

STAGE-DEPENDENT HEPATITIS C RISK AND OUTCOMES IN PRE-DIALYSIS CHRONIC KIDNEY DISEASESaira Anwar^{*1}, Imran Qaiser², Anam³^{*1,2,3} Assistant Professor, Department of Medical Sciences, University of Bahawalpur¹sairaanwar14@gmail.com, ²imranqaisar@gmail.com, ³anam765@yahoo.com**Keywords**

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Abstract

Hepatitis C virus (HCV) is a significant comorbidity in chronic kidney disease (CKD), yet its prevalence in pre-dialysis populations is underexplored. This cross-sectional study assessed 220 pre-dialysis CKD patients at a tertiary care hospital in Karachi. HCV screening was performed via ELISA, and demographic, clinical, and laboratory data were analyzed using SPSS v16. HCV prevalence was 13.2% (n=29), predominantly in Stage 4 CKD (86.2%, p<0.001), with no cases detected in Stages 2–3. Traditional metabolic risk factors, including hypertension and diabetes, were not significantly associated with HCV positivity. Lower education levels showed a marginal association (p=0.03), while blood transfusions and surgical history were non-significant. These results indicate that HCV disproportionately affects advanced pre-dialysis CKD patients, independent of conventional metabolic risks. The findings support routine HCV screening in Stage 4 CKD and timely antiviral therapy to mitigate renal decline, alongside targeted prevention strategies considering socioeconomic determinants.

INTRODUCTION

Chronic kidney disease (CKD) and hepatitis C virus (HCV) infection represent significant global health challenges, each contributing to substantial morbidity and mortality. CKD, characterized by progressive loss of renal function, affects millions worldwide, with its prevalence rising due to aging populations and increasing rates of diabetes and hypertension (1). HCV, a blood-borne pathogen, infects an estimated 170 million people globally, with a particularly high burden in developing regions like Pakistan (2). While the hepatic consequences of HCV, such as cirrhosis and hepatocellular carcinoma, are well-documented, its extrahepatic manifestations, including renal involvement, are increasingly recognized but remain understudied (3). The intersection of these two conditions poses unique clinical and public health dilemmas, particularly in resource-limited settings where

diagnostic and therapeutic options are constrained (4).

The relationship between HCV and CKD is bidirectional. HCV can directly induce glomerular injury through immune-complex deposition, cryoglobulinemia, or viral replication within renal tissue, accelerating CKD progression (5). Conversely, CKD patients, especially those on dialysis, are at heightened risk of HCV acquisition due to frequent healthcare exposures (6). Despite this interplay, most research has focused on HCV prevalence in end-stage renal disease (ESRD) patients on hemodialysis, leaving a gap in understanding its impact on pre-dialysis CKD populations (7). Early-stage CKD patients often remain asymptomatic until advanced renal impairment occurs, delaying diagnosis and intervention (8). HCV infection in this subpopulation may further hasten renal decline, yet

screening protocols are not standardized, and local data from regions like Pakistan are sparse (9). This oversight is critical, as early HCV detection and treatment could mitigate renal deterioration, reduce complications, and improve outcomes (10).

The rationale for this study stems from the urgent need to quantify HCV prevalence in pre-dialysis CKD patients and identify associated risk factors. Existing literature suggests HCV prevalence in CKD ranges from 6% to 20%, but regional variations are stark, and Pakistan's high HCV endemicity (4.8% seroprevalence) demands localized evidence (1, 9). Prior studies in tertiary care settings, such as Cavoli et al. (2011), reported a 6.25% HCV prevalence in pre-dialysis CKD, but similar data from South Asia are limited (5). Furthermore, demographic and clinical correlates, such as age, diabetes, or prior blood transfusions, remain poorly characterized in this context (3, 6). Addressing these gaps is essential to inform screening guidelines, optimize antiviral therapy timing, and allocate resources effectively (7). By examining HCV frequency in pre-dialysis CKD patients at a Karachi tertiary hospital, this study aims to provide actionable insights for clinicians and policymakers, ultimately reducing the dual burden of HCV and CKD in high-prevalence settings (10).

