Dicle Tıp Dergisi / Dicle Med J (2026) 53 (2) : 381- 388

www.diclemedj.org

Original Article / Özgün Araştırma

Allergic and Autoimmune Features in Children with Selective IgA Deficiency

Enes Çelik[1], **Hande Yüksel Bulut**[1]

1 Ankara Atatürk Sanatorium Training and Research Hospital, Division of Pediatric Allergy and Immunology, Department of Pediatrics, Ankara, Türkiye

Received: 18.02.2026; Revised: 24.04.2026; Accepted: 27.04.2026

Abstract

Background: Selective immunoglobulin A deficiency (SIgAD) represents the most frequently encountered form of primary immunodeficiency and is defined by markedly reduced circulating IgA concentrations despite normal IgG and IgM levels. This study aimed to characterize the clinical spectrum and laboratory profile of pediatric patients with SIgAD, with particular emphasis on allergic comorbidities and autoimmune manifestations.

Methods: This retrospective observational study was conducted among children aged 4–18 years who met the European Society for Immunodeficiencies (ESID) diagnostic criteria for SIgAD between April 1, 2023, and December 1, 2025. Information on demographic variables, clinical features, and laboratory measurements was collected from electronic health records and analyzed using descriptive statistics.

Results: Twenty-six pediatric patients were included (61.5% male). The median ages at symptom onset and diagnosis were 4.50 years (IQR, 3.0–6.0) and 6.97 years (IQR, 5.1–9.5), respectively. Recurrent infections affected 76.9% of patients and represented the predominant clinical manifestation. Allergic diseases were present in 73.1% of patients, most frequently asthma and allergic rhinitis; 42.3% showed aeroallergen sensitization, predominantly to pollens. Among the patients who underwent autoantibody screening, positivity was detected in approximately two-thirds; however, only two were diagnosed with autoimmune diseases (thyroiditis and vitiligo). Antibiotic prophylaxis was initiated in 7.7% of patients due to recurrent infections.

Conclusion: Although recurrent infections are the most common presentation in SIgAD, serum IgA evaluation should also be considered in patients with allergic or autoimmune conditions. Regular follow-up is necessary to monitor allergic manifestations, immunologic status, and the potential development of autoimmune diseases.

Keywords: selective IgA deficiency, allergic diseases, autoimmunity, recurrent infections, pediatric

DOI: 10.5798/dicletip.1964451

Correspondence / Yazışma Adresi: *Enes Çelik, Ankara Atatürk Sanatorium Training and Research Hospital, Division of Pediatric Allergy and Immunology, Pınarbaşı Mah. Sanatoryum Caddesi Ardahan Sok. No: 25, Keçiören, Ankara, Türkiye e-mail: drenescelik@outlook.com*

Selektif IgA Eksikliği Olan Çocuklarda Alerjik ve Otoimmün Özellikler

Öz

Amaç: Selektif immünoglobulin A eksikliği (SIgAD), en sık görülen primer immün yetmezlik olup serum IgA düzeylerinin belirgin derecede düşük olması, buna karşın IgG ve IgM düzeylerinin normal olmasıyla tanımlanır. Bu çalışmada, SIgAD tanılı pediatrik hastaların klinik spektrumu ve laboratuvar bulgularının, özellikle alerjik komorbiditeler ve otoimmün bulgulara odaklanılarak değerlendirilmesi amaçlanmıştır.

Yöntemler: Bu retrospektif gözlemsel çalışma, 1 Nisan 2023 ile 1 Aralık 2025 tarihleri arasında European Society for Immunodeficiencies (ESID) tanı kriterlerini karşılayan 4–18 yaş arası çocuklarda gerçekleştirildi. Demografik veriler, klinik özellikler ve laboratuvar ölçümleri elektronik tıbbi kayıtlarından elde edilmiş ve tanımlayıcı istatistiksel yöntemler kullanılarak analiz edildi.

