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Clinical Phenotypes and Factors Associated with Systemic Involvement in Pediatric IgA Vasculitis: A Single-Center Study from Southeastern Türkiye

Gülcan Özomay Baykal[1]

1 Mardin Training and Research Hospital, Clinic of Pediatric Rheumatology, Mardin, Türkiye

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Abstract

Objective: To characterize clinical phenotypes and laboratory indicators of systemic involvement (gastrointestinal [GI] and renal) in pediatric immunoglobulin A vasculitis (IgAV), focusing on purpura distribution, inflammatory markers, and ultrasonography (USG) findings.

Methods: We retrospectively reviewed medical records of 51 children diagnosed with IgAV according to EULAR/PRINTO/PRES 2008 criteria at a tertiary center in Mardin (March 2025–January 2026). Data were analyzed using Chi-square/Fisher's exact tests for categorical variables and Mann–Whitney U tests for continuous variables.

Results: Median age was 6.67 years; 54.9% were male. GI and renal involvement occurred in 41.2% and 43.1%, respectively. Widespread rash (33.3%) significantly correlated with GI ($p<0.001$) and renal ($p=0.012$) involvement. Trunk ($p=0.0067$) and upper extremity involvement (GI: $p=0.019$; renal: $p=0.027$) were also associated factors. Fecal occult blood (FOB) positivity (21.6%) associated with widespread rash ($p=0.0039$). USG demonstrated mesenteric lymphadenopathy (66.6%), free fluid (38.1%), bowel wall thickening (23.8%), and intussusception (14.3%). Thrombocytosis (33.3%) was more frequent in renal involvement (63.6% vs 34.5%, $p=0.04$). Elevated C-reactive protein (CRP) correlated with FOB positivity ($p=0.01$). Steroid use was linked to GI involvement ($p=0.017$), while ACE inhibitor use was strongly associated with renal involvement and higher spot urine protein/creatinine ratios ($p<0.001$).

Conclusion: In pediatric IgAV, the extent and distribution of purpura—specifically widespread rash and upper extremity involvement—serve as visible phenotypic predictors of systemic complications. Thrombocytosis and elevated CRP provide additional prognostic value, supporting early risk stratification in clinical practice.

Keywords: IgA vasculitis; Henoch–Schönlein purpura; purpura distribution; gastrointestinal involvement; renal involvement; thrombocytosis; ultrasonography

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Correspondence / Yazışma Adresi: *Gülcan Özomay Baykal, Mardin Training and Research Hospital, Clinic of Pediatric Rheumatology, Mardin, Türkiye e-mail: gulcanozomaybaykal@gmail.com*

Pediatrik İmmünoglobulin A Vaskülitinde Sistemik Tutulumla İlişkili Klinik Fenotipler ve Faktörler: Güneydoğu Türkiye'den Tek Merkezli Bir Çalışma

Öz

Amaç: Pediatrik immünoglobulin A vaskülitinde (IgAV; Henoch–Schönlein purpurası) sistemik tutulumun (gastrointestinal [Gİ] ve renal) klinik fenotiplerini ve laboratuvar göstergelerini; purpura dağılımı, fekal gizli kan (FGK), ultrasonografi (USG) bulguları ve inflamatuar belirteçlere odaklanarak karakterize etmektir.

Yöntemler: Mardin'deki bir tersiyer pediatrik romatoloji merkezinde Mart 2025 ile Ocak 2026 tarihleri arasında EULAR/PRINTO/PRES 2008 kriterlerine göre IgAV tanısı alan 51 çocuğun tıbbi kayıtları geriye dönük olarak incelendi. İstatistiksel analizlerde kategorik değişkenler için Ki-kare/Fisher'in kesinlik testleri, sürekli değişkenler için ise Mann–Whitney U testleri kullanıldı.

