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Original Article

Predicting hepatitis C infection via machine learning

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Abstract: Objective: HCV infection is frequently asymptomatic, with current diagnosis relying mainly on costly and less accessible HCV RNA testing. While HCV-Ab and biochemical markers exhibit suboptimal diagnostic performance, whether machine learning can enhance their accuracy remains unclear. Methods: This study is a retrospective study, which included data from 179 patients whose HCV-Ab levels were greater than 1.00 S/CO to explore the relationship between HCV-Ab, biochemical indicators, and HCV infection. Univariate logistic regression and restricted cubic splines (RCS) were employed to explore these associations. Machine learning integrated HCV-Ab and biochemical indicators to predict early HCV infection (undiagnosed chronic cases), with validation conducted using receiver operating characteristic curve (ROC) analysis. The machine learning approach randomly divided study participants into training and test sets at a 5:5 ratio, with the training set being used for variable selection and model construction. Results: After full adjustment, TP showed no significant association with HCV infection. Restricted cubic spline (RCS) analysis revealed nonlinear relationships between HCV-Ab, ALT, AST, mAST, GGT, A/G and HCV infection. HCV-Ab exhibited an inflection point at 11.17 (below: OR = 1.04 per unit increase; above: no association). Similar threshold patterns were observed for ALT, AST, mAST and GGT. The integrated HCV-Ab and biochemical marker model achieved excellent predictive performance (AUC = 0.977). Conclusion: TP exhibited a linear association with HCV infection, whereas HCV-Ab, ALT, AST, mAST and GGT showed nonlinear associations with distinct threshold effects. Early prediction of HCV infection using these indicators represents a cost-effective strategy.

Keywords: HCV-RNA, HCV-Ab, biochemical indicators, machine learning, restricted cubic splines

Introduction

Hepatitis C virus (HCV), a single-stranded RNA virus of the Flaviviridae family, primarily targets hepatocytes and leads to both acute and chronic hepatitis [1, 2]. Globally, approximately 58 million individuals are chronically infected with HCV, with approximately 399,000 deaths annually attributed to HCV-related complications such as cirrhosis and hepatocellular carcinoma (HCC) [3-6]. The primary modes of transmission include blood exposure (e.g., transfusion, needle sharing, unsafe medical procedures), vertical mother-to-child transmission, and sexual contact. High-risk populations include intravenous drug users, recipients of unscreened blood products, and healthcare workers exposed to blood. Undiagnosed early HCV infections frequently progress to chronic hepatitis (55%-85% of acute cases), subsequently leading to hepatic fibrosis, cirrhosis, and HCC [7-9]. Studies indicate that 15%-30% of chronic HCV patients develop cirrhosis within two decades, with an annual progression rate of 1%-5% to HCC [10-12]. HCV infection is also associated with extrahepatic manifestations, including metabolic abnormalities, cardiovascular diseases, and lymphoma [13-17]. The introduction of direct-acting antivirals (DAAs) has significantly improved treatment outcomes, achieving cure rates exceeding 95% [18, 19]. The gold standard for HCV diagnosis is the detection of HCV-RNA [20], which confirms active infection and quantifies viral load. However it exhibits inherent limitations [21]: 1) High intrinsic test cost: The cost of HCV RNA test reagents and specialized equipment (e.g., PCR machines) is substantially higher than that of HCV antibody tests or routine biochemical assays. In resource-limited settings, the cost per test can pose a significant economic barrier. 2) Infrastructure and personnel costs: RNA

