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Original Article

Predictors of pediatric Henoch-Schönlein purpura recurrence

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Abstract

Background Henoch-Schönlein purpura (HSP) is the most common systemic vasculitis in children and is usually self-limited with a favorable prognosis. However, recurrence in children is associated with a poorer prognosis, i.e., a higher risk of progressing to chronic kidney disease (CKD) as a long-term complication. In Indonesia, the recurrence rate of HSP and its predictors in children have not been well established .

Objective To estimate the incidence of recurrent HSP and determine its predictors in children at Dr. Cipto Mangunkusumo National General Hospital (RSCM).

Methods A retrospective cohort review of medical records followed children aged <18 years at RSCM for 6 months after HSP diagnosis based on the the *European League Against Rheumatism* (EULAR)/ P*aediatric Rheumatology European Society* (PRESS)/ *Paediatric Rheumatology International Trials Organization* (PRINTO) criteria. Multivariate, Cox logistic regression, and Kaplan-Meier analyses were performed.

Results This study included 116 children aged 2-17 years with HSP. Twenty-six (22.4%) of the subjects experienced recurrence, with an incidence of 3.56 per 100,000 person-years. The only statistically significant predictor for recurrence was the presence of infection after the first episode of HSP (HR 11.301; 95%CI 4.327 to 29.519; P<0.001). The cumulative survival of subjects with infection for recurrence over 6 months was 51%, with mean 5.3 months survival duration (95%CI 4.76 to 5.99; P< 0.0001). Chronic kidney disease, a long-term complication of HSP, was noted in 22 (19%) participants.

Conclusion Recurrence of HSP was observed in 22.4% of our subjects within 6 months follow up. However, subjects with a history of infection after their first episode of HSP resolution should be notified about the possibility of recurrence. Chronic kidney disease occurred in 22 participants (19%), possibly becoming a long-term complication of HSP. **[Paediatr Indones. 2025;65:307-15; DOI: https://doi.org/10.14238/pi65.4.2025.307-15].**

Keywords: *children; chronic kidney disease; Henoch-Schönlein purpura; predictor of recurrence; recurrence*

Henoch-Schönlein purpura (HSP) is the prevailing form of systemic vasculitis in children. It is distinguished by the occurrence of palpable and non-thrombocytopenic purpura, primarily in the lower extremities. It may also be accompanied by abdominal pain or gastrointestinal bleeding, arthritis or joint pain, and/or nephritis.[1] The prevalence of HSP in children is approximately 10-20 per 100,000 individuals, with the highest occurrence observed in children aged 4-6 years.[2-4] The current prevalence of HSP in Indonesia has not been determined. Nevertheless, data obtained from the Department of Child Health, Universitas Indonesia and Dr. Cipto Mangunkusumo National General Hospital (RSCM) indicate a consistent rise in the number of new cases of HSP within a decade, with an increase from 23 cases between 1998 and 2003 to 71 cases between 2009 and 2012.[5]

Henoch-Schönlein purpura is often a self-limiting condition with a favorable prognosis. However, it commonly recurs in children, particularly during the initial two years following the resolution of

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the first episode of HSP.[6,7] Recurrence is characterized by the reappearance of new symptoms of HSP following a period of at least one month without any signs or symptoms.[6] The global recurrence rate of HSP is estimated to range from 2.7% to 66.2%.[7,8] Several factors that may indicate the likelihood of recurrence have been discovered, such as extensive joint and gastrointestinal involvement at the time of diagnosis, a history of infection, treatment with steroids, and renal symptoms. However, the evidence regarding these predictors is inconclusive. The recurrence of HSP is linked to an increased risk of a chronic kidney disease (CKD), which is a long-term and irreversible complication of HSP, and has a worse prognosis.[7-9] Moreover, there is limited knowledge about the frequency and factors that contribute to HSP recurrence in Indonesia. This information is crucial to determine the appropriate duration of follow-up. We aimed to estimate the incidence, onset, and characteristics of recurrent HSP in children, and determine the predictors of recurrence. Subsequently, we determined an appropriate length of time for follow-up in children with HSP.