Bulgular: Ortanca yaş 6,67 yıl olup hastaların %54,9'u erkekti. Gİ tutulumu %41,2, renal tutulum %43,1 oranında saptandı. Yaygın döküntü varlığı (%33,3), hem Gİ ($p<0.001$) hem de renal (p=0,012) tutulumla anlamlı düzeyde ilişkiliydi. Gövde (p=0,0067) ve üst ekstremite tutulumu (Gİ: p=0,019; renal: p=0,027) sistemik tutulum için güçlü öngördürücülerdi. FGK pozitifliği (%21,6) yaygın döküntü ile ilişkili bulundu (p=0,0039). USG incelemelerinde mezenterik lenfadenopati (%66,6), serbest sıvı (%38,1), bağırsak duvar kalınlaşması (%23,8) ve invajinasyon (%14,3) gözlendi. Trombositoz (%33,3), renal tutulumu olanlarda daha sıktı (%63,6'ya karşı %34,5, p=0,04). Yüksek CRP düzeyleri FGK pozitifliği ile koreleydi (p=0,01). Steroid kullanımı Gİ tutulumuyla (p=0,017), ACE inhibitörü kullanımı ise renal tutulum ve yüksek proteinüri değerleriyle ($p<0,001$) güçlü ilişkiliydi.

Sonuç: Pediatrik IgAV'de purpuranın yaygınlığı ve anatomik dağılımı—özellikle yaygın döküntü ve üst ekstremite tutulumu—sistemik komplikasyonlar için önemli fenotipik göstergelerdir. Trombositoz ve yüksek CRP seviyeleri, klinik pratikte risk sınıflandırmasını destekleyen ek prognostik veriler sunmaktadır.

Anahtar kelimeler: immünoglobulin A vasküliti; Henoch–Schönlein purpurası; purpura dağılımı; gastrointestinal tutulum; renal tutulum; trombositoz; ultrasonografi.

INTRODUCTION

Immunoglobulin A vasculitis (IgAV), historically known as Henoch-Schönlein purpura (HSP), is the most common systemic small-vessel vasculitis in the pediatric population[1,2]. The disease is defined by the deposition of IgA1-dominant immune complexes within the vessel walls of the skin, joints, gastrointestinal (GI) tract, and kidneys[3]. Although its clinical course is generally self-limiting and benign, the multisystem nature of the disease can lead to acute complications such as intussusception or long-term morbidity related to chronic renal impairment[1,4,5].

The exact etiology of IgAV remains elusive, though environmental triggers—primarily upper respiratory tract infections—acting upon a susceptible host play a critical role[6,7]. Epidemiological data indicate a peak incidence during autumn and winter, frequently following the seasonal prevalence of respiratory pathogens[1,8,9]. While the classic tetrad involves palpable purpura, arthritis, abdominal pain, and renal findings, the severity and timing of these symptoms vary across different populations[6,10].

Renal involvement (RI) is the primary determinant of long-term prognosis, while acute morbidity is often driven by GI complications[1,5,6]. Early risk stratification is essential, and recent studies have focused on the predictive value of inflammatory markers such as the white blood cell (WBC) count, C-reactive protein (CRP), and platelet count[2,6]. Specifically, leukocytosis and thrombocytosis have been proposed as potential indicators of increased risk for renal or GI manifestations[7].

Regional differences in the clinical phenotype of IgAV are well-documented and may be

influenced by environmental factors or specific local backgrounds[3,6,10]. Although Turkey has a high disease burden, detailed clinical and laboratory data from the Southeastern region, particularly from Mardin, remain limited. The objective of this study is to evaluate the demographic and clinical features of children diagnosed with IgAV in Mardin. We specifically aim to investigate the relationship between inflammatory markers and the severity of organ involvement to identify regional prognostic indicators.

METHODS

Study Design and Setting

This retrospective cross-sectional study was conducted at the Pediatric Rheumatology Clinic in Mardin, a healthcare center in Southeastern Turkey. The study analyzed the medical records of pediatric patients diagnosed with IgAV between March 2025 and January 2026. The study protocol adhered to the principles of the Declaration of Helsinki and was approved by the ethics committee of Mardin Artuklu University (Number: 243407).

