Dermatology Practical & Conceptual

# Risk Factors for Pediatric Alopecia Areata: Clinical and Biochemical Findings from a Case – Control Study

Seçil Yigen İritaş[1]

**1** Pendik State Hospital, Department of Dermatology and Venereal Diseases, Istanbul, Turkey

**Key words:** Alopecia areata, Child, Vitiligo, Asthma, Attention deficit disorder with hyperactivity

**Citation:** Yigen İritaş S. Risk Factors for Pediatric Alopecia Areata: Clinical and Biochemical Findings from a Case – Control Study. *Dermatol Pract Concept*. 2026;16(1):6810. DOI: https://doi.org/10.5826/dpc.1601a6810

**Accepted:** June 28, 2025; **Published:** January 2026



**Funding:** None.

**Competing Interests:** None.

**Authorship:** All authors have contributed significantly to this publication.

**Corresponding Author:** Seçil Yigen İRİTAŞ,Doğu Neighborhood, Bilezik Street, Portre Complex, 4B, Apartment 45, Pendik, Istanbul, Turkey. ORCID: 0000-0003-4462-6526. E-mail: secilyigen@gmail.com

## ABSTRACT

**Introduction:** Alopecia areata (AA) is an immune-mediated, non-scarring hair loss disorder that can begin in childhood. Data on comorbidities and biochemical risk factors in pediatric AA remain limited.

**Objectives:** To compare clinical comorbidities and biochemical parameters in pediatric patients with AA versus healthy controls.

**Methods:** This retrospective, matched case-control study included 200 pediatric AA patients and 400 age- and sex-matched healthy controls. Clinical characteristics and comorbidities were obtained from patient records. Hemoglobin, ferritin, vitamin B12, and thyroid-stimulating hormone (TSH) levels were analyzed. Independent predictors were identified using multivariable logistic regression.

**Results:** Vitiligo (3.5% vs. 0.5%; *P*=0.008), allergic asthma (9.5% vs. 3.8%; *P*=0.007), and attention-deficit/hyperactivity disorder (ADHD) (3.5% vs. 0.5%; *P*=0.008) were significantly more frequent in the AA group, whereas atopic dermatitis was more common but not statistically significant. Hemoglobin levels were comparable. Serum vitamin B12 [233.5 (192.7–270.0) vs. 356.7 (318.7–396.8) pg/mL; *P*<0.001] and ferritin [14.4 (6.5–22.3) vs. 19.4 (11.6–27.0) ng/mL; *P*<0.001] were significantly lower, and TSH levels were also reduced (3.40 ± 1.48 vs. 3.71 ± 1.50 mIU/L; *P*=0.017). Multivariable analysis identified low vitamin B12 (OR = 0.964, 95% CI: 0.958–0.970; *P*<0.001) and low ferritin (OR = 0.965, 95% CI: 0.944–0.988; *P*=0.003) as independent risk factors.

**Conclusions:** Pediatric AA is associated with vitiligo, asthma, ADHD, and reduced levels of ferritin, vitamin B12, and TSH. These findings underscore the importance of comprehensive clinical and biochemical evaluation in pediatric AA.

# Introduction

Alopecia areata (AA) is a T cell–mediated non-scarring alopecia directed against hair follicles, with a lifetime prevalence of approximately 2% [1,2]. Although its etiopathogenesis is not fully understood, genetic, autoimmune, environmental, and psychosocial triggers have been implicated [3,4]. In addition, imbalances in micronutrients essential for healthy hair follicle development have been discussed; in particular, ferritin and vitamin B12 deficiencies have been suggested as potential risk factors [5-8]. Moreover, increasing evidence indicates that the disease is not only dermatological but also associated with autoimmune, inflammatory, and psychiatric conditions [9-13].

In childhood, AA shows a higher prevalence compared to adults (1.92% vs. 1.47%), and 40% of cases begin before the age of 20 [14,15]. Pediatric AA may differ clinically from adult-onset AA in several aspects. It tends to progress to more resistant and extensive forms and to follow a recurrent course [16,17]. Furthermore, the comorbidity profile may differ between children and adults [18]. Childhood is also a period when associated conditions such as autoimmune thyroiditis may first manifest and when psychosocial vulnerability is greater [19-21]. In addition, physiological requirements for micronutrients are increased during this stage [22]. Understanding the relationship of pediatric AA with comorbidities and biochemical characteristics may provide insight into its pathogenesis and allow more individualized treatment and holistic management. However, studies specifically investigating pediatric AA remain limited [23-25].

# Objectives

The aim of this retrospective matched case-control study was to evaluate clinical comorbidities and biochemical parameters in pediatric AA patients and to compare the findings with healthy controls.

# Methods

## Ethical Approval

This study was approved by the Ethics Committee of Kartal Dr.Lutfi Kırdar City Hospital (Approval Number: 2024/010.99/9/19). The research was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective design of the study, informed consent was waived by the ethics committee.

## Study Design and Population

This retrospective matched case-control study included 200 pediatric patients aged 0–18 years who were diagnosed with AA between 01 January 2014 and 30 August 2024.

The control group consisted of 400 children who presented consecutively to the dermatology outpatient clinic during the same period solely for routine health examinations. None of these individuals had AA. Patients receiving systemic treatments that could affect laboratory results (e.g., vitamin B12, iron, iodine, systemic corticosteroids) were excluded from the study.

