

Analysis of Factors Associated with the Incidence of Complications in Patients with Acute Post *Streptococcal* Glomerulonephritis

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ARTICLE INFO	ABSTRACT
<p>Keywords: complications of PSAGN; emergent hypertension; acute renal failure; congestive heart failure; encephalopathy; risk factors.</p>	<p><i>Post-streptococcal acute glomerulonephritis (PSAGN) is a form of glomerulonephritis that occurs after group A β-hemolytic Streptococcus infection and is one of the leading causes of renal impairment in children. Although the prognosis is generally good, some patients develop serious complications, such as emergent hypertension, acute renal failure (AKI), congestive heart failure (CHF), and encephalopathy. This study aims to analyze the factors associated with the incidence of complications in PSAGN patients at RSUD Ulin Banjarmasin. This study used a cross-sectional retrospective design with data from the medical records of PSAGN patients treated between January 2022 and September 2024. The results showed that emergency hypertension was significantly associated with edema and high serum creatinine levels. CHF was more common in patients with low albumin levels, while encephalopathy was found to correlate with excess nutritional status, proteinuria, and crisis hypertension. This study underscores the importance of close monitoring of PSAGN patients to detect early risk factors for complications and provide appropriate interventions to reduce morbidity.</i></p>

INTRODUCTION

Glomerulonephritis is an inflammation that causes changes in permeability, structure, and glomerular function.(Akteruzzaman et al., 2019) One of the most common kidney diseases affecting children in low- and middle-income countries is acute glomerulonephritis (AGN).(Zabara et al., 2023) Acute glomerulonephritis is an acute nephritic syndrome accompanied by sudden severe hematuria, proteinuria, oliguria, hypertension, edema, and renal insufficiency.(Zabara et al., 2023) The clinical course ranges from mild symptoms to severe disease such as anuria, hypertensive encephalopathy, and heart failure.(Ong, 2022) Serum creatinine returns to the previous *baseline* within four weeks, hematuria resolves within six months, and proteinuria falls during recovery.(Utari et al., 2024)

In children with acute glomerulonephritis, the prognosis is usually good. However, glomerulonephritis causes end-stage renal disease in those on hemodialysis with an incidence of 39% in 2000, according to data from the Indonesian Nephrology Association (INA).(Daya, 2023) The incidence of primary glomerulonephritis ranges from 0.2-2.5 per 100,000 people per year.(McGrogan et al., 2011) There is insufficient information on the epidemiology of glomerulonephritis in Indonesia. Each hypertension and renal center is committed to reporting their studies. One of the most common types of acute glomerulonephritis in children is Post *Streptococcal* Acute Glomerulonephritis (PSAGN).(Daya, 2023)With the manifestation of acute nephritic syndrome and the usual negative germ culture, a definite diagnosis of PSAGN can be made by performing supporting examinations such as microscopic hematuria, erythrocyte thoracic, proteinuria on urinalysis, increased ASTO, and decreased C3 levels.(Utari et al., 2024)

As many as 60-70% of PSAGN patients show signs of hypertension. The Shah study in Nepal, in 41 cases, showed 100% hypertension and edema of the face and limbs. The main causes of hypertension in children are renal parenchymal and renovascular diseases. *The National High Blood Pressure Education Program (NHBPEP)* issued guidelines to identify prehypertensive and hypertensive conditions in children and adolescents. Systolic or diastolic blood pressure that reaches the 95th percentile or above, measured at least three times, is known as hypertension.(Salim et al., 2021)Death from PSAGN is rare but complications can occur during the acute phase of glomerulonephritis such as *congestive heart failure*, emergent hypertension, encephalopathy and retinopathy.(Bhat et al., 2024; Ong, 2022), the researchers wanted to analyze the factors associated with the incidence of complications in patients with acute glomerulonephritis at ULIN Banjarmasin Hospital.

Previous research on Post-Streptococcal Acute Glomerulonephritis (PSAGN) has highlighted various factors influencing the development of complications in affected patients. Studies by (Ong, 2022) and (Sanni et al.,

2022) have demonstrated that PSAGN, although generally self-limiting, can lead to severe complications, such as acute kidney injury (AKI), emergent hypertension, and congestive heart failure (CHF), especially in pediatric populations. Similarly, research by (Bajracharya et al., 2024) identified edema and proteinuria as common predictors of PSAGN complications, noting that early identification of these symptoms could reduce the risk of adverse outcomes. Meanwhile, (Utari et al., 2024) focused on the progression of PSAGN to chronic glomerulonephritis, underscoring the importance of monitoring renal function post-recovery. While these studies offer valuable insights, most focus on the incidence and progression of PSAGN without a comprehensive analysis of the specific factors contributing to each complication.

Despite numerous studies investigating PSAGN and its complications, there remains a significant gap in understanding how specific clinical and laboratory factors influence the development of individual complications such as emergent hypertension, AKI, CHF, and encephalopathy. While prior research has identified general risk factors, few studies have dissected the relationship between distinct patient characteristics—such as nutritional status, proteinuria levels, and serum creatinine—and the incidence of specific complications. Additionally, there is a lack of region-specific data on PSAGN in Indonesia, particularly regarding pediatric populations treated in tertiary care centers. This gap limits the ability to tailor clinical management strategies to local patient demographics and health care settings.

