Jurnal Kedokteran dan Kesehatan: Publikasi Ilmiah Fakultas Kedokteran Universitas Sriwijaya
Volume 13, No 3. 2026/DOI: 10.32539/JKK.V13i3.1003
p-ISSN 2406-7431; e-ISSN 2614-0411
Page:390 - 399

CLINICOPATHOLOGICAL CHARACTERISTICS OF NEPHROTIC SYNDROME PATIENTS AT DR. MOHAMMAD HOESIN PALEMBANG HOSPITAL IN 1 JANUARY 2022 - 31 OCTOBER 2025

Nina Atizah[1], Maria Ulfa[2], Ika Kartika[3]
[1] Anatomical Pathology Residency Program, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia
[2] Anatomical Pathology Department, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia
[3]Anatomical Pathology Department, Faculty of Medicine, Universitas Sriwijaya and Ancillary Medical Services Department of Mohammad Hoesin General Hospital Palembang, Indonesia

ARTICLE INFO

Corresponding author :
Nina Atizah
Anatomical Pathology Residency Program, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

Email:
atizahnina@gmail.com

Kata kunci:
Sindrom nefrotik
Glomerulus
Ginjal

Keywords:
Nephrotic syndrome
Glomerulus
Kidney

Original submission:
June 11, 2026;

Accepted:
July 13, 2026;

Published:
September 20, 2026

ABSTRAK

Sindrom nefrotik merupakan kelainan ginjal akibat gangguan filtrasi glomerulus, baik primer maupun sekunder, mencakup berbagai etiologi seperti membranous nephropathy, minimal change disease, focal segmental glomerulosclerosis, membranoproliferative glomerulonephritis, diabetic nephropathy, dan lupus nephritis,masing-masing dengan karakteristik klinikopatologi tersendiri. Penelitian ini bertujuan untuk memperoleh gambaran klinikopatologi 104 pasien sindrom nefrotik di RSUP Dr. Mohammad Hoesin Palembang. Desain penelitian deskriptif, menggunakan data rekam medis periode 1 Januari 2022–31 Oktober 2025. Karakteristik dominan yang ditemukan adalah pasien dewasa, laki-laki, edema anasarka, tekanan darah <140/90 mmHg, hiperkolesterolemia, proteinuria +3, albumin >2,5 g/dL, PAS negatif, dan trichrome positif, dengan etiologi terbanyak berupa diabetic nephropathy, focal segmental glomerulosclerosis, minimal change disease, membranoproliferative glomerulonephritis, dan lupus nephritis. Karakteristik per etiologi juga ditelaah. Temuan ini merupakan rangkaian peristiwa yang saling terkait secara patofisiologi dan penting dipahami dalam merencanakan terapi pasien.

ABSTRACT

Clinicopathological Characteristics of Nephrotic Syndrome Patients at Dr. Mohammad Hoesin Palembang Hospital in 1 January 2022 – 31 October 2025. Nephrotic syndrome is a kidney disorder caused by impaired glomerular filtration, either primary or secondary, encompassing etiologies such as membranous nephropathy, minimal change disease, focal segmental glomerulosclerosis, membranoproliferative glomerulonephritis, diabetic nephropathy, and lupus nephritis. This study aims to identify clinicopathological features for more accurate therapy. This study aimed to describe the clinicopathological profile of 104 patients with nephrotic syndrome at RSUP Dr. Mohammad Hoesin Palembang. This descriptive study used medical record data from 1 January 2022 to 31 October 2025. The predominant characteristics identified were adult, male patients with anasarca edema, blood pressure <140/90 mmHg, hypercholesterolemia, +3 proteinuria, albumin >2.5 g/dL, negative PAS staining, and positive trichrome staining, with diabetic nephropathy, focal segmental glomerulosclerosis, minimal change disease, membranoproliferative glomerulonephritis, and lupus nephritis as the most common etiologies. Characteristics by etiology were also examined. These findings represent a pathophysiologically interrelated sequence of events important for treatment planning.

