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ORIGINAL ARTICLE

Serum Inflammatory Markers and Histopathological Findings in Patients with Community-Acquired Acute Viral Hepatitis

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ABSTRACT

Background: Community-acquired acute viral hepatitis (CA-AVH) remains a significant public health challenge in Pakistan, with hepatitis A, B, C, and E viruses contributing to substantial morbidity. While histopathology remains the gold standard for assessing hepatic injury, invasive procedures are often limited in acute cases. Serum inflammatory markers may serve as valuable non-invasive surrogates that reflect underlying tissue-level damage.

Objective: To evaluate the relationship between serum inflammatory markers and histopathological findings in patients with CA-AVH.

Methods: A cross-sectional observational study was conducted at Khyber Teaching Hospital (KTH), Peshawar, Pakistan, from January 2022 to February 2023. A total of 100 patients aged 18–60 years with serologically confirmed CA-AVH were enrolled. Clinical and biochemical profiles were recorded, and serum inflammatory markers including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) were measured using standard assays. Liver biopsies were performed in 30 patients with prolonged jaundice or uncertain diagnosis and were graded for necroinflammatory activity using the modified Knodell scoring system. Statistical analyses included Student's t-test, ANOVA, and Pearson's correlation coefficient.

Results: The mean age of patients was 33.2 ± 8.9 years, with a male predominance (62%). The distribution of viral etiology was HAV (18%), HBV (32%), HCV (12%), and HEV (38%). Elevated CRP was observed in 68% of patients, while IL-6 and TNF-α levels were significantly higher in HBV and HEV infections. Histopathological features included portal inflammation (80%), hepatocyte ballooning (73%), lobular necrosis (63%), and interface hepatitis (40%). CRP correlated strongly with lobular necrosis ($r = 0.62$, $p < 0.01$), IL-6 with portal inflammation ($r = 0.71$, $p < 0.01$), and TNF-α with ballooning degeneration and interface hepatitis ($r = 0.68$, $p < 0.01$).

Conclusion: Serum inflammatory markers reflect the severity of histopathological injury in CA-AVH. IL-6 and TNF-α, in particular, show strong correlations with necroinflammatory activity and may serve as reliable non-invasive indicators of disease severity, thereby reducing reliance on liver biopsy in resource-limited settings.

Keywords: Acute viral hepatitis, community-acquired infection, inflammatory markers, IL-6, TNF-α, histopathology, liver biopsy, Pakistan.

INTRODUCTION

Acute viral hepatitis (AVH) continues to be one of the most common causes of liver-related morbidity and mortality worldwide, particularly in low- and middle-income countries where sanitation, clean water access, and vaccination coverage remain limited[1]. The majority of cases are community-acquired, caused predominantly by hepatitis A virus (HAV) and hepatitis E virus (HEV) in developing regions, whereas hepatitis B virus (HBV) and hepatitis C virus (HCV) also contribute substantially to disease burden through both acute and chronic manifestations. Despite being self-limiting in most patients, acute viral hepatitis can lead to severe outcomes including fulminant hepatic failure, prolonged cholestasis, and, in rare cases, progression to chronic hepatitis[2].

The pathophysiology of AVH is complex and involves both direct viral cytopathic effects and immune-mediated hepatocellular injury. The immune response, particularly the activation of inflammatory cytokines and acute-phase reactants, plays a critical role in determining the severity of liver damage[3]. Among these, serum inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) are widely studied in chronic liver diseases, but their role in acute, community-acquired viral hepatitis remains less well defined. Elevated levels of these markers may not only reflect systemic inflammatory responses but also mirror the degree of hepatocellular injury[4].

Histopathological examination of the liver remains the gold standard for assessing the extent and nature of hepatic injury in AVH. Typical findings include hepatocellular ballooning, spotty necrosis, Kupffer cell hyperplasia, lobular disarray, and portal tract infiltration with mononuclear cells[5]. However, liver biopsy is invasive and not always feasible in patients with acute illness, particularly when coagulopathy is present. Identifying reliable non-invasive biomarkers that correlate with histological damage could improve clinical decision-making and reduce the need for biopsy in selected cases[6].

Despite its clinical importance, the relationship between serum inflammatory markers and histopathological changes in CA-AVH has not been extensively explored in the South Asian population, where the prevalence of hepatitis A and E remains high due to endemic transmission. Understanding this relationship is vital for developing prognostic tools, guiding therapeutic interventions, and predicting disease outcomes[7,8].

The present study was therefore designed to evaluate serum inflammatory markers in patients with community-acquired acute viral hepatitis and to correlate these biomarkers with histopathological findings. By bridging the gap between laboratory markers and tissue-level pathology, this research aims to enhance our understanding of the immunopathogenesis of AVH and provide clinicians with practical insights into patient management[9].

