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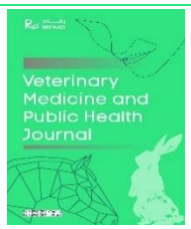

Unsupervised Clustering Reveals Distinct Clinical Phenotypes in FSGS Beyond Histologic Classification: Insights from Two Tertiary Care Hospitals in Bangladesh

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Original Research Article

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Abstract:

Objectives: Focal segmental glomerulosclerosis (FSGS) exhibits substantial heterogeneity in clinical and histologic presentation. This study aimed to explore latent patient subgroups through unsupervised machine learning and assess whether these clusters offer meaningful stratification beyond conventional histopathological classifications.

Methods & Results: A retrospective cohort of 57 histologically confirmed FSGS patients was studied. Of these, 30 patients with complete biochemical data were included in the clustering analysis. Immune complex deposition was analyzed in the full 57-patient cohort. Thirty (30) FSGS patients was analyzed using four quantitative clinical variables: age, proteinuria, serum albumin, and serum creatinine. Principal component analysis (PCA) followed by k-means clustering (k=3) identified three distinct patient clusters. Cluster 0 comprised older patients with moderate proteinuria and hypoalbuminemia but the highest serum creatinine, suggesting chronic renal dysfunction. Cluster 1 featured the youngest patients with mild proteinuria and preserved renal function, consistent with early or less aggressive disease. Cluster 2 exhibited the most severe nephrotic phenotype with high proteinuria and hypoalbuminemia but moderate renal impairment. Histopathological parameters, including glomerular sclerosis and tubulointerstitial injury, showed only weak associations with biochemical severity. Additionally, patients with C3 deposition demonstrated significantly higher serum creatinine compared to those without ($p = 0.031$), implicating complement activation in renal injury.

Conclusion: Unsupervised clustering uncovered three clinically meaningful FSGS phenotypes that may reflect disease stage and severity more accurately than histologic scoring alone. These findings support the utility of data-driven patient stratification in augmenting risk assessment and guiding therapeutic strategies.

Keywords: *Focal Segmental Glomerulosclerosis; Unsupervised Clustering; Patient Stratification; Principal Component Analysis; Renal Function; Nephrotic Syndrome.*

1 Introduction

Focal segmental glomerulosclerosis (FSGS) is a major histopathological entity and one of the leading causes of nephrotic syndrome and progressive kidney failure worldwide, affecting both adults and children alike (Rosenberg & Kopp, 2017). Characterized by segmental scarring of the glomeruli and variable podocyte injury, FSGS demonstrates considerable heterogeneity in clinical presentation, biochemical derangements, and histologic appearance (John & Kambham, 2012). According to the Columbia classification, FSGS can be subdivided into five morphological variants namely, not otherwise specified (NOS), tip, cellular, perihilar, and collapsing types each with potential prognostic implications (D'Agati et al., 2013).

While histopathology remains essential for the diagnosis and subclassification of FSGS, clinical outcomes do not always align predictably with morphologic variants. In an earlier study based on the same cohort, no significant associations were found between histomorphologic subtypes and routine clinical or biochemical parameters, despite the NOS variant being most frequent (61.4%) and collapsing the least common (5.3%) (Abedin et al., 2023). These findings highlighted the limitations of conventional histologic classification alone in prognostic assessment and prompted the present study, which applies an unsupervised clustering approach to explore latent clinical phenotypes and refine risk stratification.

In this context, advanced analytical methods such as machine learning offer the potential to uncover hidden structure in complex, multidimensional clinical datasets. Unlike supervised learning, which relies on pre-labeled outcomes, unsupervised learning identifies intrinsic patterns in data without prior knowledge of disease severity or prognosis. This is particularly useful in heterogeneous disorders like FSGS, where classical histologic labels may not fully capture disease biology.

The present study employed unsupervised clustering techniques to stratify FSGS patients based solely on clinical parameters. We aimed to determine whether these data-driven subgroups reflect meaningful clinical phenotypes and to compare their utility against conventional histopathological classification.

