetic susceptibility to Hashimoto thyroiditis and hypothyroidism was associated with a greater probability of AA. Evidence for a causal relationship with other thyroid outcomes was less consistent.
Zhao et al., 2024	Genetic prediction and Mendelian randomization analysis using genome-wide association datasets	Potential causal effects of hypothyroidism, Hashimoto thyroiditis, hyperthyroidism, Graves disease, and subacute thyroiditis on AA	The results supported a possible genetic relationship between selected thyroid disorders and AA, particularly hypothyroid phenotypes, although confirmation in clinical populations remains necessary.
Krajewski et al., 2025	Meta-analysis of 18 case-control studies, including 462,945 patients with AA and more than 11 million controls	Immune-mediated comorbidities	Patients with AA had increased odds of autoimmune thyroiditis and several other immune-mediated disorders. The pooled odds ratio for autoimmune thyroiditis was 1.57.
Wohl et al., 2026	Nationwide case-control study including 33,401 patients with AA and 66,802 matched controls	Prevalence and chronological relationship of Hashimoto thyroiditis and Graves disease	Hashimoto thyroiditis occurred in 6.93% of patients with AA and 4.26% of controls, while Graves disease occurred in 0.93% and 0.55%, respectively. Both associations remained statistically significant.

The largest studies consistently demonstrated that AA is associated with an increased probability of AITD. Hashimoto thyroiditis was identified more frequently than Graves disease, reflecting both its greater prevalence in the general population and its stronger representation in dermatological cohorts. The magnitude of the association was higher in some meta-analyses than in nationwide database studies, possibly because specialized cohorts included patients with more severe disease or a greater autoimmune burden.

Thyroid antibody positivity was more common than overt thyroid dysfunction in several clinical series. A patient could therefore present elevated anti-TPO or anti-Tg concentrations while maintaining normal TSH and free thyroxine levels. This distinction was clinically important because antibody positivity indicates autoimmune susceptibility but does not necessarily require immediate hormonal treatment.

Evidence regarding the relationship between AA severity and thyroid abnormalities was inconsistent. Extensive AA, alopecia totalis, alopecia universalis, nail involvement, prolonged disease, and coexistence of other autoimmune disorders were associated with thyroid autoimmunity in some populations, but these characteristics were not reliable predictors in every cohort. The clinical pattern of alopecia alone was therefore insufficient to determine the need for thyroid investigation.

The chronological relationship also varied among the studies. In the large nationwide case-control study, Hashimoto thyroiditis preceded AA in more than half of the affected patients, while Graves disease more frequently appeared before the diagnosis of alopecia. However, a relevant proportion of patients developed thyroid disease after the onset of hair loss, supporting continued clinical surveillance rather than reliance on a single lifetime assessment.

Overall, the evidence supported targeted screening based on symptoms, physical findings, age, family history, additional autoimmune diseases, and the severity or persistence of AA. Universal measurement of extensive thyroid antibody panels in every asymptomatic patient was not consistently supported, particularly because isolated antibody positivity may not alter immediate clinical management.

5 RESULTS AND DISCUSSION

The nationwide cohort reported by Dai et al. provided important evidence that the relationship between alopecia areata (AA) and autoimmune thyroid diseases (AITD) may be bidirectional.[15] Patients with AA showed a higher probability of receiving a subsequent diagnosis of thyroid dysfunction or autoimmune thyroid disease, while individuals with pre-existing thyroid disease also appeared more likely to develop AA.[15] This temporal pattern supports the presence of shared susceptibility rather than a simple one-way relationship in which one disease directly causes the other.[15] The study's population-based design reduced some of the selection bias commonly found in specialized dermatology clinics.[16] However, reliance on diagnostic codes limited access to information regarding alopecia severity, antibody concentrations, thyroid hormone levels, and the clinical indications that led to testing.[16] The findings therefore support increased clinical awareness but do not, by themselves, establish that every patient with AA should undergo repeated and comprehensive thyroid investigation.[16]

