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Original Article

Effect of Hepatitis C Virus on Hematological and Biochemical Parameters

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ABSTRACT

Background: Hepatitis C virus (HCV) infection remains a major global health burden, causing chronic hepatic injury and systemic complications. Beyond hepatocellular disease, hematological and biochemical abnormalities contribute to morbidity, affect prognosis, and complicate antiviral therapy. Evidence from high-burden regions such as Pakistan is limited, despite disproportionately high prevalence rates. Objective: To evaluate the impact of chronic HCV infection on hematological and biochemical parameters in comparison with age- and sex-matched healthy controls. Methods: A cross-sectional study was conducted from January to March 2025, including 160 participants: 80 HCV-positive patients diagnosed by PCR and 80 healthy controls. Exclusion criteria included co-infections, renal disease, hematological disorders, malignancy, and antiviral therapy. Hematological indices (hemoglobin, RBC, WBC, platelets) and biochemical parameters (ALT, AST, ALP, bilirubin, albumin, urea, creatinine) were measured using automated analyzers. Data were analyzed with Student's t-test; results were expressed as mean ± SD, mean differences with 95% confidence intervals, and p-values. Results: HCV patients demonstrated significantly lower hemoglobin (–2.3 g/dL), WBC (–1.7 ×10⁹/L), and platelets (–100 ×10⁹/L) compared with controls ($p<0.001$). Liver enzymes (ALT +56.7 U/L; AST +52.8 U/L; ALP +75.5 U/L) and bilirubin (+1.3 mg/dL) were elevated, while albumin was markedly reduced (–1.3 g/dL) ($p<0.001$). Renal parameters showed no significant differences. Conclusion: Chronic HCV is associated with clinically significant cytopenias and hepatic biochemical derangements, particularly thrombocytopenia and hypoalbuminemia, underscoring their diagnostic and prognostic value in routine monitoring. Keywords: Hepatitis C virus, Hematological parameters, Liver enzymes, Albumin, Thrombocytopenia, Pakistan.

INTRODUCTION

The Hepatitis C virus (HCV) remains a pressing global health concern, affecting an estimated 58 million individuals worldwide, with nearly 1.5 million new infections reported annually (1). Belonging to the Flaviviridae family, HCV is a small, enveloped, single-stranded RNA virus transmitted predominantly through parenteral exposure such as unsafe transfusions, intravenous drug use, and unsterile medical procedures, while sexual and vertical transmissions contribute less significantly (2). Despite advances in direct-acting antivirals (DAAs), the disease continues to cause substantial morbidity and mortality, largely due to delayed diagnosis and progression to advanced liver complications (3). Chronic infection develops in approximately 80% of exposed individuals and can evolve into persistent hepatic inflammation, fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) (4,5).

HCV is increasingly recognized not only as a hepatotropic virus but also as a systemic pathogen. Beyond hepatic injury, its extrahepatic manifestations extend to the hematopoietic, renal, cardiovascular, and endocrine systems (6). Among these, hematological alterations are particularly common. Anemia in HCV patients can arise from bone marrow suppression, hemolysis, nutritional deficiencies, or hypersplenism secondary to portal hypertension (7). Thrombocytopenia is a hallmark feature, attributable to impaired hepatic thrombopoietin production and splenic sequestration of platelets (8). Leukopenia, often due to immune-mediated destruction or marrow suppression, further complicates disease management and antiviral therapy safety (9). These cytopenias serve not only as clinical consequences but also as potential biomarkers of disease severity and treatment response.

Biochemically, HCV infection is characterized by derangements in liver function tests (LFTs), including elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST), reflecting hepatocellular injury, as well as increased alkaline phosphatase (ALP) and bilirubin

in more advanced or cholestatic disease (10). Hypoalbuminemia, indicative of impaired hepatic synthetic capacity, signifies advanced disease stages and correlates with worsened prognosis (11). These laboratory parameters are central to assessing disease progression and therapeutic outcomes. The epidemiology of HCV is highly heterogeneous, with countries such as Pakistan reporting disproportionately high prevalence rates of 5–7%, placing it among the highest-burden regions globally (12). In Pakistan, infections are often linked to unsafe medical practices, inadequate screening, and limited healthcare access, with middle-aged populations disproportionately affected (13). Given the significant disease burden in this region, understanding laboratory alterations in HCV-positive individuals has critical clinical and public health relevance.