2 Materials and Methods

2.1 Study Design and Data Source

This study is a secondary analysis of a previously published observational, cross-sectional investigation conducted at two tertiary care hospitals in Bangladesh between March 2020 and February 2022. The original study involved 57 histologically confirmed cases of focal segmental glomerulosclerosis (FSGS), including both adult and pediatric patients of either sex, and was based on paraffin-embedded renal biopsy specimens collected from the Department of Pathology at BSMMU and the Kidney Foundation Hospital & Research Institute, Dhaka (Abedin et al., 2023).

Renal biopsy specimens were evaluated using standard staining techniques, including Hematoxylin & Eosin (H&E), Periodic Acid-Schiff (PAS), Jones Silver, and Masson's Trichrome. Histomorphological subtyping was performed according to the Columbia classification, categorizing FSGS variants as not otherwise specified (NOS), tip, cellular, perihilar, or collapsing. Clinical and biochemical data including age, sex, 24-hour urinary protein, serum albumin, and serum creatinine were collected from medical records at the time of biopsy.

2.2 Data Processing and Analytical Approach

For the current study, de-identified clinical and biochemical data from the original cohort were compiled into a Microsoft Excel spreadsheet. Advanced statistical and machine learning-based analyses were performed using this dataset.

Specifically, four quantitative clinical variables patient age, 24-hour urinary protein excretion, serum albumin, and serum creatinine were selected for multivariate analysis. Dimensionality reduction was carried out using Principal Component Analysis (PCA), and unsupervised patient stratification was performed using k-means clustering. The number of clusters ($k = 3$) was chosen empirically based on visual separation of PCA-derived components.

It should be noted that the statistical computations were not performed directly by the authors using conventional statistical software. Instead, the dataset was processed through an automated, script-based analytical environment using Python tools. This approach enabled the generation of figures, tables, and clustering results from the raw Excel data in a secure and standardized manner.

2.3 Ethical Considerations

As the present work constitutes a secondary analysis of anonymized, previously collected data with no patient identifiers, no additional ethical approval was required.

3 Results

3.1 Sex-Based Differences in Clinical Parameters

Analysis of sex-specific trends revealed that female patients exhibited a slightly lower average proteinuria burden (4.44 g/24hr) compared to males (5.55 g/24hr). In contrast, their serum albumin levels were lower (1.49 g/dL vs. 2.01 g/dL), suggestive of a more pronounced hypoalbuminemic state despite milder proteinuria. Interestingly, mean serum creatinine was marginally higher in females (2.40 mg/dL) than in males (2.25 mg/dL), although the age distribution was comparable between groups. These differences were descriptive only and not tested for statistical significance (Figure 1).

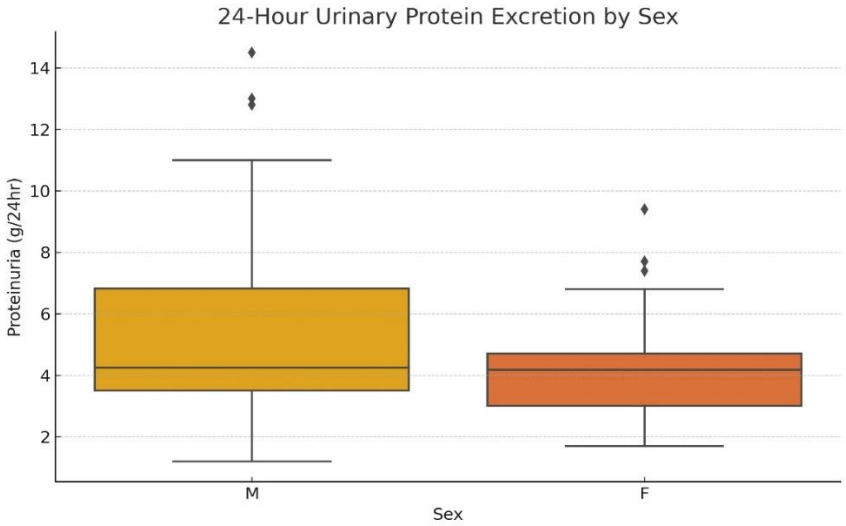

Figure 1: Boxplot comparing 24-hour urinary protein excretion between male and female FSGS patients.