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testing generally requires specialized molecular biology laboratory facilities (e.g., strict containment, specialized equipment, stable power/cold chain) and relies on trained technical personnel. Establishing and maintaining such laboratories is expensive. 3) Accessibility challenges: Molecular diagnostics laboratories are predominantly concentrated in urban centers or large tertiary hospitals, resulting in severely limited accessibility in remote, rural, or resource-poor areas [22]. In contrast, HCV antibody (HCV-Ab) and biochemical indicators assays are cost-effective and widely available in primary care facilities [23]. HCV-Ab refers to antibody produced by activated immune cells in response to HCV infection. This antibody persists in the human body and is typically detected in individuals who have achieved viral clearance (either through treatment or spontaneous resolution) as well as those with chronic active infections. The HCV-Ab test offers significant advantages: low cost, operational simplicity, rapid results, and high accessibility. The hepatitis C virus primarily infects hepatocytes, inducing hepatic injury and subsequent liver dysfunction [24]. Markedly elevated levels of biochemical indicators may indicate active liver injury and, combined with other factors, can heighten suspicion of active infection or disease activity. The combined utilization of serological HCV-Ab and biochemical detection may enhance diagnostic accuracy and improve clinical evaluation of HCV infection status. This study analyzed biochemical parameters and HCV-RNA results from 179 patients with HCV-Ab levels greater than 1.00 S/CO to investigate the dynamic changes in HCV-Ab and biochemical profiles. Utilizing machine learning techniques, we evaluated the predictive performance of combining HCV-Ab with biochemical indicators for early diagnosis of HCV.

Methods

Data collection and processing

This is a retrospective study. The primary data were collected from patients whose HCV-Ab level was greater than 1.00 S/CO, at Shidong Hospital Affiliated to University of Shanghai for Science and Technology between 2019 and 2024, with all data obtained from medical records. The Ethics Committee of Shanghai Shidong Hospital approved the study protocol (Ethics approval report ID: 2025-031-01). For all participants, biochemical indicators data from the first visit were screened and used as features for model construction. Patients with other hepatitis infections or liver/kidney impairment due to other etiologies were excluded. Detection of HCV-RNA is the gold standard for the diagnosis of HCV infection.

Specimen collection and laboratory testing

Venous blood samples were collected using sterile techniques. For serum samples, a standard serum separator tube without anticoagulants was utilized, allowing the blood to clot at room temperature for 30 minutes prior to centrifugation. The samples were then centrifuged at 3,500 RPM for 10 minutes to separate the serum.

The Abbott Alinity i diagnostic kit (America) was employed for quantitative detection of HCV antibody in human serum using the chemiluminescent microparticle immunoassay (CMIA) method according to the manufacturer's instructions. For HCV-RNA detection, the in vitro nucleic acid amplification kit (Sansure Biotech Inc., Changsha, China) was utilized, with all procedures strictly adhering to the manufacturer's protocols. Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), mitochondrial aspartate aminotransferase isoenzyme (mAST), Alkaline Phosphatase (ALP), Gamma-Glutamyl Transferase (GGT), Total Bilirubin (TB), Conjugated Bilirubin (CB), Total Protein (TP), Albumin (ALB), Albumin-to-Globulin ratio (A/G), Blood Urea Nitrogen (BUN), Creatinine (Cr), Uric Acid (UA), Glomerular Filtration Rate (GFR), and Glucose (GLU) were quantified using a clinical chemistry analyzer (Beckman5800 Chemistry System). All the reagents were provided by the respective manufacturers as part of pre-packaged kits, ensuring consistency and reliability across tests. All tests were performed in accordance with rigorous quality control protocols.

Study methods

First, trend analysis of multivariate logistic regression was used to assess the association between HCV-Ab, biochemical indicators and HCV infection. TP was introduced into the logistic regression model as a continuous variable, and the results were expressed as odds ratio (OR) and 95% confidence interval (95% CI). Three models were constructed by adjusting for

different confounding variables. In model 1 was unadjusted for variables. In model 2, confounders including gender and age were adjusted. Model 3 was further adjusted for HCV-Ab, ALT, AST, mAST, GGT, A/G. To further explore the potential nonlinear relationship between HCV-Ab, biochemical indicators and HCV infection, restricted cubic spline (RCS) regression analyses were performed. Likelihood ratio tests were used to detect nonlinearity. The threshold effect of HCV-Ab and biochemical indicators on HCV infection risk was further analyzed by a two-stage linear regression model.

