Mohammed *et. al.,* Babcock Univ. Med. J.2026 9(2):13-18
https://doi.org/10.38029/babcockuniv.med.j..v9i2.1211
eISSN: 2756-4657

RESEARCH ARTICLE OPEN ACCESS

Serum levels of C-reactive protein and interleukin-1 among patients with chronic kidney disease and the influence of hepatitis C virus infection

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Submitted: 11th October 2025
Accepted: 11th November 2025
Published: 3oth June 2026
ID: Orcid ID

Abstract
Objective: To evaluate the influence of hepatitis C virus (HCV) infection on serum interleukin-1 (IL-1) and high-sensitivity C-reactive protein (hs-CRP) levels among patients with chronic kidney disease (CKD).
Methods: A cross-sectional study was conducted on 100 CKD patients (aged 40–69 years) attending Baghdad Teaching Hospital, Iraq, between November 2024 and February 2025. Participants were categorised into two groups: Group I (CKD without HCV infection, n=50) and Group II (CKD with HCV infection, n=50). Serum IL-1 and hs-CRP levels were measured using ELISA. Statistical analysis was performed using SPSS version 25.0. Group comparisons were made using Student's t-test or Mann–Whitney test as appropriate, and ROC analysis was used to discriminate CKD+HCV from CKD–HCV patients.
Results: IL-1 and hs-CRP levels were significantly higher in CKD+HCV patients than in CKD–HCV patients ($p < 0.001$). hs-CRP suggests excellent discriminatory ability (AUC = 0.976, sensitivity = 100%, specificity = 96%), while IL-1 showed moderate performance (AUC = 0.675).
Conclusion: hs-CRP is a more reliable biomarker than IL-1 for identifying inflammation associated with HCV infection in CKD patients, supporting its potential role in cardiovascular risk stratification and monitoring.

Keywords: Chronic kidney disease, Hepatitis C virus, hs-CRP, Interleukin-1, Inflammation.

Plain English Summary

Chronic kidney disease (CKD) often leads to serious heart problems. People with CKD who also have hepatitis C virus (HCV) infection are at even greater risk. In this study, we measured two blood markers of inflammation, interleukin-1 (IL-1) and C-reactive protein (CRP), in 100 patients with CKD. We found that both markers were higher in CKD patients with HCV infection than in those without it. However, CRP was much better at detecting disease in patients with HCV infection. This means that a simple blood test for CRP could help doctors identify which CKD patients are at higher risk of heart disease. Using this test may improve treatment and reduce complications for these patients.

Introduction

Chronic kidney disease (CKD) is a major global health concern associated with a high risk of cardiovascular disease (CVD) and premature mortality. Even mild renal impairment significantly elevates cardiovascular risk (1). Among CKD patients, hepatitis C virus (HCV) infection is a frequent comorbidity that further exacerbates

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systemic inflammation, endothelial dysfunction, and cardiovascular complications (2).

Traditional risk factors such as diabetes mellitus (DM), hypertension, dyslipidaemia, and ageing contribute to CVD development in CKD; however, inflammation-related biomarkers like interleukin-1 (IL-1) and C-reactive protein (CRP) have emerged as key mediators linking CKD and cardiovascular outcomes (3, 4). Elevated hs-CRP is observed in more than 70% of CKD patients and correlates with adverse outcomes (5). In ESRD, increased serum concentration of cardiac troponin (cTn) and C-reactive protein (CRP) has been linked to higher cardiovascular mortality. Nevertheless, the association between these biomarkers and mortality among individuals with CKD is still not well established (6).

The present study aimed to investigate the influence of HCV infection on IL-1 and hs-CRP levels among CKD patients, providing insights into the inflammatory pathways and potential clinical applications of these biomarkers. We hypothesised that HCV infection increases serum IL-1 and hs-CRP levels among CKD patients.

Methods and Methods

This cross-sectional study included 100 CKD patients (aged 40–69 years) attending the kidney diseases and Transplant Centre at Baghdad Teaching Hospital, Medical City, Iraq, between November 2024 and February 2025. Patients were divided into two groups:

Group I: CKD patients without HCV infection (n = 50)

Group II: CKD patients with HCV infection (n = 50)

Patients with clinically confirmed chronic kidney disease (CKD) stages 3–5, defined according to KDIGO guidelines (eGFR <60 mL/min/1.73 m² for ≥3 months), were recruited from the Kidney Diseases and Transplant Centre. Exclusion criteria included patients with acute infections, autoimmune or malignant diseases, recent surgery, acute kidney injury, use of corticosteroids or anti-inflammatory drugs within the past four weeks, and those with other chronic viral infections such as hepatitis B or HIV.