The clinical implications are profound. HCV treatment with direct-acting antivirals (DAAs) has revolutionized outcomes, achieving cure rates >95%, but access remains uneven in low-income countries (4, 7). If HCV is identified early in CKD, antiviral therapy could preserve renal function, avert dialysis dependence, and reduce transmission risks (2, 8). Conversely, undiagnosed HCV in CKD populations may exacerbate renal injury, increase cardiovascular morbidity, and complicate future transplantation (5, 9). This study's findings will underscore the importance of integrating HCV screening into routine CKD management, aligning with global efforts to eliminate HCV as a public health threat (1, 10). By bridging the evidence gap, this research seeks to catalyze targeted interventions, improve patient prognoses, and highlight the interconnectedness of infectious and non-communicable disease burdens in vulnerable populations (3, 6).

Literature Review

Chronic kidney disease (CKD) and hepatitis C virus (HCV) infection share a complex, bidirectional relationship that significantly impacts disease progression and clinical outcomes. While HCV is primarily recognized for its hepatic effects, emerging evidence highlights its profound extrahepatic manifestations, particularly on renal function (15). The virus can directly infiltrate kidney tissue, triggering immune-mediated damage and accelerating CKD progression through various pathological mechanisms (19).

One of the hallmark renal manifestations of HCV is cryoglobulinemic vasculitis, which leads to membranoproliferative glomerulonephritis (MPGN) (16). This condition occurs when immune complexes deposit in the glomeruli, activating inflammatory pathways and causing progressive kidney damage (17). Beyond direct viral effects, HCV contributes to renal dysfunction by inducing metabolic disturbances such as insulin resistance and dyslipidemia, which exacerbate existing conditions like diabetic nephropathy (12). The virus also stimulates the production of pro-fibrotic factors that promote tubulointerstitial fibrosis, leading to irreversible nephron loss and declining glomerular filtration rates (18).

The clinical implications of HCV in CKD populations are substantial. Patients with concurrent HCV infection tend to experience more rapid CKD progression and worse outcomes compared to their non-infected counterparts (9,10). This accelerated disease course underscores the importance of early HCV detection and intervention in CKD management (11). While the relationship between HCV and end-stage renal disease has been well-documented in dialysis populations (14), the impact of HCV on earlier stages of CKD remains less understood, particularly in high-prevalence regions (13).

Effective management of HCV in CKD patients has been revolutionized by direct-acting antiviral (DAA) therapies (11). These treatments not only achieve high rates of viral eradication but also demonstrate potential renal protective effects, including reduced proteinuria and slowed GFR decline (12). However, treatment considerations must account for renal

function, as some antiviral agents require dose adjustments in advanced CKD (13). The timing of HCV therapy in CKD progression also presents clinical challenges, balancing the benefits of early intervention against potential drug-related nephrotoxicity (7,8).

Despite these advances, significant knowledge gaps persist regarding HCV's epidemiology and impact in pre-dialysis CKD populations (9,10). Variations in regional prevalence, transmission risks, and healthcare access further complicate the clinical picture (14). Addressing these gaps through targeted research is crucial for developing optimized screening protocols and treatment strategies tailored to CKD patients at different disease stages (11,12).

This evolving understanding of the HCV-CKD relationship highlights the need for integrated care approaches that address both viral infection and renal function preservation (15,16). Future research directions should focus on elucidating the mechanisms of HCV-related renal injury (17,18), optimizing treatment protocols for CKD patients (11,13), and investigating long-term outcomes following viral eradication (19). Such efforts will be instrumental in improving clinical management and outcomes for this vulnerable patient population (7,14).

The current study is built upon several key hypotheses that emerge from the complex interplay between hepatitis C virus (HCV) infection and chronic kidney disease (CKD) progression. These hypotheses are grounded in the pathophysiological mechanisms linking viral infection to renal dysfunction (19) and are further supported by clinical observations in pre-dialysis populations (20). The primary theoretical framework suggests that HCV infection acts as an independent risk factor for renal function deterioration in CKD patients, even in early disease stages (21), through both direct viral effects (15) and indirect systemic consequences (6).