This study provides a novel approach by systematically analyzing the association between various clinical and laboratory parameters and the incidence of specific complications in PSAGN patients at Ulin Banjarmasin Hospital. Unlike previous studies that often focus on general risk factors, this research delves into the nuanced relationships between variables such as serum creatinine levels, albumin concentration, nutritional status, and the occurrence of complications like emergent hypertension and encephalopathy. Moreover, the study emphasizes the Indonesian context, offering region-specific data that can enhance the understanding of PSAGN epidemiology in Southeast Asia. By identifying key predictive factors for severe complications, this research aims to contribute to early detection protocols and improve patient outcomes.

The primary objective of this study is to analyze the factors associated with the incidence of complications in PSAGN patients, focusing on emergent hypertension, acute kidney injury, congestive heart failure, and encephalopathy. By identifying these factors, the study aims to provide healthcare professionals with critical information to improve early detection and intervention strategies. The findings are expected to benefit both clinical practitioners and policymakers by informing the development of targeted management protocols for PSAGN, ultimately reducing morbidity rates. Additionally, the study offers valuable data for future research, serving as a reference point for epidemiological studies on pediatric nephrology in Indonesia and potentially guiding public health initiatives aimed at preventing streptococcal infections and their complications.

METHOD

Data analysis in this study was univariate analysis and bivariate analysis. Univariate analysis to determine the distribution of PSAGN patients based on patient profiles, clinical features, and laboratory examinations. Bivariate analysis to determine the relationship of each factor with the incidence of complications in PSAGN patients, using the Chi Square test, with a confidence degree (α) of 95% so that the p value of 0.05 means that the calculation results are meaningful or indicate a relationship between the independent variable and the dependent variable. If there is an *expected count* value that is less than 5, then the p value used is from *Fisher's Exact Test*. The study was conducted in September-December 2024 at Ulin Banjarmasin Hospital.

RESULTS AND DISCUSSION

Demographic characteristics of PSAGN patients at Ulin Banjarmasin Hospital

This study obtained 42 samples that met the inclusion criteria, data were taken from medical records from January 2022 to September 2024. Patients with PSAGN are mostly male with a total of 34 (81%) of 42 patients. The most common age group diagnosed with PSAGN 6-10 years included 17 (40.5%), in addition to the age group 1-5 years as much as 2 (4.8%), the age group 11-15 years as much as 17 (40.5%) and the age group 16-18 years amounted to 1 (2.4%). The lowest age is 5 years, the highest age is 17 years and the most age experiencing PSAGN is 8 years.

Characteristics	Frequency (n=42)	Percentage (%)
Age		
1-5 Years	2	4.8%
6-10 Years	22	52.4%
11-15 Years	17	40.5%
16-18 Years	1	2.4%
Gender		
Male	34	81%

Female	8	19%
Nutrition Status		
More nutrition	5	11.9%
Adequate nutrition	23	54.8%
Undernourishment	13	31%
Obesity	1	2.4%

Distribution of PSAGN Patients according to clinical features

In the clinical picture, 29 patients showed edema as the most common symptom (69%). In terms of blood pressure on admission, there were 15 (35.7%) grade 2 hypertension, 14 (33.3%) grade 1 hypertension, and 19% of patients presented with hypertensive crisis with 4 (9.5%) urgent hypertension and 4 (9.5%) emergent hypertension. Followed by 9 (21.4%) patients who came with *gross* hematuria, 8 (19%) with a history of pharyngitis, 7 (16.7%) with *oliguria*, 6 (14.3%) with shortness of breath, 6 (14.3%) headache, 5 (11.9%) with a history of pyoderma, 4 (9.5%) with seizures, 3 (7.1%) with vomiting, 1 (2.4%) with decreased consciousness.

Clinical Features	Frequency (n=42)	Percentage (%)
<i>Gross</i> Hematuria	9	21.4%
Blood Pressure		
No Hypertension	5	11.9%
Grade 1 Hypertension	14	33.3%
2nd Degree Hypertension	15	35.7%
Hypertension Crisis:		
Hypertension Urgency	3	7.1%
Hypertension Emergencies	5	11.9%
Decreased consciousness	1	2.4%
Shortness of breath	6	14.3%
Seizures	4	9.5%
Edem	29	69%
History of Pyoderma	5	11.9%
Headache	6	14.3%
History of Pharyngitis	8	19%
Vomiting	3	7.1%
<i>Oliguria</i>	7	16.7%

Distribution of PSAGN Patients according to laboratory examination results

The data showed that a 38 (90.5%) patients with hematuria and there were 34 (81%) proteinuria. Based on the ASTO examination, there were 33 (78.6%) positive patients and there were 35 (83.3%) decreased C3 examinations. Based on KDIGO, 4 (9.5%) had stage 3 AKI (failure).