All participants provided informed consent. Demographic and clinical data were collected through interviews and clinical records. Five millilitres of venous blood were drawn from each participant for measurement of serum IL-1 and hs-CRP using commercial ELISA kits according to the manufacturer's protocol.

Serum interleukin-1 (IL-1) and high-sensitivity C-reactive protein (hs-CRP) concentrations were measured using commercial ELISA kits (MyBioSource Inc., San Diego, USA) according to the manufacturer's protocol.

Statistical Analysis

Continuous variables were expressed as mean ± SD, and categorical variables as percentages. Normality was checked using the Kolmogorov–Smirnov test for normality. Group comparisons were analysed using the independent t-test. Correlations were examined using Pearson's coefficient. ROC curve analysis evaluated the discriminatory ability of biomarkers between CKD+HCV and CKD–HCV patients. A p-value ≤ 0.05 was considered statistically significant.

Ethical approval was obtained from the Kidney Diseases and Transplant Centre, Baghdad Teaching Hospital (Approval No: KDTC/2024/112).

Results

Demographic and Clinical Data

A total of 100 CKD patients were enrolled. The mean age was 57.5 ± 11.7 years in CKD+HCV patients and 53.6 ± 11.8 years in CKD–HCV patients (p = 0.102). No significant sex difference was found between groups. Hypertension was more frequent among CKD+HCV patients (56%) than among CKD–HCV patients (22%), whereas DM prevalence differed slightly.

Regarding sex distribution, the CKD+HCV group consisted of an equal number of males and females (50%), while the control group consisted of (62%) males and (38%) females. There was no significant difference in sex distribution between the two groups.

Biomarker Levels

Serum IL-1 levels were significantly higher in CKD+HCV patients (514.1 ± 448.3 pg/mL) compared with CKD–HCV patients (236.1 ± 216.3 pg/mL, p < 0.001). Similarly, hs-CRP levels were elevated in CKD+HCV (9.39 ± 4.73 mg/L) compared to CKD–HCV (2.53±4.22mg/L, p < 0.001), as shown in Table 1.

Table 1: Demographic and Clinical Characteristics

Groups		Mean ± SD		*P* value
Parameter		CKD–HCV	CKD+HCV	
Age		53.58±11.812	57.46±11.725	0.102
Sex N (%)	M	31 (62)	25 (50)	-

	F	19 (38)	25 (50)	-
DM N (%)	No	23 (46)	46 (92)	-
	Yes	27 (54)	4 (8)	-
Blood pressure	No	39 (78)	22 (44)	-
N (%)	Yes	11 (22)	28 (56)	-
IL-1 (pg/mL)		236.1±216.338	514.138±448.319	< 0.0001*
hs-CRP (mg/L)		2.53±4.22	9.39±4.73	< 0.0001*

**P* value ≤ 0.05

Correlation Analysis

A strong positive correlation was observed between IL-1 and hs-CRP levels in CKD–HCV patients (r = 0.987, p < 0.001). In contrast, no significant correlation was observed between IL-1 and hs-CRP in CKD+HCV patients, suggesting altered cytokine regulation during viral infection. Additionally, the correlation between age and hs-CRP was non-significant in both groups, as shown in Table 2 and Figures 1-4.

Table 2: Correlation between IL-1, hs-CRP, and Age

Parameter	hs-CRP (mg/L)			
	CKD–HCV		CKD+HCV	
	R	*P* value	R	*P* value
IL-1 (pg/mL)	0.987**	0.000	0.065	0.653
Age	0.058	0.687	-0.131	0.365

** Correlation is significant at the 0.01 level (2-tailed)

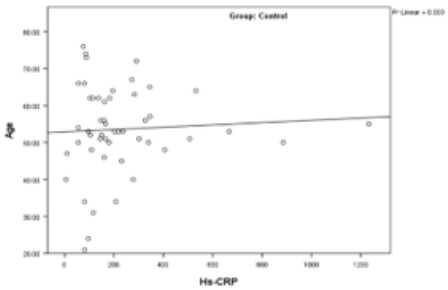


Figure 1: Scattered dot diagram of correlation between Age and Hs-CRP of the control group

