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Original Article

Clinicopathological Characteristics of Children with Focal Segmental Glomerulosclerosis

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ABSTRACT

Background: Focal segmental glomerulosclerosis (FSGS) is a major cause of childhood nephrotic syndrome, particularly steroid-resistant nephrotic syndrome (SRNS), and is associated with variable treatment response, chronic renal damage, and disease progression. **Aim:** To evaluate the clinicopathological characteristics of children with biopsy-confirmed FSGS, including clinical features, treatment patterns, renal biopsy findings, and IgM/C3 deposition. **Methods & Materials:** This retrospective observational study was conducted in the Department of Pediatric Nephrology, Bangladesh Medical University, Dhaka, from January to December 2025. Thirty-three children with biopsy-confirmed FSGS and complete clinical, treatment, histopathological, and immunofluorescence records were included. Data were collected from hospital records using a structured sheet and analyzed with SPSS version 26.0. Quantitative data were expressed as mean ± SD or median (IQR), and qualitative data as frequencies and percentages. **Results:** Among 33 children, the mean current age was 8.77 ± 4.33 years, with male predominance (21; 63.6%). SRNS was the most common clinical diagnosis (19; 57.6%). At presentation, hematuria occurred in 25 (75.8%) children, hypertension in 20 (60.6%), and nephrotic-range proteinuria in 13 (39.4%). The most frequent sequential non-steroidal immunosuppressive regimen was MMF → CNI → RTX (7; 21.2%). Treatment-related complications included hypertension (26; 78.8%), hypertrichosis (23; 69.7%), and acute kidney injury (14; 42.4%). Renal biopsy commonly showed interstitial fibrosis (23; 69.7%), enlarged glomeruli (20; 60.6%), and tubular atrophy (16; 48.5%). Immunofluorescence demonstrated IgM and/or C3 positivity in 15 (45.5%) children, with no significant clinicopathological differences compared with other/negative IF groups. **Conclusion:** Childhood FSGS was commonly associated with SRNS, male predominance, hematuria, hypertension, and chronic biopsy changes. IgM and/or C3 deposition showed no significant clinicopathological association. Frequent treatment-related complications emphasize the need for early diagnosis, careful immunosuppressive therapy, regular monitoring, and long-term follow-up.

Keywords: *Focal segmental glomerulosclerosis, Steroid-resistant nephrotic syndrome, Renal biopsy and IgM and C3 deposition*

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INTRODUCTION

Among childhood glomerular diseases, focal segmental glomerulosclerosis (FSGS) is an important clinicopathological entity characterized by focal and segmental glomerular scarring, typically reflecting podocyte injury and maladaptive glomerular repair [1,2]. Rather than representing a single disease, FSGS is now understood as a histological pattern with diverse causes, including primary, genetic, secondary, and undetermined forms [1-3]. This distinction is clinically important because treatment response, risk of progression, and recurrence after kidney transplantation vary with the underlying mechanism [1,3]. Globally, FSGS is one of the major causes of nephrotic syndrome and steroid-resistant nephrotic syndrome (SRNS) in children. Although minimal change disease remains the most common histology in steroid-sensitive childhood nephrotic syndrome, FSGS is frequently encountered in children with steroid resistance, persistent proteinuria, hematuria, hypertension, or impaired renal function [4,5]. Across pediatric cohorts, SRNS carries a high risk of chronic kidney disease and kidney failure, particularly when remission is not achieved or when biopsy shows chronic damage such as interstitial fibrosis, tubular atrophy, and glomerulosclerosis [5,6]. The study by Trautmann et al. also emphasized that long-term outcomes in childhood SRNS are strongly influenced by genetic background, treatment response, and renal histology [6]. Across South Asia, renal biopsy series have shown that FSGS contributes substantially to biopsy-proven pediatric renal disease, particularly among children selected for biopsy because of atypical nephrotic syndrome, steroid resistance, or progressive renal involvement [7,8]. The study from South Punjab, Pakistan, reported FSGS as an important cause of pediatric nephrotic syndrome and highlighted its association with steroid resistance, relapse, and variable treatment response [8]. In Bangladesh, pediatric renal biopsy data from Dhaka showed FSGS in 37 of 354 biopsied children with nephrotic syndrome (10.45%), while mesangial proliferative glomerulonephritis, minimal change disease, and IgM nephropathy were also common patterns [9]. This regional variation indicates the need for local clinicopathological data, as ethnicity, referral patterns, genetic background, infection burden, treatment access, and biopsy indications may influence the observed histological spectrum. Clinically, children with FSGS often require prolonged corticosteroid exposure and additional immunosuppressive agents, including calcineurin inhibitors, mycophenolate mofetil, cyclophosphamide, or rituximab [5,10]. Because these children are vulnerable to hypertension, growth failure, obesity, cushingoid changes, hypertrichosis, infection, and nephrotoxicity, documenting treatment-related complications is essential for balancing disease control against treatment harm [10]. In addition, immunofluorescence findings, particularly IgM and C3 deposition, have attracted