Inclusion and Exclusion Criteria

The diagnosis of IgAV was established according to the EULAR/PRINTO/PRES 2008 classification criteria[3]. Inclusion criteria required the following:

1. Being under 18 years of age at the time of diagnosis.

2. The presence of mandatory non-thrombocytopenic palpable purpura (typically with lower limb predominance).

3. The presence of at least one of the following four criteria:

• Diffuse abdominal pain,

• Histopathology showing predominant IgA deposition,

• Acute arthritis or arthralgia,

• RI, defined as the presence of proteinuria or hematuria[1,2,6].

Exclusion criteria were:

• Patients with thrombocytopenic purpura,

• Coexistence of other systemic vasculitides or systemic lupus erythematosus (SLE),

• Pre-existing chronic renal or hepatic disease,

• Patients with missing or insufficient data regarding primary clinical outcomes and laboratory parameters.

Clinical Definitions and Data Collection

Demographic, clinical, and laboratory data were retrieved from the patients' medical records[2,6]. Joint involvement was defined as the presence of arthritis (joint swelling or limitation of movement) and/or arthralgia (joint pain without swelling)[2,4]. GI was characterized by the occurrence of abdominal pain, melena, hematemesis, or the detection of occult blood in the stool[1,4].

• Cutaneous Involvement: Purpura distribution was categorized as "Localized" (limited to lower extremities and buttocks) or "Extensive" (involving the trunk, upper extremities, or face)[1,2].

• Joint Involvement: Defined as arthritis (joint swelling/limitation) or arthralgia (pain without swelling)[2,4].

• GI Involvement: GI Involvement: Defined according to the EULAR/PRINTO/PRES 2008 criteria as the presence of acute, diffuse, and typically colicky abdominal pain, melena, hematemesis, or fecal occult blood positivity. For patients presenting with abdominal pain alone, a clinical diagnosis of GI involvement was made only after excluding other potential etiologies and confirming the pain's characteristic nature in association with the IgAV clinical course[1,2,4]. Abdominal ultrasonography (USG) was performed for all patients at initial diagnosis to evaluate for both

symptomatic and subclinical gastrointestinal involvement.

• RI was assessed based on the presence of microscopic or macroscopic hematuria, the detection of proteinuria, or the deterioration of renal functions[2]. To evaluate the severity of nephropathy, proteinuria was categorized according to the spot protein/creatinine ratio (mg/mg) as follows: Normal: <0.2; Mild: 0.2–0.5; Moderate: 0.5–2.0; Nephrotic: >2.01,2,11,12.

• The emergence of a new purpuric rash or other IgAV-related symptoms and findings at least 12 weeks after the initial presentation was defined as recurrence[1,4].

Laboratory Analysis

Blood and urine samples obtained at the time of diagnosis were analyzed for WBC count, platelet (PLT) count, hemoglobin (Hb), erythrocyte sedimentation rate (ESR), and CRP.

• Inflammatory Markers: Leukocytosis was defined as WBC >11,000/mm^3 2. Thrombocytosis was defined as PLT >450,000/mm^3 6. Anemia was defined across age groups.

• Proteinuria: The spot protein/creatinine ratio was used for categorization (Normal vs. Nephrotic range)[1,2].

Statistical Analysis

Statistical analyses were performed using SPSS software (Version 26.0). Continuous variables were expressed as mean ± standard deviation (SD) or median (IQR), while categorical variables were reported as frequencies and percentages. Normal distribution was assessed using the Shapiro-Wilk or Kolmogorov-Smirnov test. For group comparisons, the Chi-square or Fisher's exact test was used for categorical data, and the Student's t-test or Mann-Whitney U test was used for continuous variables. To identify independent factors associated with systemic involvement, multivariable logistic regression analysis was performed. Variables were selected based on univariate significance and clinical relevance (age, sex, and CRP). Results were presented as odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

RESULTS

Patient population and demographic characteristics

A total of 51 pediatric patients diagnosed with IgAV (HSP) were included in the analysis. All patients were evaluated using their clinical and laboratory data and were followed longitudinally after diagnosis (Table 1). The cohort demonstrated a slight male predominance, with 28 patients (54.9%) being male and 23 (45.1%) females. The median age at diagnosis was 6.67 years [IQR 5.71–9.67], consistent with the typical school-age peak reported in pediatric HSP cohorts. At initial presentation, the most frequent symptom was rash (n=43; 84.3%), followed by abdominal pain (n=5; 9.8%) and swelling (n=3; 5.9%). Hospitalization was required in 20 patients (39.2%). Five patients (9.8%) experienced at least one relapse during follow-up period.

