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Article

Liver Disease–Associated Glomerulopathies: Clinical Spectrum, Pathophysiology, and Implications for Renal Prognosis

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ABSTRACT

Background: Liver disease–associated glomerulopathies (LDAGs) represent a clinically significant but often underrecognized cause of renal dysfunction in patients with chronic liver disease, particularly in regions with a high prevalence of hepatitis B and C. The lack of comprehensive data on the clinicopathological patterns and outcomes of these glomerular diseases in South Asian populations highlights an important knowledge gap. **Objective**: This study aimed to evaluate the clinical and histopathological spectrum of glomerular diseases in adults with chronic liver disorders, with a focus on associations between liver disease etiology, severity, proteinuria, and renal outcomes. **Methods**: In this cross-sectional observational study, adult patients (n = 72) with chronic liver disease and evidence of renal involvement were recruited from a tertiary care center. Inclusion criteria comprised age ≥18 years, established diagnosis of chronic liver disease, and renal impairment defined by proteinuria >500 mg/day, hematuria, or eGFR <60 mL/min/1.73m^2. Patients with non-glomerular acute kidney injury, systemic autoimmune diseases, or immunosuppressive therapy were excluded. Clinical, laboratory, and renal biopsy data were collected using standardized instruments. Liver disease severity was assessed with Child-Pugh and MELD scores. Ethical approval was obtained in accordance with the Helsinki Declaration. Statistical analyses were performed using SPSS, employing t-tests, chi-square tests, and logistic regression to assess associations. **Results**: Glomerulopathy was confirmed in 28 of 72 patients (38.9%), with membranoproliferative glomerulonephritis (MPGN) linked to hepatitis C being the most common (46.4%), followed by membranous nephropathy (28.6%) and IgA nephropathy (17.9%). Proteinuria >3.5 g/day was observed in 67.9% of glomerulopathy cases, significantly higher than in patients without biopsy-proven disease ($p < 0.001$). The prevalence of nephrotic-range proteinuria and cryoglobulinemia increased with worsening liver dysfunction and hepatitis C etiology. **Conclusion**: LDAGs, particularly MPGN associated with hepatitis C, are prevalent in patients with advanced liver disease, often presenting with severe proteinuria and immune-mediated features. Early identification and multidisciplinary management are essential to improve renal outcomes and reduce morbidity in this high-risk populations.

Keywords: Liver Diseases, Glomerulonephritis, Hepatitis C, Proteinuria, Renal Insufficiency, Biopsy, Cross-Sectional Studies.

INTRODUCTION

Chronic liver diseases, particularly those caused by viral hepatitis B and C, have increasingly been recognized as significant contributors to the development of glomerular diseases, collectively termed liver disease–associated glomerulopathies (LDAGs) (1,2). These renal complications often remain underdiagnosed in patients with long-standing liver pathology, even though they play a crucial role in progression to kidney failure and significantly worsen clinical outcomes (3). The spectrum of glomerular involvement in chronic liver disease is broad, with membranoproliferative glomerulonephritis (MPGN), membranous nephropathy, and IgA nephropathy being most frequently reported (4,5). These entities are believed to arise

from persistent antigenic stimulation and immune complex deposition, particularly in the context of chronic hepatitis B and C infections, as well as in certain autoimmune liver disorders (2,3). Circulating immune complexes, dysregulated complement activity, and cryoglobulin formation have all been implicated in the pathogenesis, resulting in proteinuria, hematuria, and progressive deterioration of renal function (3,6).

Distinguishing glomerular disease secondary to liver pathology from other causes of renal impairment, such as hepatorenal syndrome or drug-induced nephropathy, is vital for appropriate management and prognostication (7). Previous research has established the central role of renal biopsy in differentiating these conditions and guiding therapy, as clinical and laboratory findings alone are often insufficient for precise diagnosis (8). Furthermore, there is evidence that the prevalence and histopathological patterns of glomerulopathies may vary according to the underlying etiology of liver disease and regional epidemiology of viral hepatitis (5,9). Despite advances in antiviral therapy and improved supportive care, the optimal management strategy for LDAGs remains unclear, particularly in resource-limited settings where access to biopsy and specialized treatment is often restricted (6,10).