Laboratory Examination	Frequency (n=42)	Percentage (%)
Hematuria		
Yes	38	90.5%
No	4	9.5%
Proteinuria		
Yes	34	81%
No	8	19%
ASTO		
Positive	33	78.6%
Negative	9	21.4%
AKI (KDIGO)		
No MMR	3	7.1%
Stage 1 (risk)	31	73.8%
Stage 2 (injury)	1	2.4%
Stage 3 (failure)	7	16.7%
C3		
Down	35	83.3%

Not Down	7	16.7%
Albumin		
Normal	18	42.9%
Down	24	57.1%

Bivariate analysis of the relationship between the incidence of hypertension in PSAGN patients

Bivariate analysis was conducted to evaluate factors associated with hypertension in PSAGN. The results of this study showed that, although clinical factors such as age, gender, and nutritional status, as well as other laboratory parameters such as hematuria, ASTO, creatinine, albumin, and C3 levels did not show a significant association with the incidence of hypertension in pediatric PSAGN patients, proteinuria was found to have a significant association with hypertension. Statistical analysis showed that higher levels of proteinuria were associated with an increased prevalence of hypertension in patients, with a p value = 0.05, indicating a statistically significant effect between proteinuria and hypertension in 29 (85.3%) pediatric PSAGN.

Clinical and Laboratory	Hypertension		p	OR		
	Yes	No				
Age						
<13 years old	25	75.1	7	21.9	1.000	0.893
≥13 year	8	80	2	20		
Gender						
Male	26	76.5	8	23.5	0.662	0.464
Female	4	87.5	1	12.5		
Nutrition Status						
Adequate and deficient nutrition	29	80.6	7	19.4	0.593	0.483
Overnutrition and obesity	4	66.7	2	33.3		
Hematuria						
Positive	32	82.1	7	17.9	0.111	9.143
Negative	1	33.3	2	66.7		
Edema						
Yes	25	86.2	4	13.8	0.107	3.906
No	8	61.5	5	38.5		
Proteinuria						
Positive	29	85.3	5	14.7	0.050*	0.172
Negative	4	50	4	50		
AKI						
Yes	31	79.5	8	20.5	0.525	1.938
No	2	66.7	1	33.3		
ASTO						
Positive	26	78.8	7	21.2	1.000	1.061
Negative	7	77.8	2	22.2		
C3						
Down	28	80	7	20	0.631	1.600
Normal and Rising	5	71.4	2	28.6		
Albumin						
Normal	12	66.7	6	33.3	0.139	0.286
Down	21	87.5	3	12.5		
Pharyngitis						
Yes	6	75	2	25	1.000	0.778
No	27	79.4	7	20.6		
Pyoderma						
Yes	4	80	1	20	1.000	1.103
No	29	78.4	8	21.6		

Bivariate analysis of the relationship between the incidence of hypertension urgency in PSAGN patients

Statistically there is no relationship between clinical factors and laboratory factors with the incidence of

hypertension urgency in PSAGN patients.

Clinical and Laboratory	Hypertension Urgency				p	OR
	Yes	%	No	%		
Age						
<13 years old	3	9.4	29	90.6	1.000	0.931
≥13 year	1	10	9	90		
Gender						
Male	3	8.8	31	91.2	1.000	0.677
Female	1	12.5	7	87.5		
Nutrition Status						
Adequate and deficient nutrition	4	11.1	32	88.9	1.000	1.125
Overnutrition and obesity	0	0	6	100		
Hematuria						
Positive	3	7.7	36	92.3	0.265	0.167
Negative	1	33.3	2	66.7		
Edema						
Yes	1	3.4	28	96.6	0.080	0.119
No	3	23.1	10	76.9		
Proteinuria						
Positive	3	8.8	31	91.2	1.000	1.476
Negative	1	12.5	7	87.5		
AKI						
Yes	1	14.3	6	85.7	0.532	1.778
No	3	8.6	32	91.4		
C3						
Down	4	11.4	31	88.6	1.000	0.886
Not Down	0	0	7	100		
Albumin						
Normal	0	0	18	100	0.122	1.200
Down	4	16.7	20	83.3		
Pharyngitis						
Yes	0	0	8	100	0.572	1.133
No	4	11.8	30	88.2		
Pyoderma						
Yes	0	0	5	100	1.000	1.121
No	4	10.8	33	89.2		

Bivariate analysis of the relationship between the incidence of AKI in PSAGN patients

The results of this study showed that, although clinical factors and other laboratory parameters such as age, gender, nutritional status, creatinine, albumin, and proteinuria levels did not show a significant relationship with the incidence of *Acute Kidney Injury* (AKI) in PSAGN patients, there was a significant relationship between serum C3 component levels and the incidence of AKI, with a p value = 0.05.