growing attention. Although these deposits were once considered nonspecific trapping in sclerotic areas, recent evidence suggests that IgM and/or C3 may reflect complement activation and may have prognostic implications in some patients with FSGS [11,12]. Although FSGS is clinically important, local pediatric data integrating demographic profile, clinical presentation, treatment exposure, renal histopathology, immunofluorescence pattern, and treatment-related adverse effects remain limited. Therefore, updated institutional data are needed to guide early recognition, risk assessment, treatment planning, and follow-up strategies for children with FSGS. Accordingly, this study aimed to evaluate the clinicopathological characteristics of children with biopsy-confirmed focal segmental glomerulosclerosis, with emphasis on clinical presentation, immunosuppressive treatment patterns, adverse effects, histopathological findings, and IgM/C3 deposition pattern.

METHODS & MATERIALS

This retrospective observational study was conducted in the Department of Pediatric Nephrology, Bangladesh Medical University, Dhaka, Bangladesh. Children diagnosed with focal segmental glomerulosclerosis (FSGS) on renal biopsy during the study period of one year study period from January to December 2025 were included. A total of 33 children fulfilled the eligibility criteria and were enrolled in the final analysis. Children of either sex with biopsy-confirmed FSGS and available clinical, laboratory, treatment, histopathological, and immunofluorescence records were included. Cases with incomplete medical records, inadequate biopsy samples, or alternative primary renal diagnoses were excluded. After selection, relevant information was collected from hospital records using a structured data collection sheet. Baseline variables included current age, age at initial presentation, disease duration, sex, and current clinical diagnosis. Clinical diagnosis was categorized as infrequent relapsing nephrotic syndrome (IFRNS), steroid-dependent nephrotic syndrome (SDNS), and steroid-resistant nephrotic syndrome (SRNS). Clinical manifestations at presentation, including hypertension, hematuria, and degree of proteinuria, were recorded. Proteinuria was categorized into non-nephrotic-range and nephrotic-range proteinuria according to standard clinical assessment. Treatment-related information was reviewed, with a focus on non-steroidal immunosuppressive agents, including mycophenolate mofetil (MMF), calcineurin inhibitors (CNIs), cyclophosphamide (CYC), and rituximab (RTX). Treatment categories represented sequential exposure to these agents during the treatment course rather than simultaneous administration. For example, MMF → CNI → CYC indicated stepwise treatment modification or escalation according to clinical response and disease course. Treatment-related adverse effects and complications, such as hypertension, hypertrichosis, acute kidney injury, moon face, growth failure, acanthosis nigricans, obesity, convulsions, cataract, buffalo hump, and acne, were documented. Renal biopsy findings were reviewed from histopathology and immunofluorescence reports. Histopathological variables included interstitial fibrosis, enlarged glomeruli, tubular atrophy, increased mesangial cellularity, global sclerosis, segmental sclerosis, combined global and segmental sclerosis, and glomerular basement membrane thickening. Immunofluorescence findings included deposition of IgM, C3, IgG, C1q, kappa light chain, fibrin, IgA, and lambda light chain. For comparative analysis, patients were grouped into IgM- and/or C3-positive and other/negative immunofluorescence groups. Data were analyzed using standard statistical software (SPSS, V. 26.0). Quantitative variables were expressed as mean ± standard deviation or median with interquartile range, while qualitative variables were expressed as frequency and percentage. Group comparisons were performed using appropriate statistical tests, and a p-value <0.05 was considered statistically significant.

RESULTS

Table I summarizes the baseline demographic and clinical profile of the study population. The mean current age was 8.77 ± 4.33 years, and the median age at initial presentation was 3.55 years (IQR 6.75). The mean disease duration was 3.44 ± 3.48 years. Male children were predominant (21; 63.6%), while SRNS (19; 57.6%) was the most frequent current clinical diagnosis.

Table I
Baseline Demographic and Clinical Characteristics of the Study Population (*n* = 33).