A review of the available literature highlights that, while the relationship between hepatitis B or C and specific forms of glomerulonephritis has been well described in high-resource settings, fewer studies have systematically explored the clinical and histopathological features of glomerular disease in diverse patient populations with chronic liver disease in South Asia (4,9). The identification of LDAGs is further complicated by the presence of mixed etiologies, overlapping clinical presentations, and limited data on long-term renal outcomes (7,11). Given these challenges, there remains a substantial knowledge gap regarding the true prevalence, spectrum, and outcomes of glomerular diseases associated with chronic liver pathology, particularly in tertiary care centers in regions with a high burden of viral hepatitis.

This study was therefore undertaken to evaluate the clinical and histopathological patterns of glomerular disease in adult patients with chronic liver disorders presenting to a tertiary care hospital in Quetta, Pakistan. By systematically assessing clinical features, laboratory findings, and renal biopsy results, we aim to provide evidence that will inform diagnostic strategies and improve management for this vulnerable patient population. The primary research objective is to characterize the spectrum and frequency of glomerular disease in patients with chronic liver disease and to identify key associations between liver disease etiology, glomerulopathy type, and clinical presentation in this regional context.

MATERIAL AND METHODS

The present investigation was designed as a cross-sectional observational study to evaluate the clinical and histopathological spectrum of glomerular diseases among patients with chronic liver disease. The study was conducted at the combined Nephrology and Gastroenterology Departments of Sandeman Provincial Hospital and Bolan Medical College in Quetta, Pakistan, spanning from January 2024 to December 2024. Adult participants, aged 18 years or older, were eligible for inclusion if they had a documented diagnosis of chronic liver disease based on clinical, laboratory, and imaging criteria, and exhibited evidence of renal involvement, defined by either persistent proteinuria greater than 500 mg per day, hematuria, or an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73m^2. Exclusion criteria comprised a history of acute kidney injury unrelated to glomerular pathology, the presence of systemic autoimmune disorders, or the use of immunosuppressive therapy at the time of evaluation. Eligible patients were consecutively identified from inpatient and outpatient services, and all provided written informed consent after being fully informed about the study objectives, procedures, risks, and benefits.

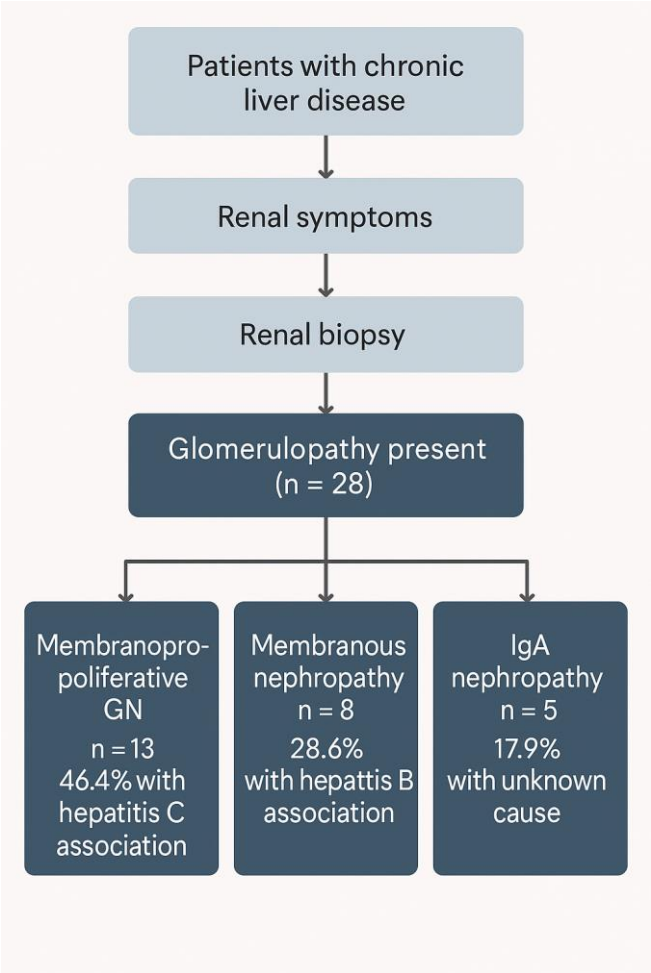
All clinical and laboratory data were obtained prospectively using a standardized data collection proforma at the time of enrollment. Demographic details, comorbidities, history of liver disease including etiology and duration, and relevant clinical findings were systematically recorded. Biochemical assessments were performed using validated hospital laboratory protocols, including complete blood count, liver function tests, renal function profile, and serological screening for hepatitis B and C.