Methods

This retrospective cohort study was approved by the Medical Ethics Committee at Universitas Indonesia in January 2022. A retrospective assessment of medical records was conducted for 116 eligible patients diagnosed with HSP at RSCM from January 2014 to October 2021. Subjects ranged in age from 1 month to <18 years and had been diagnosed with HSP for the first time. Pediatric allergy-immunologists diagnosed HSP using the criteria established by the *European League Against Rheumatism* (EULAR)[10] and the *Paediatric Rheumatology European Society* (PRES). These criteria were validated by the *Paediatric Rheumatology International Trials Organization* (PRINTO) in 2008 (EULAR/PRES/PRINTO 2008).[10] Patients with incomplete medical records (more than 20% of needed data missing) were excluded from the study. Additionally, children who had previously been diagnosed with HSP prior to coming to RSCM, but whose disease progression could not be traced, were also excluded.

The initial data obtained upon subject enrollment comprised age, gender, nutritional status, date of first HSP diagnosis, clinical symptoms of HSP, laboratory findings, and prior use of systemic corticosteroid treatment. However, not all subjects underwent supplementary procedures, including comprehensive peripheral blood tests, serum IgA level assessments, and kidney biopsies. After HSP was diagnosed by pediatric allergy-immunologists, subjects were monitored for a period of 6 months. During the follow-up, the time it took for the disease to resolve, post-HSP infection history, recurrence of HSP, and any long-term complications related to chronic renal disease were noted. We examined six potential indicators of HSP recurrence: age, gender, history of atopy, renal manifestation during the initial HSP episode, corticosteroid administration for more than 10 days during the first HSP episode, and history of infection following the initial HSP episode. The *Statistical Package for the Social Sciences* (SPSS) *version 26.0* (*SPSS Inc*, USA) software was utilized for descriptive and analytical data analysis. Statistical studies were conducted using multivariate Cox logistic regression and Kaplan-Meier analyses.

Results

Of the 176 patient records evaluated for inclusion, 21 had incomplete records, 31 had diagnoses other than HSP, and 8 presented with recurrent HSP. However, it was not possible to retrieve the initial disease history and initial laboratory examination for these individuals. Therefore, the total number of subjects evaluated was 116. Subjects had a balanced gender distribution, with an mean age of 9 years at the time of first HSP diagnosis. The youngest age at which HSP was initially diagnosed was 4 months, while the oldest age was 17 years. Most participants exhibited good nutritional status (75.9%), while all participants had normal stature.

Table 1 provides an overview of the characteristics of participants. Recurrence was observed in 26 individuals (22.4%), with an incidence rate of 3.56 per 100,000 person-years. There were more males (57.7%) with recurrence than females. The predominant demographics of participants experiencing recurrence were age 7-12 years (57.7%), with the recurrence onset at >3-6 months following the resolution of the initial

episode of HSP (57.6%). Five participants (4.4%) were lost to follow-up over the 6-month monitoring period; their recurrence status was unknown.

The clinical and laboratory manifestations of subjects diagnosed with HSP during the initial episode and subsequent recurrences are presented in **Table 2**. All subjects reported dermatological symptoms, including palpable purpura, and gastrointestinal symptoms, i.e., acute abdominal pain, as the predominant clinical manifestations. Hematuria and proteinuria were the main clinical symptoms of nephritis. Six (13%) subjects who suffered Henoch-Schönlein nephritis (HSN) underwent kidney biopsy; it was the sole target organ biopsy executed in our investigation. Four (66.7%) subjects who underwent kidney biopsy had histological characteristics of diffuse proliferative mesangial glomerulonephritis, whereas two subjects (33.3%) presented with focal proliferative mesangial glomerulonephritis. Immunohistochemical examination demonstrated IgA deposits in the glomerular mesangial cells of all patients who underwent kidney biopsy.

In total, 82 (70.7%) subjects received corticosteroid medication during their initial episode of HSP. The predominant indications for administration were renal symptoms and acute stomach pain. These 82 subjects received early corticosteroid medication (<14 days post-onset), both during their initial episode of HSP and upon recurrence. The majority of subjects received corticosteroids for more than 11 days; HSP resolved during a period beyond 2-4 weeks (**Table 3**).

Among the six possible predictors for recurrence of HSP, only one was statistically significant: a history of infection occurring post-initial HSP episode (HR 11.301; 95%CI 4.33 to 29.52; P<0.001). Among participants who contracted infection, the cumulative survival probability over 6 months was 51.1%, with a mean survival duration of 5.3 months (95%CI 4.76 to 5.99; P<0.0001). This indicates that 51.1% of participants remained free from HSP recurrence after 5.3 months. In contrast, subjects without infection had a cumulative survival rate of 94%, accompanied by a mean survival duration of 6.8 months (95%CI 6.7 to 7.1). This indicates that in subjects with no infection, 94% of participants remained free from recurrence after 6.8 months. Consequently, there was a 42.9% drop in cumulative survival probability between individuals without a history of infection and those with such a history (**Figure 1**).