Table 1: Demographic, Clinical, Environmental, and Imaging Characteristics of Patients with IgA Vasculitis

	Overall (n = 51)
Demographic characteristics Median [IQR Q1–Q3]	
Age at diagnosis, years	6.67 [5.71–9.67]
Gender [n (%)]	
Female	23 (45.1%)
Male	28 (54.9%)
Location of origin [n (%)]	
Mardin	33 (64.7%)
Şırnak	9 (17.6%)
Syria	4 (7.8%)
Gaziantep	2 (3.9%)
Kayseri	2 (3.9%)
Şanlıurfa	1 (2%)
Diyarbakır	1 (2%)
Other	3 (5.9%)
Season at disease onset [n (%)]	
Autumn	19 (37.3%)
Spring	12 (23.5%)
Summer	10 (19.6%)
Winter	10 (19.6%)
Cutaneous manifestations (Location of Purpura) [n (%)]	
Lower extremity	51 (100.0%)
Lower Extremity with Gluteal	19 (37.3%)
Upper extremity	18 (35.3%)
Widespread rash	17 (33.3%)
Trunk	8 (15.7%)
Facial	2 (3.9%)
Joint involvement [n (%)]	23 (45.1%)
Multiple joint	19 (82.6%)
Single joint	4 (17.4%)
Gastrointestinal involvement [n (%)]	21 (41.2%)
sonography findings [n (%)]	
enteric lymphadenopathy	14 (66.7%)
intraperitoneal fluid	8 (38.1%)
el wall thickening	5 (23.8%)
susception	3 (14.3%)
Renal-related findings [n (%)]	
Renal involvement	22 (43.1%)
Proteinuria	16 (31.4%)
Hematuria	13 (25.5%)
Hypertension	4 (7.8%)
Biopsy-Proven IgA Nephropathy	3 (5.9%)

Regarding geographic characteristics, most patients were from the following origins: Mardin (n=33, 64.7%), followed by Şırnak (n=9, 17.6%), Syria (n=4, 7.8%), Gaziantep (n=2, 3.9%), Kayseri (n=1, 2%), Diyarbakır (n=1, 2%), and Şanlıurfa (n=1, 2%). Disease onset occurred most frequently in autumn (n=19, 37.3%), followed by spring (n=12, 23.5%), while summer and winter accounted for 10 cases each (19.6%). No statistically significant association was observed between season of disease onset and GI. As the triggering factor for the IgAV, the distribution of infections was ordered as: Upper Respiratory Infections (n=26, 51%), Acute Gastrointestinal Infections (n=16, 31.4%), Acute Otitis Media (n=5, 9.8%) and other infections (n=4, 7.8%)

Clinical manifestations

Cutaneous involvement

Cutaneous manifestations were present in the entire cohort. All patients exhibited cutaneous manifestations involving the lower extremities. In addition to this data, patients with more than a single cutaneous involvement were numerically sorted as: the gluteal region in 19 patients (37.3%), upper extremities in 18 patients (35.3%), trunk in 8 patients (15.7%), and facial involvement in 2 patients (3.9%). A widespread rash pattern, defined as involvement beyond the classical dependent areas, was observed in 17 patients (33.3%).