At the core of the hypothesis development lies the understanding that HCV exerts nephrotoxic effects through multiple pathways. The virus is postulated to directly infect renal cells (19), leading to local inflammation and tissue damage. This direct cytopathic effect is complemented by immune-mediated injury (16), where circulating immune

complexes deposit in glomerular structures (17), triggering complement activation and subsequent inflammatory responses (18). These processes are theorized to manifest clinically as declining glomerular filtration rates and worsening proteinuria (12), even in patients who have not yet reached end-stage renal disease (9). The study specifically examines whether these effects are detectable across different CKD stages (10), with particular attention to earlier stages that have been less thoroughly investigated (21).

The second major hypothesis concerns the role of traditional risk factors in modifying the HCV-CKD relationship. Theoretical considerations suggest that common comorbidities such as diabetes and hypertension may interact synergistically with HCV infection to accelerate renal decline (6). This interaction is hypothesized to occur through several mechanisms, including exacerbation of endothelial dysfunction (15), amplification of oxidative stress (22), and potentiation of inflammatory pathways (16). The study explores whether the presence of these comorbidities results in a more pronounced effect of HCV on renal parameters compared to patients without such conditions (20). This aspect is particularly relevant for clinical practice, as it may help identify high-risk subgroups that would benefit most from targeted screening and early antiviral intervention (11,13).

A third hypothesis focuses on the potential reversibility of HCV-related renal damage. Emerging theoretical models suggest that successful viral eradication may stabilize or even improve renal function in pre-dialysis CKD patients (11,21). This hypothesis is built on the premise that removing the viral trigger could interrupt the cascade of inflammatory and fibrotic processes damaging the kidneys (17,19). The study examines this possibility by analyzing renal function parameters in relation to HCV status (12), with implications for treatment timing and the potential renal benefits of early antiviral therapy in CKD populations (13,21).

Demographic factors form another component of the theoretical framework. The hypothesis development considers potential variations in HCV's renal impact based on age, gender, and socioeconomic status (20). Theoretical models

suggest that older patients and those from disadvantaged backgrounds may experience more severe HCV-related renal consequences due to accumulated comorbidities (6), reduced immunological resilience (22), and barriers to healthcare access (20). These considerations inform the study's stratified analyses and may help explain disparities in renal outcomes among HCV-infected CKD patients (9,10).

The duration of CKD emerges as another critical variable in the hypothesis development. Longer-standing CKD is theorized to create a more vulnerable renal environment where HCV infection can exert more pronounced damaging effects (19). This hypothesis stems from the understanding that kidneys with pre-existing structural damage and reduced functional reserve may be less capable of withstanding additional insults from viral infection (17,18). The study specifically tests whether disease duration modifies the association between HCV and renal parameters (10).

Lastly, the theoretical framework incorporates the potential role of healthcare-related exposures in HCV acquisition among CKD patients (9,10). The hypothesis suggests that certain medical interventions, particularly blood transfusions and surgical procedures, may serve as important routes of HCV transmission in this population (14). This aspect of the study has important implications for infection control practices in nephrology settings (7) and may help identify modifiable risk factors for HCV infection in CKD patients (8).

These interconnected hypotheses collectively form a comprehensive theoretical model that guides the study's design and analysis (20). The model not only addresses the basic question of HCV prevalence in pre-dialysis CKD but also explores the nuanced relationships between viral infection, renal function trajectories, and modifying factors (15,19). This multifaceted approach allows for a more complete understanding of how HCV impacts CKD progression (17) and which patient subgroups are most vulnerable to its nephrotoxic effects (6,16). The theoretical development emphasizes the clinical relevance of these relationships (11,13), particularly in resource-limited settings where both HCV and CKD represent significant public health challenges

(20). By testing these hypotheses, the study aims to provide evidence that can inform screening protocols (21), treatment algorithms (12,13), and preventive strategies (7,8) for this high-risk patient population.

Methodology

This cross-sectional study was conducted at the Department of Nephrology, Abbasi Shaheed Hospital, Karachi. The target population comprised pre-dialysis CKD patients attending the outpatient clinic. A sample of 220 participants was determined using WHO sample size software, based on an expected HCV prevalence of 6.25%, a 95% confidence level, and a 3.2% margin of error. Non-probability consecutive sampling was employed to ensure representation of the routine patient flow while minimizing selection bias.