Overall, the cumulative survival probability of HSP subjects for recurrence decreased over time. The decline began in the initial month (96%), with 5 (4.3%) patients having recurrence. At the conclusion of the observation, survival diminished by 2.6-7.7% monthly, along with an escalating number of participants experiencing recurrence. The subsequent data represent the cumulative survival probability of patients with HSP throughout time (**Table 4**). The cumulative survival probability for 6 months was 77.6%(**Figure 2**).

Chronic kidney disease (CKD), a long-term complication, was found in 22 subjects (19%),

Table 1. Baseline characteristics of subjects

Characteristics	(N=116)
Gender, n (%)	
Male	58 (50)
Female	58 (50)
Age at the first HSP diagnosis, n(%)	
1 month-6 years	35 (30.2)
7-12 years	49 (42.2)
13-18 years	32 (27.6)
Mean (SD), years	9 (3.4)
History of atopy, n (%)	
Yes	110 (94.8)
No	6 (5.2)
History of infection following the initial HSP episode, n (%)	
Yes	45 (38.8)
No	71 (61.2)
Type of infection, n (%)	
Airway infection	40 (89)
Urinary tract	3 (7)
Oral and/or dental	2 (4)
Recurrence of HSP following the initial HSP episode, n (%)	
Yes	26 (22.4)
No	85 (73.2)
Unknown (lost to follow up)	5 (4.4)
Gender of subjects with recurring HSP, n (%)	
Male	15 (57.7)
Female	11 (42.3)
Age at the diagnosis of recurrent HSP, n(%)	
1 month-6 years	5 (19.1)
7-12 years	15 (57.7)
13-18 years	6 (23.2)
Mean (SD), years	10 (3.9)
Onset of recurrent HSP following the initial HSP episode, n (%)	
1-3 months	11 (42.3)
>3-6 months	15 (57.7)

Table 2. Clinical and laboratory manifestations in subjects

Clinical manifestations	Initial episode HSP (n=116)	Recurrent HSP (n=26)
Skin, n (%)		
Palpable purpura or petechiae	116 (100)	25 (96.2)
Persistent purpura	10 (8.6)	4 (15.3)
No skin manifestation	0 (0)	1 (0.8)
Joints, n (%)		
Mild-to-moderate arthralgia in lower extremity joints	43 (37.1)	6 (23.1)
Mild-to-moderate arthralgia in other joints	3 (2.6)	0 (0)
No joint manifestation	70 (60.3)	20 (76.9)
Gastrointestinal tract, n (%)		
Acute abdominal pain, colic, and nausea/vomiting	113 (97.4)	21 (80.7)
Acute abdominal pain, colic, and GI tract bleeding	3 (2.6)	0 (0)
No GI tract manifestation	0 (0)	5 (19.2)
Renal, n (%)		
Yes	46 (39.7)	14 (53.8)
Hematuria	15 (12.9)	3 (11.5)
Proteinuria	3 (2.6)	1 (3.8)
Hematuria and proteinuria	22 (18.9)	7 (26.9)
Acute nephritic syndrome	6 (5.3)	3 (11.5)
No renal manifestation	70 (60.3)	12 (46.3)
Thrombocytes, n (%)		
Normal	27 (23.4)	10 (38.5)
Thrombocytosis	59 (50.8)	16 (61.5)
No data	30 (25.8)	0 (0)
Leukocytes, n (%)		
Normal	33 (28.4)	10 (38.5)
Leukocytosis	32 (27.5)	16 (62.5)
No data	51 (44.1)	0 (0)
Serum IgA, n (%)		
Normal	10 (8.6)	5 (19.2)
Elevated	2 (1.7)	0 (0)
No data	104 (89.7)	21 (80.7)

consisting of 10 subjects who experienced recurrence and 12 subjects who did not. Chronic kidney disease complications were unknown in 5 (4.3%) subjects who were lost to follow-up. According to the *Kidney Disease: Improving Global Outcome* (KDIGO) CKD Work Group's criteria in 2012,the degree of CKD found in this study ranged from grade 1 (G1), with a glomerular filtration rate (GFR) > 90 ml/min/1.73 m^2, G2 (GFR 60-89 mL/min/1.73 m^2), to G3a (GFR 45-59 mL/min/1.73 m^2) (**Figure 3**).11The majority of subjects who experienced CKD at the end of monitoring were at stage G2, in subjects with and without recurrence.