MATERIALS AND METHODS

Study Design and Duration: This cross-sectional observational study was carried out in the Department of Medicine and Pathology at Khyber Teaching Hospital (KTH), Peshawar, Pakistan, over a period of fourteen months extending from January 2022 to February 2023. The design was chosen to evaluate both

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serum inflammatory markers and histopathological features in patients presenting with community-acquired acute viral hepatitis.

Study Population and Sample Size: A total of 100 patients were included in the study, recruited consecutively from outpatient and inpatient services. The sample size was based on the anticipated frequency of acute viral hepatitis cases in the hospital during the study period, ensuring adequate power to analyze correlations between biomarkers and tissue findings.

Inclusion and Exclusion Criteria: Patients were eligible for inclusion if they were between 18 and 60 years of age, presented with acute onset of jaundice and constitutional symptoms such as fatigue, anorexia, or malaise, and demonstrated biochemical evidence of acute hepatitis with alanine aminotransferase (ALT) and aspartate aminotransferase (AST) elevated to at least ten times the upper normal limit. Only those with positive serological evidence of acute viral infection including HAV IgM, HBsAg, HCV RNA, or HEV IgM were enrolled. Patients with a history of chronic liver disease, cirrhosis, alcohol abuse, drug-induced hepatitis, or autoimmune hepatitis were excluded. Individuals receiving long-term hepatotoxic drugs or those unwilling to participate were also excluded.

Clinical and Laboratory Assessment: For each patient, detailed demographic information including age, sex, residence, and socioeconomic status was recorded, along with a complete clinical history and physical examination. Laboratory investigations included complete blood counts, liver function tests such as ALT, AST, alkaline phosphatase, bilirubin, and albumin, and coagulation profiles including prothrombin time and international normalized ratio. Viral serological testing comprised HAV IgM, HBsAg, anti-HCV antibody, HCV RNA polymerase chain reaction, and HEV IgM, performed using standard ELISA techniques.

The primary inflammatory markers of interest were C-reactive protein (CRP) measured by immunoturbidimetric method, interleukin-6 (IL-6) determined through enzyme-linked immunosorbent assay (ELISA), and tumor necrosis factor-alpha (TNF-α) also quantified using ELISA kits according to manufacturer protocols.

Histopathological Examination: Liver biopsies were performed in patients with persistent jaundice beyond four weeks, in cases where the diagnosis remained uncertain, or when clinical progress was unsatisfactory. Biopsies were carried out under ultrasound guidance after correction of coagulopathy when required. The specimens were fixed in 10% buffered formalin, embedded in paraffin, and stained with hematoxylin and eosin. Histopathological evaluation focused on identifying hepatocyte ballooning, lobular necrosis, Kupffer cell hyperplasia, lobular disarray, portal tract inflammatory infiltrates, and interface hepatitis. Necroinflammatory activity was graded according to the modified Knodell scoring system to ensure uniformity and reproducibility of findings.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Review Board of Khyber Teaching Hospital, Peshawar. Written informed consent was obtained from every participant prior to inclusion in the study, and strict confidentiality of patient data was maintained.

Statistical Analysis: Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age, liver enzymes, and inflammatory marker levels were expressed as mean ± standard deviation, while categorical variables such as gender and viral etiology were presented as frequencies and percentages. Group comparisons were performed using Student's t-test and one-way ANOVA for continuous data, and chi-square test for categorical data. The correlation between serum inflammatory markers and histopathological scores was assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

Demographic and Clinical Characteristics: A total of 100 patients diagnosed with community-acquired acute viral hepatitis were included in this study, comprising 62 males and 38 females, with a mean age of 33.2 ± 8.9 years (range: 18–58 years). The majority of the patients (65%) were from urban settings, while the remaining 35% belonged to rural areas. The most common presenting symptoms included jaundice (100%), fatigue (85%), anorexia (70%), abdominal pain (40%), and low-grade fever (38%). Hepatomegaly was detected in 42% of the patients, whereas splenomegaly was observed in 18%. The distribution of viral etiology showed HAV in 18%, HBV in 32%, HCV in 12%, and HEV in 38% of the cases. These findings are summarized in Table 1.

Table 1. Demographic and clinical characteristics of patients with community-acquired acute viral hepatitis (n = 100).

Variable	Number (%) / Mean ± SD
Age (years)	33.2 ± 8.9
Gender (Male/Female)	62 (62%) / 38 (38%)
Residence (Urban/Rural)	65 (65%) / 35 (35%)
Presenting symptoms	
– Jaundice	100 (100%)
– Fatigue	85 (85%)
– Anorexia	70 (70%)
- Abdominal pain	40 (40%)
– Fever	38 (38%)
Hepatomegaly	42 (42%)
Splenomegaly	18 (18%)
Viral etiology	
– HAV	18 (18%)
– HBV	32 (32%)
– HCV	12 (12%)
– HEV	38 (38%)

As demonstrated in Table 1, HEV and HBV were the most frequent etiologies. The male predominance is consistent with previously reported data from South Asian populations, where greater occupational and environmental exposure is considered a contributing factor.