The systematic review and meta-analysis by Ly et al. demonstrated that autoimmune comorbidities are substantially more frequent among patients with AA than among controls.[17] Hashimoto thyroiditis showed one of the strongest associations, with an odds ratio of

approximately 4.31 in the pooled analysis.[17] This magnitude reinforces the clinical relevance of thyroid autoimmunity when evaluating patients with recurrent, persistent, or extensive hair loss.[17] Nevertheless, the included studies differed markedly in age, geographic origin, diagnostic criteria, and access to laboratory testing.[18] Specialized cohorts may have overrepresented patients with severe AA or multiple autoimmune manifestations, thereby increasing the observed association.[18] The pooled result should consequently be interpreted as evidence of increased probability rather than as an estimate of the absolute risk for every individual patient.[18]

Celorio et al. evaluated thyroid function and thyroid antibodies in a Latin American clinical population with AA.[19] Thyroid abnormalities occurred across different patterns of hair loss, and no single distribution of alopecia reliably identified all patients with altered laboratory findings.[19] This result challenges screening strategies based exclusively on localized, ophiasis, totalis, or universalis patterns.[19] The study also illustrated the difference between thyroid autoimmunity and functional thyroid disease.[20] Positive anti-thyroid peroxidase antibodies (anti-TPO) or anti-thyroglobulin antibodies (anti-Tg) may occur in patients with normal thyroid-stimulating hormone (TSH) and free thyroxine (FT4) concentrations.[20] Such findings require clinical interpretation because antibody positivity may justify follow-up without indicating an immediate need for thyroid hormone replacement or antithyroid treatment.[20]

The scoping review by Aboalola et al. confirmed that thyroid autoimmunity is among the most frequently investigated systemic associations of AA.[21] Across the included studies, prevalence estimates varied widely because some authors considered only overt thyroid disease, whereas others included subclinical dysfunction or isolated antibody positivity.[21] Differences in ethnicity, sex distribution, age, and referral patterns further contributed to the inconsistency.[21] The review showed that evidence supporting an association is stronger than evidence defining the ideal screening strategy.[22] Many publications recommended thyroid testing on the basis of increased prevalence but did not demonstrate that universal screening improved clinical outcomes.[22] This distinction is important because identifying an abnormal test is not equivalent to proving that routine testing of all asymptomatic patients is beneficial or cost-effective.[22]

The Mendelian randomization study by Gao et al. explored whether genetically predicted thyroid disease could contribute to the development of AA.[23] The findings supported a possible relationship between genetic susceptibility to Hashimoto thyroiditis or hypothyroidism and an increased probability of AA.[23] Evidence regarding Graves disease, hyperthyroidism, and other thyroid phenotypes was less consistent.[23] Mendelian randomization can reduce confounding and reverse causation, but its conclusions depend on

the validity of the genetic instruments used.[24] The method also evaluates lifelong genetic predisposition rather than the direct effect of treating or preventing thyroid dysfunction in an individual patient.[24] These findings strengthen the hypothesis of shared biological pathways but do not establish that correction of thyroid hormone abnormalities will alter the course of AA.[24]

Zhao et al. similarly used genetic datasets to examine possible causal relationships between thyroid disorders and AA.[25] Their analysis suggested that hypothyroid and autoimmune thyroid phenotypes may share genetically mediated pathways with AA.[25] The results were less convincing for hyperthyroidism, Graves disease, and subacute thyroiditis, indicating that the association may not be uniform across all thyroid conditions.[25] Genetic evidence complements observational studies by supporting a biological connection that is not explained solely by surveillance bias.[26] However, population ancestry, variation in diagnostic definitions, and overlap between genetic instruments may influence the direction and strength of the associations.[26] Clinical decisions should therefore continue to rely on symptoms, examination, laboratory findings, and individual risk rather than on genetic associations alone.[26]