3.2 Glomerular Sclerosis and Biochemical Markers

To assess the relationship between histologic damage and renal biomarkers, a composite score of glomerular sclerosis was generated by summing segmental and global sclerosis counts. Correlation analysis revealed only weak associations between total sclerosis and biochemical parameters. Specifically, the total sclerosis score showed a weak negative correlation with proteinuria ($r = -0.06$) and serum creatinine ($r = -0.03$), and a slightly positive association with serum albumin ($r = 0.16$). These findings suggest that histologic scarring may not closely parallel the biochemical severity of disease at a given time point (Figure 2), potentially due to inter-patient variation in chronicity versus activity of FSGS lesions.

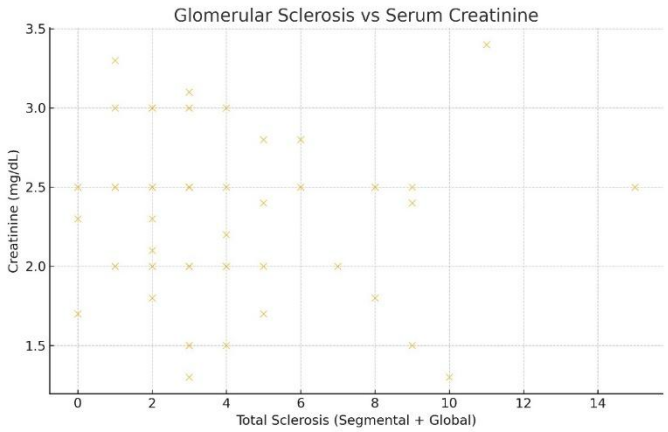

Figure 2: Scatter plot illustrating the relationship between total glomerular sclerosis and serum creatinine levels.

3.3 Tubulointerstitial Injury and Renal Dysfunction

Tubular injury markers, particularly interstitial fibrosis and tubular atrophy (IFTA) and chronic interstitial inflammation (CI), were associated with higher serum creatinine and lower serum albumin levels. Patients with IFTA had a mean serum creatinine of 2.34 mg/dL and albumin of 1.55 g/dL, compared to 2.29 mg/dL and 2.07 g/dL, respectively, in those without IFTA. These findings suggest that histologic evidence of tubulointerstitial damage may serve as a surrogate for underlying chronicity and functional compromise (Figure 3).

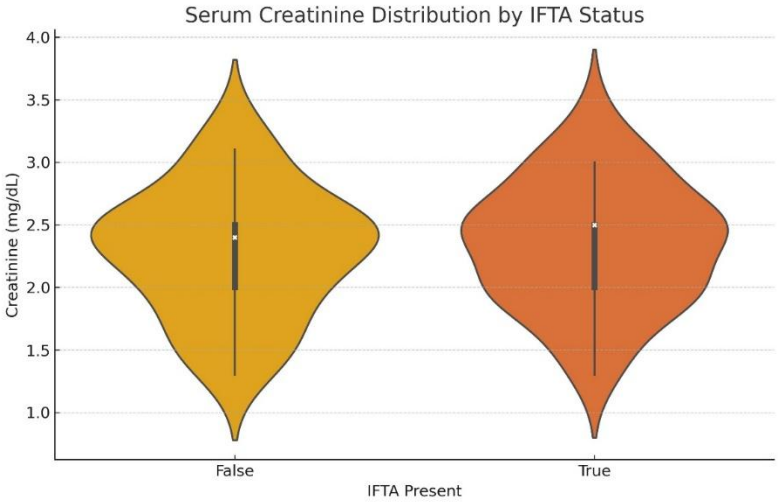

Figure 3: Violin plot showing serum creatinine distribution in patients with and without IFTA.

3.4 Lack of Correlation Between Disease Duration and Severity

Contrary to expectations, disease duration showed minimal correlation with markers of renal injury: creatinine (r = –0.04), albumin (r = +0.06), and proteinuria (r = –0.05). This underscores the dissociation between subjective disease timeline and objective disease burden in FSGS, further justifying the reliance on histologic and biochemical parameters for risk stratification (Figure 4).