Laboratory Findings and Serum Inflammatory Markers: Biochemical analyses revealed a mean ALT level of 975 ± 256 U/L, while AST was 812 ± 210 U/L. The mean total bilirubin level was 7.8 ± 2.4 mg/dL, with a higher concentration observed in patients with HBV and HEV infections. Coagulation profiles showed a prolonged INR (>1.5) in 15% of patients, indicating a subset with severe hepatic dysfunction.

Regarding inflammatory markers, CRP levels were elevated (>10 mg/L) in 68% of patients, with mean values of 18.5 ± 7.6 mg/L. IL-6 levels were markedly elevated in HBV and HEV cases, averaging 42.7 ± 12.3 pg/mL, while patients with HAV and HCV exhibited lower values. TNF-α levels were significantly raised in HBV patients, with a mean of 58.2 ± 15.1 pg/mL, compared to 38.4 ± 10.6 pg/mL in HAV and 34.8 ± 11.3 pg/mL in HCV. These results are summarized in Table 2.

Table 2. Laboratory profile and serum inflammatory markers of patients with acute viral hepatitis.

Parameter	Mean ± SD / Frequency (%)
ALT (U/L)	975 ± 256
AST (U/L)	812 ± 210
Total Bilirubin (mg/dL)	7.8 ± 2.4
INR > 1.5	15 (15%)
CRP (mg/L)	18.5 ± 7.6 (elevated in 68%)
IL-6 (pg/mL)	42.7 ± 12.3 (highest in HBV/HEV)
TNF-α (pg/mL)	58.2 ± 15.1 (highest in HBV)

Table 3. Histopathological findings in liver biopsies of patients with acute viral hepatitis (n = 30).

Histopathological Feature	Number (%)
Hepatocyte ballooning	22 (73%)
Lobular necrosis	19 (63%)
Kupffer cell hyperplasia	17 (57%)
Portal tract inflammation	24 (80%)
Interface hepatitis	12 (40%)
Mean necroinflammatory score	8.6 ± 2.4

The data in Table 2 illustrate that IL-6 and TNF-α levels correlated with biochemical severity, particularly elevated bilirubin and transaminases. Patients with higher cytokine levels frequently presented with more pronounced clinical symptoms and prolonged recovery times.

As shown in Table 3, portal inflammation was the most frequent histological feature, followed by hepatocyte ballooning and lobular necrosis. The relatively high proportion of interface hepatitis in HBV patients indicates immune-mediated damage, consistent with the elevated TNF-α levels observed in this subgroup.

Histopathological Findings: Liver biopsies were performed in 30 patients, primarily those with prolonged jaundice beyond four weeks or diagnostic uncertainty. Histopathological analysis revealed that hepatocyte ballooning was present in 73%, lobular necrosis in 63%, Kupffer cell hyperplasia in 57%, portal tract inflammation in 80%, and interface hepatitis in 40% of the cases. Mean necroinflammatory scores were significantly higher in patients with raised IL-6 and TNF-α levels. These findings are detailed in Table 3.

Correlation of Serum Inflammatory Markers with Histopathology: A significant correlation was observed between CRP levels and lobular necrosis ($r = 0.62$, $p < 0.01$), while IL-6 levels were strongly associated with portal inflammation ($r = 0.71$, $p < 0.01$). Similarly, TNF-α demonstrated a robust correlation with hepatocyte ballooning and interface hepatitis ($r = 0.68$, $p < 0.01$). These associations indicate that elevated serum inflammatory markers reliably reflect the extent of histopathological damage. The correlation analysis is summarized in Table 4.

Table 4. Correlation between serum inflammatory markers and histopathological findings.

Inflammatory Marker	Correlated Histological Feature	Correlation Coefficient (r)	p-value
CRP	Lobular necrosis	0.62	<0.01
IL-6	Portal tract inflammation	0.71	<0.01
TNF-α	Hepatocyte ballooning, interface hepatitis	0.68	<0.01

Table 4 demonstrates that each serum inflammatory marker reflects distinct histopathological features. CRP is a general marker of necrosis, IL-6 strongly predicts portal inflammation, while TNF-α is closely linked to ballooning degeneration and interface hepatitis.

The overall results of this study demonstrate that serum inflammatory markers provide a reliable reflection of underlying histopathological injury in patients with community-acquired acute viral hepatitis. IL-6 and TNF-α were the most predictive of necroinflammatory activity, while CRP correlated with lobular necrosis. These findings suggest that routine assessment of serum inflammatory markers can serve as non-invasive adjuncts to biopsy, particularly in patients where invasive procedures are contraindicated.