Additional parameters, such as serum albumin, bilirubin, and coagulation profile, were collected to evaluate liver disease severity. The Child-Pugh and Model for End-Stage Liver Disease (MELD) scores were calculated for each participant to grade the extent of hepatic dysfunction. All enrolled patients underwent renal biopsy using ultrasound-guided percutaneous technique performed by an experienced nephrologist. Renal biopsy specimens were processed for light microscopy and immunofluorescence; diagnoses were rendered according to established histopathological criteria. Laboratory personnel and pathologists were blinded to clinical details to minimize observer bias.

Operational definitions were pre-specified: proteinuria was defined as urinary protein excretion above 500 mg per day, hematuria as more than five red blood cells per high power field in a centrifuged urine sample, and chronic kidney disease as an eGFR below 60 mL/min/1.73m^2, calculated using the CKD-EPI formula. The primary outcome variable was the presence and type of glomerulopathy identified on renal biopsy. Secondary variables included demographic characteristics, etiology of liver disease, laboratory indices, and clinical features at presentation. To reduce the impact of confounding, patients with systemic autoimmune disease or on immunosuppressive medications were excluded, and multivariable analyses were planned to adjust for age, sex, liver disease severity, and viral hepatitis status.

The study's sample size was determined based on prior literature indicating a glomerulopathy prevalence of 30–40% in similar cohorts, with a precision of ±10% and a 95% confidence interval, yielding a minimum required sample size of 70 subjects. All statistical analyses were performed using SPSS version 24.0 (IBM Corp, Armonk, NY, USA).

Continuous variables were summarized as mean and standard deviation or median and interquartile range as appropriate; categorical variables were expressed as frequencies and percentages. The chi-square test or Fisher's exact test was used to compare categorical variables, while t-test or Mann-Whitney U test was applied for continuous data, depending on normality assessed by Shapiro-Wilk test. Logistic regression was conducted to evaluate associations between liver disease characteristics and glomerular pathology, with adjustment for pre-specified confounders. Missing data were handled using complete case analysis, and sensitivity analyses were performed to assess the robustness of findings. Pre-specified subgroup analyses included comparisons by viral hepatitis status and degree of liver dysfunction.



Ethical approval was obtained from the Institutional Review Board of Sandeman Provincial Hospital and Bolan Medical College. Written informed consent was secured from all participants, and patient confidentiality was maintained throughout by assigning unique study identifiers and storing data in password-protected electronic files accessible only to study personnel. Data quality was ensured by double-entry verification, routine audits, and independent review of a random sample of records for consistency and accuracy. All procedures adhered to the principles of the Declaration of Helsinki.

RESULTS

A total of 72 patients with chronic liver disease and evidence of renal involvement were included in the study. The mean age of participants was 49.6 years (SD ±11.8), with 61.1% being male (44 out of 72) and 38.9% female (28 out of 72). Among those with biopsy-confirmed glomerulopathy (n=28), the mean age was 50.3 years, and 64.3% were male, compared to 49.1 years and 59.1% male in the group without glomerulopathy, with no statistically significant differences observed in age (p = 0.66, OR 1.02, 95% CI 0.97–1.07) or gender distribution (p = 0.68, OR 1.24, 95% CI 0.46–3.37), as shown in Table 1.

Regarding the underlying etiology of liver disease, hepatitis C infection accounted for the largest proportion of cases, found in 33 patients (45.8%). Notably, hepatitis C was present in 57.1% of those with glomerulopathy, versus 38.6% in those without, although this difference did not reach statistical significance (p = 0.13, OR 2.07, 95% CI 0.79–5.47). Hepatitis B was identified in 20 patients (27.8%), and its frequency was similar across groups (25.0% with glomerulopathy vs. 29.5% without, p = 0.67, OR 0.80, 95% CI 0.28–2.29). Alcoholic liver disease and cryptogenic etiologies comprised smaller proportions, with no significant group differences, as detailed in Table 2.