INTRODUCTION

Nephrotic syndrome is a kidney disorder frequently encountered in both pediatric and adult populations. This condition arises from impaired glomerular filtration, mediated by either primary or secondary mechanisms. Year after year, the global incidence of nephrotic syndrome in adults remains relatively stable, at approximately 2.7 to 3 new cases per 100,000 people annually.[1] Meanwhile, the global prevalence of nephrotic syndrome in children ranges from 1.15 to 16.9 per 100,000 children per year.[2] In Indonesia, according to data from the Indonesian Renal Registry (IRR) in 2020, hypertensive kidney disease, followed by diabetic nephropathy and glomerulopathy, are the leading causes of chronic kidney disease. For children aged <5 years, the most common causes are congenital kidney anomalies and obstructive uropathy. Conversely, in children aged >5 years, various forms of glomerulonephritis are more predominant.[3] In Palembang, however, data regarding the prevalence of nephrotic syndrome occurrences has not yet been documented.

Nephrotic syndrome can be caused by several specific renal disorders, including membranous nephropathy, minimal change disease, focal segmental glomerulosclerosis, and membranoproliferative glomerulonephritis, as well as other systemic diseases such as diabetic nephropathy and lupus nephritis (SLE).[4] Each etiology presents its own distinct characteristics, encompassing sociodemographic factors such as age and sex; clinical manifestations such as edema and hypertension; clinical laboratory findings such as cholesterol, proteinuria, and albumin levels; and microscopic features involving PAS and trichrome staining. This entire pathological process ultimately culminates in a primary defect: disruption of the glomerular filtration barrier. [4,5]

Histopathological examination plays a crucial role, ranging from aiding in establishing a diagnosis to guiding the management of nephrotic syndrome. Histopathological evaluation, enhanced by histochemical and immunofluorescence examinations, enables pathologists to differentiate lesions with greater specificity. Along with advances in molecular pathology, diagnostic methods for nephrotic syndrome have also advanced, including fluorescence and electron microscopy. Immunological processes involving antigen-antibody interactions can cause glomerular filtration failure, as seen with antibodies against PLA2R in membranous nephropathy, which causes thickening of the basement membrane.[5]

Cases of nephrotic syndrome are expected to continue to rise. This trend is influenced by environmental factors and an increasingly sedentary lifestyle. Therefore, a deeper understanding of the clinicopathological features of nephrotic syndrome is highly required to establish specific diagnoses, which can subsequently be utilized as a consideration in selecting more personalized therapies.

METHODS

The manuscript was prepared in accordance with STROBE guidelines. This study used a descriptive approach. Data were obtained from the medical records and anatomical pathology archives of patients with nephrotic syndrome who underwent histopathological and histochemical examinations at RSUP dr. Mohammad Hoesin Palembang. Data collection was conducted at the Department of Anatomical Pathology, RS Mohammad Hoesin Palembang. The study was conducted from October to December 2025, using medical record data from nephrotic syndrome patients who underwent histopathological examinations between 1 January 2022 and 31 October 2025. This study was approved by the Ethics Committee of RSUP Dr. Mohammad Hoesin Palembang (Approval No.DP.04.03/D.XVIII.06.08/ETIK/318/2025). Patient data were anonymized prior to analysis to ensure confidentiality. The study population consisted of patients with nephrotic syndrome at RSUP

dr. Mohammad Hoesin Palembang, who had undergone both histopathological and histochemical evaluations. The study sample included all population members who met the inclusion and exclusion criteria during the period 1 January 2022 – 31 October 2025. The initial sample size was 154. Fifty samples were excluded due to incomplete medical records data that met our criteria. The final analyzable sample was 104. The inclusion criteria comprised patients diagnosed with nephrotic syndrome with complete demographic data (age and sex); clinical manifestations (edema and blood pressure); clinical laboratory results (cholesterol levels, proteinuria, and blood albumin levels); histopathological findings; and histochemical results (PAS and trichrome staining). The exclusion criteria consisted of medical records that were incomplete for any reason. All samples were then analyzed for the study variables, including age, sex, edema, systolic and diastolic blood pressure, cholesterol levels, proteinuria, PAS and trichrome histochemical staining, and histopathological examination to determine the etiology. Blood pressure variables for pediatric patients were not included in the analysis, as not all of these patients had the height data (n=26) required for blood pressure determination. Several diagnoses based on histopathological and histochemical findings will be further analyzed in separate sections for diagnoses with multiple spectrum levels. The completeness of the collected data was manually verified, and the data were subsequently processed and presented as frequency distribution tables and graphs using SPSS version 22.