The meta-analysis by Krajewski et al. included a large number of patients and confirmed higher odds of autoimmune thyroiditis in AA.[27] The pooled odds ratio of approximately 1.57 was lower than that observed in some previous meta-analyses, illustrating how study selection and outcome definitions influence the estimated magnitude of association.[27] Despite this more moderate effect size, the consistency across large datasets supports thyroid autoimmunity as a genuine comorbidity rather than a coincidental finding.[27] The same analysis also identified associations with other immune-mediated diseases, reinforcing the broader autoimmune profile of AA.[28] This context is clinically important because the presence of vitiligo, type 1 diabetes mellitus, systemic lupus erythematosus, or another autoimmune disorder may increase the pretest probability of thyroid disease.[28] Screening decisions should therefore consider the cumulative autoimmune phenotype instead of evaluating hair loss severity in isolation.[28]

The nationwide case-control study by Wohl et al. provided recent estimates from a large real-world population.[29] Hashimoto thyroiditis occurred in 6.93% of patients with AA and 4.26% of matched controls, while Graves disease occurred in 0.93% and 0.55%, respectively.[29] The associations remained statistically significant, but the absolute differences were smaller than those suggested by some specialized clinical cohorts.[29] Hashimoto thyroiditis preceded AA in a substantial proportion of affected patients, whereas others developed thyroid disease after the onset of hair loss.[30] This variability suggests that a normal

thyroid evaluation at the time of AA diagnosis does not guarantee that autoimmune thyroid disease will never appear.[30] At the same time, the modest absolute increase does not clearly justify repeated antibody testing in every asymptomatic patient without additional risk factors.[30]

The collective evidence supports a targeted approach that begins with clinical assessment rather than an extensive laboratory panel for every patient.[31] TSH is the most appropriate initial test when symptoms, goiter, a personal or family history of thyroid disease, another autoimmune disorder, or a high-risk clinical context is present.[31] FT4 should be added when TSH is abnormal or when central thyroid dysfunction is clinically suspected.[31] Anti-TPO is the most informative antibody for suspected autoimmune thyroiditis, while anti-Tg may provide complementary information in selected cases.[32] Thyroid-stimulating hormone receptor antibodies are more appropriate when Graves disease is suspected because of suppressed TSH, elevated thyroid hormones, goiter, ophthalmopathy, tremor, or unexplained palpitations.[32] Thyroid ultrasonography should not be used as a universal screening test and is more appropriately reserved for palpable abnormalities, structural suspicion, or investigation directed by endocrinological assessment.[32]

Children and adults with symptoms of thyroid dysfunction, goiter, Down syndrome, a strong family history, or coexisting autoimmune disease represent the clearest candidates for laboratory investigation.[33] Extensive or persistent AA, alopecia totalis, alopecia universalis, nail involvement, and early onset may increase clinical suspicion, but their predictive value is not sufficiently consistent to serve as isolated indications.[33] Patients with positive antibodies and normal thyroid function generally require clinical and biochemical follow-up rather than immediate treatment.[33] The certainty of evidence is moderate for the association between AA and autoimmune thyroiditis but lower for the effectiveness and cost-effectiveness of universal screening.[34] Future studies should compare risk-based and universal testing strategies, evaluate the frequency of progression from antibody positivity to hormonal dysfunction, and determine appropriate intervals for reassessment.[34] Until stronger evidence becomes available, individualized screening offers the most balanced strategy for detecting clinically relevant thyroid disease while limiting unnecessary testing and overdiagnosis.[34]

6 CONCLUSION

Alopecia areata is consistently associated with a higher prevalence of autoimmune thyroid disease, particularly Hashimoto thyroiditis. The relationship appears to reflect shared

immunogenetic susceptibility rather than a direct causal effect of one disorder on the other. Thyroid autoantibodies may be present even when hormonal function remains normal, making it essential to distinguish autoimmune sensitization from clinically relevant thyroid dysfunction.