Variable	Mean ± SD / n (%)
Current age (years)	8.77 ± 4.33
Age at initial presentation (years) [Median (IQR)]	3.55 (IQR: 6.75)
Disease duration (years)	3.44 ± 3.48
Sex	
Male	21 (63.6)
Female	12 (36.4)
Current clinical diagnosis	
IFRNS	8 (24.2)
SDNS	6 (18.2)
SRNS	19 (57.6)

Clinical manifestations at presentation are presented in *Table II*. Hematuria was the most common feature, affecting 25 (75.8%) children, followed by hypertension in 20 (60.6%). Non-nephrotic-range proteinuria was observed in 20 (60.6%) children, whereas 13 (39.4%) had nephrotic-range proteinuria.

Table II
Clinical Manifestations at Presentation in Children with FSGS (*n* = 33).

Clinical characteristic		n (%)
Hypertension		20 (60.6)
Hematuria		25 (75.8)
Proteinuria	Non-nephrotic proteinuria	20 (60.6)
	Nephrotic-range proteinuria	13 (39.4)

Regarding nonsteroid immunosuppressive therapy, *Table III* shows that the most common pattern was MMF → CNI → RTX, observed in 7 (21.2%) children, followed by CNI only in 5 (15.2%) and MMF → CNI → CYC in 4 (12.1%). Other sequential treatment patterns were used in 9 (27.3%) children, while only 1 (3.0%) received no second-line immunosuppressive drug. The treatment patterns represent sequential modification or escalation of therapy rather than simultaneous administration of the listed agents.

Table III
Non-Steroid Immunosuppressive Treatment Combinations Used in Children with FSGS.

Variable	Category	n (%)
Drug combination	MMF → CNI → RTX	7 (21.2)
	CNI only	5 (15.2)
	MMF → CNI → CYC	4 (12.1)
	MMF only	3 (9.1)
	CNI → RTX	2 (6.1)
	MMF → CNI	2 (6.1)
	Other combinations	9 (27.3)
	No second-line immunosuppressive drug	1 (3.0)

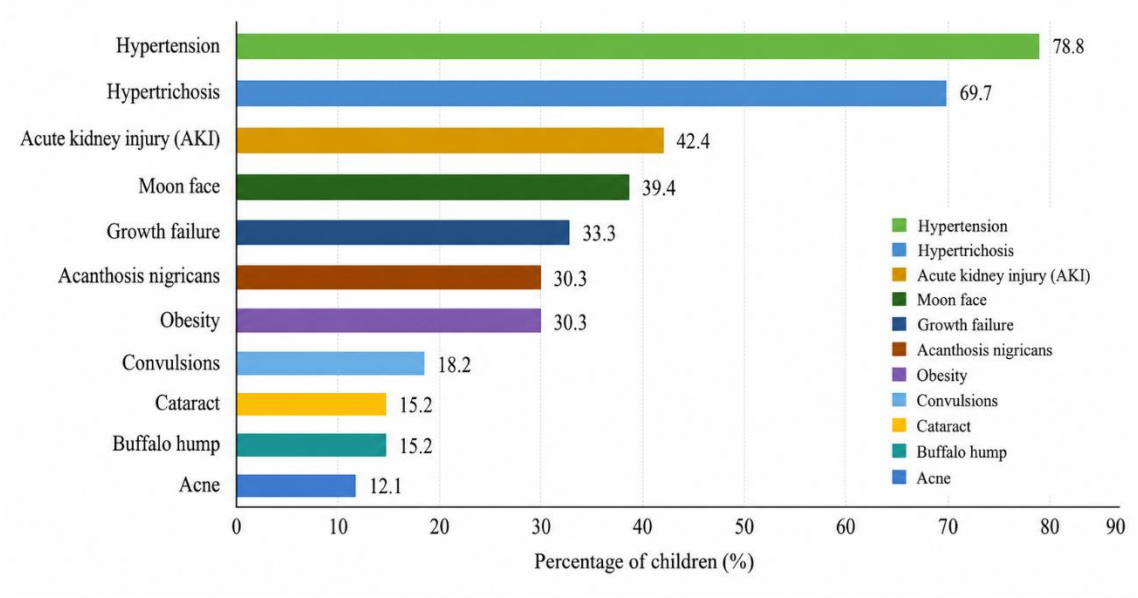

Figure 1 Treatment-Related Adverse Effects and Complications.

In *Figure 1*, treatment-related adverse effects and complications are illustrated. Hypertension was the leading adverse effect, reported in 26 (78.8%) children. Other common complications included hypertrichosis in 23 (69.7%), acute kidney injury in 14 (42.4%), moon face in 13 (39.4%), and growth failure in 11 (33.3%).

Renal biopsy findings in *Table IV* indicate that interstitial fibrosis was the most frequent abnormal histopathological feature, present in 23 (69.7%) children. Enlarged glomeruli were found in 20 (60.6%), tubular atrophy in 16 (48.5%), and increased mesangial cellularity in 14 (42.4%) cases.