RESULTS

The results of this study are classified into three sections: the clinicopathological characteristics of patients with nephrotic syndrome, those with glomerulosclerosis, and those with SLE (lupus nephritis). This classification was performed as a feedback mechanism on the study data to further analyze the clinicopathological characteristics of glomerulosclerosis, the most common etiology, and lupus nephritis, which were differentiated by class.

Clinicopathological characteristics of nephrotic syndrome patients

The study analyzed 104 patient samples, classified into 3 groups based on clinicopathological characteristics: nephrotic syndrome, glomerulosclerosis, and SLE (lupus nephritis). Results indicate 154 initial samples, of which 50 were excluded due to incomplete data.

Table 1. Clinicopathological Characteristics of Nephrotic Syndrome Patients

Variables	Category	Frequency	Percentage
Ages	>18 years	78	75.0
Sex	Male	64	61.5
Edema	Anasarca	61	58.7
Sistolic BP	Normotension	52	66.7
Diastolic BP	Normotension	50	64.1
Cholesterol	High	84	80.8
Proteinuria	Positive 3	42	40.4
Albumin	>2.50	38	36.5
PAS result	Negative	73	70.2
Trichrome result	Positive	72	69.2
Diagnosis	FSGS	62	59.6

*Patients that were initially coded for FSGS will be reclassified in table 3 into four histopathological spectrum (DGGS, DSGS, FGGS, FSGS) for further analysis.

Other sociodemographic variables, such as race, socioeconomic status, and others, could not be included because the information obtained was limited. Blood pressure measurements were calculated only for adult patients because the medical records lacked sufficient data on children's height. The clinicopathological characteristics for each diagnosis are shown in the following table.

Table 2. Clinicopathological Characteristics of Nephrotic Syndrome Patients in Each Diagnosis

Variables	Diagnoses					Total
	DMN (n=1)	FSGS (n=62)	MCD (n=24)	MPGN (n=10)	SLE (n=7)	
Age						
0-18 years	0 (0%)	17 (27,4%)	2 (8,3%)	4 (40,0%)	3 (42,8%)	26 (25,0%)
>18 years	1 (100%)	45 (72,6%)	22 (91,7%)	6 (60,0%)	4 (57,2%)	78 (75,0%)
Sex						
Male	1 (100%)	41 (66,1%)	16 (66,6%)	5 (50,0%)	1 (14,3%)	64 (61,5%)
Female	0 (0%)	21 (33,9%)	8 (33,4%)	5 (50,0%)	6 (85,7%)	40 (38,5%)
Edema						
Anasarca	1 (100%)	40 (64,5%)	13 (54,2%)	4 (40,0%)	3 (42,8%)	61 (58,6%)
Local	0 (0%)	22 (35,5%)	11 (45,8%)	6 (60,0%)	4 (57,2%)	43 (41,4%)
Systolic BP						
Normotension	1 (100%)	29 (64,4%)	15 (68,2%)	3 (50,0%)	4 (100%)	52 (66,6%)
Hypertension	0 (0%)	16 (35,6%)	7 (31,8%)	3 (50,0%)	0 (0%)	26 (33,4%)
Diastolic BP						
Normotension	1 (100%)	28 (62,2%)	15 (68,2%)	3 (50,0%)	3 (75,0%)	50 (64,1%)
Hypertension	0 (0%)	17 (37,8%	7 (31,8%)	3 (50,0%)	1 (25,0%)	28 (35,9%)
Cholesterol						
Normal	0 (0%)	7 (11,2%)	5 (20,8%)	4 (40,0%)	4 (57,2%)	20 (19,2%)
High	1 (100%)	55 (88,8%)	19 (79,2%)	6 (60,0%)	3 (42,8%)	84 (80,8%)
Proteinuria						
Trace	0 (0%)	6 (9,6%)	7 (29,2%)	1 (10,0%)	1 (14,4%)	15 (14,4%)
Positive 1	0 (0%)	7 (10,7%)	1 (4,2%)	2 (20,0%)	0 (0%)	10 (9,6%)
Positive 2	1 (100%)	23 (37,1)	8 (33,3%)	2 (20,0%)	3 (42,8%)	37 (35,6%)
Positive 3	0 (0%)	26 (41,6%)	8 (33,3%)	5 (50,0%)	3 (42,8%)	42 (40,4%)
Albumin level						
<1.6	0 (0%)	21 (33,8%)	7 (29,2%)	3 (30,0%)	2 (28,6%)	33 (31,7%)
1.6-2.0	0 (0%)	14 (22,5%)	3 (12,5%)	1 (10,0%)	0 (0%)	18 (17,3%)
2.1-2.5	0 (0%)	12 (19,4%)	2 (8,3%)	1 (10,0%)	0 (0%)	15 (14,4%)
>2.5	1 (100%)	15 (24,3%)	12 (50,0%)	5 (50,0%)	5 (71,4%)	38 (36,6%)
PAS result						
Positive	1 (100%)	13 (21%)	3 (12,5%)	9 (90,0%)	5 (71,4%)	31 (29,8%)
Negative	0 (0%)	49 (79%)	21 (87,5%)	1 (10,0%)	2 (28,6%)	73 (70,2%)
Trichrome result						
Positive	1 (100%)	57 (92%)	7 (29,2%)	2 (20,0%)	5 (71,4%)	72 (71,3%)
Negative	0 (0%)	5 (8%)	17 (70,8%)	8 (80,0%)	2 (28,6%)	32 (28,7%)

