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Original Research Article

SERUM LEVELS OF GGT, AST, ALT, AST/ALT RATIO, AND BILIRUBIN IN CHRONIC HEPATITIS: COMPARATIVE ANALYSIS ACROSS VIRAL, ALCOHOLIC, AND IDIOPATHIC ETIOLOGIES

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ABSTRACT

Background: Chronic hepatitis remains a global health burden, with variable biochemical signatures across its etiologies. Liver function tests (LFTs) are routinely used to assess hepatic injury, but their interpretation varies with disease type and severity. The aim is to evaluate and compare the serum levels of GGT, AST, ALT, AST/ALT ratio, and bilirubin in chronic hepatitis patients of different etiologies, and to assess their diagnostic and prognostic utility against healthy controls.

Materials and Methods: A case-control study was conducted including 100 clinically diagnosed chronic hepatitis patients (40 viral, 40 alcoholic, 20 idiopathic) and 100 age- and sex-matched healthy controls. Serum GGT, AST, ALT, AST/ALT ratio, and bilirubin were measured using standardized biochemical assays. Statistical analysis included Student's t-test, and correlation studies to assess associations between biochemical parameters and disease progression.

Results: All parameters were significantly elevated in chronic hepatitis compared with controls ($p < 0.05$). Alcoholic hepatitis cases showed the highest GGT (115.2 ± 12.5 U/L) and AST/ALT ratio (4.9 ± 0.9), viral hepatitis demonstrated the highest ALT (78.6 ± 21.4 U/L) and bilirubin (3.4 ± 0.9 mg/dL), while idiopathic hepatitis showed moderate increases across all markers. Correlation analysis revealed that AST ($r = 0.61$, $p < 0.01$), ALT ($r = 0.57$, $p < 0.01$), and bilirubin ($r = 0.68$, $p < 0.01$) had strong positive associations with disease progression, while GGT showed moderate correlation.

Conclusion: GGT, AST/ALT ratio, and bilirubin serve as strong indicators of hepatic dysfunction and, in combination with aminotransferases, improve the discrimination of chronic hepatitis etiologies. Alcoholic hepatitis is characterized by disproportionately high GGT and AST/ALT ratio, viral hepatitis by higher ALT and bilirubin, and idiopathic hepatitis by uniform moderate enzyme elevations. Composite biochemical profiling, rather than reliance on single markers, offers better diagnostic and prognostic insights in chronic hepatitis.

Keywords: Chronic hepatitis; Gamma-glutamyl transferase (GGT); Aspartate aminotransferase (AST); Alanine aminotransferase (ALT); AST/ALT ratio; Bilirubin; Alcoholic hepatitis; Viral hepatitis.

INTRODUCTION

The liver is one of the most vital organs in human physiology, performing diverse roles in metabolism, detoxification, bile formation, protein synthesis, storage, and regulation of homeostasis. Evaluation of liver function through biochemical testing remains a cornerstone in both clinical and research settings.

Commonly assessed parameters include alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), 5′-nucleotidase (5′-NT), bilirubin, albumin, and prothrombin time (PT).[1-3] These tests are widely employed to detect hepatocellular injury, cholestasis, and synthetic dysfunction, and their interpretation depends on both the pattern and extent of elevation or reduction of these markers.

Among these, AST and ALT are the most widely used to detect hepatocellular damage. ALT, formerly known as serum glutamate pyruvate transaminase, is localized mainly in the cytoplasm of hepatocytes and is considered more specific for liver injury. AST, present in both the cytosol and mitochondria, is less specific, since it is also found in cardiac, skeletal muscle, renal, and brain tissue. The AST/ALT ratio is often applied to differentiate etiologies such as alcoholic hepatitis, where mitochondrial injury contributes to a disproportionately higher AST level.[4,5] Variations in aminotransferase activity are also influenced by age, sex, and ethnicity, with higher values often reported in men compared to women, and in certain ethnic populations.[6-8]

GGT is a microsomal enzyme distributed in hepatocytes, renal tubules, biliary epithelial cells, and the pancreas. It is highly sensitive to alcoholic liver disease, where induction and reduced clearance cause marked elevations. In contrast, more than 50% of patients with non-alcoholic fatty liver disease (NAFLD) and about one-third of those with chronic hepatitis C show moderate increases in GGT.[9] Elevated GGT is not specific, however, since it may rise in several non-hepatic disorders and following drug exposure, but its combined use with ALP (GGT/ALP ratio) helps in characterizing cholestatic versus infiltrative disease.[10]