Joint Involvement and Joint Localization

Joint involvement was present in 23 patients (45.1%). Among these patients, when considering the number of affected joints, multiple joints were affected in 19 patients (82.6%), while only one joint was affected in 4 patients (17.4%). The most common localization was ankle involvement (n=20; 90.9%). Wrist involvement was present in 3 patients (13.6%). In 4 patients, involvement of other joints (knee, elbow, arm) was observed (18.1%). No patient developed chronic arthritis or deformity during the follow-up period.

There was no statistically significant association between joint involvement and RI (p=0.775) or GI (p=0.775).

Gastrointestinal and renal involvement

Gastrointestinal symptoms occurred in 21 patients (41.2%), including abdominal pain, vomiting, and/or gastrointestinal bleeding. Fecal occult blood (FOB) positivity was detected in 11 patients (21.6%). FOB positivity was significantly associated with widespread rash (p=0.0039). Multivariable logistic regression demonstrated that widespread rash was independently associated with a nearly 10-fold increase in the risk of GI involvement (OR: 9.68, 95% CI: 2.12–44.18, p=0.003) and a 4.5-fold increase in the risk of RI (OR: 4.53, 95% CI: 1.13–18.14, p=0.033), after adjusting for potential confounders including age, sex, and CRP levels. In patients with gastrointestinal symptoms, abdominal ultrasound revealed mesenteric lymphadenopathy in 14 (66.6%), free fluid in 8 (38.1%), bowel wall thickening in 5 (23.8%), and intussusception in 3 (14.3%). Clinical resolution occurred in these three patients following intravenous hydration alone.

RI was identified in 22 patients (43.1%), based on urinalysis abnormalities and/or elevated spot urine protein/creatinine ratios. Among patients with RI, proteinuria was present in 16 patients (31.4%), hematuria in 13 patients (25.5%), and hypertension in 4 patients (7.8%). Biopsy-proven IgA nephropathy was diagnosed in 3 patients (5.9%) due to persistent urinary abnormalities. Proteinuria was recorded as a clinical severity category with the following distribution: normal (n=22), mild (n=22), moderate (n=5), and nephrotic (n=2).

Laboratory characteristics

Laboratory parameters demonstrated a predominantly inflammatory profile (Table 2). The median white blood cell count was 12.00 $\times 10^3/\mu L$ [8.85–13.65]. The mean platelet count was 402,549 ± 114,856/μL, consistent with reactive thrombocytosis in systemic inflammation. Leukocytosis was observed in 33 patients (64.7%), while thrombocytosis was present in 17 patients (33.3%) and anemia in 12(23.5%). Thrombocytosis was more frequent among patients with RI compared to those without (63.6% vs 34.5%, p=0.04). The median hemoglobin concentration was 12.5 g/dL [11.45–13.35], and anemia was identified in 12 patients (23.5%), among patients with FOB positivity (p=0.03).

Table II: Laboratory Characteristics of Patients with IgA Vasculitis

	Overall (n = 51)
Hematologic and inflammatory parameters	
White blood cell count ($\times 10^3/\mu L$) (Median [IQR Q1–Q3])	12.00 [8.85–13.65]
Platelet count ($\times 10^3/\mu L$) (Mean ± SD)	402.5 ± 114.9
Hemoglobin (g/dL) (Median [IQR Q1–Q3])	12.50 [11.45–13.35]
Leukocytosis [n (%)]	33 (64.7%)
Thrombocytosis [n (%)]	17 (33.3%)
Anemia [n (%)]	12 (23.5%)
Inflammatory markers	
C-reactive protein (mg/L) (Median [IQR Q1–Q3])	16.0 [7.25–32.50]
Erythrocyte sedimentation rate (mm/h) (Median [IQR Q1–Q3])	20 [12–29]
Renal and Gastrointestinal parameters	
Spot urine protein/creatinine ratio (mg/mg) (Median [IQR Q1–Q3])	0.24 [0.13–0.33]
Fecal occult blood positivity [n (%)]	11 (21.6%)

The median CRP level was 16.0 mg/L [IQR 7.25–32.5], while the median ESR was 20 mm/h [12–29]. Patients with FOB positivity had significantly higher median CRP levels compared to FOB-negative patients (28 vs 11 mg/L, p=0.01).