*Patients that were initially coded for FSGS will be reclassified in table 3 into four histopathological spectrum (DGGS, DSGS, FGGS, FSGS) for further analysis.

Since only a single case of diabetic nephropathy was identified in this study, the clinicopathological characteristics are solely presented in Table 2. Further analysis was not conducted due to the inadequate sample size. Therefore, these findings cannot be used to draw a generalized conclusion.

Clinicopathological characteristics of glomerulosclerosis patients

Focal segmental glomerulosclerosis was the most common diagnosis identified in this study. Consequently, a more focused analysis was conducted on this specific diagnosis. The analysis revealed four variants in the number and location of lesions within the FSGS diagnoses in this study: Diffuse Global Glomerulosclerosis (DGGS), Diffuse Segmental Glomerulosclerosis (DSGS), Focal Global Glomerulosclerosis (FGGS), and Focal Segmental Glomerulosclerosis (FSGS). Generally, in the literature, all of these fall within the FSGS spectrum, although as the disease progresses, the term "focal" can evolve into "diffuse," and "segmental" can progress to "global."

Table 3. Clinicopathological Characteristics of Glomerulosclerosis Patients

Variables	Diagnoses				Total
	DGGS (n=3)	DSGS (n=1)	FGGS (n=2)	FSGS (n=56)	
Age					
Children	1 (33,3%)	0 (0%)	1(50%)	15(26,8%)	17 (27,4%)
Adult	2 (66,7%)	1(100%)	1(50%)	41(73,2%)	45 (72,6%)
Sex					
Male	0 (0%)	1(100%)	2(100%)	38(67,9%)	41 (66,1%)
Female	3 (100%)	0 (0%)	0 (0%)	18(32,1%)	21 (33,9%)
Edema					
Anasarca	1 (33,3%)	0 (0%)	2(100%)	37(66,1%)	40 (64,5%)
Local	2 (66,7%)	1(100%)	0 (0%)	19(33,9%)	22 (35,5%)
Sistolic BP					
Normotension	1 (50%)	1(100%)	1(100%)	26(63,4%)	29 (55,5%)
Hypertension	1 (50%)	0 (0%)	0 (0%)	15(36,6%)	16 (45,5%)
Diastolic BP					
Normotension	1 (50%)	1(100%)	1(100%)	25(60,9%)	28 (62,2%)
Hypertension	1 (50%)	0 (0%)	0 (0%)	16(39,1%)	17 (37,8%)
Cholesterol					
Normal	0 (0%)	0 (0%)	0 (0%)	7(12,5%)	7 (11,3%)
High	3 (100%)	1(100%)	2(100%)	49(87,5%)	55 (88,7%)
Proteinuria					
Trace	1 (33,3%)	0 (0%)	0 (0%)	5(8,9%)	6 (9,7%)
Positive 1	0 (0%)	0 (0%)	0 (0%)	7(12,5%)	7 (11,3%)
Positive 2	1 (33,3%)	0 (0%)	0 (0%)	22(39,3%)	23 (37,1%)
Positive 3	1 (33,3%)	1(100%)	2(100%)	22(39,3%)	26 (41,9%)
Albumin level					
<1,6	0 (0%)	0 (0%)	1 (50%)	18(32,1%)	19 (30,6%)
1,6-2,0	0 (0%)	0 (0%)	1 (50%)	15(26,8%)	16 (25,8%)
2,1-2,5	1 (33,3%)	0 (0%)	0 (0%)	10(17,9%)	11 (17,7%)
>2,5	2 (66,7%)	1(100%)	0 (0%)	13(23,2%)	16 (25,9%)
PAS result					
Positive	2 (66,7%)	0 (0%)	0 (0%)	12(21,4%)	14 (22,6%)
Negative	1 (33,3%)	1(100%)	2(100%)	44(78,6%)	48 (77,4%)
***Trichrome* result**					
Positive	3 (100%)	1(100%)	2(100%)	52(92,9%)	58 (93,5%)
Negative	0 (100%)	0 (0%)	0 (0%)	4 (7,1%)	4 (6,5%)