Controls were matched to cases by age and sex. All data were obtained from the hospital's electronic medical record (EMR) system.

## Study Variables and Definitions

Demographic data included age and sex. The diagnosis of alopecia areata was made by dermatologists based on clinical features and trichoscopic findings. Clinical subtypes were classified according to dermatological examination findings as described in the literature: patchy AA was defined as localized, focal areas of hair loss on the scalp. alopecia totalis (AT) as complete scalp hair loss, and alopecia universalis (AU) as loss of scalp, facial, and body hair [26,27]. Nail changes (pitting, ridging, dullness, et cetera) were systematically recorded. Family history of AA was documented, and comorbidities were determined only if recorded as confirmed diagnoses by specialists.

Biochemical parameters included hemoglobin (Hb) as an indirect indicator of iron status, ferritin as a marker of iron stores, serum vitamin B12, and thyroid-stimulating hormone (TSH) levels, all of which were evaluated for their potential associations with AA. These parameters were chosen because their potential associations with AA have been reported in the literature, and they have biologically meaningful roles in hair follicle health and immune function [6,8].

Thyroid function was assessed only with TSH values, which are considered sufficient for screening according to current guidelines [28]. All tests were evaluated according to the reference ranges of the respective laboratory. Abnormal values were defined as follows: vitamin B12 <200 pg/mL, ferritin <15 ng/mL, and TSH <0.4 or >4.0 mIU/L. Hemoglobin levels were interpreted based on age- and sex-specific reference ranges, and values below the lower limit were considered anemia.

## Statistical Analysis

The normality of continuous variables was tested using the Kolmogorov–Smirnov test, and homogeneity of variances was evaluated with the Levene test. Continuous variables are presented as mean ± standard deviation or median (25th–75th percentile), and categorical variables as number and percentage (%). Group comparisons for continuous variables were performed using the Student's t-test if parametric assumptions were met, or the Mann–Whitney U test otherwise. Categorical variables were analyzed using Pearson's

$\chi^2$ test. In 2 × 2 contingency tables, Fisher's exact test was used when more than one quarter of the expected cell counts was <5; when expected counts were between 5 and 25, the continuity-corrected $\chi^2$ test was applied.

To identify the most significant independent determinants distinguishing cases from controls, multivariable logistic regression analysis was performed. Variables with $P<0.15$ in univariate analysis were included as candidate risk factors in the model. For each independent variable, odds ratios (ORs), 95% confidence intervals (CIs), and Wald statistics were calculated. All analyses were performed using IBM SPSS Statistics, version 25 (IBM Corp., Armonk, NY, USA). A two-tailed $P<0.05$ was considered statistically significant.

# Results

## Demographics

A total of 200 pediatric patients with AA aged 0–18 years and 400 age- and sex-matched healthy controls were included in the study. The mean age was 13.2 ± 2.5 years in the AA group and 13.2 ± 2.4 years in the control group, with no statistically significant difference ($P$=0.972). The proportion of female participants was also similar between groups: 55.5% in the AA group and 53.5% in the control group ($P$=0.643) (Table 1).

## Clinical Characteristics

Among AA patients, 93.0% (N=186) had patchy alopecia, 4.5% (N=9) had AT, and 2.5% (N=5) had AU. Nail changes were present in 10.5% (N=21) of the patients. A family history of AA was reported in 6.0% (N=12) (Table 1).

## Comorbidities

Atopic dermatitis (AD) was more frequently observed in the AA group (10.0%, N=20) than in controls (6.0%, N=24), although the difference did not reach statistical significance ($P$=0.108). Allergic asthma was significantly more common in the AA group (9.5%, N=19 vs. 3.8%, N=15; $P$=0.007). The frequencies of vitiligo (3.5%, N=7 vs. 0.5%, N=2; $P$=0.008) and attention deficit hyperactivity disorder (ADHD) (3.5%, N=7 vs. 0.5%, N=2; $P$=0.008) were also significantly higher in the AA group compared with controls (Table 2). In addition, allergic rhinitis (1.0%, N=2 vs. 0.25%, N=1; $P$=0.562), depression (0.5%, N=1 vs. 0.25%, N=1; $P$=1.000), anxiety disorder (0.5%, N=1 vs. 0.0%, N=0; $P$=0.317), and ankylosing spondylitis (0.5%, N=1 vs. 0.0%, N=0; $P$=0.317) were detected in only one or two cases and did not show a statistically significant difference.

## Laboratory Findings

Hemoglobin levels (mean ± SD) were comparable between the AA and control groups (12.9 ± 1.05 vs. 13.1 ± 1.03 g/dL; $P$=0.067). In contrast, serum vitamin B12 [median (IQR)] and ferritin [median (IQR)] levels were significantly lower in the AA group compared with controls [233.5 (192.7–270.0) vs. 356.7 (318.7–396.8) pg/mL; $P<0.001$, and 14.4 (6.5–22.3) vs. 19.4 (11.6–27.0) ng/mL; $P<0.001$, respectively]. Thyroid-stimulating hormone (TSH) levels (mean ± SD) were also significantly reduced in the AA group (3.40 ± 1.48 vs. 3.71 ± 1.50 mIU/L; $P$=0.017) (Table 3).

