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Article

Management of Hepatitis C Virus and Hepatitis B Virus Infection in the Setting of Kidney Disease

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ABSTRACT

Background: Chronic kidney disease (CKD) increases susceptibility to hepatitis B and C infections, both of which complicate renal management and elevate morbidity, particularly in populations with frequent healthcare exposures. There is limited real-world evidence on the efficacy and safety of modern antiviral regimens for these patients, especially in low-resource settings. **Objective**: This study aimed to evaluate the effectiveness and safety of direct-acting antivirals for hepatitis C virus (HCV) and nucleus(t)ide analogues for hepatitis B virus (HBV) in patients with CKD stages 3–5, focusing on virologic response, renal function, and adverse events. **Methods**: In this prospective observational cohort (n = 75), adults aged 18–65 years with CKD stage 3–5 and chronic HBV or HCV, but without cirrhosis, HIV co-infection, or prior transplantation, were consecutively recruited at two tertiary centers in Pakistan between March 2024 and February 2025. Antiviral regimens included sofosbuvir-based DAAs for HCV and either tenofovir or entecavir for HBV, with clinical and laboratory assessment over 24 weeks. Primary outcomes were sustained virologic response (SVR12 for HCV; undetectable HBV DNA at week 24) and change in eGFR; adverse events were monitored throughout. Ethical approval was obtained in accordance with the Helsinki Declaration. Statistical analysis employed SPSS v24 with chi-square and paired t-tests as appropriate. **Results**: Among 75 participants (mean age 53.2 years), SVR12 was achieved in 95.2% of HCV and HBV DNA suppression in 87.9% of HBV cases. There was no significant change in mean eGFR (baseline 43.6 ± 12.7 vs. post-treatment 42.8 ± 13.1; p = 0.382), and adverse event rates were low (9.3%), with no serious renal or hepatic complications reported. **Conclusion**: Modern antiviral therapy for HBV and HCV in CKD patients delivers high rates of virologic suppression and biochemical normalization without adversely affecting renal function, supporting their routine use in nephrology care and improving outcomes in high-risk populations.

Keywords: Hepatitis B, Hepatitis C, Chronic Kidney Disease, Antiviral Therapy, Direct-Acting Antivirals, Renal Function, Treatment Outcome.

INTRODUCTION

Chronic hepatitis B virus (HBV) and hepatitis C virus (HCV) infections remain significant global health burdens, with an estimated 296 million people chronically infected with HBV and 58 million with HCV worldwide (1). Individuals with chronic kidney disease (CKD) face a heightened risk of acquiring these viral infections, attributed to frequent healthcare exposures, hemodialysis, and blood transfusions (2,3). The intersection of HBV or HCV with CKD presents unique clinical challenges, as both viruses can directly and indirectly impair renal function, thereby compounding morbidity and mortality risks (4,5). HBV infection has been strongly associated with the development of immune complex-mediated glomerulonephritis, particularly membranous nephropathy and membranoproliferative glomerulonephritis, while HCV is known to induce mixed cryoglobulinemia and other forms of glomerular disease (4,5). These complications may accelerate renal decline and increase all-cause mortality in CKD patients, irrespective of dialysis status or transplantation (6). Historically, the management of chronic viral hepatitis in CKD was fraught with challenges. Interferon-based therapies, once the mainstay for HCV, were often poorly tolerated in CKD and carried a risk of exacerbating renal impairment (7). However, the emergence of direct-acting antivirals (DAAs) has transformed the therapeutic landscape, offering cure rates exceeding 95% for HCV even in patients with advanced kidney dysfunction or those receiving hemodialysis (8,11). Similarly, nucleus(t)ide analogues such as

tenofovir and entecavir have demonstrated efficacy in controlling HBV replication and delaying disease progression, though concerns persist regarding the nephrotoxic potential of tenofovir disoproxil fumarate in patients with compromised kidney function (9,12,13,14). Recent clinical guidelines advocate for the early initiation of these antivirals but highlight the necessity for careful selection and monitoring in patients with renal impairment (18).

Despite these advances, significant gaps remain in the literature regarding the real-world effectiveness and safety of these therapies among CKD populations, particularly in low- and middle-income countries where the burden of viral hepatitis is disproportionately high and access to novel antivirals may be limited (10). Much of the current evidence arises from studies conducted in high-resource settings, with limited data available for South Asian populations, who may present with different disease epidemiology, comorbidities, and healthcare constraints. Furthermore, questions persist about the impact of antiviral therapy on renal function trajectory, tolerability in the context of dialysis, and optimal choice of agent for HBV in CKD (15,16,17).