Clinical and Laboratory	AKI				p	OR
	Yes	%	No	%		
Age						
<13 years old	29	90.6	3	9.4	1.000	0.906
≥13 year	10	100	0	0		
Gender						
Male	32	94.1	2	5.9	0.479	2.286
Female	7	87.5	1	12.5		
Nutrition Status						
Adequate and deficient nutrition	33	91.7	3	8.3	1.000	1.091
Overnutrition and obesity	6	100	0	0		
Hematuria						

Positive	36	82.1	3	7.7	1.000	0.923
Negative	3	100	0	0		
Edema						
Yes	23	95.8	1	4.2	0.567	2.875
No	16	88.9	2	11.1		
Proteinuria						
Positive	31	91.2	3	8.8	1.000	1.097
Negative	8	100	0	0		
Hypertension						
Yes	31	93.9	8	88.9	0.525	1.938
No	8	88.9	1	11.1		
C3						
Down	32	91.4	3	8.6	0.05*	0.914
Not Down	7	100	0	0		
Albumin						
Normal	16	88.9	2	11.1	0.567	0.348
Down	23	95.8	1	4.2		
Pharyngitis						
Yes	1	12.5	7	87.5	1.000	0.667
No	6	17.6	28	82.4		
Pyoderma						
Yes	0	0	5	100	0.569	1.233
No	7	18.9	30	81.1		

Factor analysis of the occurrence of complications in PSAGN patients

Based on the results of the analysis, it was found that *Acute Kidney Injury* stage 3 (failure) 7 (16.7%), in *Congestive Heart Failure* 6 (14.3%), in emergency hypertension occurred in 4 (9.5%) and encephalopathy 2 (4.8%) out of a total of 42. This complication distribution data provides an overview of the prevalence of each complication in pediatric PSAGN patients studied.

Complications	Frequency	
	n.	%
Hypertension Emergencies	5	11.9
<i>Acute Kidney Injury stage 3 (Failure)</i>	7	16.7
<i>Congestive Heart Failure</i>	6	14.3
Encephalopathy	2	4.8

Bivariate analysis of the relationship between emergency hypertension in PSAGN patients

Based on the analysis, the results showed that a significant association was only found between emergent hypertension with edema ($p = 0.026$) and elevated serum creatinine levels ($p = 0.025$), while other clinical factors and other laboratory parameters showed no significant association with emergent hypertension in PSAGN patients.

Clinical and Laboratory	Hypertension Emergencies				p	OR
	Yes	%	No	%		
Age	4	12.5	28	87.5	1.000	1.286
<13 years old	1	10	9	90		
≥13 year	5	14.7	29	85.3		
Gender	0	0	8	100	0.564	0.853
Male						
Female	3	8.3	33	91.7		
Nutrition Status	2	33.3	4	66.7	0.141	5.500
Adequate and deficient nutrition						
Overnutrition and obesity	1	11.1	8	88.9		
Hematuria	4	12.1	29	87.9	1.000	0.906
Positive						

Negative	1	3.4	28	96.6		
Edema	4	30.8	9	69.2	0.026*	0.080
Yes						
No	3	8.8	31	91.2		
Proteinuria	2	25	6	75	0.237	3.444
Positive	3	42.9	4	57.1		
Negative	2	5.7	33	94.3		
Serum Creatinine	5	14.3	30	85.7	0.025*	12.375
High (≥ 4 mg/dL)	0	0	7	100		
No						
C3	2	11.1	16	88.9	0.569	0.857
Down	3	12.5	21	87.5		
Not Down						
Albumin	1	12.5	7	87.5	1.000	0.875
Normal	4	11.8	30	88.2		
Down						
Pharyngitis	0	0	5	100	1.000	1.071
Yes	5	13.5	32	86.5		
No						
Pyoderma						
Yes					1.000	1.156
No						

Bivariate analysis of the relationship between stage 3 (*Failure*) AKI in PSAGN patients

Based on the results of the analysis using SPSS, there was no significant relationship between clinical factors and laboratory factors in AKI *failure*.

Clinical and Laboratory	AKI (<i>Failure</i>)				p	OR
	Yes	%	No	%		
Age						
<13 years old	5	15.6	27	84.4	1.000	0.741
≥ 13 year	2	20	8	80		
Gender						
Male	6	17.6	28	82.4	1.000	1.500
Female	1	12.5	7	87.5		
Nutrition Status						
Adequate and deficient nutrition	6	16.7	30	83.3	1.000	1.000
Overnutrition and obesity	1	16.7	5	83.3		
Hematuria						
Positive	6	15.4	33	84.6	0.430	0.364
Negative	1	33.3	2	66.7		
Edema						
Yes	4	13.8	25	86.2	0.657	0.533
No	3	23.1	10	76.9		
Proteinuria						
Positive	7	20.6	27	79.4	0.312	1.259
Negative	0	0	8	100		
C3						
Down	4	11.4	31	88.6	0.077	0.172
Not Down	3	42.9	4	57.1		
Albumin						
Normal	2	11.1	16	88.9	0.679	0.475
Down	5	20.8	19	79.2		
Hypertension						
Yes	6	18.2	27	81.8	1.000	1.778

No	1	11.1	8	88.9		
Pharyngitis						
Yes	1	12.5	7	87.5	1.000	0.667
No	6	17.6	28	82.4		
Pyoderma						
Yes	0	0	5	100	0.569	1.233
No	7	18.9	30	30.8		

Bivariate analysis of the association of CHF in PSAGN patients

Based on the results of the analysis using SPSS, it was found that only albumin levels showed a significant association with the incidence of CHF ($p= 0.029$), while other clinical factors and other laboratory parameters were not significantly associated with the development of CHF in patients ($p> 0.05$). These results indicate that decreased serum albumin levels may act as an important predictive factor in the development of CHF in children with PSAGN.