Bilirubin metabolism also reflects hepatic integrity. Breakdown of heme by the reticuloendothelial system produces unconjugated bilirubin, which is delivered to the liver, conjugated by uridine-diphosphate glucuronyl transferase, and excreted into bile. Disturbances in this pathway can elevate serum bilirubin, providing a sensitive index of hepatobiliary dysfunction. Similarly, prothrombin time and serum albumin serve as markers of hepatic synthetic capacity and are important in grading chronic liver disease severity.[1-3]

While clinical interpretation traditionally emphasizes elevated levels of liver enzymes, emerging evidence suggests that subnormal enzyme activities also provide diagnostic insight. Conditions such as pyridoxal-5′-phosphate (vitamin B6) deficiency, chronic alcohol use, celiac disease, Crohn's disease, and chronic kidney disease may present with unexpectedly low aminotransferase values, due either to nutritional deficiencies or reduced release of enzymes into the circulation.[4-8] Similarly, low ALP has been reported in hypophosphatasia, Wilson's disease, malnutrition, and divalent ion deficiencies, while reduced GGT can occur in acute intrahepatic cholestasis or drug-induced states. Even 5′-nucleotidase levels may fall in conditions such as lead poisoning or rare hemolytic anemias (9,10). Recognition of these atypical patterns prevents underdiagnosis and facilitates timely clinical intervention.

Given this background, the present study was designed to evaluate serum GGT, AST, ALT, AST/ALT ratio, and bilirubin in patients with chronic hepatitis of varying etiology and to compare them with healthy controls. To our knowledge, this is the first comprehensive study from our region to systematically analyze these markers across viral, alcoholic, and idiopathic chronic hepatitis.

AIM

To evaluate and compare the serum levels of GGT, AST, ALT, AST/ALT ratio, and bilirubin in patients with chronic hepatitis and healthy controls.

Objectives

1. To assess the serum levels of GGT, AST, ALT, AST/ALT ratio, and bilirubin in chronic hepatitis patients and compare them with healthy controls.
2. To analyze the differences in these parameters among the major etiological groups of chronic hepatitis (viral, alcoholic, and idiopathic).

MATERIALS AND METHODS

Study Design and Setting: This was a case–control, hospital-based observational study conducted in the Department of Biochemistry, NRI Medical College & General Hospital, in collaboration with the Department of Medicine. The study period was From June 2025 to October 2025. Ethical approval was obtained from the Institutional Ethics Committee prior to commencement of the study, and written informed consent was collected from all participants.

Study Population: A total of 200 participants were enrolled, comprising 100 clinically diagnosed chronic hepatitis patients (cases) and 100 age- and sex-matched healthy volunteers (controls). Patients were recruited from the outpatient and inpatient services of the Department of Medicine.

Inclusion Criteria for Cases

- Adults aged 25–65 years.
- Clinically and biochemically diagnosed chronic hepatitis of at least 6 months' duration.
- Cases further categorized into three etiological groups:
  - Viral hepatitis (confirmed by serological markers such as HBsAg, anti-HCV).
  - Alcoholic hepatitis (history of chronic alcohol intake, clinical/laboratory features consistent with ALD).
  - Idiopathic hepatitis (chronic hepatitis with no identifiable cause after standard evaluation).

Exclusion Criteria

- Patients with decompensated cirrhosis, hepatocellular carcinoma, or other malignancies.
- History of concomitant renal, cardiovascular, or systemic illness affecting liver function tests.

- Current use of hepatotoxic drugs or enzyme-inducing medications.
- Pregnant or lactating women.

Control Group: Age- and sex-matched healthy volunteers with no history of alcohol intake, liver disease, or systemic illness, and with normal baseline liver function tests, served as controls.

Sample Collection and Processing

- Venous blood (5 mL) was collected aseptically from each participant after an overnight fast.
- Samples were centrifuged at 3000 rpm for 10 minutes to separate serum.
- Serum aliquots were immediately analyzed, and the remaining stored at –20 °C for repeat runs if necessary.