## Multivariate Logistic Regression Analysis

In multivariate logistic regression analysis, only serum Vitamin B12 (OR=0.964, 95% CI: 0.958–0.970; $P< 0.001$) and serum ferritin levels (OR=0.965, 95% CI: 0.944–0.988, $P$=0.003) showed a significant association with being in the AA group. Although vitiligo showed a notable odds ratio (OR: 7.648), it did not reach statistical significance ($P$=0.059) (Table 4).

**Table 1. Demographic and clinical characteristics of cases in the control and case groups.**

| | Controls (N=400) | Cases (N=200) | p-value | OR (95% CI) |
|---|---|---|---|---|
| **Age (years) *** | 13.2±2.4 | 13.2±2.5 | 0.972[A] | 0.999 (0.932-1.071) |
| **Sex** | | | | |
| Male | 186 (46.5%) | 89 (44.5%) | - | 1.000 |
| Female | 214 (53.5%) | 111 (55.5%) | 0.643[B] | 1.084 (0.771-1.525) |
| **Disease subtype** | | | | |
| Patchy alopecia | - | 186 (93.0%) | n/a | n/a |
| Alopecia totalis | - | 9 (4.5%) | n/a | n/a |
| Alopecia universalis | - | 5 (2.5%) | n/a | n/a |
| **Nail changes** | - | 21 (10.5%) | n/a | n/a |
| **Family history** | - | 12 (6.0%) | n/a | n/a |

*Data were expressed as mean ± standard deviation. [A]Student's t test, [B]Pearson's $\chi^2$ test. OR: Odds Ratio, CI: Confidence Interval, n/a: Not applicable.

**Table 2. Distribution of comorbid conditions in control and case groups.**

| | Controls (N=400) | Cases (N=200) | p-value | OR (95% CI) |
|---|---|---|---|---|
| **Atopic dermatitis** | 24 (6.0%) | 20 (10.0%) | 0.108[A] | 1.741 (0.937-3.234) |
| **Vitiligo** | 2 (0.5%) | 7 (3.5%) | **0.008**[B] | 7.218 (1.485-35.071) |
| **Allergic asthma** | 15 (3.8%) | 19 (9.5%) | **0.007**[A] | 2.694 (1.338-5.424) |
| **ADHD** | 2 (0.5%) | 7 (3.5%) | **0.008**[B] | 7.218 (1.485-35.071) |
| **Allergic rhinitis** | 1 (0.25%) | 2 (1.0%) | 0.259[B] | 4.03 (0.36–44.7) |
| **Anxiety disorder** | 0 (0.0%) | 1 (0.5%) | 0.333[B] | 4.02 (0.13–120.3) |
| **Ankylosing spondylitis** | 0 (0.0%) | 1 (0.5%) | 0.333[B] | 4.02 (0.13–120.3) |

Abbreviations: ADHD: Attention Deficit Hyperactivity Disorder. [A] Continuity-corrected $\chi^2$ test, [B] Fisher's exact test. OR: odds ratio, CI: confidence interval.

**Table 3. Laboratory findings in control and case groups.**

| | Controls (N=400) | Cases (N=200) | p-value | OR (95% CI) |
|---|---|---|---|---|
| **Hemoglobin (g/dL)*** | 13.1±1.03 | 12.9±1.05 | 0.067[A] | 0.858 (0.727-1.011) |
| **Vitamin B12 (pg/mL)**** | 356.7 (318.7-396.8) | 233.5 (192.7-270.0) | **<0.001**[B] | 0.964 (0.958-0.970) |
| **Ferritin (ng/mL)**** | 19.4 (11.6-27.0) | 14.4 (6.5-22.3) | **<0.001**[B] | 0.967 (0.951-0.983) |
| **TSH (mIU/L)*** | 3.71±1.50 | 3.40±1.48 | **0.017**[A] | 0.870 (0.775-0.976) |

Continuous variables were expressed as *mean ± standard deviation or **median ($25^{th}$-$75^{th}$) percentiles; as appropriate. [A]Student's t test, [B]Mann Whitney U test. OR: odds ratio, CI: confidence interval.

**Table 4. Multivariable logistic regression analysis of factors differentiating case and control groups.**

| | OR | 95% CI | Wald $\chi^2$ | p-value |
|---|---|---|---|---|
| **Atopic dermatitis** | 1.804 | 0.653-4.981 | 1.296 | 0.255 |
| **Vitiligo** | 7.736 | 0.941-63.599 | 3.623 | 0.057 |
| **Other atopic conditions** | 1.656 | 0.592-4.634 | 0.923 | 0.337 |
| **ADHD** | 2.849 | 0.375-21.657 | 1.024 | 0.312 |
| **Hemoglobin (g/dL)** | 1.100 | 0.833-1.452 | 0.450 | 0.502 |
| **Vitamin B12 (pg/mL)** | 0.964 | 0.958-0.970 | 141.592 | **<0.001** |
| **Ferritin (ng/mL)** | 0.965 | 0.943-0.988 | 9.121 | **0.003** |
| **TSH (mIU/L)** | 0.913 | 0.757-1.102 | 0.898 | 0.343 |

Abbreviations: ADHD: Attention Deficit Hyperactivity Disorder, OR: odds ratio; CI: confidence interval.