The study adopted a positivist research philosophy, relying on objective clinical measurements to examine the association between HCV and CKD. Data were collected prospectively using a structured proforma after obtaining verbal consent. Inclusion criteria required patients aged 30–60 years with confirmed pre-dialysis CKD (eGFR <60 mL/min/1.73m² for >6 months). Exclusion criteria eliminated those with prior HCV/HIV/hepatitis B infection, pregnancy, thyroid disorders, congestive cardiac failure, chronic liver disease, stroke, COPD, or current antilipidemic drug use to control confounding variables.

Trained investigators collected demographic and clinical data, including age, gender, CKD stage, comorbidities (diabetes, hypertension), and HCV risk factors (blood transfusion history, surgery). HCV status was determined via ELISA testing of blood samples. Data analysis utilized SPSS v16. Descriptive statistics (mean \pm SD) summarized continuous variables (age, CKD duration), while frequencies/percentages described categorical variables (HCV prevalence, comorbidities). Chi-square tests ($p \leq 0.05$) assessed associations post-stratification by CKD stage, comorbidities, and demographic factors. The methodology prioritized internal validity through strict diagnostic criteria and standardized data collection protocols.

This approach balanced feasibility with scientific rigor, providing actionable insights into HCV

frequency in a high-risk, understudied population while controlling for major confounders. The design facilitated efficient data collection within resource constraints, aligning with the study's pragmatic objectives.

Data Analysis and Results

The collected data were analyzed using SPSS version 16, with both descriptive and inferential statistical methods applied to assess the frequency of hepatitis C (HCV) in pre-dialysis chronic kidney disease (CKD) patients. The study included 220 pre-dialysis CKD patients, with a mean age of 56.78 ± 2.81 years (range: 36–59 years). The mean duration of CKD was 5.72 ± 1.24 years. Gender

distribution revealed that 56.4% (n=124) were male, while 43.6% (n=96) were female. Among the participants, 13.2% (n=29) tested positive for HCV, while the majority (86.8%, n=191) were HCV-negative.

The data were stratified to examine associations between HCV status and key variables:

CKD Stage & HCV Prevalence

- Stage 4 CKD had the highest HCV prevalence (86.2% of HCV+ cases).
- No HCV+ cases were found in Stage 2 or 3 CKD.

Table 1 CKD Stage & HCV Prevalence

CKD Stage	HCV+ (n=29)	HCV- (n=191)	Total (n=220)
Stage 2	0 (0%)	70 (36.6%)	70 (31.8%)
Stage 3	0 (0%)	53 (27.7%)	53 (24.1%)
Stage 4	25 (86.2%)	29 (15.2%)	54 (24.5%)
Stage 5	4 (13.8%)	39 (20.4%)	43 (19.5%)
p-value	0.00		

The analysis of comorbidities and HCV status revealed that hypertension was present in 55.2% of HCV-positive patients compared to 45% of HCV-negative patients, though this difference was not statistically significant ($p=0.20$). Similarly, diabetes mellitus type II was observed in 34.5% of HCV-positive cases versus 38.7% of HCV-negative cases ($p=0.41$), indicating no significant association. Examination of potential risk factors showed that 48.3% of HCV-positive patients had a history of blood transfusion compared to 42.4% of HCV-negative patients ($p=0.34$), while surgical history was reported in 31% of HCV-positive cases versus 28.3% of HCV-negative cases ($p=0.45$), neither reaching statistical significance. Statistical analysis employed chi-square tests for categorical variables including gender, CKD stage, and comorbidities, while independent t-tests compared mean differences in age and CKD duration between HCV-positive and negative groups, with a p-value ≤ 0.05 considered statistically significant. The most notable finding was the strong association between advanced CKD (Stage 4) and HCV positivity ($p=0.00$), while no significant

relationships were detected between HCV status and diabetes, hypertension, smoking, or socioeconomic status. A marginal association was observed with educational status ($p=0.03$), showing higher HCV prevalence among less educated groups. These results underscore the particular vulnerability of late-stage CKD patients to HCV infection, suggesting the importance of routine HCV screening in pre-dialysis populations, especially those with Stage 4 disease. The absence of clear links between HCV and traditional metabolic risk factors implies that HCV may exert independent effects on renal deterioration, warranting further investigation into the specific mechanisms by which HCV influences CKD progression in this patient population.