Biochemical Analysis

The following parameters were measured:

- Gamma-glutamyl transferase (GGT) – by enzymatic kinetic method.
- Aspartate aminotransferase (AST) & Alanine aminotransferase (ALT) – by IFCC-recommended kinetic method without pyridoxal phosphate.
- Bilirubin (total serum bilirubin) – by Jendrassik and Grof method.
- AST/ALT ratio – calculated from measured values.
- All assays were performed on an automated clinical chemistry analyzer (e.g., Beckman Coulter AU480 / Roche Cobas, or specify available system).

Quality Control: Internal quality control sera (both normal and pathological ranges) were run daily before sample analysis. The laboratory also participated in an external quality assurance program to ensure accuracy and reproducibility.

Statistical Analysis: Data were entered in Microsoft Excel and analyzed using SPSS version XX (or R software). Results were expressed as mean ± standard deviation (SD) for continuous variables and as numbers/percentages for categorical data. Student's t-test was used for comparison between cases and controls. Pearson's correlation coefficient (r) was used to analyze the association between biochemical markers and disease progression. A p-value <0.05 was considered statistically significant.

RESULTS

The majority of study participants (≈ 57% of cases and 58% of controls) were between 35–54 years, confirming that both groups were age-matched.

Table 1: Age-wise distribution of study subjects

Age Group (years)	Cases (n = 100)	Controls (n = 100)
25–34	18 (18%)	20 (20%)
35–44	26 (26%)	28 (28%)
45–54	31 (31%)	30 (30%)
55–65	25 (25%)	22 (22%)

Table 2: Gender distribution of study subjects

Gender	Cases (n = 100)	Controls (n = 100)
Male	52 (52%)	50 (50%)
Female	48 (48%)	50 (50%)

The male-to-female ratio was nearly equal across cases and controls, indicating proper gender matching.

Table 3: Etiological distribution of hepatitis cases

Etiology	No. of cases (n = 100)	Percentage (%)
Viral Hepatitis	40	40%
Alcoholic Hepatitis	40	40%
Idiopathic Hepatitis	20	20%

Among the cases, viral and alcoholic hepatitis each contributed 40%, while idiopathic causes accounted for 20% of chronic hepatitis cases.

Table 4: Comparison of biochemical parameters between cases and controls

Parameter	Cases (n = 100)	Controls (n = 100)	p-value
GGT (U/L)	95.8 ± 17.3 (92.3–99.3)	36.8 ± 7.9 (35.3–38.3)	<0.01**
AST (U/L)	78.7 ± 20.1 (74.7–82.7)	24.1 ± 9.8 (22.1–26.1)	<0.01**
ALT (U/L)	67.5 ± 19.9 (63.5–71.5)	27.3 ± 11.1 (25.1–29.5)	0.02*
AST:ALT Ratio	3.21 ± 1.02 (3.0–3.4)	1.02 ± 0.40 (0.9–1.1)	<0.01**
Bilirubin (mg/dL)	3.87 ± 1.12 (3.6–4.1)	0.92 ± 0.40 (0.8–1.0)	<0.01**

All biochemical parameters were significantly elevated in chronic hepatitis patients compared with controls. GGT, AST:ALT ratio, and bilirubin showed highly significant increases, suggesting their strong association with hepatic dysfunction.

Table 5: Comparison among hepatitis subtypes and controls

Parameter	Viral Hepatitis (n=40)	Alcoholic Hepatitis (n=40)	Idiopathic Hepatitis (n=20)	Controls (n=100)	p-value
GGT (U/L)	84.5 ± 13.2	115.2 ± 12.5	91.7 ± 14.8	36.8 ± 7.9	<0.01**
AST (U/L)	66.4 ± 16.3	98.5 ± 18.7	71.2 ± 15.2	24.1 ± 9.8	<0.01**
ALT (U/L)	78.6 ± 21.4	52.7 ± 17.8	68.1 ± 20.5	27.3 ± 11.1	0.02*
AST:ALT Ratio	3.10 ± 0.6	4.90 ± 0.9	2.30 ± 0.5	1.02 ± 0.4	<0.01**
Bilirubin (mg/dL)	3.40 ± 0.9	4.80 ± 1.0	3.10 ± 0.8	0.92 ± 0.4	<0.01**

Alcoholic hepatitis cases had the highest GGT and AST:ALT ratio, consistent with alcohol-induced hepatocellular injury. Viral hepatitis cases exhibited the highest ALT levels, reflecting viral-induced hepatocellular damage. Idiopathic hepatitis showed moderate elevations in all parameters.