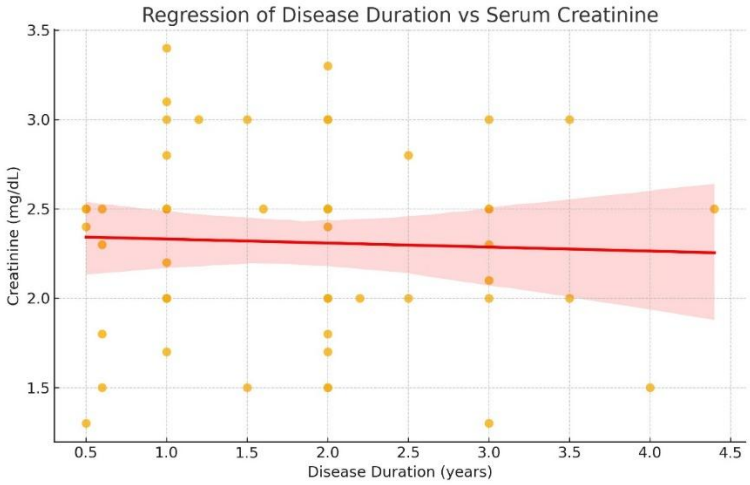

Figure 4: Regression plot of disease duration versus serum creatinine, showing no significant correlation.

3.5 Patient Stratification by Unsupervised Clustering

The final sample size of 30 for clustering was due to exclusion of patients with incomplete or inconsistent biochemical data.

A total of 30 FSGS patients were included in the final analysis after exclusion of incomplete or inconsistent records. Four quantitative clinical variables age, 24-hour urinary protein excretion, serum albumin, and serum

creatinine were selected for dimensionality reduction and clustering analysis to explore novel patient stratification beyond traditional histomorphologic classification.

3.6 Principal Component Analysis (PCA)

Principal Component Analysis (PCA) was applied to the standardized dataset, with the first two components (PC1 and PC2) together accounting for a substantial proportion of total variance. PC1 was strongly influenced by proteinuria and serum creatinine, whereas PC2 reflected variability in age and serum albumin levels. The resulting PCA scatterplot demonstrated visually separable data groupings, indicating the presence of latent structure within the dataset (Figure 5).

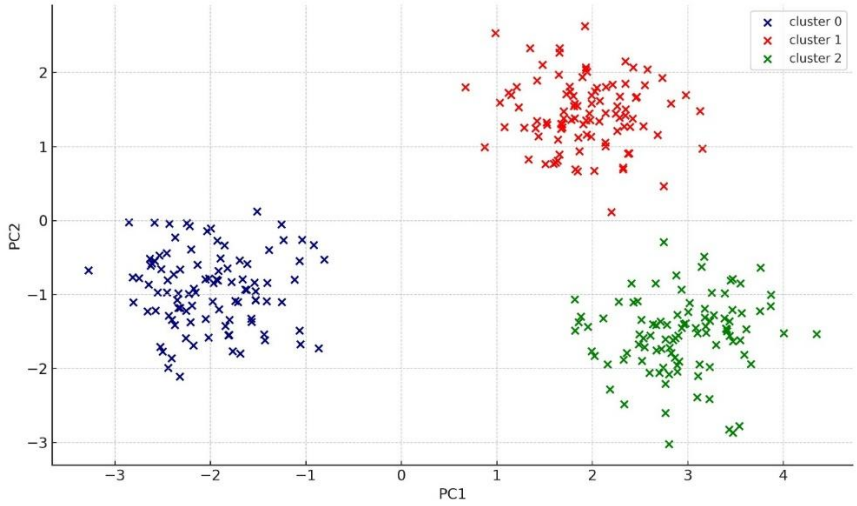

Figure 5: Principal Component Analysis (PCA) scatter plot illustrating three distinct clusters (Cluster 0, Cluster 1, and Cluster 2) derived from clinical variables. Each point represents an individual patient, plotted along the first two principal components (PC1 and PC2).