Discussion

Most subjects experiencing recurrent HSP fell within the 7-12 years age range (57.7%), with an mean age of 10 (SD 3.9) years. Subjects with recurrence had a higher mean age than subjects who did not [9 (SD 3.4 years]. The number of male and female subjects was balanced. However, more recurrent subjects were male (57.7%) than female (42.3%). The role of gender in the inflammatory process is linked to the development of immune responses in humans. In prepubertal children, the immune response of inflammatory cytokines is higher in boys than in girls.[8,12] All subjects had purpura and/or palpable petechiae, primarily in the lower extremities. Acute and diffuse abdominal pain, accompanied by nausea/vomiting, was the most common gastrointestinal symptom found in 80.7% of subjects experiencing recurrence. Mild-to-moderate arthralgia in the lower extremities was found in 23.1% of subjects with recurrence. In recurrent cases, the skin, gastrointestinal, and joint symptoms were similar to those seen in the first HSP episode. This finding was

Table 3. Corticosteroid treatment profile and response

Treatment profiles	Initial episode of HSP (n=116)	Recurrent HSP (n=26)
Treatment indications, n (%)		
Renal manifestations	46 (56)	14 (53.8)
Mild-to-moderate abdominal pain	16 (19.5)	6 (23.1)
Severe abdominal pain	11 (13.4)	2 (7.6)
Mild-to-moderate arthralgia	6 (7.3)	4 (15.4)
GI tract bleeding	3 (3.7)	0 (0)
Type of corticosteroid, n (%)		
Methylprednisolone	72 (87.9)	23 (88.5)
Prednisone	10 (12.1)	3 (11.5)
Treatment initiation, n (%)		
Not administered	34 (29.3)	1 (3.2)
Early (<14 days)	82 (70.7)	25 (96.8)
Late (>14 days)	0 (0)	0 (0)
Treatment duration, n (%)		
Not administered	34 (29.3)	0 (0)
<10 days	20 (17.3)	1 (3.2)
>11 days	62 (53.4)	25 (96.8)
Duration of HSP resolution, n (%)		
1-2 weeks	56 (48.3)	4 (15.4)
>2 to 4 weeks	42 (36.2)	16 (61.5)
>4 weeks	11 (9.5)	6 (23.1)
Unresolved HSP at the end of follow-up	7 (6.0)	0 (0)

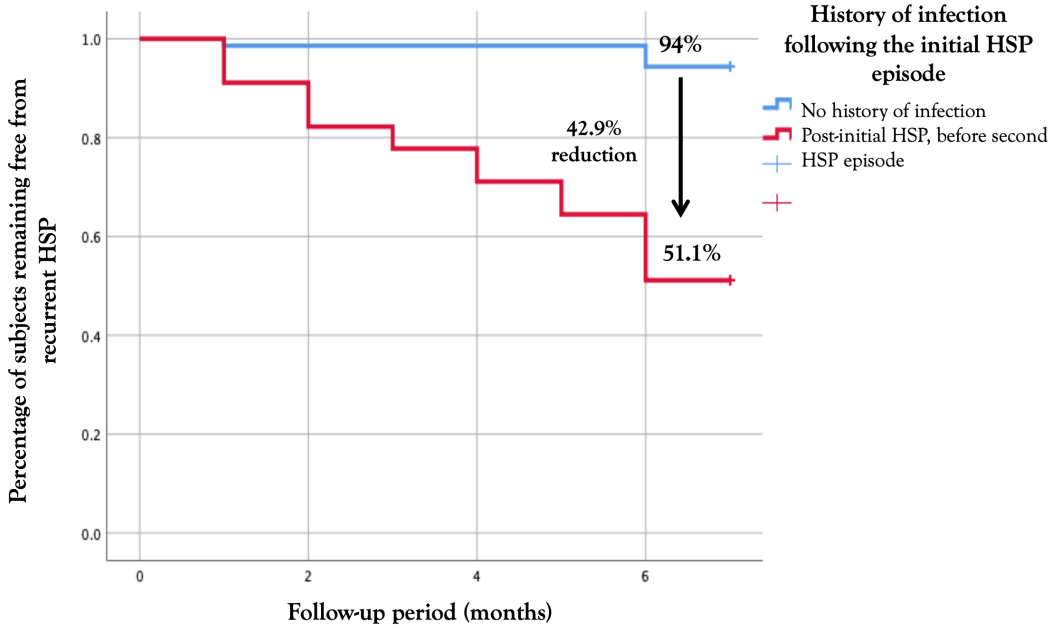

Figure 1. Kaplan-Meier curve of history of infection against cumulative survival during 6 months

in line with the results of previous studies that found a general similarity between the clinical symptoms during recurrence and the first HSP episode.[7-9,12]