The glomerulosclerosis identified in this study spans several disease severity levels and involves 62 patients. However, the distribution of patients across diagnoses was uneven. The FSGS diagnosis predominated across almost every variable. Therefore, due to the limited sample size, further discussion regarding other diagnoses could not be conducted, and the findings did not represent general characteristics.

Clinicopathological characteristics of SLE (Lupus Nephritis) patients

In addition to FSGS, SLE (lupus nephritis) was also classified into several classes in this study. Therefore, lupus nephritis will be further discussed to better understand its characteristics. There were 7 cases of lupus nephritis, with the specific details of each presented in the table below.

Table 4. Clinicopathological characteristics of SLE (Lupus Nephritis) patients

No.	Age	Sex	Edema	BP	Cholesterol	Proteinuria	Albumin	PAS	*Trichrome*	Diagnosis
1	29	F	Local	138/104	146	Positive 3	3.9	Positive	Positive	*Class III*
2	46	F	Anasarca	110/70	127	Positive 2	2,6	Negative	Positive	*Class III*
3	20	F	Anasarca	122/81	110	*Trace*	3,9	Positive	Negative	*Class I*
4	23	M	Local	118/87	318	Positive 3	1,3	Positive	Positive	*Class IIIA*
5	16	F	Local	120/80	395	Positive 2	2.6	Positive	Positive	*Class IV*
6	17	F	Local	90/60	154	Positive 2	2.8	Negative	Negative	*Class I*
7	10	F	Anasarca	120/90	227	Positive 3	1.4	Positive	Positive	*Class II*

DISCUSSION

Clinicopathological characteristics of nephrotic syndrome patients

The study summarized from 104 samples in the table shows the demographic characteristics of the patients, where the most common age group was adults (75%) and the predominant sex was male (61.5%). This is consistent with global epidemiological research indicating that while nephrotic syndrome can affect any age of onset, it occurs more frequently in adulthood and is predominantly male (2:1).[2,6,7] Kidney damage resulting from systemic and autoimmune diseases can accumulate and begin to manifest clinically in adulthood. The majority of adult nephrotic syndrome patients present with primary glomerular diseases, such as FSGS, membranous nephropathy, and MCD.[8]

The correlation between sex and the incidence of nephrotic syndrome has not yet been clearly established. However, there is a general hypothesis attributing this to differences in sex hormones, such as estrogen, progesterone, and testosterone.[9] Estrogen and progesterone are known to exert protective effects on blood flow, glomerular filtration rate, and the process of ion and water reabsorption within the nephron. Conversely, testosterone exhibits the opposite effects.[9,10]

The majority of nephrotic syndrome patients in this study experienced anasarca edema due to massive proteinuria that leads to hypoalbuminemia and decrease oncotic pressure.[2,5] Meanwhile, patients with MCD exhibited a nearly balanced distribution of edema severity. When linked to the degree of glomerular damage in MCD, localized edema is highly plausible.[11] A study conducted by Son HE et al. in 2023 stated that 73.4% of patients with MCD achieved complete remission, which is why histopathological examination often detects minimal or completely unnoticeable damage.[12] The false hypovolemia induced by hypoalbuminemia triggers compensatory mechanisms via the renin-angiotensin-aldosterone system (RAAS) and an increase in vasopressin. Consequently, salt and water retention occurs, which subsequently increases blood volume and blood pressure in adequate time is required to cause a failure in body homeostasis before the RAAS can be activated.[2,13]