Clinical and Laboratory	Congestive Heart Failure				p	OR
	Yes	%	No	%		
Age						
<13 years old	3	9.4	29	90.6	0.135	0.241
≥13 year	3	30	7	70		
Gender						
Male	3	8.8	31	91.2	0.072	0.161
Female	3	37.5	5	62.5		
Nutrition Status						
Adequate and deficient nutrition	6	16.7	30	83.3	0.569	1.200
Overnutrition and obesity	0	0	6	100		
Hematuria						
Positive	1	11.1	8	88.9	1.000	0.700
Negative	5	15.2	28	84.8		
Edema						
Yes	4	16.7	20	83.3	0.685	1.600
No	2	11.1	16	88.9		
Proteinuria						
Positive	4	11.8	30	29.1	0.319	2.500
Negative	2	25	6	75		
C3						
Down	6	17.1	29	82.9	0.567	0.829
Not Down	0	0	7	100		
Albumin						
Normal	0	0	18	100	0.029*	1.333
Down	6	25	18	75		
Hypertension						
Yes	5	15.2	28	84.8	1.000	1.429
No	1	11.1	8	88.9		
Serum Creatinine						
High (≥4mg/dL) No	0	0	7	100	0.567	1.207
	6	17.1	29	82.9		
Pharyngitis Yes						
No	2	25	6	75	0.319	2.500
	4	11.8	30	88.2		
Pyoderma Yes						
No	0	0	5	100	1.000	1.194
	6	16.2	31	83.8		

Bivariate analysis of the association of encephalopathy in PSAGN patients

Based on the results of the analysis using SPSS, it was found that only nutritional status and proteinuria showed a significant relationship with the incidence of encephalopathy ($p < 0.05$), while other clinical factors and other laboratory parameters did not show a significant relationship ($p > 0.05$). These results indicate that poor (excessive) nutritional status and high levels of proteinuria can increase the risk of encephalopathy in PSAGN patients.

Clinical and Laboratory	Encephalopathy				p	OR
	Yes	%	No	%		
Age						
<13 years old	2	6.3	30	93.8	1.000	0.938
≥13 year	0	0	10	100		
Gender						
Male	2	5.9	32	94.1	1.000	0.941
Female	0	0	8	100		
Nutrition Status	0	0	36	100		
Adequate and deficient nutrition	2	33.3	4	66.7	0.017*	0.667
Overnutrition and obesity	2	5.1	37	94.9		
Hematuria	0	0	3	100	1.000	0.949
Positive	0	0	29	100		
Negative	2	15.4	11	84.6	0.091	1.182
Edema	0	0	34	100		
Yes	2	25	6	75		
No	0	0	7	100	0.033*	0.750
Proteinuria	2	5.7	33	94.3		
Positive	0	0	33	100		
Negative	2	5.7	33	94.3		
Serum Creatinine						
High (≥4mg/dL)	2	5.7	33	94.3	1.000	1.061
No	0	0	7	100		
C3						
Down	1	5.6	17	94.4	1.000	0.943
Not Down	1	4.2	23	95.8		
Albumin						
Normal	1	12.5	7	87.5	1.000	1.353
Down	1	2.9	33	97.1		
Pharyngitis	0	0	5	100		
Yes	2	5.4	35	94.6	0.348	4.714
No	2	25	6	75		
Pyoderma	0	0	34	100	1.000	1.057
Yes						
No						
Hypertension Crisis					0.033*	0.705
Yes						
No						

Demographic characteristics of PSAGN patients at Ulin Banjarmasin Hospital

This study showed that the age distribution of respondents varied, with the majority of patients falling within the age range of 6-10 years. This observation is in accordance with other studies where the majority of cases belong to the age group above 5 years. This is because at school age children have begun to often be outside the home and are more active so that they are easily exposed to germs that cause PSAGN. The low rate of glomerulonephritis at a very early age has been attributed to immature immune responses at an early age. (Shrestha et al., 2024)

Analysis of gender demographics showed that the percentage of men was higher than women, 81% men and 19% women with a ratio of 4.2:1. This finding is in line with the literature which shows that PSAGN is more common in men than women. Males have high risk factors for developing acute glomerulonephritis, while females have risk factors for developing lupus nephritis. According to Lufyan, boys are generally more active than girls, making them more likely to be exposed to the environment and infected with germs. There is no definitive

research on this, but most PSAGN patients are male.(Adhikari et al., 2022)

In this study, nutritional status analysis showed that 54.8% of children had adequate nutritional status, 31% were undernourished, 11.9% were overnourished and 2.4% were obese. Smith et al. mentioned malnutrition or poor nutrition is one of the risk factors for PSAGN, but Pirania et al. mentioned differently that good nutrition is also at risk of PSAGN.(Tatipang et al., 2017)

Distribution of PSAGN Patients according to clinical features

Nine literatures suggest there are four typical symptoms of PSAGN such as hematuria, hypertension, edema, and oliguria in accordance with the IDAI Nephrology UKK Consensus.(Zabara et al., 2023) In other literature, oliguria was not found as one of the four typical symptoms of PSAGN. The most common typical clinical picture found in children diagnosed with PSAGN is edema.(Utari et al., 2024) This is in line with the theory that states that edema is described in 65-90% of cases. Edema usually occurs suddenly and is first seen in the orbital region.