Subjects experiencing recurrence (53.8%) were more likely to have HSN than those experiencing the first HSP episode (39.7%). In subjects with recurrence, 26.9% had hematuria and proteinuria (**Table 2**). Microscopic or gross hematuria was reported to be the most common manifestation of HSN.[13,14] The recurrence of HSP is a significant predictor of

Table 4. Cumulative survival probability of HSP subjects throughout time

Time	Number of subjects with recurrent HSP	Cumulative survival (%)	95%CI
1 month	5	95.7	89.9 to 98.1
2 months	4	92.2	85.6 to 96.8
3 months	2	90.5	83.5 to 94.6
4 months	3	87.9	80.5 to 92.6
5 months	3	85.3	77.5 to 90.6
6 months	9	77.6	68.9 to 84.1

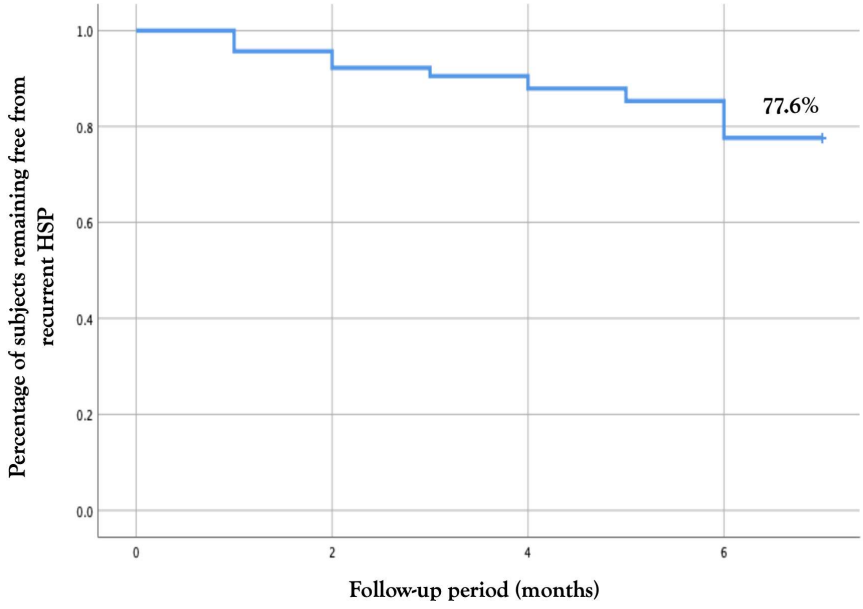

Figure 2. Kaplan-Meier survival curve during 6 months of follow-up

nephritis, which is postulated POSSIBLY due to recurrence being associated with a more severe degree of disease.[13,15] Corticosteroid therapy was administered to 96.8% of subjects experiencing recurrence. In subjects experiencing recurrence, the indications for administration and type of corticosteroids were the same as during the first HSP episode. The majority of subjects with recurrence (53.8%) received corticosteroids due to kidney involvement and acute abdominal pain (23.1%). We did not evaluate the range of corticosteroid doses given. A study found that methylprednisolone doses at RSCM were between 0.2 and 3 mg/kg/day, prednisone doses were between 0.5 and 2 mg/kg/day, and triamcinolone doses were 0.5 mg/kg/day.[16] According to the 2012 KDIGO criteria, 22 (19%) subjects experienced CKD at the end of the monitoring period of this study. The incidence of CKD was higher in subjects with recurrence (38.4%) compared to subjects without recurrence (13.3%). In general, the majority of subjects (54.5%) experienced CKD stage G2 (GFR 60-89 mL/min/1.73 m^2). Subjects experiencing recurrence showed similar results, with 70% exhibiting CKD stage 2. The results obtained in this study were generally more severe than reported by previous studies conducted at RSCM. A study found that 40% of HSP cases with kidney involvement that progressed to CKD were classified as CKD G1 (GFR >90 mL/min/1.73 m2).[16] Our study's more severe CKD degree profile could have stemmed from the association between HSP recurrence and more severe disease, thereby increasing the likelihood of CKD progression.[9-12,17]