Table IV
Abnormal Histopathological Findings in Renal Biopsy Specimens of Children with Focal Segmental Glomerulosclerosis (*n* = 33).

Variable	Category	n (%)
Abnormal histopathological finding	Interstitial fibrosis	23 (69.7)
	Enlarged glomeruli	20 (60.6)
	Tubular atrophy	16 (48.5)
	Increased mesangial cellularity	14 (42.4)
	Global sclerosis	11 (33.3)
	Segmental sclerosis	11 (33.3)
	Combined global and segmental sclerosis	11 (33.3)
	GBM thickening	8 (24.2)

The immunofluorescence findings are detailed in *Table V*. Any immunofluorescence deposition was detected in 16 (48.5%) children. IgM deposition was found in 13 (39.4%), and C3 deposition in 10 (30.3%). Overall, an IgM and/or C3-positive pattern was observed in 15 (45.5%) children, while 18 (54.5%) had other or negative immunofluorescence findings.

Table V
Immunofluorescence Findings and IgM/C3 Deposition Pattern in Renal Biopsies of Children with Focal Segmental Glomerulosclerosis (*n* = 33).

Variable	Category	n (%)
Immunofluorescence finding	Any immunofluorescence deposition	16 (48.5)
	IgM deposition	13 (39.4)
	C3 deposition	10 (30.3)
	IgG deposition	2 (6.1)
	C1q deposition	1 (3.0)
	Kappa (κ) light chain deposition	1 (3.0)
	Fibrin deposition	1 (3.0)
	IgA deposition	0 (0.0)
	Lambda (λ) light chain deposition	0 (0.0)
	IgM and/or C3-positive pattern	15 (45.5)
	Other/negative immunofluorescence findings	18 (54.5)

The comparison based on immunofluorescence patterns is shown in Table 6. No statistically significant difference was found between the IgM and/or C3-positive group and the other/negative IF group. SRNS was common in both groups, 9 (60.0%) and 10 (55.6%), respectively. Hypertension was observed in 7 (46.7%) versus 13 (72.2%), hematuria in 11 (73.3%) versus 14 (77.8%), and nephrotic-range proteinuria in 5 (33.3%) versus 8 (44.4%). All comparisons were not statistically significant ($p > 0.05$).

Table VI
Comparison of Clinical and Histopathological Characteristics According to Immunofluorescence Pattern (*n* = 33).

Variable	IgM and/or C3 Positive (n = 15)	Other/negative IF findings (n = 18)	P value
Current clinical diagnosis			
IFRNS	4 (26.7)	4 (22.2)	0.798
SDNS	2 (13.3)	4 (22.2)	
SRNS	9 (60.0)	10 (55.6)	
Hypertension			
Present	7 (35.0)	13 (65.0)	0.135
Absent	8 (61.5)	5 (38.5)	
Hematuria			
Present	11 (44.0)	14 (56.0)	1
Absent	4 (50.0)	4 (50.0)	
Proteinuria			
Non-nephrotic/Absent	10 (50.0)	10 (50.0)	0.515
Nephrotic	5 (38.5)	8 (61.5)	
Sclerosis pattern			
Global sclerosis	4 (26.7)	7 (38.9)	0.693
Segmental sclerosis	5 (33.3)	6 (33.3)	
Both global and segmental sclerosis	6 (40.0)	5 (27.8)	