Bulgular: Çalışmaya 26 pediatrik hasta dahil edildi (%61,5 erkek). Semptom başlangıç yaşı ortancası 4,50 yıl (IQR, 3,0–6,0), tanı yaşı ortancası ise 6,97 yıl (IQR, 5,1–9,5) olarak saptandı. Hastaların %76,9'unda rekürren enfeksiyonlar görülmüş olup, en sık klinik başvuru nedeniydi. Hastaların %73,1'inde en az bir alerjik hastalık (en sık astım ve alerjik rinit) saptanmış, %42,3'ünde ise en az bir aeroalerjen duyarlılığı gösterilmiş olup polen duyarlılığı ön plandaydı. Otoantikor taraması yapılan hastaların yaklaşık üçte ikisinde pozitiflik saptanmıştı; ancak yalnızca iki hastaya otoimmün hastalık (tiroidit ve vitiligo) tanısı konuldu. Tekrarlayan enfeksiyonlar nedeniyle hastaların %7,7'sine antibiyotik profilaksisi başlandığı tespit edildi.

Sonuç: Tekrarlayan enfeksiyonlar SIgAD'de en sık klinik başvuru nedeni olmakla birlikte, alerjik veya otoimmün bulgularla başvuran hastalarda da serum IgA düzeyinin değerlendirilmesi düşünülmelidir. Alerjik bulguların, immünolojik durumun ve olası otoimmün hastalık gelişiminin izlenebilmesi için düzenli takip gereklidir.

Anahtar kelimeler: selektif IgA eksikliği, alerjik hastalıklar, otoimmünite, tekrarlayan enfeksiyonlar, çocukluk çağı.

INTRODUCTION

Selective immunoglobulin A deficiency (SIgAD) represents the most frequently encountered primary immunodeficiency worldwide[1]. Its reported prevalence varies substantially across populations, ranging from approximately 1:3,000 to 1:150 individuals, reflecting geographic and ethnic differences in distribution[1,2]. In Türkiye, the estimated frequency has been reported as 1:188[3]. SIgAD is diagnosed in individuals older than four years who demonstrate serum IgA levels below 7 mg/dL despite normal IgG and IgM concentrations. The diagnosis requires exclusion of secondary causes of hypogammaglobulinemia, confirmation of intact T-cell immunity, and evidence of preserved specific antibody responses to vaccinations[1,4,5].

Although numerous studies have explored the underlying mechanisms of SIgAD, its precise pathogenesis remains incompletely understood. The condition is considered heterogeneous, suggesting that multiple etiologic pathways may contribute to its development. Proposed mechanisms include intrinsic defects in B-cell differentiation and maturation, abnormalities in T-cell regulation, and dysregulation of cytokine networks involved in immunoglobulin production[6].

A substantial proportion of individuals with SIgAD—estimated to be as high as 75%—remain clinically asymptomatic and are often identified incidentally during laboratory investigations performed for unrelated reasons. In contrast, symptomatic patients may present with recurrent infections, particularly affecting the respiratory tract, as well as allergic or autoimmune manifestations[1].

In some patients, allergic disorders constitute the earliest or even the only clinical expression of IgA deficiency. The reported prevalence of

allergic diseases in SIgAD ranges between 25% and 50%, and allergic symptoms may become more pronounced over time. The most commonly associated allergic diseases include rhinitis, conjunctivitis, urticaria, atopic dermatitis, food allergy, and asthma[6].

Compared with the general population, individuals with SIgAD demonstrate an increased tendency toward autoimmune phenomena. Published series report autoimmune disease frequencies varying between 5% and 30%, depending on age and cohort characteristics. Commonly described associations include celiac disease, immune thrombocytopenia, autoimmune thyroiditis, autoimmune hemolytic anemia, and systemic lupus erythematosus[1].

The present study aimed to characterize the clinical spectrum and laboratory profile of pediatric patients with SIgAD, with particular focus on allergic comorbidities and autoimmune features.

METHODS

This retrospective observational study was conducted at the Division of Pediatric Allergy and Immunology, Ankara Atatürk Sanatorium Training and Research Hospital, between April 1, 2023, and December 1, 2025. Children aged 4–18 years who met the European Society for Immunodeficiencies (ESID) diagnostic criteria for SIgAD were included[5].