Recurrent HSP occurred in 22.4% of subjects, with an incidence rate of 3.56 per 100,000 person-years. The recurrence rate seen in this study aligned with the extensive range of HSP recurrence incidence

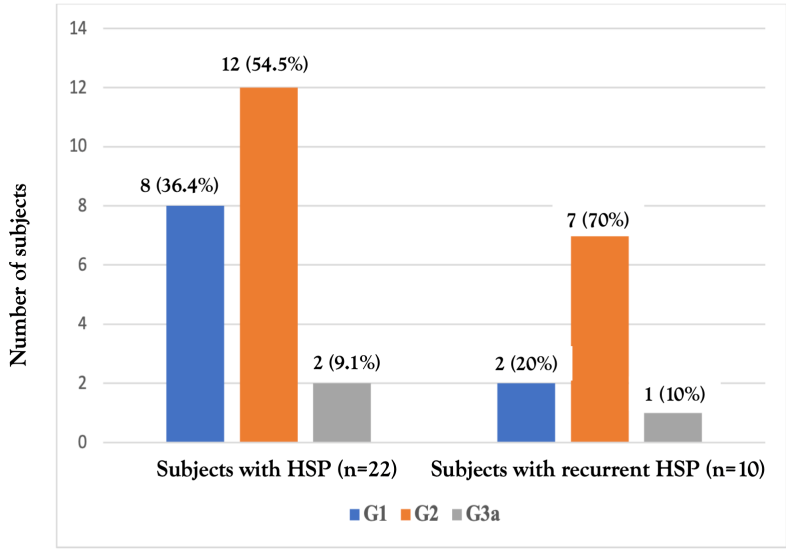

Figure 3. Stages of CKD in study subjects who developed CKD

reported in the literature, which spanned from 2.7-51.7% and 2.7-66.2%.[13-15,18] The recurrence status of 5 (4.4%) individuals who were lost to follow-up during the 6-month monitoring period remains uncertain. We noted a greater recurrence rate than in another investigation at RSCM, which documented recurrence in 5 of 71 individuals (7%) utilizing a randomized study design.[5] The increased recurrence rate in our study may be attributed to the larger sample size and the retrospective cohort design with a 6-month observation period, facilitating the detection of more recurrence episodes. Our study revealed that participants sent to RSCM reported a higher recurrence rate than in earlier studies, namely 20 subjects (17.2%).

In our study, 57.6% of participants suffered a recurrence more than 3-6 months after the resolution of the initial episode of HSP. In our study, recurrence was later than the three preceding studies conducted in subtropical nations, which determined that the majority of recurrences transpired within 1-3 months following the resolution of the initial HSP episode.[10,12,18,19] The subtropical environment of these nations may hasten the recurrence reported in the three prior investigations, given the elevated incidence of HSP correlates with autumn and winter.[19-21] This may be related to viruses being more prevalent in the autumn and winter seasons. Viruses such as influenza, respiratory syncytial virus (RSV), rhinovirus, adenovirus, and norovirus are recognized for initiating the HSP immunopathogenesis cascade.[19-21] Our study revealed that the cumulative probability of recurrence-free state in our participants was decreasing in the sixth month of follow up, while the incidence of recurrent HSP was at its peak. The cumulative survival rate was 77.6%, which means 77.6% of the participants in our study remained free from HSP recurrence for a duration of up to 6 months.

The only statistically significant predictor of HSP recurrence was an infection history following the resolution of the initial HSP episode. A total of 45 individuals (38.8%) developed an infection following the resolution of the initial HSP episode prior to experiencing a recurrence. Only 51.1% of individuals with a history of infection were free from HSP recurrence for 5.3 months, whereas 94% of individuals without such a history experienced a cumulative recurrence-free survival of 6.8 months. The predominant infection identified was acute respiratory system infection (89%), followed by urinary tract infection (7%) and oral-dental infection (4%). Respiratory tract infection was the predominant infection linked with HSP, occurring in either the first episode or recurrence, with Group A Streptococcus being the most commonly detected pathogen.[22,23] Pathogen clearance explains the correlation between the history of infection, especially streptococcus infection, and the frequency of HSP recurrence.[23-25]

A reduced likelihood of recurrence is associated with effective pathogen clearance in streptococcal infections. This occurs due to a reduced number of microorganisms that initiate inflammatory reactions, hence, triggering the HSP pathogenesis cascade once more.[25]