Machine learning algorithms to construct HCV infection prediction model

In this study, multiple machine learning methods were used to investigate the application and predictive value of HCV-Ab and biochemical indicators in the diagnosis of HCV infection. The study participants were randomly divided into a train set and a test set in a ratio of 5:5. The train set was used to screen the variables and construct the model. The test set was used to evaluate the performance of the final model. The diagnostic performance of each model was evaluated by calculating the area under the curve (AUC) value. The optimal model was selected based on the AUC value to plot the receiver operating characteristic curve (ROC) [25].

Statistical analysis

Continuous variables with non-normal distributions were assessed using the Mann-Whitney U test and expressed as medians (interquartile range [Q1, Q3]). Categorical variables were compared using the chi-square test and reported as counts (percentages). All statistical analyses were performed using R software (version 4.4.0), and statistical significance was defined as $P < 0.05$.

Results

Baseline clinical characteristics of participants

This study included a total of 179 participants, with 130 cases (72.63%) in the HCV control group and 49 cases (27.37%) in the HCV disease group. Statistically significant differences ($P < 0.05$) were observed between the two groups in Age, HCV-Ab, ALT, AST, mAST, GGT, TP, A/G ratio, and Gender; whereas no significant differences ($P > 0.05$) were found in ALP, TB, CB, ALB, BUN, Cr, UA, GFR, or GLU (**Table 1**).

A linear relationship between TP and the risk of HCV infection

Three models were constructed by adjusting for different confounding variables to evaluate the associations between TP with the risk of HCV infection. After adjusting for all confounding variables, in the final model, the relationship between TP (OR = 1.05, 95% CI: 0.98-1.13, P = 0.149) and HCV infection was not significant. However, the *p*-value showed a trend toward significance (**Table 2**).

A nonlinear relationship and threshold effect were observed between HCV-Ab, partial biochemical indicators, and the risk of HCV infection

Comparative analysis was performed of HCV-Ab and biochemical indicators between the HCV Control group and Disease group (**Figure 1A-F**). To further ensure the robustness of the results, the potential nonlinear relationship between HCV-Ab, partial biochemical indicators and the risk of HCV infection was examined. In the RCS regression model, after adjusting for all confounding factors, significant nonlinear associations were observed between HCV-Ab, ALT, AST, mAST, GGT, A/G and HCV infection (nonlinear $P < 0.05$) (**Figure 2A-F**).

In **Table 3**, further analysis revealed a threshold effect in the association between HCV-Ab and the risk of HCV infection (P for likelihood ratio test < 0.001), with an inflection point at 11.17. When HCV-Ab were below 11.17, a positive correlation exists between HCV-Ab and the risk of HCV infection (OR = 2.04, 95% CI: 1.34-3.10, P < 0.001). Each unit increase was associated with a 1.04-fold increase in the risk of HCV infection. However, when HCV-Ab exceeded 11.17, no significant association with the risk of HCV infection was observed (OR = 0.77, 95% CI: 0.59-1.01, P = 0.057). Additionally, a threshold effect was identified for ALT (P for likelihood ratio test = 0.017) with an inflection point at 54.00, and for AST (P for likelihood ratio test = 0.007) with an inflection point at 39.00, and for mAST (P for likelihood ratio test = 0.005) with an inflection point at 7.00. Overall, a positive correlation was observed between GGT and HCV infection (OR = 1.00, 95% CI: 1.00-1.01, P = 0.026). A threshold effect was identi-