Information on demographic variables, clinical features, and laboratory measurements was collected from the institutional electronic health records. Because many patients were newly diagnosed during the later part of the study period, follow-up duration varied among patients and was calculated from the date of SIgAD diagnosis to the last available clinical evaluation. Laboratory evaluations included complete blood count, serum immunoglobulin levels measured by nephelometry, lymphocyte subset analysis, autoantibody testing, and assessment of vaccine-specific antibody responses. The autoantibody tests included antinuclear antibodies (ANA), anti–double-stranded DNA (anti-dsDNA), anti-thyroglobulin (anti-TG), anti-thyroid peroxidase (anti-TPO), anti–tissue transglutaminase IgG (tTG-IgG), and anti–tissue transglutaminase IgA (tTG-IgA). Vaccine-specific antibody responses were evaluated by measuring hepatitis B surface antibody (anti-HBs) levels. Autoantibody tests were mainly performed as part of screening for autoimmune diseases in patients with SIgAD; however, not all patients underwent every test due to the retrospective design of the study and variations in clinical evaluation.

The diagnosis of allergic diseases was established according to internationally accepted guidelines, including the Global Initiative for Asthma (GINA) recommendations for asthma[7], the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines for allergic rhinitis[8], the Hanifin and Rajka criteria for atopic dermatitis[9], and the EAACI/GA^{2}LEN/EuroGuiDerm/APAAACI guideline for urticaria[10]. Drug allergy and food allergy were evaluated according to international consensus guidelines[11,12].

Allergic evaluation was performed using skin prick testing (SPT) and/or allergen-specific IgE measurement. SPTs were performed using standardized allergen extracts (Lofarma, Milan, Italy). The aeroallergen panel included house dust mites, grass pollen mix, cereal pollen mix, tree pollen mix, Cupressus, Plantago, Artemisia, Ambrosia, Chenopodium, Parietaria, Alternaria, Aspergillus, cat, dog, and cockroach allergens. A SPT result was considered positive when the wheal diameter exceeded the negative control by ≥3 mm. Allergen-specific IgE measurements were performed for house dust mite, grass pollen mix, mold mix, and animal dander mix, and concentrations ≥0.35 kU/L were considered indicative of sensitization. Total and specific IgE measurements were obtained using

a chemiluminescent immunoassay platform (IMMULITE 2000 XPI, Siemens, Germany).

Approval for the study was granted by the Scientific Research Ethics Committee of Ankara Atatürk Sanatorium Training and Research Hospital (Approval No: 2024-BÇEK/450).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics (version 22.0). Distribution normality was assessed with both Kolmogorov–Smirnov and Shapiro–Wilk tests. Normally distributed continuous variables are presented as mean ± standard deviation, whereas non-normally distributed variables are reported as median with interquartile range (IQR). Categorical data are expressed as counts and percentages.

RESULTS

Twenty-six pediatric patients formed the study population, with males representing 61.5% (n=16) and females 38.5% (n=10). Symptoms first appeared at a median age of 4.50 years (IQR, 3.0–6.0), whereas diagnosis was established at a median age of 6.97 years (IQR, 5.1–9.5). The median follow-up period was 5.5 months (IQR, 2.4–12.0). All children were born at term, and their immunizations were administered according to the age-appropriate vaccination schedule. Family history revealed atopy in 7 patients (26.9%) and autoimmune disease in 2 (7.7%). Parental consanguinity was documented in 3 cases (11.5%), whereas no family history of inborn errors of immunity was identified. Exposure to tobacco smoke was reported in 14 patients (53.8%).

A history of recurrent infections was present in 20 patients (76.9%), with a median annual number of infections of 6 (IQR, 5–8). Three patients (11.5%) had a history of hospitalization due to infections. The indications for serum IgA testing included recurrent upper respiratory tract infections in 20 patients (76.9%), allergic symptoms in 13 (50.0%), recurrent lower respiratory tract infections in 6 (23.1%), and celiac disease screening in 5 (19.2%). Several patients had multiple indications for testing. Table I provides an overview of the demographic and clinical profile of the study population.