The synthesis of albumin also induces the body to synthesize lipids. This mechanism causes cholesterol levels in nephrotic syndrome patients to exceed 200 mg/dL in the majority of cases.[2] Proteinuria was observed in 42 (40.4%) patients who experienced protein leakage equivalent to a 3+ result on the dipstick test, containing various proteins that leaked into the urine, such as albumin, transferrin, and immunoglobulins. Albumin levels of <1.6 g/dL is already sufficient to cause a loss of oncotic pressure within blood vessels, thereby leading to the development of anasarca edema.[2,5,13,14]

The PAS and trichrome examinations yielded contrasting results in this study. These two stains are fundamentally complementary. PAS staining can identify carbohydrates such as glycogen and glycoproteins, whereas Masson's Trichrome staining is used to differentiate collagen fibers from muscle fibers, cytoplasm, and other tissue components.[15] Both PAS and trichrome staining typically yield positive results in glomerular basement membrane abnormalities, as observed in most cases of nephrotic syndrome.[15,16]

A single case of diabetic nephropathy was identified. The patient was an adult male, which aligns with findings from several studies demonstrating a male predominance over females. Other clinical characteristics were consistent with the general pathophysiology of nephrotic syndrome. Meanwhile, the histopathological and histochemical characteristics exhibited microscopic features of capillary wall thickening of varying severity, as evidenced by positive Periodic Acid-Schiff (PAS) and trichrome staining.[17]

Clinicopathological characteristics of glomerulosclerosis patients

In this study, there were 15 pediatric patients with FSGS and 41 adult patients. Males were more frequently encountered in this study. This is consistent with global research findings indicating that FSGS, especially in adulthood, is more common in males, with a 1.5- to 2-fold higher incidence than in females.[11] When linked to race and ethnicity, FSGS occurs more frequently in Black and Hispanic males than in White males.[11,18]

Anasarca edema was the most common presentation found in patients diagnosed with FSGS. This aligns with the theory stating that FSGS exhibits a severe degree of glomerular damage and frequently progresses to end-stage renal disease, which is characterized, among other things, by the development of advanced (anasarca) edema.[19] Patients with elevated blood pressure, both systolic and diastolic, were most frequently identified in the FSGS diagnosis. This might be due to the tendency of glomerular damage in FSGS to be more severe. Furthermore, the distribution of data across diagnoses was uneven, with the highest concentration in FSGS patients.

Albumin synthesis by the liver is a compensatory mechanism of the body to maintain homeostasis in the face of albumin and other protein leakage through proteinuria.[9] Upon further analysis, LDL levels represent the component of total cholesterol that increases the most. This is associated with increased synthesis of apolipoprotein B-100 (apoB-100) in fatty acid transport due to decreased serum albumin synthesis.[2,9] In line with this process, the leakage of the filtration barrier is also reflected by the predominance of 3+ proteinuria. The severity of hypoalbuminemia occurring in FSGS patients also reflects a poorer prognosis. [10]

A positive PAS examination result indicates abnormalities in the glomerular basement membrane. In FSGS, abnormalities are confined to the glomerulus, including the basement membrane. Ideally, PAS staining should yield a positive result in FSGS diagnoses. The negative results obtained in this study may be attributed to non-representative kidney biopsy samples. Focal areas of sclerosis, particularly in the early stages (segmental), can complicate the interpretation of PAS results. On the other hand, the trichrome staining results were predominantly positive. Masson's Trichrome staining is used to differentiate collagen fibers from muscle fibers, cytoplasm,

and other tissue components.[15,16] The positive results in these FSGS cases indicate glomerular collagen deposition. PAS and trichrome staining should ideally provide consistent findings, as both are intended to complement each other to provide a comprehensive view of a tissue. Although these methods are complementary, they yielded inconsistent results in this study, necessitating in-depth, case-by-case analysis with more detailed medical record information to determine the precise causes, including the possibility of sampling error in focal lesions, technical staining factors, and inter-observer variability.

The measurement of cholesterol, proteinuria, and blood albumin levels in this study requires more detail. The assessment of these levels after therapy, rather than at initial presentation (prior to treatment), was not clearly documented in the patients' medical records. However, elevated cholesterol levels, proteinuria, and low albumin levels across the spectrum of glomerulosclerosis diagnoses indicate that the underlying glomerular damage has exceeded the body's compensatory threshold for maintaining oncotic pressure, thereby necessitating massive albumin synthesis (followed by cholesterol synthesis) by the liver.