Table 1. Clinical characteristics

Variables	Total (n = 179)	Control (n = 130)	Disease (n = 49)	Statistic	*P*
Age, M (Q_1, Q_3)	63.00 (55.50, 69.00)	64.50 (56.25, 69.75)	60.00 (54.00, 64.00)	Z = -2.22	0.026
HCV-Ab, M (Q_1, Q_3)	5.56 (1.54, 14.18)	2.27 (1.31, 8.27)	13.84 (11.85, 15.45)	Z = -7.03	< .001
ALT, M (Q_1, Q_3)	21.00 (14.50, 50.00)	18.00 (13.00, 25.75)	70.00 (48.00, 135.00)	Z = -7.75	< .001
AST, M (Q_1, Q_3)	26.00 (19.00, 47.00)	22.00 (18.00, 28.00)	68.00 (37.00, 99.00)	Z = -7.55	< .001
mAST, M (Q_1, Q_3)	3.00 (2.70, 5.25)	3.00 (2.40, 4.00)	5.00 (3.00, 8.20)	Z = -3.84	< .001
ALP, M (Q_1, Q_3)	82.00 (68.50, 100.00)	82.00 (68.50, 102.75)	82.00 (69.00, 98.00)	Z = -0.26	0.797
GGT, M (Q_1, Q_3)	30.50 (21.00, 63.00)	30.00 (18.00, 46.75)	44.00 (30.50, 103.00)	Z = -3.80	< .001
TB, M (Q_1, Q_3)	14.00 (10.90, 17.30)	14.00 (11.20, 16.45)	14.00 (10.20, 19.00)	Z = -0.75	0.454
CB, M (Q_1, Q_3)	2.80 (2.30, 3.90)	2.80 (2.23, 3.50)	2.80 (2.60, 4.90)	Z = -1.53	0.127
TP, M (Q_1, Q_3)	69.00 (64.55, 73.40)	69.00 (63.62, 72.72)	70.40 (65.90, 75.00)	Z = -2.32	0.020
ALB, M (Q_1, Q_3)	39.30 (35.75, 41.95)	39.30 (35.70, 42.18)	39.20 (36.10, 40.70)	Z = -0.51	0.611
A/G, M (Q_1, Q_3)	1.33 (1.18, 1.50)	1.33 (1.19, 1.55)	1.26 (1.16, 1.33)	Z = -2.44	0.015
BUN, M (Q_1, Q_3)	5.35 (4.60, 6.95)	5.35 (4.48, 6.88)	5.35 (4.80, 7.30)	Z = -0.50	0.619
Cr, M (Q_1, Q_3)	68.60 (58.10, 89.45)	68.60 (58.05, 88.20)	68.60 (58.30, 91.00)	Z = -0.02	0.987
UA, M (Q_1, Q_3)	339.30 (270.00, 399.65)	339.30 (269.50, 395.70)	339.30 (287.60, 415.50)	Z = -0.43	0.669
GFR, M (Q_1, Q_3)	97.80 (74.25, 107.30)	97.80 (73.77, 107.12)	97.80 (78.70, 111.40)	Z = -0.63	0.528
GLU, M (Q_1, Q_3)	5.36 (4.99, 6.08)	5.36 (4.97, 5.96)	5.36 (5.16, 6.26)	Z = -1.20	0.230
Gender, n (%)				X^2 = 5.65	0.017
Female	88 (49.16)	71 (54.62)	17 (34.69)		
Male	91 (50.84)	59 (45.38)	32 (65.31)		

Z, Mann-Whitney test, χ^2, Chi-square test; M (Q_1, Q_3), Median (1st Quartile, 3st Quartile); n (%), numbers (percentages); HCV-Ab, hepatitis C virus antibody; ALT, alanine aminotransferase; AST, aspartate aminotransferase; mAST, mitochondrial aspartate aminotransferase isoenzyme; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; TB, total bilirubin; CB, conjugated bilirubin; TP, total protein; ALB, albumin; A/G, albumin-to-globulin ratio; BUN, blood urea nitrogen; Cr, creatinine; UA, uric acid; GFR, glomerular filtration rate; GLU, glucose.

Table 2. The relationship between TP and the risk of HCV infection

Variables	Model 1		Model 2		Model 3	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
TP	1.06 (1.01-1.11)	0.030	1.06 (1.01-1.11)	0.032	1.05 (0.98-1.13)	0.149
TP (median)						
1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
2	2.07 (1.03-4.15)	0.042	2.23 (1.06-4.69)	0.035	1.79 (0.58-5.52)	0.314
P for trend		0.042		0.035		0.314

Model 1: Crude, Model 2: Adjust: gender, age, Model 3: Adjust: gender, age, HCV-Ab, ALT, AST, mAST, GGT, AG. OR, Odds Ratio; CI, Confidence Interval; TP, total protein; HCV-Ab, hepatitis C virus antibody; ALT, alanine aminotransferase; AST, aspartate aminotransferase; mAST, mitochondrial aspartate aminotransferase isoenzyme; GGT, gamma-glutamyl transferase; A/G, albumin-to-globulin ratio.

fied for GGT (P for likelihood ratio test = 0.017). However, no significant association was found when GGT was below or above 28.00.