Table I: Demographic and clinical profile of the study population

Characteristic	
Sex (female/male), n (%)	10 (38.5) / 16 (61.5)
Age at symptom onset (years), median (IQR)	4.50 (3.0-6.0)
Age at diagnosis (years), median (IQR)	6.97 (5.1-9.5)
Follow-up duration (months), median (IQR)	5.5 (2.4-12.0)
Gestational age category, n (%)	
Term	26 (100)
Immunization status (age-appropriate), n (%)	
Up-to-date	26 (100)
Family history of atopy, n (%)	7 (26.9)
Family history of autoimmune disease, n (%)	2 (7.7)
Family history of inborn errors of immunity, n (%)	0
Consanguineous marriage, n (%)	3 (11.5)
Exposure to tobacco smoke, n (%)	14 (53.8)
Pet ownership, n (%)	4 (15.4)
History of recurrent infections, n (%)	20 (76.9)
Number of infections per year, median (IQR)	6 (5-8)
Hospitalization due to infection, n (%)	3 (11.5)
Indications for serum IgA testing, n (%)*	
Recurrent upper respiratory tract infections	20 (76.9)
Recurrent lower respiratory tract infections	6 (23.1)
Allergic symptoms	13 (50.0)
Celiac disease screening	5 (19.2)

Abbreviations: IQR (interquartile range).

**Patients may have had more than one indication; therefore, percentages do not total 100%.*

Allergic diseases were present in 19 patients (73.1%). Asthma was identified in 12 patients (46.2%), and allergic rhinitis in 11 (42.3%). Multiple allergic diseases were observed in 8 patients (30.8%). Aeroallergen sensitization was detected in 11 patients (42.3%), with

pollen sensitization being the most common, observed in 9 patients (34.6%). The allergic comorbidities and aeroallergen sensitization are summarized in Table II.

Table II: Allergic comorbidities and aeroallergen sensitization

Characteristic	n (%)
Allergic diseases, n (%)	19 (73.1)
Asthma	12 (46.2)
Allergic rhinitis	11 (42.3)
Atopic dermatitis	2 (7.7)
Urticaria	1 (3.8)
Drug allergy	1 (3.8)
Food allergy	0
Multiple allergic diseases	8 (30.8)
Aeroallergen sensitization, n (%)	11 (42.3)
Pollen	9 (34.6)
House dust mite	2 (7.7)
Pet dander	2 (7.7)
Mold	1 (3.8)
Polysensitization	3 (11.5)

Thyroid autoantibody screening was performed in 20 patients, of whom 9 (45.0%) tested positive for anti-thyroglobulin antibodies; however, Hashimoto thyroiditis was diagnosed in only one of these patients. ANA testing was conducted in 20 patients, and positivity was detected in 11 (55.0%), although none were diagnosed with a rheumatologic disease. Celiac disease screening was performed in 22 patients, and tTG-IgG positivity was identified in 2 (9.1%); however, none received a diagnosis of celiac disease. Among the 22 patients who underwent autoantibody testing, 15 (68.2%) had positivity for at least one autoantibody. Vitiligo was identified in one patient. The autoantibody profile of the patients is presented in Table III.

Table III: Autoantibody findings in the study population*

	Anti-TG	Anti-TPO	ANA	Anti-dsDNA	tTG-IgA	tTG-IgG
Patients were tested for autoantibodies	20	20	20	11	22	22
Autoantibody positivity, n (%)	9 (45)	-	11 (55)	1 (9.1)	-	2 (9.1)
Autoimmune diseases, n (%)	1 (5)	-	-	-	-	-

Abbreviations: ANA (antinuclear antibody), anti-dsDNA (anti–double-stranded DNA), TG (thyroglobulin), TPO (thyroid peroxidase), and tTG (tissue transglutaminase).

**Certain patients demonstrated positivity for more than one autoantibody.*

The median values were as follows: serum IgA 2.5 mg/dL (IQR, 1–5), eosinophil count 180 cells/μL (IQR, 130–370), eosinophil percentage 2.3% (IQR, 1.7–4.5), and total IgE 49.2 IU/mL (IQR, 11–125). Serum IgG and IgM concentrations and lymphocyte subset values were consistent with age-specific reference intervals. The laboratory findings are presented in Table IV.