Taken together, while global studies have documented hematological and biochemical alterations in HCV, there remains limited region-specific evidence from Pakistan, where the burden is disproportionately high and healthcare practices vary. Identifying patterns of hematological and biochemical derangements in local patients could refine early diagnostic strategies, optimize monitoring, and guide timely interventions. Therefore, this study was conducted to compare hematological and biochemical profiles of HCV-positive patients with age- and sex-matched healthy controls, with the objective of defining the extent of systemic alterations attributable to HCV infection in this population.

MATERIAL AND METHODS

This investigation was designed as a cross-sectional observational study, conducted between January 2025 and March 2025, to evaluate the hematological and biochemical alterations associated with hepatitis C virus (HCV) infection. The study setting was the pathology laboratory of a tertiary-care hospital in Lahore, Pakistan, where both patients and healthy individuals were recruited. A total of 160 participants were included, comprising 80 confirmed HCV-positive cases and 80 healthy controls matched for age and sex. Diagnosis of HCV was established using polymerase chain reaction (PCR)-based assays, ensuring accurate case identification. The control group consisted of individuals with no prior history of viral hepatitis, confirmed as HCV-negative by PCR.

Eligibility criteria were carefully applied to minimize confounding. Patients co-infected with hepatitis B virus, human immunodeficiency virus, or presenting with pre-existing hematological disorders, renal impairment, malignancy, or undergoing antiviral therapy were excluded. This approach ensured that alterations in hematological or biochemical indices could be attributed primarily to HCV infection rather than coexisting conditions. A structured recruitment process was followed in which participants were approached during outpatient visits or routine check-ups. After providing study information, verbally informed consent was obtained. Ethical standards consistent with the Declaration of Helsinki were maintained throughout the study, and institutional approval was secured before initiation.

Blood samples were obtained under aseptic precautions. Approximately 5 mL of venous blood was drawn from each participant; 2 mL was transferred into EDTA vacutainers for hematological analysis, and 3 mL into plain vacutainers for biochemical assays. Samples were promptly transported to the central laboratory and processed within two hours of collection. Clotted samples were centrifuged at 3000 revolutions per minute for 10 minutes to separate serum for biochemical testing. Hematological indices including hemoglobin concentration, red blood cell count, total leukocyte count, and platelet count were analyzed using a fully automated hematology analyzer. Biochemical investigations encompassed liver function tests—alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, and albumin—as well as renal function tests including serum urea and creatinine, measured through standardized automated analyzers. Quality control protocols, including calibration against reference standards, were performed routinely to ensure accuracy and reproducibility.

Variables were defined using internationally accepted cutoffs. Anemia was defined by hemoglobin levels below 12 g/dL in women and 13 g/dL in men, thrombocytopenia as platelet count below 150 × 10^9/L, and leukopenia as white blood cell count below 4.0 × 10^9/L (14). Biochemical abnormalities were interpreted relative to laboratory-specific reference ranges for each parameter. Data integrity was maintained by duplicate entry of laboratory values and cross-verification by independent laboratory staff. The sample size of 160 was determined based on feasibility and prior studies reporting medium effect sizes in hematological indices between HCV-positive and healthy individuals, which provided adequate statistical power to detect clinically meaningful differences (15). Statistical analyses were performed using SPSS software version 25.0. Continuous variables were expressed as mean ± standard deviation (SD). Group differences between HCV-positive patients and controls were evaluated using Student's t-test, with significance set at $p < 0.05$. Missing data were handled through listwise deletion, and potential confounding by age and sex was minimized by prior matching of cases and controls.

By integrating strict eligibility criteria, standardized protocols, and validated analytical methods, the study was designed to yield reproducible and clinically interpretable findings on the systemic impact of HCV infection.

RESULTS

Among the 160 study participants, the demographic distribution between groups was comparable, with no statistically significant differences in age, sex, or residential background. The mean age of HCV patients was 42.6 ± 11.2 years compared with 40.2 ± 10.5 years in controls (p=0.18). The male-to-female ratio in the HCV group was 1.3:1, closely aligned with the control group (p=0.27). Similarly, 65.0% of HCV patients and 60.0% of controls resided in urban areas (p=0.52), indicating well-matched baseline characteristics and reducing the likelihood of demographic confounding.