Prediction of HCV infection risk by ALT, AST and HCV-Ab changes

A total of 179 samples were randomly divided into train and test sets in a 5:5 ratio. No statistically significant differences were observed between the two groups. The model developed with Gradient Boosting Machine (GBM) demonstrated the best performance in predicting the risk of HCV infection, achieving an AUC of 0.997 in the train set, 0.953 in the test set and 0.977 for the total samples (**Figure 3A-C**). This performance significantly outperformed that of individual indicators, suggesting that the scoring model can effectively identify the risk of developing HCV infection during early infection.

Discussion

This study established a Gradient Boosting Machine (GBM)-based predictive model integrating serological and biochemical indicators

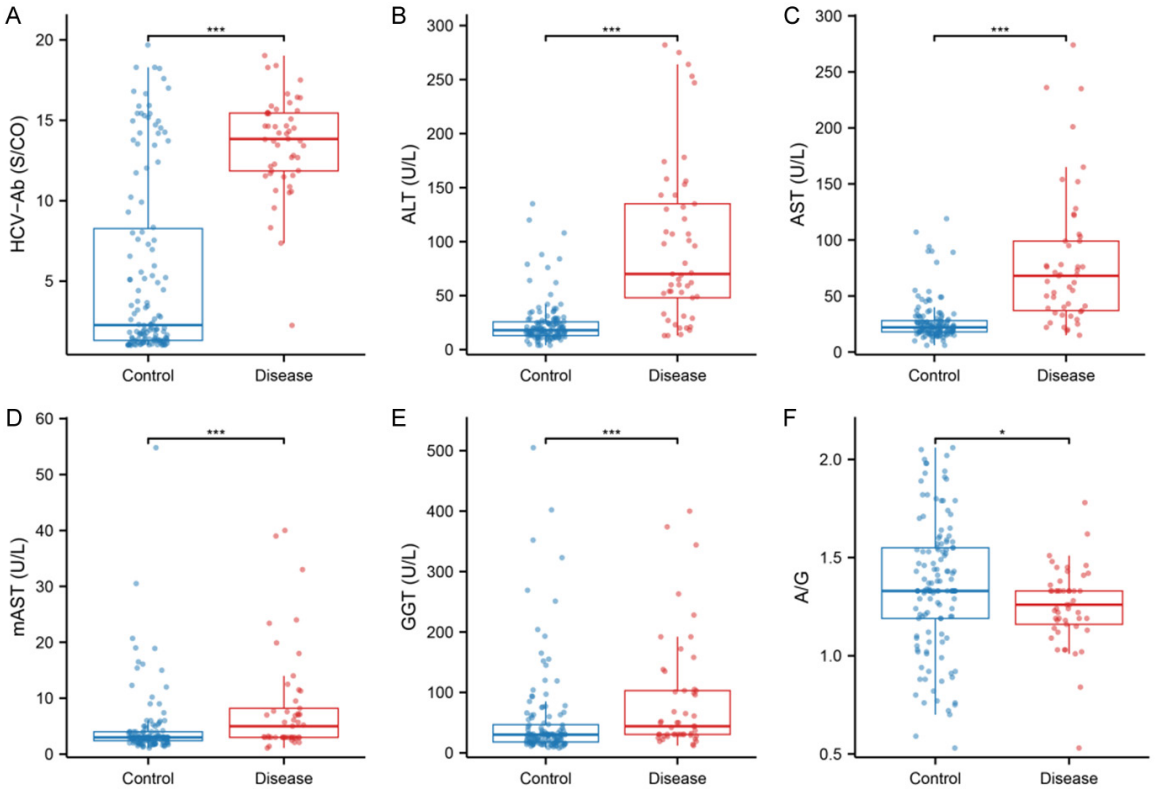