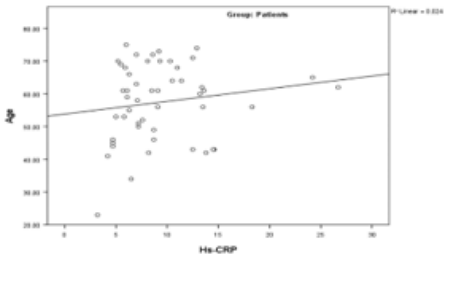


Figure 2: Scattered dot diagram of the correlation between Age and Hs-CRP of the patient group

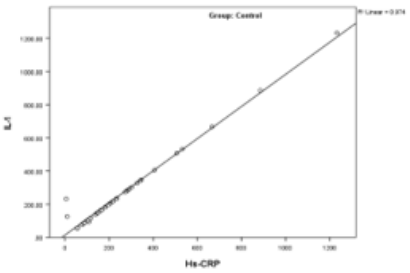


Figure 3: Scattered dot diagram of correlation between IL-1 and Hs-CRP of the control group

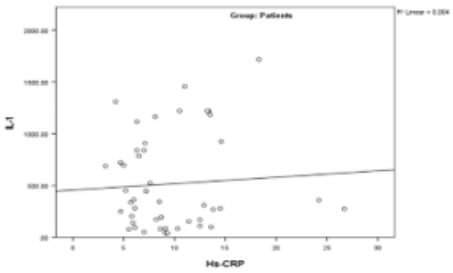


Figure 4: Scattered dot diagram of the correlation between IL-1 and Hs-CRP of the patient group

Diagnostic Accuracy (ROC Analysis)

To evaluate the diagnostic accuracy of IL-1 and hs-CRP in discriminating HCV-positive vs HCV-negative CKD patients, a receiver operating characteristic (ROC) curve analysis is used. The ROC curve provides a graphical tool that demonstrates the diagnostic capacity of a biomarker by plotting sensitivity against 1-specificity.

As shown in Table 3 and Figure 5, hs-CRP demonstrated superior diagnostic performance (AUC = 0.976, sensitivity 100%, specificity 96%, cutoff > 26.7 mg/L) compared to IL-1 (AUC = 0.675,

sensitivity 48%, specificity 88%, cutoff > 346 pg/mL).

Table 3: Diagnostic Accuracy (ROC) of IL-1 and hs-CRP.

Variables	Area	Cut off value	*P* value	Sensitivity	Specificity
IL-1	0.675	> 346	0.002	48%	88%
hs-CRP	0.976	> 26.7	<0.001	100%	96%

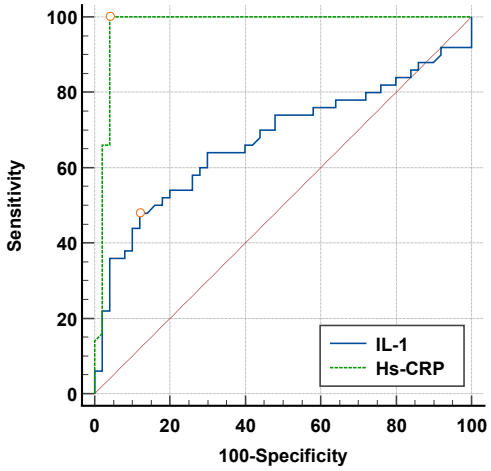


Figure 5: ROC diagram of IL-1 and hs-CRP

The optimal cutoff value of hs-CRP (> 26.7 mg/L) identified in this study was determined from the ROC curve using the Youden index to maximise combined sensitivity and specificity. Although this cutoff is higher than typical reference values, CKD patients often exhibit persistently elevated baseline hs-CRP due to chronic systemic inflammation. Therefore, this higher threshold likely reflects the additional inflammatory impact of HCV infection within a population already characterised by heightened inflammatory activity.

Discussion

The current study provides important insights into the role of inflammatory mediators in chronic kidney disease (CKD) and the impact of hepatitis C virus (HCV) infection on systemic inflammation. The elevated serum levels of interleukin-1 (IL-1) and high-sensitivity C-reactive protein (hs-CRP) observed among CKD patients with concurrent HCV infection underscore the synergistic effect of both conditions in promoting chronic inflammation and cardiovascular risk.