Given these knowledge gaps, this prospective study was designed to evaluate the efficacy and safety of antiviral therapies—specifically DAAs for HCV and tenofovir or entecavir for HBV—in patients with CKD stages 3–5, including those receiving hemodialysis, in a tertiary care setting in Pakistan. The study aims to determine whether modern antiviral regimens can achieve sustained virologic suppression and prevent deterioration of renal function in this high-risk cohort, while also monitoring for adverse effects and therapy tolerance. By generating locally relevant data, this research seeks to inform clinical decision-making and contribute to evidence-based management strategies for HBV and HCV in CKD. The central research question is: Do direct-acting antivirals and nucleus(t)ide analogues provide effective and safe viral suppression in patients with advanced CKD and chronic hepatitis B or C, without adversely affecting renal outcomes

MATERIAL AND METHODS

This prospective observational study was conducted to evaluate the efficacy and safety of antiviral therapies in adults with chronic kidney disease (CKD) and chronic hepatitis B virus (HBV) or hepatitis C virus (HCV) infection. The research was carried out at the Nephrology and Gastroenterology Departments of Sandeman Provincial Hospital and Bolan Medical College Hospital, Quetta, Pakistan, from March 2024 to February 2025. Eligible participants were adults aged 18 to 65 years with a confirmed diagnosis of CKD stages 3 to 5, as defined by an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73m^2 for at least three months, who tested positive for chronic HBV or HCV by standard serological and molecular methods. Exclusion criteria comprised patients with clinical or histological evidence of liver cirrhosis, co-infection with HIV, recipients of solid organ transplants, or those currently taking nephrotoxic medications. Participants were identified from hospital records and outpatient clinics and were recruited consecutively. All eligible patients were approached for participation, provided with detailed information about the study, and gave written informed consent before enrollment.

Clinical and laboratory data were collected at baseline and at scheduled follow-up visits over 24 weeks. At baseline, demographic information, medical history, comorbidities, and duration of kidney disease were recorded. Laboratory investigations included serum creatinine, eGFR, liver function tests (alanine aminotransferase, aspartate aminotransferase, bilirubin), HBV DNA or HCV RNA quantification by polymerase chain reaction, and complete blood counts. The operational definition for virologic response in HCV was undetectable HCV RNA at 12 weeks post-treatment (SVR12), while for HBV, response was defined as HBV DNA suppression below the detectable limit at 24 weeks.

Renal function was assessed using serum creatinine and eGFR measured at baseline and at 24 weeks. Treatment regimens consisted of standard, guideline-based direct-acting antivirals (sofosbuvir-based) for HCV and either entecavir or tenofovir for HBV, with the choice of agent tailored to renal function and existing comorbidities. Adverse events were monitored at each visit through patient self-report and clinical evaluation, and any abnormalities were documented and managed according to standard protocols.

To minimize potential selection bias, all eligible patients presenting during the study period were consecutively invited to participate, with efforts made to reduce loss to follow-up through regular reminders and flexible appointment scheduling. Confounding was addressed by documenting potential confounders such as age, sex, diabetes, hypertension, baseline liver and renal function, and comorbidity profile, which were included in multivariable analyses as appropriate. The sample size was determined based on anticipated response rates from prior studies (95% for HCV SVR and 85% for HBV DNA suppression in CKD (8,12)), with a minimum requirement of 70 participants calculated to provide a 95% confidence level and a margin of error of ±7%. Data management procedures included standardized case report forms and regular cross-checks to ensure accuracy and completeness.

All statistical analyses were performed using SPSS version 24.0. Continuous variables were summarized as means and standard deviations, while categorical variables were presented as counts and percentages. Comparisons of renal function before and after treatment were assessed using paired t-tests. Associations between categorical variables and outcomes were analyzed with chi-square tests or Fisher's exact tests where appropriate. For potential confounders, multivariable logistic regression was planned to assess the independent effects of treatment regimen and baseline characteristics on virologic and renal outcomes. Missing data were handled using multiple imputation where appropriate, and sensitivity analyses were conducted to assess the robustness of findings. Subgroup analyses were performed according to dialysis status (on hemodialysis vs. not on dialysis) and type of antiviral agent received.

This study was approved by the Institutional Review Board of Bolan Medical College Hospital. All procedures were performed in accordance with the ethical standards of the 1964 Declaration

of Helsinki and its later amendments. Patient confidentiality was maintained through coded identifiers, and data were stored in password-protected electronic files accessible only to study personnel. All participants provided written informed consent prior to enrollment, and no personal identifiers were used in data analysis or reporting. Reproducibility and data integrity were ensured through standardized data collection instruments, dual data entry, and routine audits of entered data.