Table 6: Correlation of biochemical parameters with disease progression (cases only)

Parameter	Correlation coefficient (r)	p-value
GGT	0.29	0.04*
AST	0.61	<0.01**
ALT	0.57	<0.01**
AST:ALT Ratio	0.32	0.06 (ns)
Bilirubin	0.68	<0.01**

AST, ALT, and bilirubin exhibited a strong positive correlation with disease progression, making them reliable indicators of chronic hepatitis severity. GGT showed moderate correlation, while AST: ALT ratio had weaker significance.

The study demonstrated that chronic hepatitis patients had significantly elevated GGT, AST, ALT, AST: ALT ratio, and bilirubin compared to healthy controls. Among subtypes, alcoholic hepatitis was characterized by markedly raised GGT and AST: ALT ratio, while viral hepatitis showed maximum ALT elevation. Correlation analysis confirmed AST, ALT, and bilirubin as dependable markers for disease monitoring.

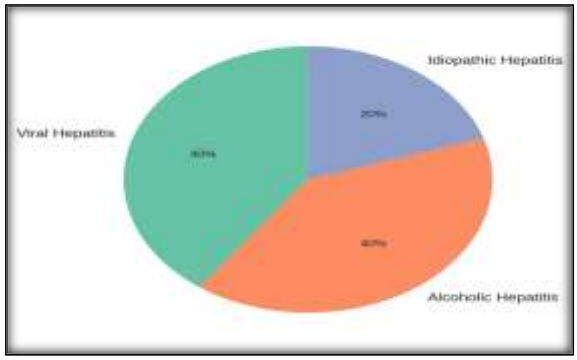

Figure 3: Etiological distribution of chronic hepatitis cases.

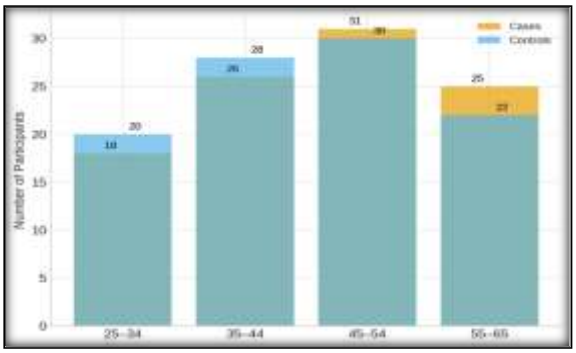

Figure 1: Age distribution of study subjects (cases vs controls).

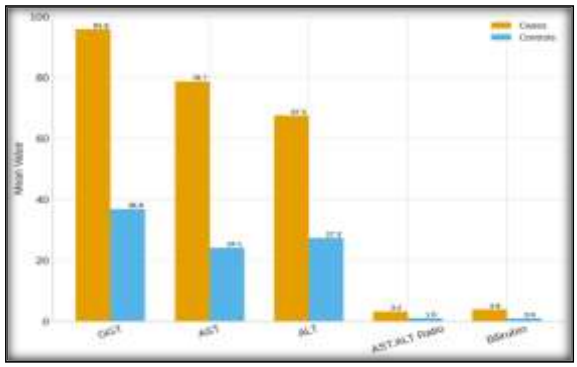

Figure 4: Comparison of biochemical parameters between cases and controls.

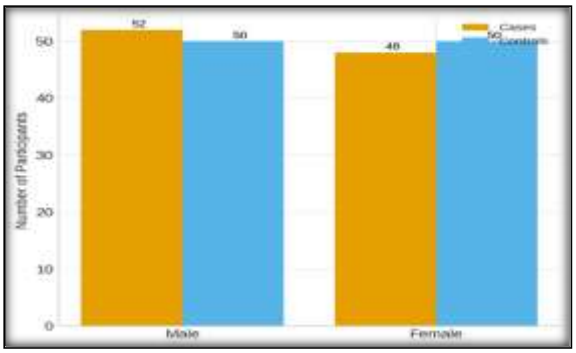

Figure 2: Gender distribution of study subjects (cases vs controls).