HCV infection is known to induce persistent immune activation and hepatic as well as extrahepatic inflammation. In patients with CKD, this inflammatory milieu is further amplified due to impaired cytokine clearance and oxidative stress. HCV triggers the release of pro-inflammatory cytokines such as IL-1, IL-6, and TNF-α through activation of toll-like receptors and NF-κB signalling pathways in hepatocytes and immune cells (7, 8). These mechanisms contribute to systemic inflammation, vascular dysfunction, and progression of renal injury. The elevated IL-1 levels observed in the present study support this pathway, reflecting enhanced cytokine release and impaired renal clearance of inflammatory mediators.

hs-CRP, a sensitive acute-phase protein synthesised by the liver in response to IL-1 and IL-6 stimulation, serves as an integrative marker of systemic inflammation (9). Elevated hs-CRP in CKD+HCV patients likely results from both hepatic induction by viral infection and the chronic uremic inflammatory state. Numerous studies have established hs-CRP as a strong predictor of cardiovascular morbidity and mortality in CKD populations (1, 2). Inflammatory processes mediated by CRP can promote endothelial dysfunction, plaque instability, and thrombogenesis, thereby accelerating cardiovascular complications (10). This aligns with the current findings that hs-CRP exhibits superior

diagnostic performance compared to IL-1, suggesting its greater clinical relevance in identifying high-risk CKD+HCV patients.

Regional studies from Iraq and neighbouring countries have similarly reported increased inflammatory markers in CKD and HCV populations. For instance, Al-Mayah et al. (2023, Iraq) demonstrated that CKD patients with viral co-infections showed significantly elevated CRP and IL-6 levels, correlating with increased cardiovascular risk (11). Comparable findings from Egypt and Iran also confirmed that HCV infection exacerbates inflammation and worsens renal and cardiovascular outcomes in CKD cohorts (12, 13). These consistent regional data reinforce the reliability of the current observations.

Clinical relevance of hs-CRP

From a clinical perspective, the use of hs-CRP as a routine biomarker could offer practical benefits in monitoring CKD patients, particularly those with HCV infection. Regular assessment of hs-CRP may help identify individuals at elevated risk for cardiovascular complications, enabling early interventions such as intensified blood pressure control, lipid management, and anti-inflammatory therapies. Although IL-1 is mechanistically important, its diagnostic variability and limited specificity make hs-CRP more suitable for clinical application.

Study limitations and future directions

The study is limited by its cross-sectional design, which precludes causal inference, and by its modest sample size. Future longitudinal studies should explore whether reducing hs-CRP levels through antiviral or anti-inflammatory therapy translates into improved cardiovascular outcomes in CKD+HCV patients.

Conclusion

hs-CRP appears to be a more sensitive and specific biomarker for detecting HCV-related inflammation in CKD patients. IL-1 is also elevated, but less reliable diagnostically. Routine hs-CRP assessment may support early cardiovascular risk detection and management in CKD+HCV populations. In conclusion, HCV infection potentiates inflammatory activation in CKD, with hs-CRP emerging as a robust biomarker reflecting the cumulative burden of inflammation. Integrating hs-CRP measurement into the clinical evaluation of CKD+HCV patients could enhance risk stratification and guide preventive strategies against cardiovascular disease.

Declarations

Ethical approval and consent to participate

Ethical approval was obtained from the Kidney Diseases and Transplant Centre, Baghdad Teaching Hospital, Medical City, Iraq. Informed consent was obtained from all participants.

Consent for publication

All the author(s) gave consent for the publication of the work under the Creative Commons Attribution-Non-Commercial 4.0 license.

Availability of data and materials

The data and materials associated with this review will be made available by the corresponding author upon reasonable request.

Conflict of Interest

None declared.

Funding

No specific funding was received.

Authors' Contributions

M.H.K. designed and performed the laboratory work. I.B.M. performed statistical analysis. Both authors contributed to manuscript writing and approved the final version.

Acknowledgements

The authors thank the staff of the Kidney Diseases and Transplant Centre, Baghdad Teaching Hospital, for their support.

List of Abbreviations

CKD: Chronic kidney disease
HCV: Hepatitis C virus
hs-CRP: High-sensitivity C-reactive protein
IL-1: Interleukin-1
ESRD: End-stage renal disease
CVD: Cardiovascular disease
ROC: Receiver operating characteristic
AUC: Area under the curve

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