This is the first study in Indonesia to examine the frequency, clinical features, and predictors of HSP recurrence in children. The findings derived from this study are expected to assist clinicians in managing HSP, enhance knowledge of recurrence potential, and serve as a foundation for referral decisions in cases of recurrence. Our findings can be used to inform referral health centers' corticosteroid treatment, renal function monitoring, and recognition of CKD as a long-term HSP complication. Furthermore, all subjects were included simultaneously at the initial diagnosis of HSP (cohort inception). The main weaknesses of this study were its retrospective method, taking into account the limited number of HSP cases. In addition, not all subjects underwent supplementary procedures, including comprehensive peripheral blood tests, serum IgA level assessments, and kidney biopsies. Consequently, the characteristics of the findings from these examinations may have been underestimated. A further drawback was the 5 (4.3%) participants lost to follow-up during the 6-month monitoring period, potentially leading to migration bias that may have influenced the incidence of recurrent HSP.

In conclusion, we observed a recurrence incidence of 22.4% within six months of the initial episode. Infection history, particularly acute respiratory infections following disease resolution, emerged as the sole significant predictor of recurrence. The clinical manifestations of recurrent episodes closely paralleled those of the initial presentation. These findings highlight the necessity for post-HSP resolution monitoring, especially for infectious triggers, to mitigate recurrence risk. This study contributes novel epidemiological insights into HSP recurrence in an Indonesian setting and highlights the need for extended prospective studies to further delineate long-term outcomes.

Conflict of interest

None declared.

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References

1. Jennette JC, Falk RJ, Bacon PA, Basu N, Cid MC, Ferrario F, *et al.* 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculities. Arthritis Rheum. 2013;65:1-11. DOI: https://doi.org/10.1002/art.37715
2. Trnka P. Henoch-Schönlein purpura in children. J Paediatr Child Health. 2013;49:995-1003. DOI: https://doi.org/10.1111/jpc.12403
3. Piram M, Mahrs A. Epidemiology of immunoglobulin A vasculities (Henoch-Schönlein): current state of knowledge. Curr Opin Rheumatol. 2013;25:171-5. DOI: https://doi.org/10.1097/BOR.0b013e32835d8e2a
4. Gardner-Medwin JM. Dolezalova P, Cummins C, Southwood TR. Incidence of Henoch-Schönlein purpura, Kawasaki disease and rare vasculities in children of ethnic origins. Lancet. 2002;360:1197-201. DOI: https://doi.org/10.1016/S0140-6736(02)11279-7
5. Sugianti I, Akib AAP, Soedjatmiko. Karakteristik purpura Henoch-Schönlein pada anak di Rumah Sakit Cipto Mangunkusumo. Sari Pediatri. 2014;16:128-33. DOI: https://doi.org//10.14238/sp16.2.2014.128-35
6. Alfredo CS, Nunes NA, Len CA, Barbosa CMP, Terreri MTRA, Hilario MCOE. Henoch-Schönlein purpura: recurrence and chronicity. J Pediatr (Rio J). 2007;83:177-80. DOI: https://doi.org/10.2223/JPED.1595
7. Prais D, Amir J, Nussinovitch M. Recurrent Henoch-Schönlein purpura in children. J Clin Rheumatol. 2007;13:25-8. DOI: https://doi.org/10.1097/01.rhu.0000255692.46165.19
8. Calvo-Rio V, Hernandez JL, Ortiz-Sanjuan F, Loricera J, Palmou-Fontana N, Gonzalez-Vela MC, *et al.* Relapses in patients with Henoch-Schonleon purpura: analysis of 417

patients from a single center. Medicine. 2016;95:e4217. DOI: https://doi.org/10.1097/MD.0000000000004217