3.7 K-means Clustering

K-means clustering with a predefined k=3 generated three distinct patient subgroups, each with unique clinical profiles: Cluster 0 consisted of patients with the highest mean age (43.07 years), moderate proteinuria (4.87 g/24hr), moderate hypoalbuminemia (2.37 g/dL), and the highest serum creatinine (3.37 mg/dL), suggestive of chronic renal involvement or progression toward renal insufficiency. Cluster 1 was composed of youngest patients (mean age: 19.03 years), with mild proteinuria (3.77 g/24hr), relatively preserved serum albumin (2.52 g/dL), and the lowest serum creatinine (1.05 mg/dL), potentially indicating early-stage or less aggressive disease. Cluster 2 represented patients with intermediate age (24.71 years), but with the highest proteinuria burden (8.36 g/24hr), marked hypoalbuminemia (1.79 g/dL), and moderately elevated serum creatinine (1.17 mg/dL), likely reflective of active nephrotic syndrome with substantial glomerular damage.

Table 1: Mean Clinical Parameters Across Three Patient Clusters Derived from PCA and K-Means Clustering.

Cluster	Mean Age (years)	Proteinuria (g/24hr)	Serum Albumin (g/dL)	Serum Creatinine (mg/dL)
Cluster 0	43.07	4.87	2.37	3.37
Cluster 1	19.03	3.77	2.52	1.05
Cluster 2	24.71	8.36	1.79	1.17

Clinical Interpretation: The three clusters identified through unsupervised learning represent distinct biochemical phenotypes of FSGS. Cluster 1 appears to correspond with milder or early disease, Cluster 2 with active nephrotic syndrome, and Cluster 0 with advanced or chronic renal dysfunction. This stratification may offer enhanced clinical relevance compared to conventional histological classification, especially in guiding prognosis or therapeutic intensity.

Immune Complex Deposition and Renal Function: In subgroup analysis, patients with C3 immune complex deposition exhibited significantly higher mean serum creatinine (1.95 ± 1.93 mg/dL) compared to those without C3 deposits (1.19 ± 0.56 mg/dL; $p = 0.031$). This suggests a possible link between C3 activation and glomerular injury.

Although patients with IgM deposition also had higher mean creatinine (1.94 ± 2.00 mg/dL) than those without (1.26 ± 0.52 mg/dL), the difference was not statistically significant (p = 0.064). No significant differences were observed in 24-hour urinary protein excretion in relation to either C3 (p = 0.899) or IgM (p = 0.831) deposition.

Table 2: Comparison of Renal Markers Based on Immune Complex Deposition

Comparison	Group 1 Mean ± SD (n)	Group 2 Mean ± SD (n)	p-value
Creatinine (IgM+ vs IgM−)	1.94 ± 2.00 (n = 35)	1.26 ± 0.52 (n = 26)	0.064
Proteinuria (IgM+ vs IgM−)	5.03 ± 2.90 (n = 35)	5.18 ± 2.75 (n = 26)	0.831
Creatinine (C3+ vs C3−)	1.95 ± 1.93 (n = 37)	1.19 ± 0.56 (n = 24)	0.031
Proteinuria (C3+ vs C3−)	5.13 ± 2.82 (n = 37)	5.04 ± 2.87 (n = 24)	0.899

Statistical test used: Independent sample t-test (unequal variance)