One case of DGGS in the pediatric population and two cases in adults are presented in Table 3. The presentation of DGGS resembles the generalized sclerotic changes typically observed in end-stage renal disease. Generally, FSGS progresses to renal failure within 5 to 10 years, although this timeline depends on various factors.[20] If diagnosed at an early stage, the progression of the disease can be slowed. In this study, 17 patients were diagnosed with FSGS during childhood. With appropriate management, these patients are not expected to progress to DGGS or end-stage renal disease over the next 5 to 10 years. Furthermore, table 3 shows that the number of male patients with FSGS is nearly twice that of females, whereas all three patients with DGGS are female. This aligns with global data indicating a higher prevalence of chronic kidney disease among females, although end-stage kidney failure is predominantly observed in males.[21] However, the small number of glomerulosclerosis spectrum cases in this study may limit the general interpretation. Further investigation into larger samples number was suggested.

Clinicopathological characteristics of SLE *(Lupus Nephritis)* patients

Lupus nephritis is a renal manifestation of systemic lupus erythematosus (SLE) that typically develops within the first year following diagnosis. Compared to the involvement of other organs, lupus nephritis carries the highest risk of morbidity and mortality. This risk of morbidity and mortality is directly related to irreversible kidney damage that ultimately progresses to ESRD. Consequently, managing this condition eventually requires kidney transplantation or dialysis.[20] Furthermore, aggressive therapy with immunosuppressive agents is also required. This, in turn, can elevate the risk of severe infections and other complications, ultimately increasing the morbidity and mortality associated with lupus nephritis.[22] It was observed that patients with class III lupus nephritis still maintained normal blood pressure and relatively normal cholesterol levels. This is consistent with findings by nephrologists that clinical manifestations frequently do not reflect the true extent of underlying glomerular damage. Given this diversity, nephrologists classify lupus nephritis into six distinct classes based on different morphological features, as previously explained in the literature review section.

Therefore, this research had several limitations: (1) Retrospective design. Because the data were extracted from historical records, we were unable to establish a definitive temporal sequence of events, thereby limiting our findings to associations. (2) The exclusion of 32.5% of the initial cohort represents a significant methodological vulnerability. It may introduce selection bias as the final analytical sample may no longer accurately reflect the demographic and clinical heterogeneity of the broader patient population; (3) Absence of immunofluorescence and electron microscopy

(instrumentation limitation). Routine light microscopy often lacks the required granularity to identify ultra-structural anomalies or specific immune-complex depositions, which lead to less precise histopathological categorizations; (4) Absence of treatment status at time of laboratory measurement. The lack of temporal alignment between medication logs and laboratory test results introduces a confounding variable, making it challenging to determine whether abnormal values are directly linked to disease pathogenesis or secondary to therapeutic interventions. (5) Single-center design constrains the generalizability of the results. (6) Blood pressure data are unavailable for pediatric patients. The unavailability of blood pressure data for the pediatric subset precluded an in-depth analysis of hemodynamic factors in this demographic.

CONCLUSION

The identified clinicopathological characteristics represent a sequence of pathophysiologically interrelated events that must be thoroughly understood, particularly in planning optimal treatment strategies for patients.