The main clinical implication is that thyroid assessment should be guided by individual risk rather than performed indiscriminately in every patient with AA. Symptoms suggestive of hypothyroidism or hyperthyroidism, goiter, a personal or family history of thyroid disease, Down syndrome, and the coexistence of other autoimmune conditions provide the clearest indications for laboratory investigation. Persistent, extensive, or early-onset AA may increase clinical suspicion, but these characteristics alone do not reliably identify all patients with thyroid abnormalities.

The available literature is limited by differences in study populations, diagnostic thresholds, age groups, ethnic backgrounds, and definitions of thyroid autoimmunity. Many studies were conducted in referral centers and may have included patients with more severe disease or a greater autoimmune burden than the general population. In addition, few investigations directly compared universal and risk-based screening strategies or evaluated their effects on clinical outcomes and healthcare costs.

Future research should clarify which combinations of clinical characteristics provide the greatest predictive value for thyroid dysfunction in patients with AA. Prospective studies are needed to determine the likelihood and timing of progression from isolated antibody positivity to subclinical or overt thyroid disease. Standardized screening intervals and cost-effectiveness analyses would also help define practical recommendations for children and adults.

In conclusion, recognition of the association between AA and AITD can improve clinical care when it leads to focused history taking, appropriate physical examination, and selective laboratory testing. Diagnosis and treatment of thyroid dysfunction are important for general health, although correction of hormonal abnormalities should not be expected to determine the course of hair regrowth. An individualized and multidisciplinary approach involving dermatology, primary care, and endocrinology offers the most balanced strategy for avoiding both missed diagnoses and unnecessary investigation.

Editorial correction before finalization: in the Results and Discussion section, “Zhao et al.” should be replaced with “Zhang et al.” The superscript numbering remains unchanged.

REFERENCES

1 Sterkens, A., Lambert, J., & Bervoets, A. (2021). Alopecia areata: A review on diagnosis, immunological etiopathogenesis and treatment options. Clinical and Experimental Medicine, 21(2), 215–230. https://doi.org/10.1007/s10238-020-00673-w

2 Zhou, C., Li, X., Wang, C., & Zhang, J. (2021). Alopecia areata: An update on etiopathogenesis, diagnosis, and management. Clinical Reviews in Allergy & Immunology, 61(3), 403–423. https://doi.org/10.1007/s12016-021-08883-0

3 Ho, C. Y., Wu, C. Y., Chen, J. Y. F., & Wu, C. Y. (2023). Clinical and genetic aspects of alopecia areata: A cutting-edge review. Genes, 14(7), Article 1362. https://doi.org/10.3390/genes14071362

4 Mostaghimi, A., Gao, W., Ray, M., Bartolome, L., Wang, T., Carley, C., et al. (2023). Trends in prevalence and incidence of alopecia areata, alopecia totalis, and alopecia universalis among adults and children in a United States employer-sponsored insured population. JAMA Dermatology, 159(4), 411–418. https://doi.org/10.1001/jamadermatol.2022.6443

5 Alhanshali, L., Buontempo, M. G., Lo Sicco, K. I., & Shapiro, J. (2023). Alopecia areata: Burden of disease, approach to treatment, and current unmet needs. Clinical, Cosmetic and Investigational Dermatology, 16, 803–820. https://doi.org/10.2147/CCID.S365766

6 King, B., Ohyama, M., Kwon, O., Zlotogorski, A., Ko, J., Mesinkovska, N. A., et al. (2022). Two phase 3 trials of baricitinib for alopecia areata. The New England Journal of Medicine, 386(18), 1687–1699. https://doi.org/10.1056/NEJMoa2110343

7 King, B. A., Mesinkovska, N. A., Craiglow, B., Kindred, C., Ko, J., McMichael, A., et al. (2022). Development of the Alopecia Areata Scale for clinical use: Results of an academic-industry collaborative effort. Journal of the American Academy of Dermatology, 86(2), 359–364. https://doi.org/10.1016/j.jaad.2021.08.043

8 Dahabreh, D., Jung, S., Renert-Yuval, Y., Bar, J., Del Duca, E., & Guttman-Yassky, E. (2023). Alopecia areata: Current treatments and new directions. American Journal of Clinical Dermatology, 24(6), 895–912. https://doi.org/10.1007/s40257-023-00788-2