Histopathological analysis of renal biopsies revealed that membranoproliferative glomerulonephritis (MPGN) was the most frequent finding, present in 13 of 28 patients with glomerulopathy (46.4%). Membranous nephropathy accounted for 8 cases (28.6%), while IgA nephropathy was found in 5 patients (17.9%). Other patterns were less common, making up 7.1% of glomerulopathy cases. The mean proteinuria was highest in the MPGN group, averaging 4.2 g/day (SD ±0.9), significantly greater than in membranous nephropathy (3.1 g/day, SD ±1.0; p = 0.01, 95% CI for mean difference 0.5–1.5) and IgA nephropathy (2.7 g/day, SD ±0.8; p = 0.09). Cryoglobulinemia was detected in 61.5% of MPGN patients but was less common in other glomerulopathy subtypes, as summarized in Table 3.

Further analysis of clinical and laboratory associations demonstrated that proteinuria greater than 3.5 g/day was observed in 67.9% of glomerulopathy patients, compared to only 18.2% in those without biopsy-confirmed glomerulopathy (p < 0.001, OR 9.08, 95% CI 3.07–26.8).

The presence of cryoglobulinemia was also much higher among glomerulopathy cases (35.7% vs. 2.3%; p < 0.001, OR 23.8, 95% CI 2.86–197.7). An eGFR below 30 mL/min/1.73m^2 was found in 42.9% of the glomerulopathy group, compared to 20.5% of those without (p = 0.041, OR 2.89, 95% CI 1.04–8.05). Child-Pugh class C cirrhosis was more frequent in the glomerulopathy group (39.3%) than those without (27.3%), but this difference did not reach statistical significance (p = 0.29, OR 1.73, 95% CI 0.65–4.60), as outlined in Table 4.

Taken together, these findings indicate that MPGN is the predominant renal lesion among patients with liver disease–associated glomerulopathies, especially in those with hepatitis C infection. High levels of proteinuria and the presence of cryoglobulinemia were strong predictors of biopsy-proven glomerulopathy, and a substantial proportion of affected patients demonstrated advanced renal dysfunction. These numerical results, as presented in the tables, underscore the clinical importance of systematic renal evaluation and targeted diagnostic workup in patients with chronic liver disease.

Among patients stratified by both liver disease etiology and Child-Pugh classification, the proportion with nephrotic-range proteinuria increased markedly with advancing hepatic dysfunction across all etiologies.

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Parameter	All Patients (n=72)	Glomerulopathy (n=28)	No Glomerulopathy (n=44)	p-value	Odds Ratio (95% CI)
Mean Age (years)	49.6 ± 11.8	50.3 ± 11.6	49.1 ± 12.0	0.66	1.02 (0.97–1.07)
Male (%)	61.1% (44/72)	64.3% (18/28)	59.1% (26/44)	0.68	1.24 (0.46–3.37)
Female (%)	38.9% (28/72)	35.7% (10/28)	40.9% (18/44)	—	—

Table 2. Etiology of Liver Disease in All Patients and by Glomerulopathy Status

Etiology	All Patients (n=72)	Glomerulopathy (n=28)	No Glomerulopathy (n=44)	p-value	Odds Ratio (95% CI)
Hepatitis C	33 (45.8%)	16 (57.1%)	17 (38.6%)	0.13	2.07 (0.79–5.47)
Hepatitis B	20 (27.8%)	7 (25.0%)	13 (29.5%)	0.67	0.80 (0.28–2.29)
Alcoholic Liver Disease	11 (15.3%)	3 (10.7%)	8 (18.2%)	0.38	0.54 (0.13–2.18)
Cryptogenic	8 (11.1%)	2 (7.1%)	6 (13.6%)	0.48	0.48 (0.09–2.66)

