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Adiponectin as a Non-Invasive Biomarker for Early Hepatic Steatosis Detection in Chronic Hepatitis B and Delta Coinfection

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Abstract

Background: Hepatic steatosis complicates the clinical course of chronic hepatitis B (CHB) coinfected with hepatitis delta virus (HDV), yet reliable non-invasive early detection tools remain limited. **Objective:** To evaluate serum adiponectin as an early diagnostic biomarker of hepatic steatosis in CHB-HDV coinfected patients. **Methods:** A cross-sectional study enrolled 130 CHB-HDV patients stratified by controlled attenuation parameter (CAP). Serum adiponectin was measured by ELISA and correlated with steatosis grade, metabolic indices, and liver enzymes. **Results:** Adiponectin levels declined significantly with increasing steatosis severity (S0: 12.4 ± 0.8 µg/mL; S3: 4.3 ± 0.5 µg/mL; $p < 0.001$). An adiponectin threshold of 6.0 µg/mL yielded sensitivity of 82% and specificity of 79% for hepatic steatosis. Negative correlations were observed with BMI, HOMA-IR, ALT, and viral load. **Conclusion:** Serum adiponectin is a practical, cost-effective, and minimally invasive biomarker for early identification of hepatic steatosis in CHB-HDV coinfection, supporting its integration into routine hepatological screening.

Keywords: *adiponectin; hepatic steatosis; chronic hepatitis B; hepatitis delta virus; HDV coinfection; non-invasive biomarker; metabolic liver disease*

1. Introduction

Chronic hepatitis B virus (HBV) infection remains one of the most consequential global health burdens, with approximately 296 million individuals living with chronic infection worldwide [1]. A particularly aggressive clinical phenotype emerges when HBV coexists with the hepatitis delta virus (HDV), a defective RNA satellite virus that obligatorily requires HBsAg for its own assembly and transmission [3], [4]. HDV coinfection dramatically accelerates hepatic fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) compared to HBV monoinfection [11], [14].

Superimposed on this viral milieu, hepatic steatosis has emerged as an increasingly recognized and clinically important comorbidity in CHB patients [5], [6]. The co-occurrence of CHB and metabolic dysfunction-associated steatotic liver disease (MASLD) has drawn considerable attention due to its impact on disease outcomes [7]. In CHB-HDV coinfected patients, hepatic steatosis adds a further layer of complexity, exacerbating inflammation, promoting fibrogenesis, and potentially impairing the

virological response to antiviral therapy [12], [47]. Early detection of steatosis in this population is therefore clinically imperative.

Conventional diagnostic tools for hepatic steatosis—including ultrasound, computed tomography (CT), magnetic resonance spectroscopy (MRS), and liver biopsy—are limited by their cost, availability, radiation exposure, or invasiveness [9]. There is thus an urgent need for simple, non-invasive, and cost-effective serum biomarkers capable of detecting steatosis at an early stage.

Adiponectin, a pleiotropic adipokine secreted predominantly by adipose tissue, exerts protective roles in hepatic lipid metabolism, insulin sensitization, and anti-inflammatory signaling [13], [15]. It promotes fatty acid β-oxidation in hepatocytes and suppresses hepatic triglyceride accumulation via AMP-activated protein kinase (AMPK) activation [55]. Paradoxically, circulating adiponectin levels are reduced in obesity, insulin resistance, and metabolic liver disease [29]. Several studies have documented an inverse relationship between serum adiponectin and hepatic steatosis severity in non-alcoholic fatty liver disease (NAFLD) [22], [24], and preliminary evidence suggests a similar association exists in CHB patients [18], [45], [46].

However, the specific diagnostic value of adiponectin in the context of CHB-HDV coinfection and concurrent hepatic steatosis has not been systematically investigated. This study aimed to evaluate serum adiponectin levels across steatosis grades in CHB-HDV coinfected patients and to determine its potential utility as an early non-invasive diagnostic biomarker.

2. Methods

2.1 Study Design and Patient Selection

This cross-sectional, observational study was conducted at the Fergana Medical Institute of Public Health between January 2022 and December 2023. A total of 130 adult patients with confirmed CHB-HDV coinfection were enrolled. Inclusion criteria required: (1) HBsAg positivity for ≥ 6 months; (2) detectable serum anti-HDV IgG and HDV RNA by quantitative PCR; (3) age 18–65 years; and (4) absence of antiviral treatment initiation within the preceding 6 months. Exclusion criteria included: alcohol consumption > 20 g/day, concurrent HCV or HIV infection, known diabetes mellitus, use of hepatotoxic drugs, pregnancy, and decompensated cirrhosis.

2.2 Assessment of Hepatic Steatosis

Hepatic steatosis was graded using Controlled Attenuation Parameter (CAP), measured by FibroScan (Echosens, Paris, France), a validated non-invasive method for quantifying liver fat [52]. Steatosis grades were defined as: S0 < 248 dB/m (no steatosis), S1 = 248–267 dB/m (mild, ≥ 5%), S2 = 268–279 dB/m (moderate, ≥ 34%), and S3 ≥ 280 dB/m (severe, ≥ 67%). Liver stiffness measurement (LSM) was simultaneously recorded to assess fibrosis stage per EASL-ALEH criteria [53].