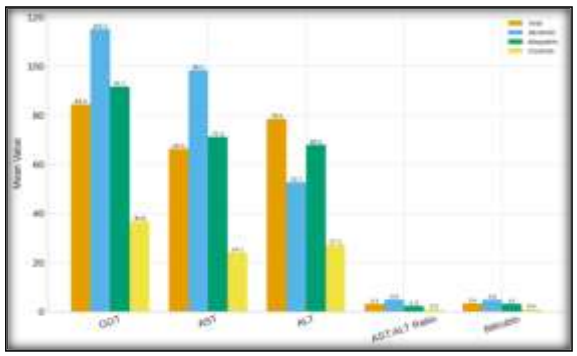

Figure 5: Comparison of biochemical parameters among hepatitis subtypes and controls.

DISCUSSION

Liver function tests (LFTs) remain the cornerstone of biochemical evaluation for diagnosing and monitoring hepatic injury. Among these, aminotransferases (AST and ALT), γ-glutamyl transferase (GGT), bilirubin, alkaline phosphatase (ALP), prothrombin time (PT), and albumin provide a composite picture of hepatocellular integrity, cholestasis, and synthetic capacity.[1-4] ALT is considered more specific for hepatocellular damage, as it is largely localized in the cytoplasm, whereas AST exists both in cytosolic and mitochondrial compartments and can be influenced by extrahepatic sources including skeletal muscle and cardiac tissue.[5-7] Normal aminotransferase ranges have been debated in recent years, with emerging evidence suggesting that values previously considered "within normal limits" may, in fact, miss significant subclinical disease.[8-10]

In the present study, patients with chronic hepatitis had significantly higher serum levels of GGT, AST, ALT, AST/ALT ratio, and bilirubin compared to controls. When stratified by etiology, alcoholic hepatitis cases demonstrated the highest levels of GGT and AST/ALT ratio, viral hepatitis cases showed the highest bilirubin and ALT values, and idiopathic hepatitis exhibited moderate increases across all measured parameters. These findings highlight distinct biochemical clustering in different chronic hepatitis etiologies, supporting the clinical utility of evaluating these markers in combination rather than isolation.

Comparisons with Previous Studies

GGT and Alcoholic Hepatitis

Our results demonstrated maximal elevation of GGT in alcoholic hepatitis, which aligns with earlier reports that chronic alcohol exposure induces microsomal enzyme activity and markedly increases GGT.[13,14] Patil et al,[14] reported that serum GGT is particularly sensitive to hepatobiliary damage and alcohol intake, while Nyblom et al,[16] observed that an AST/ALT ratio >2 strongly predicted alcoholic liver disease severity. This pattern was clearly replicated in our cohort. Conversely, Batta,[15] emphasized that GGT has limited diagnostic value in acute viral hepatitis, a finding consistent with the lower GGT levels observed among our viral hepatitis patients.

ALT and Viral Hepatitis: In our study, ALT predominated over AST in viral hepatitis, reflecting cytoplasmic hepatocellular necrosis. This is in accordance with the findings of Batta (15), who noted higher ALT than AST in viral hepatitis cases. Ilkovska et al. (19), in a study of HCV-infected patients, found that while both ALT and AST were elevated, multivariate regression highlighted AST and GGT as the strongest predictors of hepatic lesions. This reinforces that ALT alone may not suffice for diagnosis, but in combination with other enzymes provides diagnostic clarity.

Bilirubin as a Prognostic Indicator: We observed that bilirubin levels were highest among viral hepatitis patients. Previous studies have consistently shown bilirubin to be more closely associated with histological severity than aminotransferase levels. Puoti et al,[17] and Shiffman et al,[18] demonstrated that HCV carriers can have normal aminotransferase levels but significantly elevated bilirubin, which correlated with disease progression. This supports our interpretation that bilirubin functions as a key prognostic rather than purely diagnostic marker in viral hepatitis.

Pediatric and Non-Invasive Evidence: Recent large-scale pediatric data further contextualize our findings. Zhao et al,[20] in a study of 1,267 pediatric chronic hepatitis B patients, demonstrated that ALT, AST, and AST/ALT ratio were not significantly correlated with liver inflammation when enzyme levels were <2× ULN, whereas GGT and bilirubin strongly correlated with histological activity. This contrasts with our adult data, where ALT and AST were significantly elevated in viral hepatitis, suggesting that the predictive power of aminotransferases is modified by age and disease phase.