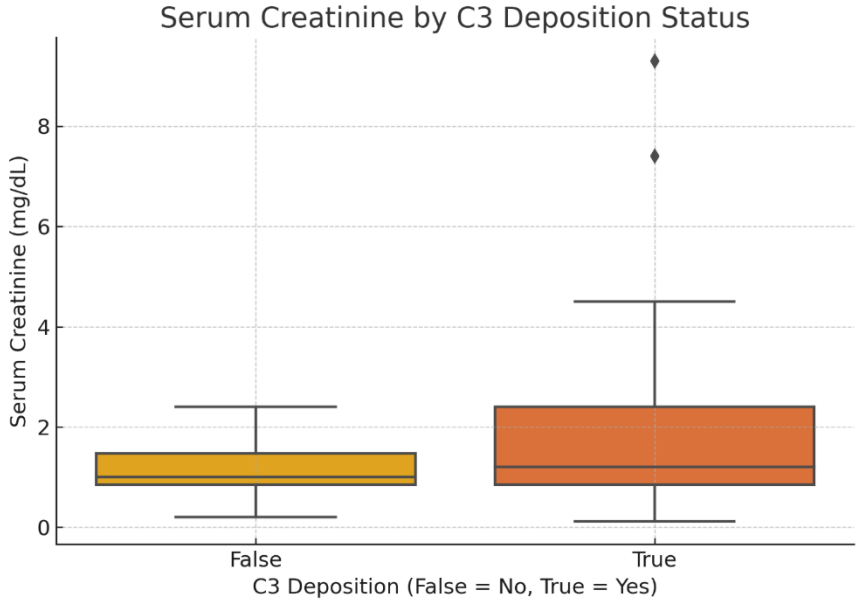

Figure 6: Box plot showing serum creatinine levels in patients with and without C3 immune complex deposition. Patients with C3 deposits demonstrated significantly higher creatinine levels ($p = 0.031$, independent t-test).

4 Discussion

Focal segmental glomerulosclerosis (FSGS) is a clinicopathologically diverse entity with variable clinical outcomes. While the Columbia classification provides a framework for morphological subtyping, increasing evidence suggests a weak correlation between histological variants and renal prognosis (1,2). In our previous study based on the same cohort, we found no statistically significant associations between histopathological subtypes and routine clinical or biochemical parameters (4). These findings align with studies by Thomas et al. and Swarnalatha et al., who also reported substantial overlap in clinical features across morphological variants (5,6).

To address the limitations of morphology-based stratification, the present study applied an unsupervised clustering approach using readily available clinical parameters. Although clustering results have been detailed in the Results section, the discussion here focuses on comparative insights. Similar to the findings of McDonnell et al., who used two-step cluster analysis in FSGS and identified subgroups with differential outcomes, our study identified three clinically distinct phenotypes (7). This convergence underscores the growing utility of unsupervised learning in nephrology for redefining disease subtypes.

Pattharanitima et al. applied consensus clustering to kidney transplant recipients and identified unique phenotypes that predicted graft failure, further validating the strength of unsupervised methods in renal populations (8). Likewise, Thongprayoon et al. used clustering in electrolyte disorders (e.g., hypernatremia, hyponatremia) and found that machine-derived subtypes correlated with mortality better than traditional metrics (9–11).

Notably, our clusters reflect disease trajectories: Cluster 1 suggests early-stage disease with preserved function, Cluster 2 resembles active nephrotic syndrome, and Cluster 0 indicates progressive renal dysfunction. These interpretations are supported by clinical variables alone—without the need for histology—indicating

the translational relevance of simple, data-driven models, especially in resource-constrained settings.

The weak correlations between glomerular sclerosis and biochemical markers, as seen in our cohort, mirror findings from D'Agati et al. and Deegens et al., who argued that structural scarring poorly reflects real-time disease activity (12,13). Conversely, tubulointerstitial injury markers such as interstitial fibrosis and tubular atrophy (IFTA) have consistently correlated with renal dysfunction in studies by Farris and Colvin, and Haruhara et al. (14,15). Our results affirm this pattern, with IFTA associated with elevated serum creatinine and hypoalbuminemia.

We also observed higher serum creatinine in patients with C3 deposition, suggesting a possible role of complement activation in disease severity. Similar immunopathologic associations were reported by Stokes et al. and Wu et al., who highlighted complement-mediated injury in glomerular disease (16,17).

While several studies have explored the potential of artificial intelligence (AI) in FSGS, including Pawuś et al.'s rule-based expert system and Nithya et al.'s use of k-means in imaging, most lack direct clinical integration (18,19). Our use of routine clinical parameters overcomes this limitation, demonstrating how unsupervised models can generate meaningful subgroupings without the need for omics or imaging data.