REFERENCES

1. Fratila VG, Lupușoru G, Sorohan BM, Obrișcă B, Mocanu V, Lupușoru M, et al. Nephrotic syndrome: from pathophysiology to novel therapeutic approaches. *Biomedicines* 2024; 12(3): 569-90.
2. Cheong HI. Immunopathogenesis of childhood idiopathic nephrotic syndrome. *Child Kidney Dis* 2023; 27(1): 1-10.
3. Kementerian Kesehatan Republik Indonesia. *Pedoman Nasional Pelayanan Kedokteran Tata Laksana Penyakit Ginjal Kronik 2023*. Jakarta: Kementerian Kesehatan Republik Indonesia; 2023.
4. Pawlina W. Urinary system. In: Pawlina W, editor. Histology: a text and atlas with correlated cell and molecular biology. 9th ed. Philadelphia: Wolters Kluwer Health; 2024. p.698-721.
5. Alpers C.E, Chang A. The kidney. In: Kumar V, Abbas AK, Aster JC, editors. Robbins and Cotran pathology basic of disease V. 10th ed. Philadelphia: Saunders Elsevier; 2021 p.914-26.
6. Nogueira PCK, Feltran LS, Camargo MF, Martins DV, Pesquero JB, Varela P, et al. Epidemiology of pediatric nephrotic syndrome: real-world data from brazil. *Glomerular dis* 2025; 5(1): 403-13.
7. Anggorowati R.H, Panjaitan H.R, Yusanto A, Annisah R. Nephrotic syndrome in pediatrics: a case report. *S J Ped* 2023; 1(1): 62-5.
8. Go AS, Tan TC, Chertow GM, Ordonez JD, Fan D, Law D, et al. Primary nephrotic syndrome and risks of ESKD, cardiovascular events, and death: the kaiser permanente nephrotic syndrome study. *J Am Soc Nephrol* 2021; 32(9): 2303-14.
9. Ward ES, Gelinas D, Dreesen E, Van Santbergen J, Andersen JT, Silvestri NJ, Kiss JE, et al. Clinical significance of serum albumin and implications of fcrn inhibitor treatment in IgG-mediated autoimmune disorders. *Front Immunol* 2022; 13(1): 1-13.
10. Maslyennikov Y, Pralea IE, Bărar AA, Rusu CC, Moldovan DT, Potra AR, et al. Pathogenesis of focal segmental glomerulosclerosis and minimal change disease: insights from glomerular proteomics. *Life (Basel)* 2025; 15(4): 1-17.
11. Cathain D.N, Reidy D, Bagnasco S, Kant S. Focal and segmental glomerulosclerosis: a comprehensive state-of-the-art review. *Sclerosis* 2025; 2(3): 1-15.
12. Son HE, Yun G, Kwon EJ, Park S, Jeong JC, Kim S, et al. Outcomes of minimal change disease

without nephrotic range proteinuria. *PLoS One* 2023;18(8):1-14.
13. Ponticelli C, Moroni G. Nephrotic syndrome: pathophysiology and consequences. *J nephrol* 2023; 36(8): 2179-90.
14. Mizdrak M. Understanding the pathophysiology of nephrotic syndrome and its implications for therapeutic development. *J Nephrol Ther* 2024; 14(2): 1-2.
15. Dey P. Special stains for the carbohydrate, protein, lipid, nucleic acid and pigments. In: Dey P, editor. Basic and advanced laboratory techniques in histopathology and cytology. 2nd ed. Singapore: Springer; 2022. p.81-97.
16. Bancroft JD, Layton C. Carbohydrates. In: Suvarna SK, Layton C, Bancroft JD, editors. Bancroft's theory and practice of histological techniques. 8th ed. China: Elsevier; 2018. p.221-24.
17. Bogdanovi´c L, Babi´c I, Prvanovi´c M, Mijaˇc D, Markovi´c AM, Popovi´c D, et al. Uncommon factors leading to nephrotic syndrome. *Biomedicines* 2025; 13(8): 1-19.
18. Drexler Y, Athavale A, Smith AR, Liu Q, Zee J, Mariani LH, Lafayette RA. Clinical Outcomes in the nephrotic syndrome study network : disease burden and treatment patterns over time. *Glomerular Dis* 2025; 5(1): 288–303.
19. Stokes MB, D'Agati VD. Focal segmental glomerulosclerosis. In: Jennette JC, Olson JL, Silva FG, D'Agati VD, editors. Heptinstall pathology of the kidney. 7th ed. Philadelphia: Wolters Kluwer Health; 2014.p.397-400.
20. Gou W, Tuo YH. Comparison of mortality and its causes in patients with complicated systemic lupus erythematosus on hemodialysis versus peritoneal dialysis: A meta-analysis. *Medicine (Baltimore)* 2022; 101(32):1-6.
21. Filippone E.J, Gulati R, Farber J. L. Contemporary review of IgA nephropathy. *Front Immunol* 2024; 15(3): 1-22.
22. Aldana-Pérez SE, García-Bañol DF, Arias-Choles AM, Aroca-Martínez GJ, Musso CG, Dominguez-Vargas A, et al. Infectious complications and prognostic factors of mortality in patients with lupus nephritis admitted to intensive care units. *J Clin Med* 2025; 14(21): 1-15.