DISCUSSION

In the present study, children with biopsy-confirmed FSGS had a mean current age of 8.77 ± 4.33 years, median age at presentation of 3.55 years, and male predominance of 63.6%. This age profile was similar to a cohort reported by Safdar et al., in which the mean age was 8.83 ± 3.05 years and males accounted for 51.6% [8]. By contrast, the Tunisian pediatric cohort reported by Boussetta et al. had a median age of 4.5 years and 54.3% boys [13]. The current proportion of SRNS (57.6%) was higher than the 29.0% steroid resistance reported by Boussetta et al., but is consistent with enrichment of FSGS among difficult nephrotic syndrome referrals [6,13]. The Bangladeshi biopsy series by Alam et al. found FSGS in 37 of 354 biopsied children (10.45%), supporting its local relevance [9]. Clinically, hematuria (75.8%) and hypertension (60.6%) were prominent. These frequencies were higher than the Tunisian series, where macroscopic hematuria was found in 4 of 35 cases and hypertension in 8 of 35 cases [13]. They were also higher than those reported in the Turkish registry by Kurultak et al., in which hematuria and hypertension were 32.6% and 34.1%, respectively [14]. This difference may reflect longer disease duration (3.44 ± 3.48 years), a higher SRNS burden, and referral of more complicated patients. Similar concerns were emphasized by Lee et al., who described SRNS as a heterogeneous condition with a poor prognosis, and by Trautmann et al., who reported lower 10-year ESRD-free survival among children with FSGS than in those with minimal change disease (52% versus 79%) [6,15]. The review by Rosenberg and Kopp also supports interpreting FSGS as a clinicopathological pattern rather than a single uniform disease [1]. The pattern of nonsteroid immunosuppressive use reflected difficult-to-treat disease. The most common specific sequential treatment pattern was MMF → CNI → RTX (21.2%), followed by CNI alone (15.2%). This is compatible with IPNA guidance supporting CNI-based treatment for nongenetic SRNS, with additional agents for CNI-dependent, CNI-resistant, or frequently relapsing disease [5]. In the study by Sinha et al., tacrolimus was superior to MMF for sustaining remission in idiopathic SRNS, while the network meta-analysis by Li et al. supported the central role of CNIs in pediatric SRNS treatment [16,17]. The use of rituximab was also understandable, as the study by Gaur et al. evaluated the combination of rituximab and MMF in CNI-resistant FSGS [18]. Treatment-related morbidity was substantial. Hypertension was the most common adverse effect or complication (78.8%), followed by hypertrichosis (69.7%), acute kidney injury (42.4%), moon face (39.4%), and growth failure (33.3%). These findings are consistent with CNI and steroid toxicities. The pharmacovigilance analysis by Liu et al. linked cyclosporine to nephrotoxicity, acute kidney injury, hypertension, and hirsutism, whereas tacrolimus was more strongly associated with metabolic and renal

fibrosis signals [19]. Thus, the high rates of hypertrichosis and AKI likely reflect CNI exposure, while moon face, obesity, cataract, and growth failure indicate cumulative corticosteroid burden. Histopathologically, interstitial fibrosis (69.7%), enlarged glomeruli (60.6%), tubular atrophy (48.5%), and increased mesangial cellularity (42.4%) were common. These findings indicate chronic structural injury and are close to the Turkish adult registry, where interstitial fibrosis was 65.6%, tubular atrophy 67.5%, and mesangial proliferation 53.5% [14]. The NEPTUNE analysis by Zee et al. further supports the importance of these findings, as interstitial fibrosis, tubular atrophy, global sclerosis, and related biopsy descriptors were among features most predictive of outcome in MCD/FSGS [20]. Therefore, biopsy chronicity in this cohort has practical prognostic value. On immunofluorescence, IgM deposition was observed in 39.4%, C3 in 30.3%, and IgM and/or C3 positivity in 45.5%. These rates were comparable to the Turkish adult registry, where IgM and C3 positivity were 32.7% and 32.9%, respectively [14], but higher than the 28.3% IgM and/or C3 deposition reported by Amer et al. [21]. Although the present study found no significant association between IgM/C3 positivity and clinical or histological variables, previous findings remain mixed. In the adult studies by Zhang et al. and Mirioglu et al., IgM/C3 co-deposition was associated with worse renal outcomes, whereas the pediatric study by Peng et al. showed that capillary C3 independently predicted poor renal outcome [11,12,22]. These differences suggest that deposit location, intensity, sample size, and disease chronicity may be more important than simple positivity.

LIMITATIONS

The study was conducted in a single center with a small sample size, which may limit the generalizability of the findings. The retrospective nature of data collection may have caused missing or incomplete information. Long-term renal outcomes, treatment response, and progression to chronic kidney disease were not evaluated. Genetic testing was also not performed, so genetic forms of FSGS could not be separately assessed.

CONCLUSION

The study concluded that childhood FSGS was commonly associated with SRNS, male predominance, and frequent clinical manifestations such as hematuria and hypertension. Renal biopsy showed substantial chronic histopathological changes, particularly interstitial fibrosis, enlarged glomeruli, and tubular atrophy. Nearly half of the children had IgM and/or C3 deposition, but this pattern was not significantly associated with clinical or histopathological differences. Treatment-related complications were frequent, underscoring the need for early diagnosis, careful use of immunosuppressants, regular monitoring, and long-term follow-up.

RECOMMENDATIONS

Children with FSGS should receive early diagnosis, regular follow-up, and close monitoring of blood pressure, renal function, proteinuria, growth, and treatment-related complications. Immunosuppressive therapy should be used cautiously with toxicity surveillance. Larger multicenter studies with long-term follow-up are recommended to assess renal outcomes and the prognostic role of IgM/C3 deposition.

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CONFLICT OF INTEREST

None declared

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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