Complement levels

All patients had normal C3 and C4 levels.

Association between cutaneous distribution and systemic involvement

Cutaneous distribution patterns demonstrated significant associations with systemic involvement. Patients with widespread rash had a markedly higher frequency of GI compared to those with localized rash (76.5% vs 24.2%, p<0.001). Similarly, RI was

significantly more common among patients with widespread rash (70.6% vs 29.4%, p=0.012).

Trunk involvement was strongly associated with gastrointestinal manifestations, observed in 87.5% of patients with trunk involvement compared to 33.3% without (p=0.0067). Upper extremity involvement was also associated with both GI (66.7% vs 28.1%, p=0.019) and RI (66.7% vs 30.3%, p=0.027). FOB positivity was significantly more frequent among patients with widespread rash (64.7% vs 9.1%, p<0.001).

Treatment modalities and associated factors

Nonsteroidal anti-inflammatory drugs (NSAIDs) were administered to 39 patients (76.5%) systemic corticosteroids to 28 patients (54.9%) and angiotensin-converting enzyme inhibitors (ACEi) to 8 patients (15.7%).

Systemic corticosteroid use was significantly associated with hospitalization (67.9% vs 4.3%, p<0.001) and GI (59.3% vs 21.7%, p=0.017). Median CRP levels were higher in steroid-treated patients (24.85 vs 10.00 mg/L, p=0.017). Among patients with nephrotic proteinuria, immunosuppressive treatment has been administered in addition to systemic steroids.

ACEi use was strongly associated with RI (100% vs 32.6%, p<0.001) and higher spot urine protein/creatinine ratios (0.55 vs 0.19 mg/mg, p<0.001).

DISCUSSION

The current study presents a detailed evaluation of clinical phenotypes and laboratory predictors of systemic involvement in a cohort of 51 pediatric patients with IgAV in the Southeastern region of Türkiye. Our findings indicate that while the demographic and seasonal characteristics of the disease largely align with general trends in the literature, the anatomical distribution of purpura serves as a critical "visible biomarker" for systemic complications.

In our cohort, a slight male predominance was observed, which is consistent with the global trend of the disease. The observed male-to-female ratio in our study aligns with the literature, showing figures similar to those reported by Ağır et.al.[2] and slightly lower than the ratios observed in other cohorts[4]. International studies, such as those conducted in Italy, have reported a more pronounced male predominance[13]. The median age at diagnosis in our cohort was 6.67 years, which aligns closely with the peak incidence age reported in large-scale epidemiological data from Korea and other international reports[8,9]. Our patients were younger than the mean age observed in some other regional studies[14].

Seasonality remains a hallmark of IgAV. Our study found a distinct peak in autumn, followed by spring. The total ratio of autumn and spring, which can be considered transitional seasons, showed consistency with the findings of Ece et al.[14]. The autumn predominance is mirrored in the cohort reported by Ağır et.al.[2].

As noted by Reamy[5], upper respiratory infections precede up to 50% of cases closely in line with our findings. This mechanism, detailed by Trnka[15], is thought to result from abnormal IgA1 glycosylation triggered by mucosal antigen exposure.

While all of our patients had lower extremity involvement, we observed a high frequency of upper extremity distribution compared to the trunk/face involvement reported in the literature[1]. Joint involvement was seen in 45.1% of our patients, primarily affecting the ankles. This is lower than the incidence reported in some other Turkish and US cohorts[2,9]. Despite this lower frequency, our finding that only a minority had multiple joint involvement confirms the transient and typically oligoarticular nature of IgAV arthritis, which

does not result in chronic deformity, as emphasized in the literature[5].

GI symptoms were consistent with previous reports[4,16]. In our cohort, FOB positivity was significantly associated with a widespread rash. This statistical correlation suggests that more intense small-vessel vasculitis in the skin mirrors the severity of mucosal damage in the gut. The rates of hematuria and proteinuria in our study were similar to those obtained in nearby regions[14]. When RI is considered, our findings are closer to the rates reported in the Italian cohort[13]. This disparity may be due to the retrospective nature of some studies or differing definitions of hematuria. The severe proteinuria in our study was comparable to the ratio of nephrotic findings in other Turkish studies[14].