Table IV: Laboratory findings of the study population

	Median (IQR)
Complete blood count parameters	
White blood cell count (cells/μL)	7800 (6700-8850)
Neutrophil count (cells/μL)	3020 (3020-5280)
Lymphocyte count (cells/μL)	2700 (2095-3590)
Eosinophil count (cells/μL)	180 (130-370)
Eosinophils (%)	2.3 (1.7-4.5)
Hemoglobin (g/dL)	12.8 (11.8-13.8)
Platelet count (cells/μL)	323000 (276000-428000)
Immunoglobulin levels	
IgG (mg/dL)	1302 (1069-1573)
IgM (mg/dL)	94 (75-115)
IgA (mg/dL)	2.5 (1-5)
Total IgE (IU/mL)	49.2 (11-125)
Lymphocyte subsets, %	
CD3+ T cells	72 (67-74)
CD4+ T cells	38 (34-40)
CD8+ T cells	27 (25-30)
CD19+ B cells	18 (15-21)
CD16+56+ NK cells	8 (6-10)
Anti-HBs antibody positivity, n (%)	22 (84.6)

Abbreviations: IQR (interquartile range).

Antibiotic (trimethoprim–sulfamethoxazole) prophylaxis was initiated in 2 of the 26 patients (7.7%) due to recurrent infections.

DISCUSSION

In this single-center study, the clinical and laboratory characteristics, as well as the allergic and autoimmune profiles, of children with SIgAD were evaluated. Allergic diseases were

present in approximately three-quarters of patients with SIgAD, and nearly half demonstrated sensitization to aeroallergens. Although only two patients had a clinically diagnosed autoimmune disease, more than half exhibited markers suggestive of autoimmune predisposition.

In our cohort, recurrent infections were identified in 76.9% of patients and represented the predominant reason for clinical evaluation and subsequent diagnosis of SIgAD. In the study by Selmanoğlu et al.[13], respiratory tract symptoms constituted the predominant reason for referral to the pediatric allergy department and were most commonly related to infectious or allergic etiologies. Although most individuals with SIgAD are reported to be asymptomatic, likely, symptomatic patients were more frequently referred to our clinic, which may explain the high rate of infectious manifestations observed. Recurrent infections were reported in 71–83.9% of cases in previous studies, consistent with our findings[2,14,15]. Population-based screening studies may identify a higher proportion of asymptomatic individuals with SIgAD, thereby providing a more accurate estimate of the true clinical spectrum of the disease.

Allergic comorbidity was present in 73.1% of the cohort, with asthma and allergic rhinitis representing the most frequent conditions. Reports from Türkiye indicate that allergic disorders are common in SIgAD, with published frequencies ranging from 43.2% to 86.4%, markedly higher than background population estimates[14,16–20]. Aeroallergen sensitization was detected in 42.3% of the patients, similar to the findings reported by Güngören et al.[18]. In their study, house dust mite sensitization was the most frequently identified allergen, whereas pollen sensitization predominated in our cohort. Similarly, in a study from our region, Yıldız et al.[21] reported pollen sensitization in 78.6% of children with aeroallergen sensitization, whereas house dust mite sensitization was detected in 36.6%. Geographic heterogeneity in environmental allergen exposure may account for the variability observed between cohorts.

In the present study, at least one autoimmune marker was detected in approximately two-thirds of the patients who underwent autoantibody screening; however, only two patients were diagnosed with autoimmune diseases, including one case of thyroiditis and one case of vitiligo. In the study by Jacob et al.[22], nearly half of the patients exhibited evidence of autoantibody reactivity; however, only about one-quarter of these individuals were diagnosed with an autoimmune disease. Lougaris et al.[23]. reported that longitudinal monitoring of children with SIgAD revealed the emergence of autoimmune disorders, with celiac disease occurring at a frequency of 14%. Published data indicate that autoimmune conditions occur in approximately 5% to 30% of individuals with SIgAD. Autoimmune manifestations appear to be age-related, with a higher frequency reported in older patients, particularly during the second decade of life[6]. Cinicola et al.[2] reported autoimmune involvement in 13% of patients at initial assessment, rising to 25% after a median observation period of 3 years. In our study, most patients were in the first decade of life and the median follow-up duration was 5.5 months; therefore, the frequency of autoimmune diseases may increase with longer follow-up.