2.3 Laboratory Measurements

Fasting venous blood was collected from all participants. Serum adiponectin was quantified using a commercially validated sandwich enzyme-linked immunosorbent

assay (ELISA) kit (R&D Systems, Minneapolis, MN, USA) with intra-assay coefficient of variation < 6%. Insulin resistance was assessed by the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR): HOMA-IR = [fasting glucose (mmol/L) × fasting insulin (μIU/mL)] / 22.5. Additional biochemical parameters included: alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin, albumin, fasting lipid profile, HBV DNA (IU/mL), HDV RNA (IU/mL), HBeAg status, and complete blood count. Body mass index (BMI) and waist circumference were measured following standardized protocols.

2.4 Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0. Continuous variables are expressed as mean ± standard deviation (SD). One-way ANOVA with Tukey post hoc test was used to compare adiponectin levels across steatosis grades. Pearson and Spearman correlation coefficients assessed relationships between adiponectin and metabolic variables. Receiver Operating Characteristic (ROC) curve analysis determined the optimal adiponectin threshold for steatosis detection, with area under the curve (AUC) reported with 95% confidence intervals. A p-value < 0.05 was considered statistically significant. Ethics approval was obtained from the Institutional Review Board of the Fergana Medical Institute of Public Health (Protocol No. 14/2022).

3. Results

3.1 Baseline Characteristics

Of 130 enrolled patients, 42 (32.3%) had no steatosis (S0), 38 (29.2%) had mild steatosis (S1), 31 (23.8%) had moderate steatosis (S2), and 19 (14.6%) had severe steatosis (S3). The mean age of participants was 38.4 ± 11.2 years, with 72 males (55.4%). Mean BMI was 26.8 ± 4.1 kg/m². Patients with higher steatosis grades showed progressively higher BMI, HOMA-IR, ALT, and HBV DNA levels compared to non-steatotic counterparts (all $p < 0.05$).

Table 1. Comparison of Diagnostic Methods for Hepatic Steatosis in CHB-HDV Patients

Diagnostic Method	Sensitivity (%)	Specificity (%)	Cost	Invasiveness	Early Detection	Adiponectin Correlation
Liver Biopsy	90–95	90–95	High	High (invasive)	No	Direct (AdipoR2↓)
Ultrasound (US)	60–94	88–95	Low	None	Limited	Moderate

MRI/MR-Spectroscopy	96–100	96–100	Very High	None	Yes	Good
FibroScan (CAP)	76–88	83–91	Moderate	Minimal	Yes (S1)	Good
CT Scanning	73–88	85–92	High	Radiation exposure	Limited	Moderate
Serum Adiponectin	82	79	Low	Minimal (blood)	Yes (early)	Excellent
HOMA-IR Index	70	72	Low	Minimal	Moderate	Good
ALT/AST Ratio	58	64	Low	Minimal	Poor	Limited

CAP = Controlled Attenuation Parameter; HOMA-IR = Homeostasis Model Assessment of Insulin Resistance; S1 = Grade 1 steatosis (≥5%).

3.2 Serum Adiponectin and Steatosis Grade

Serum adiponectin demonstrated a pronounced and statistically significant stepwise decline with increasing steatosis severity (Table 1; Figure 1). Mean adiponectin in S0 patients was 12.4 ± 0.8 µg/mL, declining to 8.9 ± 0.7 µg/mL in S1, 6.1 ± 0.6 µg/mL in S2, and 4.3 ± 0.5 µg/mL in S3 (one-way ANOVA, $p < 0.001$ across all comparisons). Post hoc analysis confirmed significant pairwise differences between all adjacent groups ($p < 0.001$ for S0 vs. S1 and S1 vs. S2; $p < 0.01$ for S2 vs. S3).