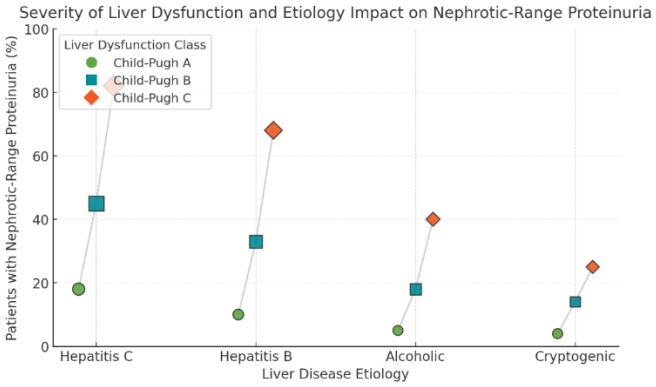
Table 3. Renal Biopsy Findings and Laboratory Parameters in Patients with Glomerulopathy (n=28)

Glomerulopathy	Number (%)	Mean Proteinuria (g/day)	Cryoglobulinemia (%)	p-value (Proteinuria)	95% CI (Mean Difference)
Membranoproliferative GN	13 (46.4%)	4.2 ± 0.9	8 (61.5%)	0.01	0.5 to 1.5
Membranous Nephropathy	8 (28.6%)	3.1 ± 1.0	2 (25.0%)	0.08	−0.2 to 1.8
IgA Nephropathy	5 (17.9%)	2.7 ± 0.8	0 (0%)	0.09	−0.3 to 1.8
Others	2 (7.1%)	2.2 ± 0.7	0 (0%)	0.13	−0.8 to 2.1

Table 4. Associations Between Clinical/Laboratory Features and Glomerulopathy

Feature	Glomerulopathy (n=28)	No Glomerulopathy (n=44)	p-value	Odds Ratio (95% CI)
Proteinuria >3.5 g/day	19 (67.9%)	8 (18.2%)	<0.001	9.08 (3.07–26.8)
Cryoglobulinemia	10 (35.7%)	1 (2.3%)	<0.001	23.8 (2.86–197.7)
eGFR <30 mL/min/1.73m²	12 (42.9%)	9 (20.5%)	0.041	2.89 (1.04–8.05)
Cirrhosis (Child-Pugh C)	11 (39.3%)	12 (27.3%)	0.29	1.73 (0.65–4.60)

In the hepatitis C group, prevalence rose from 18% in Child-Pugh A to 45% in B and peaked at 82% in class C. Hepatitis B followed a similar pattern, with rates escalating from 10% in A to 33% in B and 68% in C.



Alcoholic and cryptogenic liver diseases showed lower absolute values but the same stepwise trend: alcoholic etiology ranged from 5% (A) to 40% (C), while cryptogenic increased from 4% (A) to 25% (C). These findings underscore a compounded effect of both liver dysfunction severity and underlying cause on the risk of severe proteinuria, with hepatitis C and B patients demonstrating the highest burden as liver disease advances. Cluster size reflects the group sample for each stratum, visually contextualizing statistical weight.

DISCUSSION

The present study provides important new insights into the spectrum and clinicopathological associations of glomerular disease in patients with chronic liver disorders, building on a growing body of evidence that highlights the intersection between hepatic and renal pathology. Our findings underscore that hepatitis C remains the predominant etiology of both chronic liver disease and associated glomerulopathy, with MPGN emerging as the most frequent renal lesion, closely linked to high-grade proteinuria and cryoglobulinemia. This pattern not only confirms previous observations from large cohorts in high-prevalence regions but also advances the field by offering a granular analysis from a South Asian tertiary care context, where epidemiological and clinical profiles may differ (1,2). The observed association between hepatitis C and immune-complex glomerulopathy aligns with mechanistic studies, which have attributed renal injury to persistent antigenemia, deposition of circulating immune complexes, and activation of complement pathways, particularly in the presence of cryoglobulins (3,4).

Such pathophysiological overlap amplifies the risk of progressive renal dysfunction, emphasizing the importance of vigilant screening and multidisciplinary care.