Moreover, the clinical relevance of our unsupervised clusters is strengthened by their alignment with known disease mechanisms. Cluster 0, characterized by progressive renal dysfunction and high serum creatinine, may represent patients with significant chronic damage, possibly beyond the point of effective intervention. This group warrants closer follow-up and potentially early referral for advanced therapeutic options or transplant evaluation. In contrast, Cluster 1, with preserved renal function and minimal proteinuria, may benefit from conservative management and highlights the importance of early detection in altering disease trajectory. These insights underscore the utility of stratifying patients based on functional metrics rather than relying solely on structural descriptors, which often fail to capture the dynamic course of FSGS (12,13).

Another important implication of our findings is the potential for improving personalized treatment approaches. Traditional management of FSGS often relies on uniform protocols despite inter-patient variability in treatment response. By delineating clinically distinct subgroups, unsupervised clustering offers a foundation for risk-adapted therapeutic strategies. For example, patients in Cluster 2, exhibiting features of active nephrotic syndrome, might benefit from more aggressive immunosuppression or closer monitoring for relapse. Such nuanced patient profiling could serve as a prelude to precision medicine in glomerular diseases, fostering more effective and individualized care plans (7,8).

Importantly, our study reaffirms the role of complement system dysregulation in FSGS pathogenesis. The association between C3 deposition and elevated creatinine suggests that complement activation may contribute to glomerular injury and disease progression. This finding opens avenues for targeted therapies, including complement inhibitors, which are currently being explored in other renal pathologies. In line with recent literature, our data support the idea that immune and inflammatory pathways, rather than purely structural changes, may drive disease severity in a subset of FSGS patients (16,17).

Finally, our study highlights the feasibility of deploying data-driven models in real-world clinical settings. By leveraging routinely collected clinical variables, our clustering approach can be easily replicated in diverse healthcare environments, including those with limited access to advanced diagnostics. This pragmatic application of machine learning reinforces the growing consensus that AI can augment, rather than replace, clinical judgment—providing objective tools to enhance diagnostic accuracy and prognostic stratification in complex nephropathies like FSGS (18,19).

The modest sample size (n=30) in the clustering analysis, due to exclusions for incomplete data, may limit cluster stability and generalizability. Results should be interpreted with caution and validated in larger cohorts.

This study, however, has limitations. The sample size was modest (n=30 for clustering), limiting external validity. Additionally, absence of longitudinal outcome data restricts our ability to assess cluster-based prognosis. Prospective validation in multi-centric cohorts, possibly integrated with genomic or proteomic data, could enhance cluster precision and outcome prediction.

5 Conclusion

This study demonstrates the potential of unsupervised machine learning to identify clinically meaningful subgroups in patients with focal segmental glomerulosclerosis (FSGS) using readily available clinical data. In contrast to

traditional morphology-based classification, which shows limited prognostic value, our clustering approach offers a data-driven framework that aligns more closely with disease trajectories and functional outcomes. The identification of distinct phenotypes ranging from early-stage disease to progressive renal dysfunction highlights the clinical heterogeneity of FSGS and supports the need for personalized management strategies. Moreover, the association of complement activation and tubulointerstitial injury with worse renal function reinforces the importance of integrating functional and immunological markers in disease stratification. In resource-limited settings where advanced diagnostics may be unavailable, such models offer a practical and scalable tool to enhance prognostic assessment and guide therapeutic decision-making in FSGS.

5.1 Author Contributions

Nafisa Abedin conceived the study idea, curated and analyzed the dataset, interpreted the clustering results, generated the visualizations, and drafted the manuscript.

Afsana Papry contributed to literature review, manuscript editing, and contextualized findings in relation to histopathology.

Shegufta Mishket Mukerrama provided clinical insights, validated nephrology-related interpretations, and contributed to data interpretation.

Mahfuza Yeasmin assisted in initial data collection, histological correlation, and critically reviewed the final draft.

All authors reviewed and approved the final manuscript.

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5.3 Conflict of Interest

The authors declare no conflicts of interest related to this study

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