At the time of diagnosis, leukocytosis in our patients was present at a rate higher than in some studies, though similar to reports from northwestern Spain[17]. We found that thrombocytosis was significantly more frequent in patients with RI than in those without. This validates the findings of Akça et.al.[6], where thrombocytosis was identified as a primary risk factor for renal manifestations. Patients with FOB positivity had significantly higher CRP levels compared to FOB-negative cases. This association between higher acute-phase reactants and visceral involvement is also mentioned by Sönmez et.al.[12], who noted that patients with comorbid autoinflammatory backgrounds tend to exhibit even higher median CRP values and increased treatment requirements.

The strongest indicator of systemic severity in our study was cutaneous distribution. Patients with a widespread rash had a markedly higher frequency of GI involvement and RI. This statistically significant association suggests that the anatomical extensiveness of purpura serves as a "visible biomarker" for the intensity of the underlying vasculitis. Specifically, upper extremity cutaneous involvement was significantly associated with renal findings. Borakay and Yiğit[4] noted that prognosis depends on RI; our data supports this notion and the relevant studies in the literature[13].

We found that steroid use was strongly associated with hospitalization and high CRP levels. This clinical decision-making aligns with European consensus-based recommendations, which suggest corticosteroids for severe GI and renal manifestations despite their lack of efficacy in preventing nephritis onset, as also noted in other studies[18,19]. ACE-inhibitor (ACEi) use in our cohort was strictly associated with RI and higher spot urine protein/creatinine ratios. This reflects the nephroprotective strategies advocated and supported by the results of Söylemezoğlu et.al.[18,20].

Limitations of the Study

The limitations of this study, include its retrospective design, single-center setting, and relatively small sample size, which may affect the generalizability of the findings. Additionally, although multivariable analysis was performed, the number of included variables was limited by the sample size. The potential for referral bias at our tertiary center should also be considered.

As a strength of our study, being the first pediatric rheumatology center in the city of Mardin, it contributes valuable regional data to the existing literature on pediatric IgAV.

CONCLUSION

This study provides a comprehensive characterization of pediatric IgAV in Southeastern Türkiye, identifying key clinical and laboratory predictors of systemic involvement. Our findings demonstrate that the anatomical distribution and extensiveness of cutaneous purpura serve as a "visible biomarker"; specifically, widespread rash and upper extremity involvement are potent

indicators of both gastrointestinal and renal involvement.

The anatomical distribution and extensiveness of purpura in pediatric IgAV serve as simple yet effective clinical tools for predicting systemic complications. Laboratory parameters such as thrombocytosis and elevated CRP provide additional prognostic insights, supporting early risk stratification. The regional data obtained from the Mardin area contributes valuable insights that can help optimize the clinical management of pediatric IgAV in populations with similar demographic profiles.

Ethics Committee Approval: The study protocol adhered to the principles of the Declaration of Helsinki and was approved by the ethics committee of Mardin Artuklu University (Number: 243407).

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

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REFERENCES

1. Koçak M, Büyükkaragöz B, Kuraş Can Y, et al. The Epidemiological, Clinical And Laboratory Features Of 91 Children with Henoch-Schönlein Purpura. Abant Medical Journal. 2015;4(2):134–40.

2. Ağır MA, Güngörer V, Yorulmaz A, Arslan Ş. İmmünglobulin A Vasküliti Tanısı Konulmuş Pediyatrik Hastaların Değerlendirilmesi: Tek Merkez Deneyimi. Journal of General Medicine/Genel Tıp Dergisi. 2021;31(4).

3. Ozen S, Pistorio A, Iusan SM, et al. EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. Ann Rheum Dis. 2010 May;69(5):798–806.