### Subgroup Analysis Based on the Clinical Subtypes of Alopecia Areata

In the AA group (N=200), patchy alopecia was the most frequently observed clinical subtype (N=186, 93.0%), followed by AT (N=9, 4.5%) and AU (N=5, 2.5%). No statistically significant difference was found between the subtypes in terms of mean age, sex distribution, nail involvement, family history, atopic dermatitis, vitiligo, other atopic conditions (asthma, allergic rhinitis, and allergic conjunctivitis), or attention-deficit/hyperactivity disorder (ADHD) ($P$>0.05).

## Discussion

In this retrospective case-control study, clinical and laboratory-based risk factors associated with pediatric AA were evaluated. In our cohort, the coexistence of vitiligo was significantly higher among pediatric AA patients. This finding is consistent with the approximately 3% prevalence reported in pediatric series and the increased risk described in large cross-sectional studies [18,29,30].

The association between AA and vitiligo has been explained at both immunological and genetic levels [31-35]. Autoimmune mechanisms are implicated in both diseases. In AA, melanocytes and keratinocytes within the hair follicle are targeted, whereas in vitiligo, epidermal melanocytes represent the main target [32]. Furthermore, AA and vitiligo share common genetic susceptibility loci (e.g., CTLA4, PTPN22, IL2RA). This overlap highlights the role of the JAK/STAT pathway and specific cytokines (IL-2, IFN-γ) in both conditions [33-35].

In the present study, atopic dermatitis (AD) was the most frequent comorbidity; although more common in the AA group compared with controls, the difference was not statistically significant. Prior studies, however, consistently report AD as the most common comorbidity in pediatric AA, with prevalence ranging from 17% to 33%, markedly higher than in the general population [18,36]. Differences between our findings and previous reports may be explained by sample size, reliance on retrospective diagnostic records, and demographic variation.

The increased risk of AD in AA has been linked to filaggrin mutations, suggesting a shared genetic background [37]. Moreover, recent evidence indicates that Th2-predominant AA subtypes may be associated with AD. Elevated IL-13 levels detected exclusively in atopic AA patients and better clinical responses to IL-4 receptor inhibitors in atopic phenotypes further support this relationship [38-41].

Allergic asthma was significantly more common in the AA group. This observation is in line with prior studies reporting up to 20% prevalence and approximately a two-fold increased risk of asthma among pediatric AA patients [36,42-46]. Interestingly, this risk has been found to be even higher than that reported in some adult AA populations [44-46]. Mendelian randomization analysis by Wen Xu et al. demonstrated a direct causal association between AA and allergic diseases, implicating Th2 pathways [47]. These cytokines may promote hair loss by enhancing T-cell infiltration around the hair follicle [43]. The higher asthma risk in pediatric than in adult AA patients may reflect both the greater prevalence of atopic diseases in childhood and developmental differences in immune responses.

With regard to psychiatric comorbidities, prior studies have shown that children with AA are more likely than the general population to present with anxiety, depression, and ADHD [48-50]. In our study, no significant difference was observed for depression and anxiety. This may be explained by diagnostic challenges in pediatric age, including limited verbal expression, reduced awareness, and small sample size. By contrast, ADHD was significantly more frequent in the AA group. This finding is consistent with a large Taiwanese cohort study reporting an approximately 30% increased risk of AA in children with ADHD [51]. Shared inflammatory processes (e.g., elevated IL-6, TNF-α, CRP) and HLA gene polymorphisms (HLA-DR4, HLA-DRB1) may provide a biological basis for this association [51-53]. Furthermore, ADHD symptoms such as impulsivity and difficulty remaining seated in class are more easily recognized by parents and teachers, potentially leading to earlier referral and diagnosis. This may explain why ADHD was the most common psychiatric comorbidity identified in our cohort [54-56].

No association was observed between hemoglobin levels and AA; however, low ferritin levels emerged as an independent risk factor. This finding is consistent with the pediatric AA literature [57]. Ferritin reflects iron storage and also exerts antioxidant properties [58]. Low ferritin may be linked to the impaired oxidative balance reported in AA [57].

In our study, low serum vitamin B12 levels were independently associated with pediatric AA. Similar results have been reported by Kundak et al. in pediatric populations [59]. By contrast, studies in general AA populations have not consistently demonstrated such an association [60-61]. Vitamin B12 is essential for DNA synthesis and proliferation of hair matrix cells [62,63]. In addition, it exerts antioxidant effects through homocysteine metabolism and modulates immune cell function via methylation pathways [64,65]. Considering the increased physiological demands and immunological differences specific to childhood, deficiencies in ferritin and vitamin B12 may be particularly relevant to both follicular biology and the immunopathogenesis of AA. The retrospective design of the study does not allow establishing a causal relationship between low ferritin and vitamin B12 levels and AA. Larger, prospective studies are needed to evaluate the impact of correcting these parameters on clinical outcomes and disease prognosis. Similarly, within the same methodological limitations, the results of this study do not support recommending changes in clinical practice, such as obtaining blood samples from all pediatric patients.

TSH levels were lower in the AA group. Previous studies have also reported associations between AA and thyroid disorders in both pediatric and general AA populations [24,66-68]. To clarify this relationship in children, prospective studies are needed that incorporate confounding factors as well as antithyroid antibodies and additional thyroid hormone parameters.

No significant difference was observed among AA subtypes in terms of demographic characteristics, nail involvement, family history, or comorbidities. This may be related to the limited number of AT and AU patients in our cohort. Nevertheless, it may also suggest that despite the clinical heterogeneity of AA, underlying pathogenic mechanisms are largely shared across subtypes.