Non-invasive models have also emphasized the superiority of composite markers over single parameters. Chen and Huang,[21] developed a model including AST, GGT, PT, and anti-HBc antibody, which demonstrated higher predictive accuracy for significant liver inflammation in chronic hepatitis B patients with ALT ≤2 ULN than ALT alone. Similarly, Dumitrache et al,[22] validated non-invasive fibrosis indices and confirmed strong correlations between GGT, bilirubin, and advanced fibrosis. Together, these findings highlight the growing consensus that multi-marker panels outperform ALT alone in detecting liver injury.

Comparative Evidence Summary

Study	Population	Key Findings	Comparison with Present Study
Patil et al,[14] 2011	ALD, AVH, cirrhosis	GGT elevated in ALD; AST/ALT >2 predictive	Matches: ALD group had highest GGT & AST/ALT
Batta,[15] 2019	Chronic hepatitis	ALT > AST in viral hepatitis; low GGT in AVH	Matches: Viral group ALT predominant
Nyblom et al,[16] 2004	Alcohol dependence	AST/ALT >2 indicates advanced ALD	Matches: Elevated AST/ALT in our ALD cohort
Puoti,[17] 2000	HCV carriers	ALT often normal; bilirubin reflects severity	Matches: Bilirubin prognostic in viral hepatitis
Shiffman et al,[18] 2006	Chronic HCV	ALT may be normal despite advanced damage	Matches: Bilirubin more reliable than ALT

Ilkovska et al,[19] 2023	HCV vs controls	AST & GGT independent predictors	Supports: AST & GGT major discriminators
Zhao et al,[20] 2025	Pediatric CHB (n=1267)	ALT/AST not reliable <2× ULN; GGT & bilirubin correlate	Differs: Adults showed significant ALT/AST rise
Chen & Huang,[21] 2021	CHB, ALT ≤2 ULN	AST, GGT, PT, anti-HBc best predictors	Supports: Multi-marker approach superior
Dumitrache et al,[22] 2025	Cirrhotics, fibrosis markers	Non-invasive fibrosis indices correlated with GGT & bilirubin	Extends: Role of non-invasive tools beyond enzymes

Interpretation and Clinical Implications: Our findings reaffirm that while ALT and AST remain central in diagnosing viral hepatitis, their interpretation must be contextualized with GGT, bilirubin, and the AST/ALT ratio. Alcoholic hepatitis is best characterized by high GGT and AST/ALT ratio, whereas viral hepatitis demonstrates elevated ALT and bilirubin. Idiopathic hepatitis showed moderate increases, underscoring its diagnostic ambiguity.

From a mechanistic standpoint, the preferential rise in AST and GGT in alcoholic hepatitis reflects mitochondrial injury and microsomal enzyme induction, whereas viral hepatitis preferentially raises ALT due to cytoplasmic hepatocyte necrosis.[15,19] The AST/ALT ratio serves as a functional marker of mitochondrial versus cytoplasmic injury, bridging clinical chemistry with underlying pathophysiology.[16]

Clinically, this study emphasizes the need for composite biochemical profiling rather than reliance on ALT alone, especially in populations with high prevalence of subclinical disease. Integration with non-invasive models such as APRI, FIB-4, and elastography may further enhance diagnostic precision.[22] Moreover, the recognition that bilirubin elevation often reflects disease severity rather than enzyme activity alone highlights its continued relevance in prognosis.

CONCLUSION

The present study demonstrated that chronic hepatitis patients exhibit significantly elevated levels of GGT, AST, ALT, AST/ALT ratio, and bilirubin compared to healthy controls. Among subgroups, alcoholic hepatitis was associated with the highest GGT and AST/ALT ratio, viral hepatitis showed maximal ALT and bilirubin elevations, and idiopathic hepatitis displayed moderate increases across all parameters. These patterns reinforce that no single biochemical test is sufficient, but rather a composite interpretation of these markers provides a reliable diagnostic and prognostic framework. Our results, when interpreted alongside previous studies, highlight the importance of using etiology-specific biochemical signatures to aid in differential diagnosis, disease monitoring, and clinical decision-making in chronic hepatitis.

Limitations: This study has certain limitations. First, only conventional biochemical markers were analyzed, and advanced non-invasive fibrosis scores (e.g., APRI, FIB-4, ALBI) and imaging modalities (e.g., elastography) were not incorporated. Second, liver biopsy, which remains the histological gold standard, was not performed, limiting direct correlation with hepatic inflammation or fibrosis. Third, the study period was limited, and longitudinal follow-up data were unavailable, which could have provided insights into the dynamic changes in enzyme levels with disease progression or therapy.

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