In our study, antibiotic prophylaxis was initiated in two patients (7.7%). Current evidence suggests that patients with SIgAD who develop occasional mild infections can be managed with standard antibiotic therapy, as would be recommended for immunocompetent individuals during acute episodes. However, for patients experiencing recurrent and frequent infections, prophylactic antibiotic therapy—particularly during high-risk seasons such as

autumn and winter—may be warranted[1,16]. Only patients with significant clinical and immunologic impairment related to SIgAD are candidates for immunoglobulin replacement therapy; none of the patients in our cohort required such treatment. A proportion of patients with marked clinical severity in SIgAD have been shown to develop common variable immunodeficiency (CVID) over time, reinforcing the value of structured long-term surveillance[1]. Moreover, the risk of progression to CVID appears to be higher in patients with concomitant autoimmune manifestations[24].

The principal limitations of this study include its retrospective design, limited sample size, and relatively short follow-up duration. Because autoimmune diseases may develop later in life, the current follow-up period may underestimate the true frequency of autoimmune manifestations. In addition, because this study was conducted in a pediatric allergy and immunology clinic, the prevalence of allergic diseases in our cohort may be overestimated due to referral bias. Furthermore, a considerable proportion of individuals with SIgAD in the general population may remain asymptomatic. Despite these limitations, the assessment of allergic sensitization and autoantibody profiles strengthens the contribution of this cohort to the literature on pediatric SIgAD.

CONCLUSION

Recurrent infections constitute the predominant clinical expression of SIgAD; however, allergic manifestations and autoantibody positivity are also frequently encountered in affected children. Measurement of IgA concentrations may therefore be informative not only in patients with recurrent infections but also in those exhibiting allergic or autoimmune features. Given the potential for evolving immune dysregulation and the risk of autoimmune complications, structured long-term surveillance is advisable in pediatric patients with SIgAD.

Ethics Committee Approval: Approval for the study was granted by the Scientific Research Ethics Committee of Ankara Atatürk Sanatorium Training and Research Hospital (Approval No: 2024-BÇEK/450).

Conflict of Interest: The author(s) declare that there is no financial conflict of interest related to this article.

Financial Disclosure: The authors have stated that no financial support was provided for this study.

REFERENCES

1. Yazdani R, Latif A, Tabassomi F, et al. Clinical phenotype classification for selective immunoglobulin A deficiency. Expert Rev Clin Immunol. 2015;11(11):1245-54. doi:10.1586/1744666X.2015.1081565

2. Cinicola BL, Brindisi G, Capponi M, et al. The Allergic Phenotype of Children and Adolescents with Selective IgA Deficiency: A Longitudinal Monocentric Study. J Clin Med. 2022;11(19). doi:10.3390/jcm11195705

3. Baştürk B, Sari S, Aral A, Dalgiç B. Prevalence of selective immunoglobulin A deficiency in healthy Turkish school children. Turk J Pediatr. 2011;53(4):364-8.

4. Yel L. Selective IgA deficiency. J Clin Immunol. 2010;30(1):10-6. doi:10.1007/s10875-009-9357-x

5. European Society for Immunodeficiencies (ESID). IgA deficiency – diagnostic criteria. 2024 [cited 2026 Feb 14]. Available from: https://esid.org/wp-content/uploads/2024/04/IgA-Deficiency.doc.

6. Yazdani R, Azizi G, Abolhassani H, Aghamohammadi A. Selective IgA Deficiency: Epidemiology, Pathogenesis, Clinical Phenotype, Diagnosis, Prognosis and Management. Scand J Immunol. 2017;85(1):3-12. doi:10.1111/sji.12499

7. Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention. 2023 update. Available from: https://ginasthma.org.