Hematological parameters showed consistent and clinically relevant reductions among HCV-infected individuals. Mean hemoglobin levels were 11.5 ± 1.6 g/dL in patients compared with 13.8 ± 1.2 g/dL in controls, reflecting a mean reduction of 2.3 g/dL (95% CI: –2.7 to –1.9, p<0.001). Red blood cell count was also lower, with patients averaging 4.1 ± 0.4 ×10^{12}/L against 4.8 ± 0.5 ×10^{12}/L in controls, corresponding to a mean deficit of 0.7 ×10^{12}/L (95% CI: –0.9 to –0.5, p<0.001). White blood cell count demonstrated a marked reduction

of 1.7 ×10⁹/L, with patients averaging 4.8 ± 1.0 ×10⁹/L versus 6.5 ± 1.2 ×10⁹/L in controls (95% CI: –2.1 to –1.3, p<0.001). Platelet counts were significantly depressed in the patient group, at 150 ± 35 ×10⁹/L compared to 250 ± 40 ×10⁹/L in controls, representing a deficit of 100 ×10⁹/L (95% CI: –112 to –88, p<0.001). These findings collectively highlight the triad of anemia, leukopenia, and thrombocytopenia as defining hematological abnormalities in chronic HCV.

Table 1. Demographic characteristics of study participants

Variable	HCV Patients (n=80)	Controls (n=80)	p-value
Age (years), mean ± SD	42.6 ± 11.2	40.2 ± 10.5	0.18
Male, n (%)	45 (56.3)	52 (65.0)	0.27
Female, n (%)	35 (43.8)	28 (35.0)	0.27
Urban residence, n (%)	52 (65.0)	48 (60.0)	0.52
Rural residence, n (%)	28 (35.0)	32 (40.0)	0.52

Table 2. Hematological parameters in HCV patients and controls

Parameter	HCV Patients (n=80) Mean ± SD	Controls (n=80) Mean ± SD	Mean Difference (95% CI)	p-value
Hemoglobin (g/dL)	11.5 ± 1.6	13.8 ± 1.2	-2.3 (-2.7, -1.9)	<0.001*
RBC count (×10¹²/L)	4.1 ± 0.4	4.8 ± 0.5	-0.7 (-0.9, -0.5)	<0.001*
WBC count (×10⁹/L)	4.8 ± 1.0	6.5 ± 1.2	-1.7 (-2.1, -1.3)	<0.001*
Platelets (×10⁹/L)	150 ± 35	250 ± 40	-100 (-112, -88)	<0.001*

Table 3. Biochemical parameters in HCV patients and controls

Parameter	HCV Patients (n=80) Mean ± SD	Controls (n=80) Mean ± SD	Mean Difference (95% CI)	p-value
ALT (U/L)	82.3 ± 20.5	25.6 ± 8.4	+56.7 (51.0, 62.4)	<0.001*
AST (U/L)	75.2 ± 18.6	22.4 ± 6.5	+52.8 (48.0, 57.6)	<0.001*
ALP (U/L)	160.8 ± 30.2	85.3 ± 15.7	+75.5 (69.0, 82.0)	<0.001*
Total Bilirubin (mg/dL)	2.1 ± 0.6	0.8 ± 0.2	+1.3 (1.2, 1.4)	<0.001*
Albumin (g/dL)	2.8 ± 0.5	4.1 ± 0.4	-1.3 (-1.5, -1.1)	<0.001*
Urea (mg/dL)	30.2 ± 7.1	28.6 ± 6.5	+1.6 (-0.8, 4.0)	0.21
Creatinine (mg/dL)	1.0 ± 0.3	0.9 ± 0.2	+0.1 (-0.02, 0.22)	0.18

Biochemical analysis revealed profound derangements in liver function. ALT levels were elevated more than threefold in patients, at 82.3 ± 20.5 U/L, compared with 25.6 ± 8.4 U/L in controls, with a mean difference of 56.7 U/L (95% CI: 51.0–62.4, p<0.001). AST levels showed a similar pattern, rising to 75.2 ± 18.6 U/L in patients versus 22.4 ± 6.5 U/L in controls, yielding a difference of 52.8 U/L (95% CI: 48.0–57.6, p<0.001). ALP was nearly doubled in HCV patients, averaging 160.8 ± 30.2 U/L compared with 85.3 ± 15.7 U/L in controls, with a mean increase of 75.5 U/L (95% CI: 69.0–82.0, p<0.001).