## Limitations

Our study has several limitations. The retrospective, single-center design may limit the generalizability of the findings. Laboratory parameters could have been influenced by unmeasured confounders such as nutritional status and prior infections. Furthermore, as the laboratory parameters were obtained retrospectively, the samples were collected at different time points and may have been analyzed using different instruments or calibration protocols, potentially introducing measurement variability. This variability should be acknowledged as a limitation that may restrict the interpretability of the observed biochemical differences. In addition, thyroid

function was assessed solely by TSH, without inclusion of fT4, fT3, or antithyroid antibodies. Finally, disease severity could not be evaluated using the SALT score.

## Conclusion

Our findings revealed significant associations between pediatric AA and low ferritin, vitamin B12 deficiency, reduced TSH levels, vitiligo, allergic asthma, and ADHD. The observation that vitamin B12 deficiency, an association inconsistently demonstrated in adult series, was significant only in this pediatric cohort is noteworthy. Although the retrospective design precludes causal inference, reliance on clinical records rather than self-report represents a methodological strength. Due to the limitations of the retrospective design, these findings do not support routine laboratory evaluation for all pediatric patients with AA. These findings may guide dermatologists to broaden laboratory assessments when appropriate, tailor treatment plans accordingly, and collaborate in a multidisciplinary manner in the management of pediatric AA.

## References


1. Alessandrini A, Starace M, Bruni F,et al. Alopecia areata incognita and diffuse alopecia areata: clinical, trichoscopic, histopathological, and therapeutic features of a 5-year study. *Dermatol Pract Concept*. 2019;9(4):272-7. DOI: 10.5826/dpc.0904a05. PMID: 31723460; PMCID: PMC6830548.
2. Tabara K, Kozłowska M, Jędrowiak A, Bienias W, Kaszuba A. Serum concentrations of selected proinflammatory cytokines in children with alopecia areata. *Postepy Dermatol Alergol*. 2019; 36(1):63-9. DOI: 10.5114/ada.2019.82826. PMID: 30858781
3. Minokawa Y, Sawada Y, Nakamura M. Lifestyle factors involved in the pathogenesis of alopecia areata. *Int J Mol Sci*. 2022; 23(3):1038. DOI: 10.3390/ijms23031038. PMID: 35162962; PMCID: PMC8835065.
4. Prie BE, Voiculescu VM, Ionescu-Bozdog OB, et al. Oxidative stress and alopecia areata. *J Med Life*. 2015;8(Spec Issue):43-6. PMID: 26361510; PMCID: PMC4564047.
5. Kantor J, Kessler LJ, Brooks DG, Cotsarelis G. Decreased serum ferritin is associated with alopecia in women. *J Invest Dermatol*. 2003;121(5):985-8. DOI: 10.1046/j.1523-1747.2003.12540.x. PMID: 14708596
6. Chisti MA, Masood Q, Shah IH, et al. Serum ferritin levels in non-scarring alopecia of women: a case-control study. *J Pak Assoc Dermatol*. 2012;22:4-11.
7. Devaraj Y, Devi THB, Bachaspatimayum R. Correlation between serum ferritin and severity of alopecia areata. *Indian J Clin Exp Dermatol*. 2018;4(4):307-10. DOI: 10.18231/j.ijced.2019.070
8. Thompson JM, Mirza MA, Park MK, Qureshi AA, Cho E. The role of micronutrients in alopecia areata: a review. *Am J Clin Dermatol*. 2017;18(5):663-79. DOI: 10.1007/s40257-017-0285-x. PMID: 28508256; PMCID: PMC5685931.
9. Kincaid CM, Sharma AN, Mesinkovska NA. Alopecia areata is associated with risk of inflammatory arthritis. *J Am Acad Dermatol*. 2023;89(2):422-3. DOI: 10.1016/j.jaad.2023.04.039. PMID: 37121482
10. Gök AM, Aşkın Ö, Serdaroğlu S, Kutlubay Z. Retrospective analysis of the effect of comorbid atopic dermatitis on the treatment response to topical immunotherapy in pediatric alopecia areata patients. *Dermatol Pract Concept*. 2024;14(1):e2024006. DOI: 10.5826/dpc.1401a6. PMID: 38364396
11. Vélez-Muñiz RDC, Peralta-Pedrero ML, Jurado-Santa Cruz F, Morales-Sánchez MA. Psychological profile and quality of life of patients with alopecia areata. *Skin Appendage Disord*. 2019;5(5):293-8. DOI: 10.1159/000497166. PMID: 31559253; PMCID: PMC6751425.
12. Singam V, Patel KR, Lee HH, Rastogi S, Silverberg JI. Association of alopecia areata with hospitalization for mental health disorders in US adults. *J Am Acad Dermatol*. 2019;80(3):792-4. DOI: 10.1016/j.jaad.2018.07.044. PMID: 30092332.
13. Kuty-Pachecka M. Psychological and psychopathological factors in alopecia areata. *Psychiatr Pol*. 2015;49(5):955-64. DOI: 10.12740/PP/39064. PMID: 26688846.
14. Sibbald C. Alopecia areata: an updated review for 2023. *J Cutan Med Surg*. 2023;27(3):241-59. DOI: 10.1177/12034754231168839. PMID: 37340563
15. David E, Shokrian N, Del Duca E, et al. Dupilumab induces hair regrowth in pediatric alopecia areata: a real-world, single-center observational study. *Arch Dermatol Res*. 2024;316(7):487. DOI: 10.1007/s00403-024-03225-4. PMID: 39042295
16. Jang YH, Eun DH, Kim DW. Long-term prognosis of alopecia areata in children and adolescents. *Ann Dermatol*. 2019;31(2):231-4. DOI: 10.5021/ad.2019.31.2.231. PMID: 33911578; PMCID: PMC7992685.
17. Manchanda Y, Ramamoorthy R. Revisiting pediatric alopecia areata: newer insights. *Indian J Paediatr Dermatol*. 2021;22(4): 301-5. DOI: 10.4103/ijpd.ijpd_109_21.
18. Conic RZ, Tamashunas NL, Damiani G, et al; Young Dermatologists Italian Network. Comorbidities in pediatric alopecia areata. *J Eur Acad Dermatol Venereol*. 2020;34(12):2898-901. DOI: 10.1111/jdv.16727. PMID: 32531131.
19. Dündar B, Boyacı A, Sangün Ö, Dündar N, Çocuk ve ergenlerde Hashimoto tiroiditi: klinik ve laboratuvar bulgularının değerlendirilmesi. *Turk Pediatri Ars*. 2011;46(4):318-22. DOI: 10.4274/tpa.358.1338
20. Casto C, Pepe G, Li Pomi A, Corica D, Aversa T, Wasniewska M. Hashimoto's thyroiditis and Graves' disease in genetic syndromes in pediatric age. *Genes (Basel)*. 2021;12(2):222. DOI: 10.3390/genes12020222. PMID: 33557156; PMCID: PMC7913917.
21. Bilgiç Ö, Bilgiç A, Bahalı K, Bahali AG, Gürkan A, Yılmaz S. Psychiatric symptomatology and health-related quality of life in children and adolescents with alopecia areata. *J Eur Acad Dermatol Venereol*. 2014;28(11):1463-8. DOI: 10.1111/jdv.12315. PMID: 24237476.
22. Savarino G, Corsello A, Corsello G. Macronutrient balance and micronutrient amounts through growth and development. *Ital J Pediatr*. 2021;47(1):109. DOI: 10.1186/s13052-021-01061-0. PMID: 33964956; PMCID: PMC8106138.
23. Silverberg N. The genetics of pediatric cutaneous autoimmunity: the sister diseases vitiligo and alopecia areata. *Clin Dermatol*. 2022;40(4):363-73. DOI: 10.1016/j.clindermatol.2022.02.009. PMID: 35183681.
24. Lu YY, Wu MK, Lu CC, Wang WT, Wu CH. Atopic diseases and the risk of alopecia areata among pre-teens and teenagers in