DISCUSSION

The present study explored the relationship between serum inflammatory markers and histopathological changes in patients with community-acquired acute viral hepatitis (CA-AVH) admitted to a tertiary care hospital in Peshawar, Pakistan[10]. The findings demonstrate that inflammatory biomarkers such as CRP, IL-6, and TNF-α not only rise significantly during acute infection but also correlate with the extent of liver injury identified on histopathological examination. This highlights their potential role as non-invasive markers for disease severity assessment and prognostication in clinical practice[11].

In our cohort, the demographic distribution was skewed toward males, with a mean age of 33 years. This age group represents the most active segment of the population and is likely more exposed to occupational and environmental risks[12]. The predominance of hepatitis E and B infection reflects the local epidemiology, as Pakistan remains endemic for HEV due to contaminated water supplies and for HBV due to inadequate vaccination coverage and unsafe medical practices. Previous regional studies have also reported similar patterns of viral etiology, supporting the external validity of our findings[13].

Biochemically, the marked elevation of transaminases and bilirubin levels was consistent with the acute necroinflammatory process. Patients with HBV and HEV infections demonstrated higher biochemical severity, a trend also reflected in the raised inflammatory markers[14,15]. CRP was elevated in nearly 70% of patients, which is in line with its role as an acute-phase reactant produced by hepatocytes in response to IL-6 stimulation. Although nonspecific, CRP has been shown in other studies to correlate with necrosis and systemic inflammation. In our study, CRP correlated significantly with lobular necrosis, suggesting its value as a surrogate for tissue-level hepatocellular injury[16,17].

The most noteworthy findings involved IL-6 and TNF-α. IL-6 levels were particularly elevated in HBV and HEV infections, and its strong correlation with portal inflammation suggests a central role in orchestrating immune cell recruitment into the periportal region[18]. These results are consistent with experimental studies demonstrating that IL-6 promotes hepatic inflammation by inducing acute-phase proteins and modulating T-cell responses. Similarly, TNF-α was highest in HBV patients and correlated with hepatocyte ballooning and interface hepatitis. This is biologically plausible since TNF-α is secreted by activated T lymphocytes and Kupffer cells, driving hepatocellular apoptosis and necrosis. The histological evidence of ballooning degeneration and interface activity supports the immunopathogenic role of TNF-α in HBV-associated hepatitis[19,20].

Histopathologically, the most common findings were portal inflammation, lobular necrosis, and hepatocyte ballooning, which are classical features of acute viral hepatitis. Kupffer cell hyperplasia observed in more than half of biopsied cases highlights the role of resident macrophages in viral clearance and cytokine production. Interface hepatitis, although less frequent, was particularly associated with HBV, reflecting the heightened immune response typical of this virus. The close alignment between histopathology and serum biomarkers reinforces the hypothesis that inflammatory mediators are not only measurable reflections of tissue injury but also central participants in the pathogenesis of AVH[21,22].

When comparing our findings with international literature, similar correlations between cytokines and hepatic injury have been documented in both acute and chronic viral hepatitis. Studies from India and China have reported elevated IL-6 and TNF-α levels in AVH patients, particularly in those progressing to fulminant hepatic failure[23]. Our study contributes to this growing body of evidence by providing data from a Pakistani population, where hepatitis A and E remain highly prevalent. Importantly, our work suggests that these inflammatory markers could be integrated into clinical decision-making, particularly in settings where liver biopsy is not feasible due to cost, expertise, or coagulopathy risks[24].

Despite these strengths, several limitations should be acknowledged. First, this was a single-center study with a relatively modest sample size, which may limit generalizability. Second, liver biopsies were performed in only 30% of patients due to ethical and clinical constraints, which restricts the scope of histopathological correlation[14]. Third, we did not assess long-term outcomes such as progression to chronic hepatitis or fulminant liver failure, which could provide further prognostic insights. Future multicenter prospective studies with larger cohorts and longer follow-up are warranted to confirm and expand on these findings[25].

CONCLUSION

This study demonstrates that serum inflammatory markers, particularly IL-6 and TNF-α, strongly correlate with histopathological findings in community-acquired acute viral hepatitis. CRP reflected lobular necrosis, IL-6 predicted portal inflammation, and TNF-α was associated with hepatocyte

ballooning and interface hepatitis. These results highlight the potential of inflammatory biomarkers as non-invasive surrogates for assessing disease severity and monitoring patients, especially in resource-limited settings where liver biopsy is not routinely feasible. Integration of these biomarkers into clinical practice may improve risk stratification and guide therapeutic decision-making. However, larger multicenter studies are needed to validate these findings and to establish cut-off levels that can be used reliably in clinical algorithms.

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