Figure 1. Comparative analysis of HCV-Ab and biochemical indicators between HCV Control group and Disease group. *P < 0.05; ***P < 0.001. A. HCV-Ab; B. ALT; C. AST; D. mAST; E. GGT; F. A/G. HCV-Ab, hepatitis C antibody; ALT, alanine aminotransferase; AST, aspartate aminotransferase; mAST, mitochondrial aspartate aminotransferase isoenzyme; GGT, gamma-glutamyl transferase; A/G, albumin-to-globulin ratio.

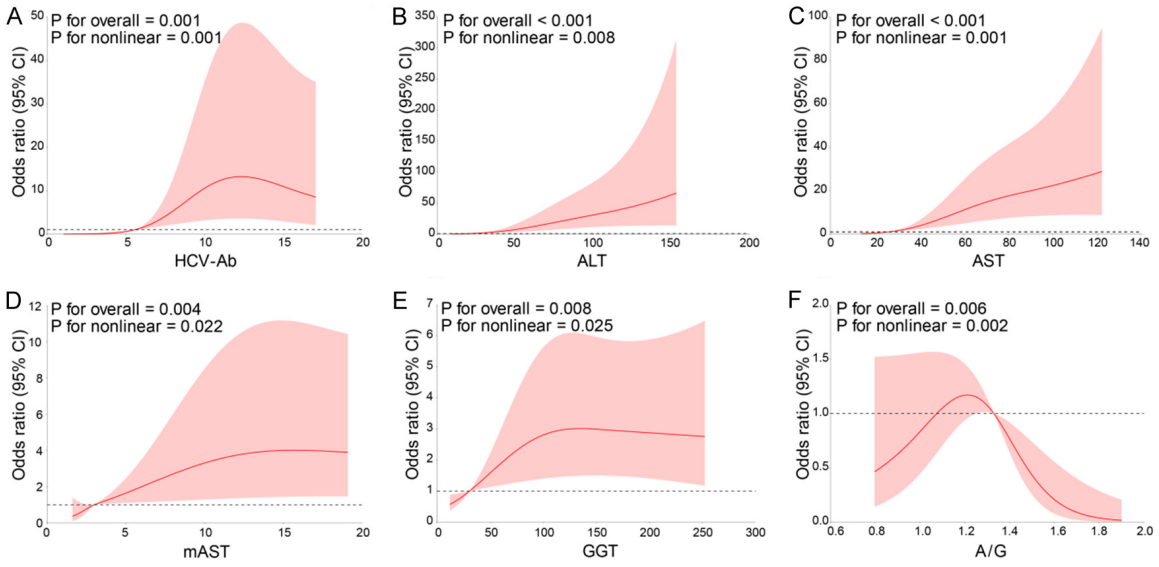

Figure 2. Restricted cubic spline analyses the association of *HCV-Ab and* biochemical indicators (A. HCV-Ab; B. ALT; C. AST; D. mAST; E. GGT; F. A/G) with HCV infection. HCV-Ab, hepatitis C antibody; ALT, alanine aminotransferase; AST, aspartate aminotransferase; mAST, mitochondrial aspartate aminotransferase isoenzyme; GGT, gamma-glutamyl transferase; A/G, albumin-to-globulin ratio.