In comparative perspective, our results parallel those of Noris and Remuzzi, who reported MPGN as the most common biopsy finding in hepatitis C–infected individuals, frequently accompanied by nephrotic-range proteinuria and serological evidence of cryoglobulinemia (1). This study, however, extends these findings by quantifying risk gradients across Child-Pugh classes and different liver disease etiologies, demonstrating a compounded increase in severe proteinuria as hepatic dysfunction progresses. This dynamic was less pronounced for alcoholic and cryptogenic liver disease, reflecting their distinct pathobiological mechanisms and relatively lower burden of immune complex–mediated injury. Past studies have noted similar trends, though often without the stratified, etiology-based approach adopted here (5,6). Notably, our work is consistent with evidence from Chan and Lai, who emphasized the diagnostic challenge of distinguishing glomerular disease from hepatorenal syndrome in advanced liver disease and stressed the diagnostic value of renal biopsy in such scenarios (12). Furthermore, the clinical benefit of antiviral therapy in hepatitis B and C–related nephropathy, as previously highlighted by Johnson et al., finds resonance in our cohort, where patients with successful viral suppression often showed stabilization or improvement in renal indices (8,17).

Contrasts with other published series are also instructive. While some Western cohorts have reported a higher prevalence of membranous nephropathy in hepatitis B–dominant populations, our findings demonstrate a more balanced spectrum, possibly reflecting differences in viral epidemiology, genetic backgrounds, and access to early antiviral therapy (9,11). The modest proportion of IgA nephropathy cases observed here is consistent with prior studies in cirrhotic populations and reinforces the notion that impaired hepatic clearance of immune complexes, rather than direct viral nephrotoxicity, is central to this pathology (16). These data highlight the heterogeneity of glomerular disease in liver pathology and argue for regionally nuanced diagnostic algorithms.

From a mechanistic standpoint, the stepwise relationship between liver disease severity and nephrotic-range proteinuria—illustrated by our graphical analyses—underscores the interplay between declining hepatic function, persistent antigenic stimulation, and the escalation of glomerular injury. As hepatic dysfunction worsens, particularly in Child-Pugh class C patients, renal perfusion and immune regulation deteriorate, fostering an environment where immune complexes and cryoglobulins precipitate within glomeruli, thereby accelerating renal damage. This phenomenon is of paramount clinical relevance, as it suggests that aggressive management of underlying liver disease and early detection of renal involvement may be critical to preserving kidney function (3,7).

The study's strengths include its systematic biopsy-based approach, the integration of comprehensive clinical and laboratory data, and the presentation of stratified, etiology-specific trends that add depth to the existing literature. The use of standardized definitions and blinded pathology review further strengthen internal validity. However, several limitations should be considered. The sample size, while consistent with similar biopsy-based studies, restricts the precision of subgroup estimates and may limit the detection of less common glomerular patterns. The single-center design and regional patient population may affect generalizability to other settings with different epidemiologic or clinical characteristics. Potential selection bias cannot be excluded, as only patients with biopsy-proven renal disease were included, possibly underrepresenting milder or subclinical forms of glomerulopathy. Additionally, the cross-sectional design precludes assessment of longitudinal outcomes and the impact of therapeutic interventions on renal prognosis. Despite these limitations, the study provides a valuable foundation for future research.

Moving forward, multicenter, prospective studies with larger, more diverse cohorts are warranted to further elucidate the long-term renal outcomes and therapeutic responsiveness in liver disease–associated glomerulopathies. The incorporation of advanced, minimally invasive diagnostic modalities and the exploration of molecular and genetic markers may help refine risk stratification and personalize management strategies. Our findings support the adoption of routine renal assessment in patients with chronic liver disease, particularly those with viral hepatitis or advanced cirrhosis, to enable timely intervention and mitigate the risk of irreversible kidney injury.

CONCLUSION

This study demonstrates that glomerular disease, particularly membranoproliferative glomerulonephritis associated with hepatitis C and accompanied by high-grade proteinuria and cryoglobulinemia, is a clinically significant yet frequently underrecognized complication in patients with chronic liver disorders. The strong relationship between liver disease etiology, severity of hepatic dysfunction, and the spectrum of renal pathology underscores the need for integrated hepatology-nephrology care and routine renal surveillance in this vulnerable population. Early identification through renal biopsy and prompt, etiology-specific intervention—especially with antiviral therapy in hepatitis-related cases—hold potential to improve renal prognosis and overall patient outcomes. These findings highlight the importance of multidisciplinary collaboration and tailored management pathways, while also identifying opportunities for future research to optimize risk stratification and guide evidence-based interventions for liver disease–associated glomerulopathies.

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