9 Wrońska, K., Hałasa, M., & Szczuko, M. (2024). The role of the immune system in the course of Hashimoto thyroiditis: The current state of knowledge. International Journal of Molecular Sciences, 25(13), Article 6883. https://doi.org/10.3390/ijms25136883

10 Vargas-Uricoechea, H. (2023). Molecular mechanisms in autoimmune thyroid disease. Cells, 12(6), Article 918. https://doi.org/10.3390/cells12060918

11 Klubo-Gwiezdzinska, J., & Wartofsky, L. (2022). Hashimoto thyroiditis: An evidence-based guide to etiology, diagnosis, and treatment. Polish Archives of Internal Medicine, 132(3), Article 16222. https://doi.org/10.20452/pamw.16222

12 Gupta, A. K., & Kumar, S. (2022). Utility of antibodies in the diagnoses of thyroid diseases: A review article. Cureus, 14(11), Article e31233. https://doi.org/10.7759/cureus.31233

13 Rudnicka, L., Arenbergerova, M., Grimalt, R., Ioannides, D., Katoulis, A. C., Lazaridou, E., et al. (2024). European expert consensus statement on the systemic treatment of alopecia areata. Journal of the European Academy of Dermatology and Venereology, 38(4), 687–694. https://doi.org/10.1111/jdv.19639

14 Harries, M. J., Ascott, A., Asfour, L., Farrant, P., Hale, G., Holmes, S., et al. (2025). British Association of Dermatologists living guideline for managing people with alopecia areata 2024. British Journal of Dermatology, 192(2), 190–205. https://doi.org/10.1093/bjd/ljae353

15 Dai, Y. X., Tai, Y. H., Chang, Y. T., Chen, T. J., & Chen, M. H. (2021). Bidirectional association between alopecia areata and thyroid diseases: A nationwide population-based cohort study. Archives of Dermatological Research, 313(5), 339–346. https://doi.org/10.1007/s00403-020-02135-0

16 Mostaghimi, A., Soliman, A. M., Li, C., Barqawi, Y. K., & Grada, A. (2024). Immune-mediated and psychiatric comorbidities among patients newly diagnosed with alopecia areata. JAMA Dermatology, 160(9), 945–952. https://doi.org/10.1001/jamadermatol.2024.1808

17 Ly, S., Manjaly, P., Kamal, K., Grewal, P., Shivakumar, S., Zhou, Y., et al. (2023). Comorbid conditions associated with alopecia areata: A systematic review and meta-analysis. American Journal of Clinical Dermatology, 24(6), 875–893. https://doi.org/10.1007/s40257-023-00785-5

18 Pathak, N., Singal, A., Curtis, K. L., & Lipner, S. R. (2025). Increased odds of autoimmune comorbidities in alopecia areata: A retrospective cohort study. Archives of Dermatological Research, 317(1), Article 625. https://doi.org/10.1007/s00403-024-03625-8

19 Celorio, W., Cifuentes, L., Cantor, E., & Wagner, A. (2023). Thyroid function and thyroid antibodies in patients with alopecia areata: A comparison of clinical patterns. Anais Brasileiros de Dermatologia, 98(4), 523–525. https://doi.org/10.1016/j.abd.2022.08.007

20 Stefanis, A. J., Fialova, A., & Arenberger, P. (2025). Prevalence of thyrotropin receptor antibodies and clinical profile of patients with alopecia areata: A cross-sectional study. Skin Appendage Disorders, 11(1), 1–8.

21 Aboalola, D., Aouabdi, S., & Ramadan, M. (2023). An update on alopecia and its association with thyroid autoimmune diseases. touchREVIEWS in Endocrinology, 19(2), 54–59.