Table 3. The threshold effect of HCV-Ab and biochemical indicators on HCV infection was analyzed using a two-stage phased regression model

Variables	Models	Adjusted OR (95% CI)	P
HCV-Ab (S/CO)	Model 1 Fitting model by standard linear regression	1.30 (1.20-1.42)	< .001
	Model 2 Fitting model by two-piecewise linear regression		
	Inflection point	11.17	
	< 11.17	2.04 (1.34-3.10)	< .001
	≥ 11.17	0.77 (0.59-1.01)	0.057
	P for likelihood test		< .001
ALT (U/L)	Model 1 Fitting model by standard linear regression	1.04 (1.03-1.06)	< .001
	Model 2 Fitting model by two-piecewise linear regression		
	Inflection point	54.00	
	< 54.00	1.09 (1.04-1.14)	< .001
	≥ 54.00	1.02 (1.00-1.04)	0.112
	P for likelihood test		0.017
AST (U/L)	Model 1 Fitting model by standard linear regression	1.05 (1.03-1.07)	< .001
	Model 2 Fitting model by two-piecewise linear regression		
	Inflection point	39.00	
	< 39.00	1.16 (1.05-1.28)	0.004
	≥ 39.00	1.02 (1.00-1.04)	0.086
	P for likelihood test		0.007
mAST (U/L)	Model 1 Fitting model by standard linear regression	1.06 (1.01-1.11)	0.016
	Model 2 Fitting model by two-piecewise linear regression		
	Inflection point	7.00	
	< 7.00	1.56 (1.14-2.13)	0.006
	≥ 7.00	1.00 (0.93-1.07)	0.983
	P for likelihood test		0.005
GGT (U/L)	Model 1 Fitting model by standard linear regression	1.00 (1.00-1.01)	0.026
	Model 2 Fitting model by two-piecewise linear regression		
	Inflection point	28.00	
	< 28.00	1.14 (0.99-1.30)	0.071
	≥ 28.00	1.00 (1.00-1.01)	0.290
	P for likelihood test		0.017

OR, odds ratio; CI, confidence interval; HCV-Ab, hepatitis C antibody; ALT, alanine aminotransferase; AST, aspartate aminotransferase; mAST, mitochondrial aspartate aminotransferase isoenzyme; GGT, gamma-glutamyl transferase.

using clinical data from 179 patients with high HCV-Ab titers (S/CO > 1.00). The multi-parameter model demonstrated superior diagnostic accuracy compared to single-marker approaches (AUC = 0.977), offering new insights for optimizing early HCV screening strategies.

As the cornerstone of HCV screening, HCV-Ab testing provides rapid and cost-effective population-level surveillance. However, its limitations are notable: 1) The prolonged seroconversion window (2-6 months) may delay early diagnosis; 2) 15%-30% of virologically cured patients maintain persistent antibodies, complicating differentiation between active and resolved infections [22, 26]; 3) F false-positive results may occur due to rheumatoid factor interference or immunosuppressive conditions [27, 28]. Our threshold effect analysis revealed a nonlinear relationship between HCV-Ab titers and infection probability. At HCV-Ab < 11.17 S/CO, each unit increase correlated with 30% elevated infection risk (OR = 1.3, 95% CI: 1.20-1.42, P < 0.001), whereas no significant correlation was observed above this threshold. This phenomenon aligns with antigen-antibody complex dynamics: subthreshold titers (< 11.17 S/CO) potentially indicate insufficient neutralizing