Discussion

The findings of this study reveal several important insights into the relationship between HCV infection and pre-dialysis CKD. The overall HCV prevalence of 13.2% in our cohort aligns closely with previous reports from similar settings, though it appears higher than the 6.25% prevalence reported

by Cavoli et al. [18] in their study of pre-dialysis CKD patients. This discrepancy may reflect regional variations in HCV epidemiology or differences in study populations.

The most striking observation was the significant association between advanced CKD (Stage 4) and HCV positivity ($p=0.00$). This finding supports the growing body of evidence suggesting that HCV may accelerate renal function decline [19,22]. The absence of HCV-positive cases in Stages 2 and 3 CKD is particularly noteworthy and may indicate either a true biological phenomenon where HCV preferentially affects more compromised kidneys, or possibly a detection bias where viral effects become more apparent in advanced disease.

Contrary to some previous reports [6,15], we found no significant associations between HCV status and traditional comorbidities like diabetes ($p=0.41$) or hypertension ($p=0.20$). This suggests that HCV may influence renal progression through mechanisms independent of these metabolic factors, possibly through direct viral effects on renal tissue or immune-mediated pathways [17,19]. The marginal association with lower educational status ($p=0.03$) echoes findings by Ahmad et al. [10] and may reflect socioeconomic determinants of healthcare access and infection risk.

The lack of significant associations with recognized HCV risk factors like blood transfusions ($p=0.34$) or surgical history ($p=0.45$) was somewhat unexpected, given established literature on these transmission routes [9,10]. This could indicate changing patterns of HCV acquisition in our setting or possibly limitations in patient recall of historical exposures.

These findings have important clinical implications. First, they support routine HCV screening in pre-dialysis CKD populations, particularly those with advanced disease. Second, the potential for HCV to independently worsen renal function underscores the importance of early antiviral therapy, especially considering recent evidence that DAA treatment can stabilize renal function in CKD patients [11,12,21]. Third, the socioeconomic patterns observed suggest targeted screening and education programs may be warranted in vulnerable populations.

Several limitations should be acknowledged. The single-center design may limit generalizability, and

the cross-sectional nature prevents causal inferences. Additionally, we lacked histological data to explore specific mechanisms of HCV-related renal injury. Future longitudinal studies with detailed virological and histological characterization would help clarify the temporal relationship between HCV infection and CKD progression.

Conclusion

This study highlights a significant HCV burden (13.2%) among pre-dialysis CKD patients, particularly in advanced stages (Stage 4 CKD). The strong association between HCV and late-stage renal disease, without clear links to traditional risk factors like diabetes or hypertension, suggests HCV may independently worsen kidney function. The marginal link to lower education levels points to socioeconomic influences on infection risk. These findings reinforce the need for routine HCV screening in CKD populations, especially as early antiviral therapy could potentially slow renal decline. While the cross-sectional design limits causal interpretations, the results underscore HCV as a modifiable risk factor in CKD progression, warranting closer integration of hepatology and nephrology care.

Clinicians should prioritize HCV screening for all pre-dialysis CKD patients, particularly those with Stage 4 disease, given the high prevalence and potential for renal deterioration. Early detection and treatment with DAAs may preserve kidney function and improve outcomes. The lack of association with typical metabolic risk factors suggests HCV's role may be underrecognized, prompting a shift toward viral testing even in patients without traditional risk profiles. Additionally, socioeconomic factors (e.g., education level) should inform targeted patient education and prevention strategies. Nephrologists and hepatologists should collaborate to optimize HCV management in CKD, balancing antiviral efficacy with renal safety.

Future studies should employ longitudinal designs to clarify HCV's causal role in CKD progression and assess whether DAA therapy mitigates renal decline. Research should explore mechanisms (e.g., viral persistence in renal tissue, immune-mediated injury) through biopsy-based studies. Larger, multicenter

cohorts could validate socioeconomic risk factors and regional prevalence patterns. Trials comparing early versus deferred HCV treatment in CKD stages 1–3 would help define optimal intervention timing. Finally, cost-effectiveness analyses of universal HCV screening in CKD populations are needed, particularly in resource-limited settings with high HCV burden. Such evidence could refine guidelines and improve outcomes for this high-risk group.

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