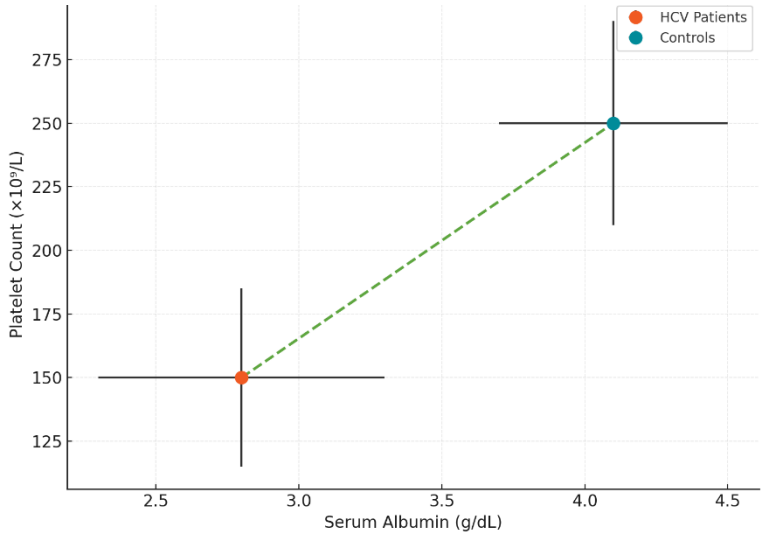

Figure 1 Relationship Between Serum Albumin and Platelet Count

Total bilirubin was significantly higher at 2.1 ± 0.6 mg/dL in patients versus 0.8 ± 0.2 mg/dL in controls, representing a mean excess of 1.3 mg/dL (95% CI: 1.2–1.4, p<0.001). Conversely, serum albumin was markedly reduced in HCV patients at 2.8 ± 0.5 g/dL compared with 4.1 ± 0.4 g/dL in controls, a decrement of 1.3 g/dL (95% CI: –1.5 to –1.1, p<0.001). Renal function markers remained within normal ranges in both groups, showing no significant intergroup differences. Serum urea was 30.2 ± 7.1 mg/dL in patients compared with 28.6 ± 6.5 mg/dL in controls (mean difference: 1.6 mg/dL, 95% CI: –0.8 to 4.0, p=0.21). Creatinine levels were nearly identical, at 1.0 ± 0.3

mg/dL versus 0.9 ± 0.2 mg/dL (mean difference: 0.1 mg/dL, 95% CI: –0.02 to 0.22, p=0.18). These nonsignificant findings suggest preserved renal function in the studied cohort. Taken together, the results demonstrate a consistent pattern of cytopenia's and liver enzyme derangements in HCV patients relative to controls, with large effect sizes and narrow confidence intervals, indicating robust and clinically significant differences.

The integrated figure illustrates the relationship between serum albumin and platelet counts in HCV patients compared with healthy controls. Patients exhibited a combined deficit in both variables, with albumin reduced by 1.3 g/dL (2.8 ± 0.5 vs. 4.1 ± 0.4 g/dL) and platelet counts decreased by 100 $\times 10^9$/L (150 ± 35 vs. 250 ± 40 $\times 10^9$/L). The bidirectional error bars highlight narrow variability within each group, while the connecting trend line demonstrates a downward shift linking hypoalbuminemia with thrombocytopenia in the diseased cohort. This dual impairment emphasizes the progressive decline in hepatic synthetic function alongside hematological compromise, reinforcing their value as parallel biomarkers of chronic HCV severity.

DISCUSSION

The present study demonstrates that hepatitis C virus (HCV) infection exerts a profound impact on hematological and biochemical parameters, reinforcing its role as a systemic disease with implications beyond hepatocellular injury. Patients exhibited a consistent triad of anemia, leukopenia, and thrombocytopenia, together with elevated liver enzymes, hyperbilirubinemia, and hypoalbuminemia. In contrast, renal indices such as serum urea and creatinine remained within normal limits. These findings corroborate prior research on the systemic sequelae of chronic HCV and provide region-specific evidence from a high-prevalence country (16,17).