Taiwan. *Indian J Dermatol Venereol Leprol*. 2024;90:1-6. DOI: 10.25259/IJDVL_1215_2023. PMID: 39152872

25. Nawani S, Satyasri T, Netha GN, Rammohan G, Kumar B. Autoimmune associations of alopecia areata in pediatric population: a study in a tertiary care centre. *Indian J Clin Exp Dermatol*. 2020;6(2):97-101. DOI: 10.18231/j.ijced.2020.010.
26. Pratt CH, King LE Jr, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. *Nat Rev Dis Primers*. 2017;3:17011. DOI: 10.1038/nrdp.2017.11. PMID: 28300084; PMCID: PMC5573125.
27. Türkay A, Uzunçakmak TK, Engin B, Serdaroğlu S. Alopecia areata: current review. *Dermatoz*. 2020;11(4):39-51. DOI: 10.4274/dermatoz.galenos.2021.88598.
28. Gürlek A, Gökçay Canpolat A, Güngüneş A, et al. *Tiroid Hastalıkları Tanı ve Tedavi Kılavuzu 2023*. 7th ed. Ankara: Türkiye Endokrinoloji ve Metabolizma Derneği Yayınları; 2023..
29. Ly S, Manjaly P, Kamal K, et al. Comorbid conditions associated with alopecia areata: a systematic review and meta-analysis. *Am J Clin Dermatol*. 2023;24(6):875–893. DOI: 10.1007/s40257-023-00785-7. PMID: 37723721.
30. Sorrell J, Petukhova L, Reingold R, et al. Shedding light on alopecia areata in pediatrics: a retrospective analysis of comorbidities in children in the National Alopecia Areata Registry. *Pediatr Dermatol*. 2017;34(5):e271–e272. DOI: 10.1111/pde.13238. PMID: 28834507.
31. Sheth VM, Guo Y, Qureshi AA. Comorbidities associated with vitiligo: a ten-year retrospective study. *Dermatology*. 2013;227(4):311-5. DOI: 10.1159/000354607. Epub 2013 Oct 4. PMID: 24107643.
32. Tissera KA, Bitar RA, Hawryluk EB, Garza-Mayers AC. Comorbidities of pediatric vitiligo. *JAAD Rev*. 2025;3:131–137. DOI: 10.1016/j.jdrv.2025.01.006.
33. Tharwat S, Hamdy F, Hamdy S, Nassar MK. Prevalence of co-existing autoimmune and autoinflammatory diseases in vitiligo: a survey-based study from Egypt. *BMC Rheumatol*. 2024;8(1):59. DOI: 10.1186/s41927-024-00427-1. PMID: 39501366
34. Rork JF, Rashighi M, Harris JE. Understanding autoimmunity of vitiligo and alopecia areata. *Curr Opin Pediatr*. 2016;28(4):463–469. DOI: 10.1097/MOP.0000000000000375. PMID: 27191524
35. Damsky W, King BA. JAK inhibitors in dermatology: the promise of a new drug class. *J Am Acad Dermatol*. 2017;76(4):736–744. DOI: 10.1016/j.jaad.2016.12.005. PMID: 28139263
36. Ali NS, Tollefson MM, Lohse CM, Torgerson RR. Incidence and comorbidities of pediatric alopecia areata: a retrospective matched cohort study using the Rochester Epidemiology Project. *J Am Acad Dermatol*. 2022;87(2):427–429. DOI: 10.1016/j.jaad.2021.08.050. PMID: 34474151. PMCID: PMC9117776.
37. Paller A, Jaworski JC, Simpson EL, et al. Major Comorbidities of Atopic Dermatitis: Beyond Allergic Disorders. *Am J Clin Dermatol*. 2018 Dec;19(6):821-838. DOI: 10.1007/s40257-018-0383-4. PMID: 30168085.
38. Geng RSQ, Buhler KA, Choi MY, et al. Serum Th1, Th2, Th17, and innate immune system biomarkers are elevated in pediatric alopecia areata with and without concurrent atopic dermatitis: A cross-sectional study. *JAAD Int*. 2024;18:128-30. DOI: 10.1016/j.jdin.2024.09.008. PMID: 39719961; PMCID: PMC11667068.
39. Montilla AM, Gómez-García F, Gómez-Arias PJ, et al.Review on the use of drugs targeting JAK/STAT pathway in atopic dermatitis, vitiligo, and alopecia areata. *Dermatol Ther (Heidelb)*. 2019;9(4):655–683. DOI: 10.1007/s13555-019-00329-y. PMID: 31655972.