Together with edema, hypertension is a typical clinical picture of PSAGN that is common in children. In a study conducted by Lufyan et al, hypertension was found in almost all (96.7%) pediatric patients diagnosed with PSAGN, and most of the cases (61%) were patients with grade 2 hypertension. The next clinical picture of PSAGN, namely hematuria both macroscopic which can be seen directly and microscopic found through laboratory examinations. According to Van DeVoorde, hematuria is seen in almost all patients with PSAGN, but only one third of cases have *gross* hematuria.(Zabara et al., 2023) The source of PSAGN infection generally comes from pharyngitis compared to pyoderma or impetigo. This finding differs from the study by Rawla et al., where nephrogenic strains of group A beta hemolytic *Streptococcus* causing PSAGN were predominantly from pyoderma compared to pharyngitis.(Zabara et al., 2023)

Distribution of PSAGN Patients according to laboratory examination results

Laboratory results play an important role in the diagnosis and management of acute glomerulonephritis in children. In this study, laboratory analysis showed some typical findings indicative of impaired renal function. First, urine analysis of the patients showed the presence of hematuria, which was seen in 38 (90.5%) patients. Hematuria is an important indicator of glomerular damage. In addition, proteinuria was also detected in 34 (81%) patients, proteinuria is the result of two mechanisms, one of which is related to abnormal glomerular filtration membrane structure and function due to increased permeability of the glomerular capillary wall as a result of immune complex deposition, release of cytokines and inflammatory mediators, and damage to glomerular capillaries. The mechanism that follows is impaired reabsorption in the proximal tubules so that a large amount of protein cannot be reabsorbed and induces proteinuria.(Zabara et al., 2023)

Antistreptolysin O (ASTO) examination results showed that 33 (78.6%) patients with acute glomerulonephritis had positive ASTO values. ASTO examination is an international standard test that is widely used to detect the presence of group A streptococcal infection (SGA). SGA produces streptococcal hemolysins or streptolysins, including streptolysin O which acts as an antigen that will trigger specific B cells to produce antibodies called antistreptolysin O (ASO) and cause an increase in ASTO examination. In studies by Khalaf et al. and Kilic et al. found an increase in ASTO occurred in almost all pediatric patients with PSAGN, which amounted to 98.6% and 97.3% respectively. Usually ASTO does not increase in skin infections due to the binding of streptolysin by lipids in the skin.(Zabara et al., 2023)

The results showed that 39 (92.9%) patients showed glomerular filtration rate (LFG) values that were below normal, indicating impaired renal function. The occurrence of inflammation in the glomerulus can interfere with the glomerular filtration rate. A decrease in LFG is generally followed by water and sodium retention which can lead to an increase in extracellular fluid volume, resulting in symptoms such as edema and hypertension.(Zabara et al., 2023)

The analysis showed that decreased C3 levels were significantly associated with the diagnosis of acute glomerulonephritis, with 35 (83.3%) patients showing C3 levels that were below the normal range. Examination of serum complement C3 levels is a test of diagnostic value in the diagnosis of PSAGN, as well as most post-infectious glomerulonephritic diseases as C3 is a component of the pathogenesis of these diseases. Complement is a compound in the blood that plays a role in the body's defense system. Increased disease activity in a tissue leads to decreased complement levels.40 The low C3 complement levels found in PSAGN indicate aggressive complement activation. After patients are infected with streptococcal bacteria, activation of the complement system, especially C3, will induce an inflammatory process in patients with PSAGN. The response from excessive immunological processes causes the formation of immune complexes and results in damage to the endothelium and glomerular basement membrane.(Zabara et al., 2023)The analysis showed that 24 (57.1%) patients had hypoalbumin, where serum albumin levels were below 3.5 g/dL. This finding suggests that hypoalbumin correlates with decreased LFG.(McGrogan et al., 2011)

Bivariate analysis of the relationship between the incidence of hypertension in PSAGN patients