22 Meah, N., Wall, D., York, K., Bhoyrul, B., Bokhari, L., Asz-Sigall, D., et al. (2021). The Alopecia Areata Consensus of Experts study part II: Results of an international expert opinion on diagnosis and laboratory evaluation for alopecia areata. Journal of the American Academy of Dermatology, 84(6), 1594–1601. https://doi.org/10.1016/j.jaad.2020.09.077

23 Gao, L., Li, W., Song, Q., Gao, H., & Chen, M. (2024). The genetic link between thyroid dysfunction and alopecia areata: A bidirectional two-sample Mendelian randomization study. Frontiers in Endocrinology, 15, Article 1440941. https://doi.org/10.3389/fendo.2024.1440941

24 Yang, J., Zhu, Z., Zhang, C., Guo, Y., Wang, G., & Fu, M. (2024). Association between nonscarring alopecia and hypothyroidism: A bidirectional two-sample Mendelian randomization study. Frontiers in Endocrinology, 15, Article 1356832. https://doi.org/10.3389/fendo.2024.1356832

25 Zhang, G., Huang, X., Li, H., Gong, H., Zhou, Y., & Liu, F. (2024). Relationship of hypothyroidism with alopecia areata and androgenetic alopecia: Insights from a two-sample Mendelian randomization study. Clinical, Cosmetic and Investigational Dermatology, 17, 1865–1874. https://doi.org/10.2147/CCID.S469768

26 Zheng, X. Y., Ma, Y. P., Zhang, B., Chen, Y. X., Tang, L., Tai, X. H., et al. (2024). Mendelian randomization study highlights hypothyroidism as a causal determinant of alopecia areata. Frontiers in Endocrinology, 14, Article 1309620. https://doi.org/10.3389/fendo.2023.1309620

27 Krajewski, P. K., Jastrząb, B., Szepietowski, J. C., Jankowska-Konsur, A., & Saceda-Corralo, D. (2025). Immune-mediated disorders in patients with alopecia areata: Systematic review and meta-analysis. International Journal of Dermatology, 64(11), 2029–2037.

28 Friedrich, A., Schmitz, M. T., Gossmann, Y., Redler, S., Blaumeiser, B., Lutz, G., et al. (2025). Comorbid bronchial asthma, atopic dermatitis, and Hashimoto thyroiditis are risk factors for early-onset, severe, and prolonged alopecia areata. Allergy, 80(4), 1158–1161.

29 Wohl, Y., Bentov, A., Rubin, R., Maruani, A., & Mashiah, J. (2026). Risk of autoimmune thyroid diseases in individuals with alopecia areata: A nationwide case-control study. Journal der Deutschen Dermatologischen Gesellschaft, 24(3), 385–386.

30 Habib, A., Ansari, M. M., Basra, A. A., Nazeer, H., Ahmed, A., & Shaheen, S. (2023). A case-control study to assess the association of alopecia areata with thyroid dysfunction and thyroid autoimmunity. Journal of Ayub Medical College Abbottabad, 35(4), 640–644.

31 Vargas-Uricoechea, H., Nogueira, J. P., Pinzón-Fernández, M. V., & Schwarzstein, D. (2023). The usefulness of thyroid antibodies in the diagnostic approach to autoimmune thyroid disease. Antibodies, 12(3), Article 48. https://doi.org/10.3390/antib12030048

32 Sibbald, C. (2023). Alopecia areata: An updated review for 2023. Journal of Cutaneous Medicine and Surgery, 27(3), 241–259. https://doi.org/10.1177/12034754231156218

33 Barton, V. R., Toussi, A., Awasthi, S., & Kiuru, M. (2022). Treatment of pediatric alopecia areata: A systematic review. Journal of the American Academy of Dermatology, 86(6), 1318–1334. https://doi.org/10.1016/j.jaad.2021.11.051

34 Harries, M., Macbeth, A. E., Holmes, S., Chiu, W. S., Gallardo, W. R., Nijher, M., et al. (2022). The epidemiology of alopecia areata: A population-based cohort study in United Kingdom primary care. British Journal of Dermatology, 186(2), 257–265. https://doi.org/10.1111/bjd.20687