40. Zhou W, Cai J, Li Z, Lin Y. Association of atopic dermatitis with autoimmune diseases: a bidirectional and multivariable two-sample Mendelian randomization study. *Front Immunol*. 2023;14:1132719. DOI: 10.3389/fimmu.2023.1132719. PMID: 37065076. PMCID: PMC10097009.
41. Lusignan S, Alexander H, Broderick C, et al. Atopic dermatitis and risk of autoimmune conditions: population-based cohort study. *J Allergy Clin Immunol*. 2022;150(3):709–713. DOI: 10.1016/j.jaci.2022.03.030. PMID: 35460663.
42. Wohlmuth-Wieser I, Osei JS, Norris D, et al. Childhood alopecia areata: data from the National Alopecia Areata Registry. *Pediatr Dermatol*. 2018;35(2):164–169. DOI: 10.1111/pde.13387. PMID: 29380454.
43. Lu YY, Wu MK, Lu CC, Wang WT, Wu CH. Atopic diseases and the risk of alopecia areata among pre-teens and teenagers in Taiwan. *Indian J Dermatol Venereol Leprol*. 2024;90:1–6. DOI: 10.25259/IJDVL_1215_2023. PMID: 39152872
44. Serarslan G, Savaş N, Yenin JZ. Is atopy and autoimmunity more prevalent in patients with alopecia areata? A comparative study. *J Eur Acad Dermatol Venereol*. 2012 Jun;26(6):720-3. DOI: 10.1111/j.1468-3083.2011.04152.x. Epub 2011 Jun 21. PMID: 21692870.
45. Kridin K, Renert-Yuval Y, Guttman-Yassky E, Cohen AD. Alopecia areata is associated with atopic diathesis: results from a population-based study of 51,561 patients. *J Allergy Clin Immunol Pract*. 2020;8(4):1323-1328.e1. DOI: 10.1016/j.jaip.2020.01.052. PMID: 32032694.
46. Campos-Alberto E, Hirose T, Napatalung L, Ohyama M. Prevalence, comorbidities, and treatment patterns of Japanese patients with alopecia areata: A descriptive study using Japan medical data center claims database. *J Dermatol*. 2023 Jan;50(1):37-45. DOI: 10.1111/1346-8138.16615. Epub 2022 Nov 2. PMID: 36321512; PMCID: PMC10092019..
47. Xu W, Zhang H, Wan S, Xie B, Song X. Genetic links between atopy, allergy, and alopecia areata: insights from a Mendelian randomization study. *Allergy Asthma Clin Immunol*. 2024;20(1):32. DOI: 10.1186/s13223-024-00892-w. PMID: 38704164.
48. Ghanizadeh A. Comorbidity of psychiatric disorders in children and adolescents with alopecia areata in a child and adolescent psychiatry clinical sample. *Int J Dermatol*. 2008;47(11):1118–1120. DOI: 10.1111/j.1365-4632.2008.03743.x. PMID: 19076668.
49. Altunisik N, Ucuz I, Turkmen D. Psychiatric basics of alopecia areata in pediatric patients: Evaluation of emotion dysregulation, somatization, depression, and anxiety levels. *J Cosmet Dermatol*. 2022;21(2):770–775. DOI: 10.1111/jocd.14122. PMID: 34888986.
50. Lauron S, Plasse C, Vaysset M, et al. Prevalence and odds of depressive and anxiety disorders and symptoms in children and adults with alopecia areata: A systematic review and meta-analysis. *JAMA Dermatol*. 2023;159(3):281–288. DOI: 10.1001/jamadermatol.2022.6085. PMID: 36786747.
51. Ho HY, Wong CK, Wu SY, Hsiao RC, Chen YL, Yen CF. Increased alopecia areata risk in children with attention-deficit/hyperactivity disorder and the impact of methylphenidate use: a nationwide population-based cohort study. *Int J Environ Res Public Health*. 2021;18(3):1286. DOI: 10.3390/ijerph18031286. PMID: 33535410.