9. Lei WT, Tsai PL, Chu SH, Kao YH, Lin CY, Fang LC, *et al.* Incidence and risk factors for recurrent Henoch-Schonlein purpura in children from a 16-year nationwide database. Ped Rheumatol. 2018;16:25. DOI: https://doi.org/10.1186/s12969-018-0247-8
10. Ruperto N, Ozen S, Pistorio A, Dolezalova P, Brogan P, Cabral DA, *et al.*; Paediatric Rheumatology International Trials Organisation (PRINTO). EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood poluarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part I: overall methodology and clinical characterisation. Am Rheum Dis. 2010;69:790-7. DOI: https://doi.org/10.1136/ard.2009.116624
11. Levey AS, Eckardt KU, Dorman NM, Christiansen SL, Hoorn EJ, Ingelfinger JR, *et al.* Nomenclature for kidney function and disease: report of a kidney disease: Improving Global Outcomes (KDIGO) Consensus Conference. Kidney Int. 2020;97:1117-29. DOI: https://doi.org/10.1016/j.kint.2020.02.010
12. Klein SL, Flanagan KL. Sex differences in immune responses. Nat Rev Immunol. 2016;16:626-38. DOI: https://doi.org/10.1038/nri.2016.90
13. Shin J, Park JM, Shin YH, Hwang DH, Kim JH, Lee JS. Predictive factors for nephritis, relapse, and significant proteinuria in childhood Henoch-Schönlein purpura. Scand J Rheumatol. 2006;35:56-60. DOI: https://doi.org/10.1080/03009740510026841
14. Kaku Y, Nohara K, Honda S. Renal involvement in Henoch-Schönlein purpura: a multivariate analysis of prognostic factors. Kidney Int. 1998;53:1755-9. DOI: https://doi.org/10.1046/j.1523-1755.1998.00915.x
15. Assadi F. Childhood Henoch-Schönlein nephritis. A multivariate analysis of clinical features and renal morphology at disease onset. Iran J Kidney Dis. 2009;3:17-21. DOI: https://doi.org/10.1088/2058-7058/8/6/5
16. Yanrismet Y, Hidayati EL, Munasir Z, Yuliarti K, Fahriyani A. Incidence and risk factors of nephritis in childhood Henoch-Schönlein purpura. Pediatr Indones. 2023;64:304-14. DOI: https://doi.org/10.14238/pi63.4.2023.304-14
17. Trapani S, Micheli A, Grisolia F, Resti M, Chiappini E, Falcini F, *et al.* Henoch- Schönlein purpura in childhood: epidemiological and clinical analysis of 150 cases over a 5-year period and review of literature. Semin Arthritis Rheum. 2005;35:143-9. DOI: https://doi.org/10.1016/j.semarthrit.2005.08.007
18. Ronkainen J, Koskimies O, Ala-Houhala M, Antikainen M, Merenmies J, Rajantie J, *et al.* Early prednisone therapy in Henoch-Schonlein purpura: a randomized, double-blind, placebo-controlled trial. J Pediatr. 2006;149:241-7. DOI: https://doi.org/10.1016/j.jpeds.2006.03.024
19. Chen AC, Lin CL, Shen TC, Li TC, Sung FC, Wei CC. Association between allergic diseases and risks of HSP and HSP nephritis: a population study. Pediatr Res. 2016;79:559-64. DOI: https://doi.org/10.1038/pr.2015.271
20. Oni L, Sampath S. Childhood IgA vasculitis (Henoch-Schönlein purpura) - advances and knowledge gaps. Front Pediatr. 2019;7:257. DOI: https://doi.org/10.3389/fped.2019.00257
21. Jung DY, Kwon YR, Yu MY, Namgoong MK. Clinical features and prognosis of Henoch-Schönlein purpura in children and adults: a 13-year retrospective study at a single centre. Child Kidney Dis. 2017;21:61-8. DOI: https://doi.org/10.3339/jkspn.2017.21.2.61
22. Yang YH, Chuang YH, Wang LC, Huang HY, Gershwin ME, Chiang BL. The immunobiology of Henoch-Schönlein purpura. Autoimm Rev. 2008;7:179-84. DOI: https://doi.org/10.1016/j.autrev.2007.11.012
23. Gonzalez-Gay MA, Calvino MC, Vazquez-Lopez ME, Garcia-Porrua C, Fernandez-Iglesias JL, Dierssen T, Llorca J. Implications of upper respiratory tract infections and drugs in the clinical of Henoch-Schönlein purpura in children. Clin Exp Rheumatol. 2004;22:781-4. PMID: 15638057.
24. Fan GZ, Li RX, Jiang Q, Niu MM, Qiu Z, Chen WX, *et al.* Streptococcal infection in childhood Henoch-Schönlein purpura: a 5-year retrospective study from a single tertiary medical center in China, 2015-2019. Pediatr Rheumatol. 2021;19:79. DOI: https://doi.org/10.1186/s12969-021-00569-3
25. Wang JJ, Xu Y, Liu FF, Wu Y, Samadli S, Wu YF, *et al*. Association of the infectious triggers with childhood Henoch-Schönlein purpura in Anhui Province, China. J Infect Public Health. 2020;13:110-7. DOI: https://doi.org/10.1016/j.jiph.2019.07.004