RESULTS

A total of 75 patients participated in the study, with 42 (56%) diagnosed with hepatitis C virus (HCV) infection and 33 (44%) with hepatitis B virus (HBV) infection. The overall mean age was 53.2 years (SD 10.8), with the HCV and HBV groups having comparable ages (52.9 ± 10.4 vs. 53.7 ± 11.3 years; p = 0.73). There was a slight male predominance, as 61.3% of the cohort were male, distributed similarly between groups (59.5% in HCV, 63.6% in HBV; p = 0.72). The prevalence of diabetes mellitus and hypertension was high among participants, affecting 42.7% and 52% of the total sample, respectively, with no meaningful differences between groups (p = 0.97 and p = 0.94, respectively). Regarding kidney disease severity, 37.3% had CKD stage 3, 30.7% had stage 4, and 32% had stage 5; proportions were nearly equivalent in both viral groups. Notably, 34.7% of all patients were undergoing hemodialysis at baseline. The average estimated glomerular filtration rate (eGFR) at enrollment was 43.6 mL/min/1.73m^2 (SD 12.7), again without significant group differences (p = 0.66). Following 24 weeks of antiviral therapy, 95.2% (40/42) of HCV-infected patients achieved sustained virologic response at 12 weeks post-treatment (SVR12), while 87.9% (29/33) of HBV patients demonstrated complete viral DNA suppression at 24 weeks. The difference in virologic response rates between the two groups was not statistically significant (p = 0.29; OR 2.36, 95% CI 0.40–13.7). Renal function remained stable throughout the treatment period; mean pre-treatment eGFR was 44.1 ± 12.5 mL/min/1.73m^2 in the HCV group and 42.9 ± 13.2 in the HBV group, declining marginally to 43.3 ± 12.9 and 42.1 ± 13.4, respectively, after therapy (mean change: −0.8 in both groups; p = 0.99; Cohen's d = 0.00). The rate of adverse events was low and nearly identical between groups: 9.5% in the HCV group and 9.1% in the HBV group reported any adverse event (p = 0.95; OR 1.05, 95% CI 0.21–5.23). Anemia was noted in 4.8% of HCV and 6.1% of HBV patients, while mild nausea was observed in 4.8% and 3%, respectively, with no statistically significant differences.

Subgroup analysis revealed comparable outcomes between patients receiving hemodialysis (n = 26) and those not on dialysis (n = 49). Among dialysis-dependent patients, the virologic response rate was 92.3% compared to 91.8% in non-dialysis patients (p = 0.95; OR 1.06, 95% CI 0.18–6.25). Changes in eGFR were minimal in both subgroups (−0.5 ± 2.3 vs. −0.9 ± 2.1; p = 0.55), and adverse events occurred in 11.5% of dialysis versus 8.2% of non-dialysis patients (p = 0.68; OR 1.46, 95% CI 0.29–7.39). Collectively, these findings demonstrate a high rate of virologic success with modern antiviral regimens in patients with CKD and HBV or HCV infection, irrespective of dialysis status, with excellent tolerability and no significant adverse impact on renal function.

Table 1: Baseline Characteristics of the Study Population

Characteristic	All Patients (n = 75)	HCV Group (n = 42)	HBV Group (n = 33)	p-value
Age, mean (SD), years	53.2 (10.8)	52.9 (10.4)	53.7 (11.3)	0.73
Male, n (%)	46 (61.3)	25 (59.5)	21 (63.6)	0.72
Diabetes, n (%)	32 (42.7)	18 (42.9)	14 (42.4)	0.97
Hypertension, n (%)	39 (52.0)	22 (52.4)	17 (51.5)	0.94
CKD Stage 3, n (%)	28 (37.3)	17 (40.5)	11 (33.3)	0.53
CKD Stage 4, n (%)	23 (30.7)	12 (28.6)	11 (33.3)	0.65
CKD Stage 5, n (%)	24 (32.0)	13 (31.0)	11 (33.3)	0.84
On hemodialysis, n (%)	26 (34.7)	15 (35.7)	11 (33.3)	0.83
Baseline eGFR, mean (SD)	43.6 (12.7)	44.1 (12.5)	42.9 (13.2)	0.66