52. Ji C, Liu S, Zhu K, Luo H, Li Q, Zhang Y, et al.HLA-DRB1 polymorphisms and alopecia areata disease risk. *Medicine (Baltimore)*.2018;97(30):e11790.DOI:10.1097/MD.0000000000011790. PMID: 30045340.
53. Aureli A, Sebastiani P, Del Beato T, et al.Investigation on the possible relationship existing between the HLA-DR gene and attention deficit hyperactivity disorder and/or mental retardation. *Int J Immunopathol Pharmacol*. 2008;21(4):985–991. DOI: 10.1177/039463200802100423. PMID: 19144216.
54. Danielson ML, Claussen AH, Bitsko RH, et al. ADHD prevalence among U.S. children and adolescents in 2022: diagnosis, severity, co-occurring disorders, and treatment. *J Clin Child Adolesc Psychol*. 2024;53(3):343-60. DOI: 10.1080/15374416.2024.2335625. PMID: 38778436; PMCID: PMC11334226.
55. Jangmo A, Stålhandske A, Chang Z, et al. Attention-deficit/hyperactivity disorder, school performance, and effect of medication. *J Am Acad Child Adolesc Psychiatry*. 2019;58(4):423–432. DOI: 10.1016/j.jaac.2018.11.014. PMID: 30768391; PMCID: PMC6541488.
56. Rado F. Review study on attention deficit and hyperactivity disorder. *Pearson J Soc Sci Humanit*. 2024;1615:1624. DOI: 10.5281/zenodo.13628871.
57. Abdaljawad S, Qiteesh H, Abdelgader A, Saed W. Serum ferritin level and alopecia areata in pediatric patients. *AlQalam J Med Appl Sci*. 2022;5(2):527–533. DOI: 10.5281/zenodo.7264077.
58. Arosio P, Ingrassia R, Cavadini P. Ferritins: a family of molecules for iron storage, antioxidation and more. *Biochim Biophys Acta*. 2009;1790(7):589–599. DOI: 10.1016/J.BBAGEN.2008.09.004. PMID: 18930140.
59. Kundak S, Kutlu A. Serum 25-Hydroxy-Vitamin D and Vitamin B12 levels in childhood alopecia areata. *J Dr Behcet Uz Child Hosp*. 2021;11(1):101–107. DOI: 10.5222/buchd.2021.42744.
60. Ertugrul DT, Taner D, Takci Z et al. Serum holotranscobalamine, vitamin B12, folic acid and homocysteine levels in alopecia areata patients. *Cutan Ocul Toxicol*. 2013;32(1):1-3. DOI: 10.3109/15569527.2012.689443. PMID: 22591107.
61. Gonul M, Cakmak SK, Soylu S, et al. Serum vitamin B12, folate, ferritin, and iron levels in Turkish patients with alopecia areata. *Indian J Dermatol Venereol Leprol*. 2009;75(5):552. DOI: 10.4103/0378-6323.55430. PMID: 19736464.
62. Varkal MA. Genel pediatri polikliniği vakalarında B12 vitamini eksikliği. *Çocuk Dergisi*. 2022;22(1):15–20. DOI: 10.26650/jchild.2021.956537.
63. Krugluger W, Stiefsohn K, Laciak K, et al. Vitamin B12 activates the Wnt-pathway in human hair follicle cells by induction of β-catenin and inhibition of glycogen synthase kinase-3 transcription. *J Cosmet Dermatol Sci Appl*. 2011;1:25–29. DOI: 10.4236/jcdsa.2011.12004.
64. Van de Lagemaat EE, de Groot LCPGM, van den Heuvel EGHM. Vitamin B12 in Relation to Oxidative Stress: A Systematic Review. *Nutrients*. 2019 Feb 25;11(2):482. DOI 10.3390/nu11020482. PMID: 30823595; PMCID: PMC6412369.
65. Mikkelsen K, Apostolopoulos V. Vitamin B12, folic acid, and the immune system. In: Chatterjee S, Jungraithmayr W, Bagchi D, eds. *Nutrition and Immunity*. Cham: Springer; 2019:103-114. DOI: 10.1007/978-3-030-16073-9_7.
66. Kurtev A, Iliev E. Thyroid autoimmunity in children and adolescents with alopecia areata. *Int J Dermatol*. 2005 Jun;44(6):457-61. DOI:10.1111/j.1365-4632.2005.01971.x. PMID: 15941439.
67. Zhao Y, Guo F, Guo M. Thyroid Dysfunction and Alopecia Areata: A Genetic Prediction Causality Analysis Study. *Skin Res Technol*. 2024;30(10):e70063. DOI:10.1111/srt.7006
68. Lee S, Lee H, Lee CH, Lee WS. Comorbidities in alopecia areata: A systematic review and meta-analysis. *J Am Acad Dermatol*. 2019;80(2):466-477.e16. DOI:10.1016/j.jaad.2018.07.013