The analysis showed that there was a significant relationship between proteinuria and hypertension (p=0.05). Glomerular basement membrane has a thickness of 300-350 nm, and is a very important part of the filtration barrier that regulates fluid transfer. If there is structural damage to the glomerular basement membrane,

for example in podocytes, various pathological conditions can occur with manifestations of proteinuria and microscopic hematuria. Proteinuria in PSAGN occurs due to damage to the glomerular capillary wall, so that protein fails to be filtrated and enters the urine. The more severe the damage to the glomerulus, the more severe the proteinuria that can occur. When associated with hypertension, due to the inflammatory response to the glomerulus until perfusion to the kidneys decreases, there will be feedback on juxtaglomerular cells, until the renin-angiotensin system is activated, which then causes vasoconstriction and increased blood pressure in PSAGN patients.(Akteruzzaman et al., 2019)

Bivariate analysis of the relationship between the incidence of hypertension urgency in PSAGN patients

Overall, although factors such as age, gender, nutritional status, hematuria, edema, proteinuria, serum creatinine, albumin, C3, pharyngitis, and pyoderma play a role in the pathogenesis of glomerulonephritis or other renal diseases, they do not necessarily have a significant direct relationship with hypertension in every case. Hypertension in patients with renal disease is more often influenced by mechanisms of renal impairment, such as fluid retention, activation of the Renin-Angiotensin-Aldosterone (SRAA) System, and vascular dysfunction, which may not always be influenced by such factors in a given clinical setting. Various vasoactive substrates play an important role in the regulation of BP (Blood Pressure). The Renin- Angiotensin-Aldosterone system is the major hormonal system in the regulation of BP. Renin is produced by juxtaglomerular cells in renal afferent arterioles. Stimuli that trigger increased renin secretion include insufficient perfusion to the glomerulus, insufficient sodium intake, and sympathetic nerve activity. Renin further plays a role in converting angiotensinogen into angiotensin I. Furthermore, angiotensin I is converted into angiotensin II by *angiotensin-converting enzyme* (ACE) in the lung. ACE also degrades bradykinin, a potent vasodilator, into its metabolites. Angiotensin II is a strong vasoconstrictor that will increase BP. Angiotensin II also stimulates the release of aldosterone from the zona glomerulosa of the adrenal gland. Subsequently, aldosterone causes water and sodium retention which adds to hyperesthesia.(Indonesia, 2017)

Bivariate analysis of the relationship between the incidence of AKI in PSAGN patients

One of the important features in the pathophysiology of PSAGN is the activation of the complement system, particularly the C3 component of the complement system, which plays a role in the development of *Acute Kidney Injury (AKI)*. Decreased C3 levels are used as an indicator in the diagnosis and assessment of the severity of acute glomerulonephritis.(Hakroush et al., 2021) After patients are infected with streptococcal bacteria, activation of the complement system, especially C3, will induce an inflammatory process in patients with PSAGN. The response of excessive immunologic processes causes the formation of immune complexes and results in damage to the endothelium and glomerular basement membrane.(Zabara et al., 2023)(Hakroush et al., 2021)

Factor analysis of the occurrence of complications in PSAGN patients

Several studies have shown that complications can occur in the acute phase of glomerulonephritis, including *congestive heart failure*, *acute kidney injury*, emergency hypertension, and encephalopathy. Complications in acute glomerulonephritis can occur due to kidney damage caused by glomerular inflammation, uncontrolled activation of the immune system, and disturbances in fluid and electrolyte balance.(Akteruzzaman et al., 2019)(Ong, 2022)(Indonesia, 2017)

Bivariate analysis of the relationship between emergency hypertension in PSAGN patients

Inflammation that occurs in the glomerulus due to the presence of immune complexes will affect capillary perfusion. The pressure in glomerular capillaries is four times higher than other capillaries, so immune complex deposits tend to occur, then the lumen of the blood vessels shrinks until glomerular filtration decreases. Then proximal tubule reabsorption will occur which in turn makes distal tubule reabsorption increase, and water and sodium will be retained. This results in an increase in extracellular fluid volume and the manifestation of edema in the patient.(Akteruzzaman et al., 2019)

The decrease in LFG is due to an inflammatory response to immune complexes located in the glomerulus. The decrease in renal filtration ability varies depending on the extent of damage to the kidneys. Decreased renal perfusion can be caused by various factors, one of which is the diameter of the lumen of the renal blood vessels which shrinks due to complement deposits and endothelial damage that occurs. This will increase the blood pressure of PSAGN patients. addition, decreased renal perfusion will signal the renin-angiotensin system to increase fluid reabsorption which will eventually increase the patient's blood pressure again.(Akteruzzaman et al., 2019)

Bivariate analysis of the relationship between stage 3 (Failure) AKI in PSAGN patients

Scientifically, the absence of a significant association between age, gender, nutritional status, hematuria, edema, proteinuria, serum creatinine, C3, albumin, pharyngitis, and pyoderma with AKI *failure* in PSAGN can be explained by focusing on the more complex pathophysiology of immune reactions and renal vascular disorders. Although these factors play a role in the clinical and diagnostic manifestations of glomerulonephritis, AKI *failure* is more influenced by the inflammatory process and impaired renal perfusion. AKI is divided into primary and secondary AKI. Primary AKI is caused by kidney disease, including acute glomerulonephritis or hemolytic uremic syndrome. Secondary AKI is caused by systemic diseases such as sepsis and shock. In the past, AKI was mostly caused by primary kidney disease. Recently, secondary AKI is more often found due to sepsis, nephrotoxic drugs or renal ischemia in children who have undergone bone marrow, stem *cell* or other organ transplants such as liver

or post-heart surgery, especially in tertiary hospitals.(Indonesia, 2017)