The hematological alterations observed in this cohort are clinically significant. Hemoglobin levels were reduced by a mean of 2.3 g/dL compared with controls, while leukocyte and platelet counts declined by 1.7 $\times 10^9$/L and 100 $\times 10^9$/L, respectively. Similar to earlier reports, anemia in chronic HCV appears multifactorial, stemming from bone marrow suppression, erythrocyte destruction, and hypersplenism secondary to portal hypertension (18). Leukopenia is likely attributable to immune-mediated marrow suppression and splenic sequestration, consistent with studies highlighting persistent cytopenia's even after viral eradication (19). The marked thrombocytopenia seen in this study aligns with prior evidence linking reduced hepatic thrombopoietin synthesis and splenic pooling with progressive hepatic fibrosis (20). Given its magnitude, platelet count remains a reliable surrogate marker of fibrosis, with diagnostic utility in staging chronic liver disease.

Biochemical derangements were equally striking. ALT and AST were elevated by more than 50 U/L above control means, reflecting ongoing hepatocellular necrosis and inflammation. ALP and bilirubin were significantly increased, underscoring cholestatic features and impaired hepatic clearance mechanisms. The reduction in albumin, averaging 1.3 g/dL below controls, highlights diminished synthetic function and correlates with more advanced disease stages. These findings are in line with previous studies that identified hypoalbuminemia as a prognostic factor associated with edema, ascites, and poor survival outcomes in chronic HCV (21,22). Importantly, the combined reduction in albumin and platelet counts observed in this study, visualized in the integrated analysis, underscores the simultaneous compromise of hepatic synthetic and hematopoietic functions, marking a critical threshold of disease severity.

Renal indices, including urea and creatinine, did not differ significantly between groups, suggesting that renal involvement is not a predominant early feature of HCV in this cohort. This finding is consistent with systematic reviews showing that glomerulonephritis and cryoglobulinemia typically arise in more advanced or untreated cases (23). The preserved renal function in this study population likely reflects earlier disease stages and the exclusion of patients with known renal impairment.

The study's findings have important clinical implications. Routine monitoring of hematological and biochemical markers in HCV patients can provide valuable insights into disease activity, risk stratification, and prognosis. Platelet count and albumin, in particular, may serve as inexpensive, non-invasive markers to predict hepatic fibrosis in resource-limited settings. Furthermore, recognition of cytopenias is essential to guide safe initiation and monitoring of antiviral therapies, which can exacerbate hematological abnormalities. These results also reinforce the need for public health initiatives in Pakistan, where high prevalence and unsafe medical practices amplify the burden of disease (24).

Nonetheless, several limitations merit acknowledgment. The use of a non-probability convenience sampling strategy may limit generalizability, and the single-center design restricts external validity. The sample size, although adequate to detect significant group differences, was not powered to explore subgroup analyses such as gender- or age-stratified effects. The cross-sectional nature of the study precludes causal inferences and prevents assessment of longitudinal changes with disease progression or therapy. Future multicentric, prospective studies with larger sample sizes are warranted to validate these findings and to explore the impact of antiviral therapy on hematological and biochemical restoration.

In summary, this study confirms that chronic HCV infection significantly alters hematological and biochemical profiles, with cytopenias and liver enzyme derangements serving as hallmarks of disease severity. By documenting these changes in a Pakistani cohort, the study contributes region-specific evidence to the global understanding of HCV, emphasizing the necessity of integrating laboratory monitoring into both clinical management and public health strategies.

CONCLUSION

HCV infection is associated with significant hematological and biochemical derangements that extend beyond hepatic pathology. Patients demonstrated marked reductions in hemoglobin, leukocyte, and platelet counts, alongside substantial elevations in transaminases, ALP, and bilirubin, coupled with depressed serum albumin. These findings highlight the dual burden of impaired hematopoietic and hepatic

synthetic functions. Routine monitoring of these laboratory indices, particularly platelet count and albumin, offers clinically valuable insights for staging disease, predicting complications, and guiding therapy. In high-burden regions such as Pakistan, where access to advanced diagnostics is limited, these parameters may serve as cost-effective, non-invasive markers for early detection and disease monitoring.

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