4. Borakay D, Yiğit Ö. Henoch-Schönlein Purpurali Çocukların Sistem Tutulumlarının Klinik Değerlendirilmesi. Acta Medica Nicomedia. 2024 Oct 27;7(3):252–6.

5. Reamy B V, Servey JT, Williams PM. Henoch-Schönlein Purpura (IgA Vasculitis): Rapid Evidence Review. Am Fam Physician [Internet]. 2020 Aug 15;102(4):229–33. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32803924

6. Akça Ü, Akça G, Nalcıoğlu H, Genç G, Özkaya O. Henoch Schönlein purpuralı hastalarda epidemiyolojik, klinik ve laboratuvar bulguların değerlendirilmesi. Turkish Journal of Family Practice. 2020 Jun 15;24(2):87–94.

7. Yang YH, Yu HH, Chiang BL. The diagnosis and classification of Henoch-Schönlein purpura: An updated review. Vol. 13, Autoimmunity Reviews. Elsevier; 2014. p. 355–8.

8. Hwang HH, Lim IS, Choi BS, Yi DY. Analysis of seasonal tendencies in pediatric Henoch–Schönlein purpura and comparison with outbreak of infectious diseases. Medicine (United States). 2018 Sep 1;97(36).

9. Saulsbury FT. Henoch-Schönlein Purpura in Children: Report of 100 Patients and Review of the Literature. Medicine [Internet]. 1999 Nov;78(6):395–409. Available from: http://journals.lww.com/00005792-199911000-00005

10. Açarı C, Bayram M, Yıldız G, Kavukçu S, Soylu A. Effect of MEFV variants on the presentation and clinical course of Henoch-Schonlein purpura in children? Dokuz Eylül Üniversitesi Tıp Fakültesi Dergisi. 2023 Jan 27;36(3):245–55.

11. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. Kidney Int [Internet]. 2021 Oct;100(4S):S1–276. Available from: http://www.ncbi.nlm.nih.gov/pubmed/34556256

12. Sönmez HE, Batu ED, Bilginer Y. İmmünoglobülin A vasküliti (Henoch-Schönlein purpura) tanısı olan çocuk hastalarda artmış ailevi Akdeniz ateşi sıklığı. Güncel Pediatri. 2018;16(1):35–46.

13. Trapani S, Micheli A, Grisolia F, et al. Henoch Schonlein purpura in childhood: Epidemiological and clinical analysis of 150 cases over a 5-year period and review of literature. Semin Arthritis Rheum. 2005;35(3):143–53.

14. Ece A, Yolbaş İ, Balık H, et al. Henoch-Schönlein purpura in childhood: Review of 214 patients. Journal of Clinical and Experimental Investigations. 2012 Dec 12;3(1).

15. Trnka P. Henoch-Schönlein purpura in children. Vol. 49, Journal of Paediatrics and Child Health. 2013. p. 995–1003.

16. Uçak K, Şahin N, Sönmez HE. Çocukluk çağı vasküliti ile takipli hastaların demografik verileri ve klinik izlemleri. Acta Medica Nicomedia [Internet]. 2024;7(2):214–8. Available from: https://dergipark.org.tr/tr/pub/actamednicomedia/article/1390118

17. Calviño MC, Llorca J, García-Porrúa C, et al. Henoch-Schönlein purpura in children from northwestern Spain: a 20-year epidemiologic and clinical study. Medicine [Internet]. 2001 Sep;80(5):279–90. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11552081

18. Ozen S, Marks SD, Brogan P, et al. European consensus-based recommendations for diagnosis and treatment of immunoglobulin A vasculitis-the SHARE initiative. Rheumatology (United Kingdom). 2019 Sep 1;58(9):1607–16.

19. Davin JC, Coppo R. Henoch-Schönlein purpura nephritis in children. Vol. 10, Nature Reviews Nephrology. Nature Publishing Group; 2014. p. 563–73.

20. Soylemezoglu O, Ozkaya O, Ozen S, et al. Henoch-schönlein nephritis: A nationwide study. Nephron Clin Pract. 2009 Jun;112(3).