Bivariate analysis of the association of CHF in PSAGN patients

Cardiac output is the result of both shock volume and heart rate. Most of the mechanisms of persistent hypertension are predominantly related to an increase in shock volume and only a few are related to heart rate. Nevertheless, increased heart rate has important prognostic significance in cardiovascular disease. An increase in shock volume is usually caused by an increase in intravascular volume. This is related to excessive fluid retention or fluid transfer from the extravascular to intravascular space. Sodium retention from both excess salt intake and increased reabsorption in the renal tubules is a major contributor to increased intravascular volume. Likewise, increased sympathetic activity will stimulate myocardial contractility and stimulate renin release.(Pratama et al., 2024) A decrease in renal glomerular function, in this case the glomerular filtration rate, results in increased aldosterone secretion resulting in water and sodium retention which can cause fluid overload syndrome in the form of edema on the face and legs. In , an increase in non-enzymatic glycosylation products, an increase in the polyol pathway, glucotoxicity (an increase in blood sugar levels) can cause kidney damage, where there is a process of changes in the glomerular basement membrane, namely the proliferation of mesangium cells so that there is a change in the permeability of the glomerular basement membrane characterized by proteinuria (loss of protein through urine) which can cause a decrease in albumin levels.(Setiadi et al., 2020)The incidence of cardiac complications is higher in some studies, although the incidence of cardiac complications is higher in other studies, but there are no deaths due to the possibility of early presentation in the hospital, timely treatment, and good control of hypertension.(Shrestha et al., 2024)

Bivariate analysis of the association of encephalopathy in PSAGN patients

Obesity will affect the findings of hypertension in PSAGN patients, because children with obesity tend to have primary hypertension. In people who are obese, there can be selective resistance to insulin, which will result in hyperinsulinemia, which can then have several impacts, such as impaired vascular function, impaired ion transport, sodium retention, and increased sympathetic nerve activity which ultimately results in increased blood pressure.(Akteruzzaman et al., 2019)Obesity is probably the most powerful and modifiable risk factor for hypertension during childhood. In some studies it has been shown that the risk of hypertension is higher in adolescents with overweight and obesity, as a risk factor for hypertension. With increasing evidence that target organ damage may become irreversible regardless of blood pressure control, preventing prolonged hypertension and associated target organ damage in children is critical.(Koebnick et al., 2023) Meanwhile, secondary hypertension (hypertension with a known cause) is the most common cause of hypertension in infants and young children, with the primary cause being renal disease, both renal parenchymal and renovascular. Kidney diseases that can cause manifestations of hypertension in children include glomerulonephritis both acute and chronic, scarring of the renal parenchyma, and chronic renal failure in children. In , vascular-related diseases such as renal artery stenosis and coarctation of the aorta can also trigger hypertension in children. In addition, endocrine diseases such as congenital adrenal hyperplasia, hyperaldosteronism, feochromocytoma, neuroblastoma, and cushing syndrome can also manifest as pediatric hypertension. Secondary hypertension usually shows a much higher blood pressure when compared to primary hypertension. Complaints of headache, dizziness, epistaxis, anorexia, visual disturbances, and seizures may occur. addition, children with secondary hypertension may show symptoms and signs according to the underlying disease, such as hematuria in patients with glomerulonephritis.(Ngantung et al., 2022)

Hemodynamic changes in glomerular blood flow can also result in proteinuria. A reduced number of functioning nephrons, as occurs in chronic renal failure, leads to increased protein filtration in the remaining nephrons and causes proteinuria. Other conditions that cause proteinuria by altering glomerular hemodynamics include exercise, fever, seizures, epinephrine use, and emotional stress. Proteinuria occurs when the plasma concentration of certain small proteins exceeds the capacity of the tubules to reabsorb the filtered proteins. Examples include the presence of immunoglobulin light chains in the urine in multiple myeloma, hemoglobinuria in intravascular hemolysis, myoglobinuria in rbdomyolysis, and amylaseuria in acute pancreatitis.(Leung et al., 2017)

CONCLUSION

Based on the results of the research that has been done, it can be concluded that the general age experiencing PSAGN is 5-16 years, and more men experience PSAGN, and more nutritional status is good nutrition. Clinically obtained edema and hypertension in PSAGN patients and laboratory hematuria is most common in PSAGN patients. Complications that occur in PSAGN are emergency hypertension, *AKI failure*, *Congestive Heart Failure* (CHF), and encephalopathy. In emergency hypertension this is related to edema and high serum creatinine that PSAGN patients have, in CHF related to low albumin in PSAGN patients, and in encephalopathy related to overnutrition and obesity, proteinuria, and hypertensive crisis that occurs in PSAGN patients. Therefore the need for regular follow-up and monitoring of these patients to avoid residual morbidity.

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