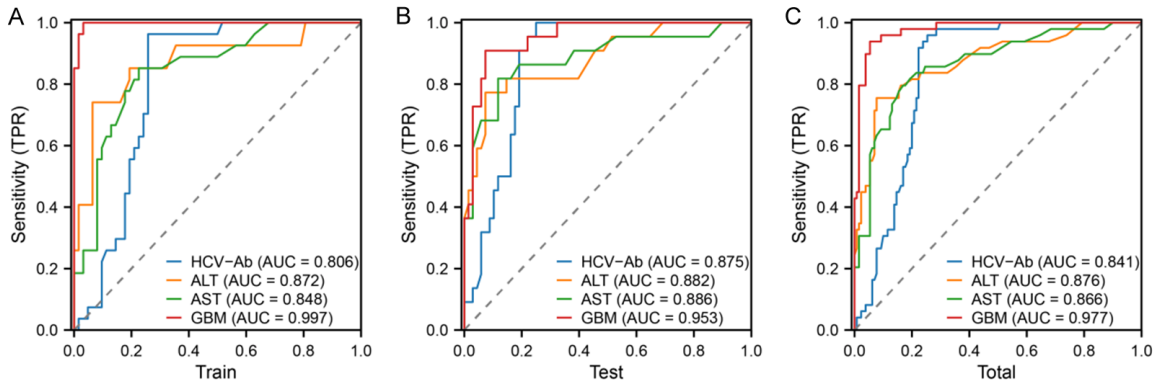

Figure 3. Machine learning algorithms to construct HCV infection prediction models. A. The ROC curves of HCV-Ab, ALT and AST (GBM) model and the HCV infection prediction model in the train group. B. The ROC curves of HCV-Ab, ALT and AST (GBM) model and the HCV infection prediction model in the test group. C. The ROC curves of HCV-Ab, ALT and AST (GBM) model and the HCV infection prediction model in the total group.

capacity, allowing viral replication, whereas suprathreshold levels (> 11.17 S/CO) may represent either effective immune containment or chronic infection states - a phenomenon analogous to "antigen trap" mechanism observed in HIV affinity maturation [29, 30].

Chronic HCV infection induces progressive hepatic injury through inflammation-fibrosis cascades, as evidenced by perturbations in serum biomarkers including TP, ALB, ALT, AST, mAST, ALP, GGT, TB, and CB [31-34]. Our threshold analysis identified nonlinear associations for liver enzymes: ALT < 54 U/L (OR = 1.04, P < 0.001), AST < 39 U/L (OR = 1.05, P < 0.001), and mAST < 7 U/L (OR = 1.06, P = 0.016) showed positive correlations with infection risk, with diminishing effects beyond these cutoffs. This may reflect progression from early to late liver injury [35-37]. While GGT showed statistical association (P = 0.026), its nonspecific elevation in alcoholic liver disease and cholestatic conditions limits diagnostic specificity. TP showed linear association in unadjusted models (P = 0.030), but significance attenuated after multivariable adjustment (Model 3: P = 0.149), likely confounded by compensatory hepatic synthesis mechanisms.

Conventional linear models inadequately capture complex biomarker interactions. Our GBM-based machine learning model demonstrated robust performance across training, testing, and pooled datasets, outperforming conventional methods through automated feature engineering and nonlinear relationship modeling.

Several limitations should be acknowledged. First, sample size constraints necessitate multicenter validation for generalizability. Second, exclusion of emerging markers like HCV core antigen (HCV-cAg), detectable within 7-10 days post-infection, could address HCV-Ab's window period limitations [38, 39]. Future studies should explore "HCV-Ab + ALT + HCV-cAg" triage protocols integrated with deep learning algorithms for dynamic prediction. Third, the inclusion criterion of selecting patients with HCV-Ab > 1.00 S/CO may introduce potential selection bias. Although this threshold facilitates the identification of suspected infections with definitive serological evidence, it may skew the results toward reflecting clinical characteristics of patients with elevated HCV-Ab levels, potentially underrepresenting populations with borderline antibody values or those in early seroconversion phases. This limitation could compromise the applicability of our conclusions to broader hepatitis C virus-infected populations, particularly in clinical scenarios involving equivocal serological status. Lastly, all sample data were derived from a single-center cohort. Despite rigorous standardization of data collection protocols, the generalizability of findings may be constrained by regional homogeneity in healthcare practices, demographic profiles, and diagnostic-therapeutic expertise. To enhance external validity and elucidate the mechanistic associations between HCV-Ab dynamics and clinical outcomes, future investigations should employ multi-center, large-scale designs incorporating patients with diverse antibody levels and geographical distributions.

Such efforts will enable systematic validation of our observations while advancing understanding of HCV serological evolution in relation to disease progression.

This study proposes two optimization pathways for resource-limited settings: 1) A stepwise "HCV-Ab screening → liver function retesting" cascade to reduce unnecessary HCV-RNA testing; 2) Portable GBM model deployment for real-time risk assessment in primary care. Furthermore, patients with mild ALT elevation (40-80 U/L) and HCV-Ab positivity should prioritize antiviral therapy to mitigate fibrosis progression.

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Disclosure of conflict of interest

None.

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