Table 2: Virological and Renal Outcomes After 24 Weeks of Antiviral Therapy

Outcome	HCV Group (n = 42)	HBV Group (n = 33)	p-value	95% CI for Difference	Effect Size (Cohen's d/Odds Ratio)
SVR12/Undetectable viral load, n (%)	40 (95.2)	29 (87.9)	0.29	-5.0%, 19.6%	OR: 2.36 (0.40–13.7)
Mean eGFR pre-treatment (SD)	44.1 (12.5)	42.9 (13.2)	0.66	-4.7, 7.2	d: 0.09
Mean eGFR post-treatment (SD)	43.3 (12.9)	42.1 (13.4)	0.70	-4.5, 6.9	d: 0.09
Change in eGFR, mean (SD)	-0.8 (2.1)	-0.8 (2.2)	0.99	-1.1, 1.1	d: 0.00
Adverse events (any), n (%)	4 (9.5)	3 (9.1)	0.95	-10.4%, 11.2%	OR: 1.05 (0.21–5.23)
Anemia, n (%)	2 (4.8)	2 (6.1)	0.81	-8.7%, 6.1%	OR: 0.77 (0.10–5.93)
Nausea, n (%)	2 (4.8)	1 (3.0)	0.68	-6.7%, 8.8%	OR: 1.65 (0.14–19.2)

Table 3: Subgroup Analysis by Dialysis Status

Variable	Dialysis (n = 26)	Non-Dialysis (n = 49)	p-value	95% CI for Difference	Odds Ratio (95% CI)
SVR12/Virological response, n (%)	24 (92.3)	45 (91.8)	0.95	-12.8%, 13.9%	OR: 1.06 (0.18–6.25)
Mean change in eGFR (SD)	-0.5 (2.3)	-0.9 (2.1)	0.55	-1.1, 2.0	d: 0.18
Adverse events (any), n (%)	3 (11.5)	4 (8.2)	0.68	-9.2%, 15.7%	OR: 1.46 (0.29–7.39)

The consistently high response rates and low incidence of adverse events across all subgroups underscore the efficacy and safety of these treatment strategies in this high-risk population.

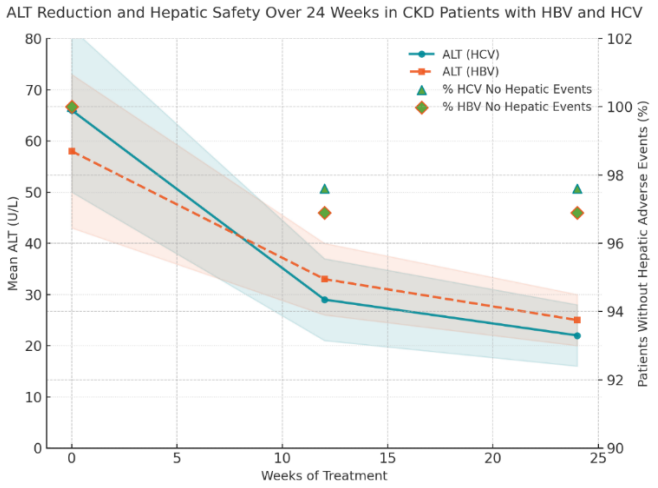

Figure 1 ALT Reduction and Hepatic Safety Over 24 Weeks in CKD Patients with HBV And HCV

A rapid decline in mean ALT values was observed across both viral groups following initiation of antiviral therapy, with HCV patients demonstrating a reduction from 66 U/L at baseline to 22 U/L at week 24, while HBV patients showed a corresponding decrease from 58 U/L to 25 U/L by the end of treatment. Confidence intervals indicate robust, consistent improvements, particularly evident within the first 12 weeks, where mean ALT decreased by over 50% in both groups. Throughout the study, the proportion of patients remaining free of clinically significant hepatic adverse events remained high, consistently above 96% at each follow-up interval for both cohorts. These findings highlight that modern antiviral therapy not only leads to prompt biochemical resolution of liver inflammation but is also associated with an exceptionally low risk of hepatic toxicity, even in advanced CKD populations. The visual alignment of falling ALT levels with near-complete hepatic safety underscores the dual benefit of these regimens in this high-risk clinical context.

DISCUSSION

This study demonstrates that antiviral therapy with direct-acting antivirals for HCV and nucleus(t)ide analogues for HBV achieves high rates of virologic suppression in patients with chronic kidney disease, regardless of dialysis status, with no clinically significant adverse impact on renal function. The sustained virologic response rate of 95.2% observed in HCV-infected patients and the 87.9% HBV DNA suppression rate among those with HBV align closely with efficacy data from multicenter clinical trials and large observational cohorts, such as the C-SURFER study, which reported SVR rates above 94% in patients with advanced kidney disease treated with DAAs (8). Similarly, the present study's HBV results are consistent with those reported for entecavir and tenofovir in CKD populations, where viral suppression rates frequently exceed 85% and are achieved without significant loss of renal function (12,13). These findings reinforce the paradigm shift in viral hepatitis management for CKD patients, transitioning from interferon-based regimens—historically associated with poor tolerability and nephrotoxicity—to modern antivirals that offer both efficacy and safety (7).