8. Bousquet J, Khaltaev N, Cruz AA, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health

Organization, GA(2)LEN and AllerGen). Allergy. 2008;63 Suppl 86:8-160. doi:10.1111/j.1398-9995.2007.01620.x

9. Rothe MJ, Grant-Kels JM. Diagnostic criteria for atopic dermatitis. Lancet (London, England). 1996;348(9030):769-70. doi:10.1016/S0140-6736(05)65206-3

10. Zuberbier T, Abdul Latiff AH, Abuzakouk M, et al. The international EAACI/GA2LEN/EuroGuiDerm/APAAACI guideline for the definition, classification, diagnosis, and management of urticaria. Allergy. 2022;77(3):734-766. doi:10.1111/all.15090

11. Demoly P, Adkinson NF, Brockow K, et al. International Consensus on drug allergy. Allergy. 2014;69(4):420-37. doi:10.1111/all.12350

12. Muraro A, Werfel T, Hoffmann-Sommergruber K, et al. EAACI food allergy and anaphylaxis guidelines: diagnosis and management of food allergy. Allergy. 2014;69(8):1008-25. doi:10.1111/all.12429

13. Selmanoğlu A, Yörüsün G, Genis C, et al. Inpatient Pediatric Allergy Referrals: A One-Year Review of Consultation Trends. Dicle Medical Journal. 2025;52(3):569-76. doi:10.5798/dicletip.1785132.

14. Aytekin C, Tuygun N, Gokce S, Dogu F, Ikinciogullari A. Selective IgA deficiency: clinical and laboratory features of 118 children in Turkey. J Clin Immunol. 2012;32(5):961-6. doi:10.1007/s10875-012-9702-3

15. Çevik S, Altaş U, Bozkurt HB, Özkars MY. Retrospective evaluation of patients with selective IgA deficiency. Alergol Pol - Polish J Allergol. 2024;11(4):286-91. doi:10.5114/pja.2024.145188

16. Artaç H, Keleş S, Karagöl C, Göktürk B, Reisli İ. İmmünglobulin A eksikliğinde atopi ve allerjik hastalıklar. Asthma Allergy Immunol. 2012;10:25-30.

17. Erkoçoğlu M, Metin A, Kaya A, et al. Allergic and autoimmune disorders in families with selective IgA deficiency. Turkish J Med Sci. 2017;47(2):592-8. doi:10.3906/sag-1605-50

18. Güngören EY, Çalışkan N, Özkalkan B, Karadağ İK, Özçeker D, Çimen SS. Insights into the clinical spectrum of selective IgA deficiency: Data from two centers. Allergol Immunopathol (Madr). 2025;53(6):32-7. doi:10.15586/aei.v53i6.1472

19. Ağralı N, Asilsoy S, Anal Ö, Köse SS. İmmünglobulin A Eksikliğinde Atopik Hastalıkların Sıklığı. İzmir Dr. Behçet Uz Çocuk Hastanesi Dergisi. 2020; 10(1): 15-21. 10.5222/buchd.2020.26879.

20. Ulaş S, Al S, Naiboğlu S, et al. Selective IgA deficiency and allergic diseases: Clinical and immunological evaluation. Allergol Immunopathol (Madr). 2025;53(5):78-86. doi:10.15586/aei.v53i5.1374

21. Yildiz YK, Yaytokgil SB, Vezir E. Evaluation of clinical and demographic characteristics in children with aeroallergen sensitization: A study based on skin prick test results. Allergol Immunopathol (Madr). 2025;53(6):46-54. doi:10.15586/aei.v53i6.1412

22. Jacob CMA, Pastorino AC, Fahl K, Carneiro-Sampaio M, Monteiro RC. Autoimmunity in IgA deficiency: revisiting the role of IgA as a silent housekeeper. J Clin Immunol. 2008;28 Suppl 1:S56-61. doi:10.1007/s10875-007-9163-2

23. Lougaris V, Sorlini A, Monfredini C, et al. Clinical and Laboratory Features of 184 Italian Pediatric Patients Affected with Selective IgA Deficiency (SIgAD): a Longitudinal Single-Center Study. J Clin Immunol. 2019;39(5):470-5. doi:10.1007/s10875-019-00647-y

24. Abolhassani H, Gharib B, Shahinpour S, et al. Autoimmunity in patients with selective IgA deficiency. J Investig Allergol Clin Immunol. 2015;25(2):112-9.