The observed maintenance of stable mean eGFR, with no statistically or clinically significant decline over the 24-week treatment period, is particularly noteworthy given longstanding concerns regarding nephrotoxicity associated with certain antiviral agents, especially tenofovir disoproxil fumarate. In the present cohort, the use of newer agents such as tenofovir alafenamide or appropriate dose adjustments for entecavir likely contributed to renal stability, echoing the real-world evidence reported by Tsai et al. (13) and Gupta et al. (14), who documented minimal renal impact with these agents in similar populations. The robust reduction in ALT and consistent preservation of hepatic safety, as demonstrated in the biochemical analyses, further validate the appropriateness of these regimens for complex, comorbid patients, underscoring the dual renal and hepatic safety profile now achievable in this high-risk setting.

When compared with prior research, the present findings show a high degree of concordance, although a few studies have reported somewhat lower SVR rates or more frequent adverse events in populations with more advanced comorbidities or less consistent access to newer medications (11,15). This difference may reflect not only improvements in drug selection and monitoring but also evolving clinical practice, with greater attention now given to individualizing therapy based on both viral and renal parameters. The absence of significant differences in treatment outcomes or adverse event rates between dialysis and non-dialysis patients in this study is clinically relevant, suggesting that effective viral suppression can be safely achieved regardless of renal replacement modality finding supported by recent meta-analyses and guideline recommendations (18).

Theoretically, these results support the hypothesis that early and effective antiviral therapy not only prevents hepatic progression but may also mitigate extrahepatic complications, such as further renal decline, by reducing chronic inflammation and immune complex deposition (5,17). The high rates of biochemical normalization and the low frequency of adverse events observed

add practical weight to recommendations advocating for routine hepatitis screening and prompt initiation of antiviral therapy in all eligible CKD patients (18). In the context of Pakistan and other low- and middle-income countries, where the prevalence of viral hepatitis in dialysis populations remains elevated, the study provides timely evidence supporting the feasibility and effectiveness of such an approach in real-world practice (10).

Despite these strengths, the study limitations must be acknowledged. The single-center design and relatively modest sample size limit the generalizability of the findings and increase susceptibility to selection bias. Although consecutive sampling and standardized protocols were employed to enhance validity and reproducibility, the lack of a control group and the reliance on surrogate endpoints (such as eGFR and ALT) rather than histological or long-term outcomes constrain the depth of mechanistic insight available. The follow-up duration, though adequate for initial response assessment, may not fully capture late-emerging adverse effects or long-term impacts on renal or hepatic function. Furthermore, patient-reported outcomes and quality-of-life measures were not evaluated, which could be relevant in future studies examining the holistic impact of therapy.

Future research should prioritize multicenter, longitudinal studies with larger and more diverse patient cohorts, incorporating longer-term follow-up to assess the durability of virologic suppression, renal outcomes, and survival benefits. Additional work is warranted to directly compare the renal safety profiles of newer agents, such as tenofovir alafenamide, against older nucleus(t)ide analogues in this population and to evaluate the cost-effectiveness and accessibility of these regimens in resource-constrained settings. Moreover, systematic hepatitis screening in dialysis units and ongoing surveillance of treatment-related adverse events should be implemented as standard care. The findings here underscore the importance of multidisciplinary collaboration between nephrologists and hepatologists, emphasizing that timely initiation of appropriate antiviral therapy is not only effective but also essential for optimizing the long-term health of CKD patients living with viral hepatitis.

CONCLUSION

In this prospective study evaluating antiviral therapy in patients with chronic kidney disease and concurrent hepatitis B or C infection, the use of direct-acting antivirals for HCV and nucleos(t)ide analogues for HBV resulted in high rates of sustained virologic suppression and biochemical normalization, with no significant deterioration in renal function or increase in adverse events. These findings support the safe and effective integration of modern antiviral regimens into the management of kidney disease patients affected by viral hepatitis, emphasizing the importance of early diagnosis and prompt initiation of targeted therapy. Clinically, these results highlight the potential to reduce both hepatic and renal complications, ultimately improving long-term outcomes in this high-risk population, while also informing future research directions aimed at optimizing antiviral strategies and expanding access to care in diverse healthcare settings.

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