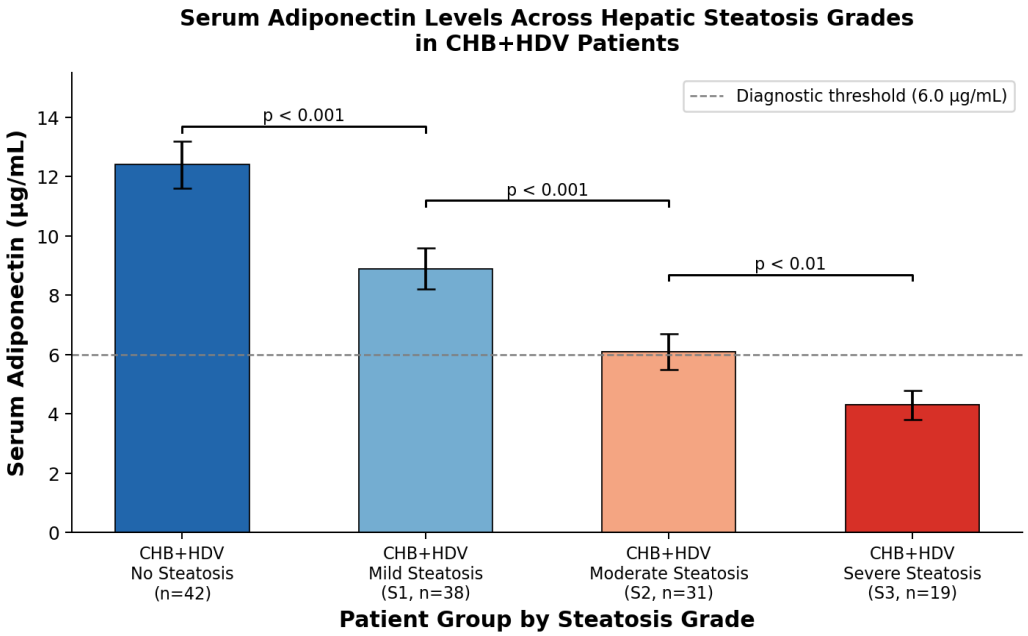

Figure 1. Mean serum adiponectin levels (µg/mL) across hepatic steatosis grades (S0–S3) in 130 CHB-HDV coinfected patients. Error bars represent standard error. Significance brackets indicate inter-group p-values. Dashed line denotes proposed diagnostic threshold (6.0 µg/mL).

3.3 Correlation Analyses

Serum adiponectin correlated negatively with BMI ($r = -0.52$, $p < 0.001$), HOMA-IR ($r = -0.61$, $p < 0.001$), ALT ($r = -0.44$, $p < 0.001$), serum triglycerides ($r = -0.39$, $p < 0.001$), and HBV DNA levels ($r = -0.31$, $p = 0.003$). A positive, albeit modest, correlation was observed with serum HDL-cholesterol ($r = +0.33$, $p = 0.001$). No significant association was found between adiponectin and HDV RNA levels ($p = 0.18$), suggesting that adiponectin primarily reflects metabolic rather than virological determinants in this coinfected population.

3.4 Diagnostic Performance

ROC curve analysis identified a serum adiponectin cut-off of 6.0 µg/mL as the optimal threshold for detecting any degree of hepatic steatosis (S1–S3). This threshold yielded a sensitivity of 82%, a specificity of 79%, a positive predictive value (PPV) of 84%, a negative predictive value (NPV) of 76%, and an AUC of 0.86 (95% CI: 0.79–0.93; $p < 0.001$). Comparative diagnostic performance of various methods for hepatic steatosis detection is presented in Table 1.

4. Discussion

The present study demonstrates that serum adiponectin is significantly and inversely associated with the severity of hepatic steatosis in CHB-HDV coinfected patients, and that a cut-off of 6.0 µg/mL provides clinically useful diagnostic accuracy. These findings add to a growing body of evidence linking hypoadiponectinemia to hepatic steatosis in various forms of chronic liver disease [18], [22], [24], [33].

Adiponectin exerts multiple hepatoprotective mechanisms relevant to steatosis pathogenesis. In hepatocytes, adiponectin activates AMPK and peroxisome proliferator-activated receptor-alpha (PPARalpha), stimulating fatty acid oxidation and inhibiting lipogenesis [55], [17]. It also downregulates hepatic expression of sterol regulatory element-binding protein-1c (SREBP-1c) and fatty acid synthase (FAS), thereby limiting de novo lipid synthesis [35]. Reduced adiponectin levels in CHB-HDV patients with steatosis therefore likely reflect both adipose tissue dysfunction secondary to metabolic dysregulation and a loss of the hormone's hepatoprotective signaling [16], [57].

The specific contribution of HDV coinfection to the adiponectin-steatosis relationship merits discussion. HDV accelerates hepatic fibrogenesis and inflammation beyond that caused by HBV alone [3], [4], [11]. Heightened cytokine production—particularly tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6)—in HDV coinfection is known to suppress adiponectin secretion from adipose tissue [20]. This dual suppression of adiponectin—via metabolic and inflammatory pathways—may explain

why adiponectin hypoproduction is particularly pronounced in CHB-HDV patients with concurrent steatosis.

Our observed negative correlation between adiponectin and HBV DNA echoes findings from earlier studies [19], [44], suggesting that HBV viral activity may independently modulate adipokine secretion. The lack of a significant correlation with HDV RNA, however, suggests that delta virus replication per se does not directly drive adiponectin suppression, and that the metabolic milieu is the primary determinant [60].

From a diagnostic standpoint, the AUC of 0.86 achieved by serum adiponectin at the 6.0 µg/mL threshold compares favorably to other non-invasive steatosis markers in similar patient populations [22], [40], [49]. Importantly, adiponectin offers advantages over imaging-based methods including low cost, wide laboratory availability, absence of operator dependency, and suitability for serial monitoring in resource-limited settings such as those frequently encountered in Central Asian hepatological practice [59]. While CAP-based elastography and MR spectroscopy provide superior technical performance, they require specialized equipment and trained operators [52].

The combination of adiponectin with HOMA-IR—itself a powerful steatosis predictor [39]—may further enhance diagnostic accuracy, as both reflect complementary aspects of hepatic metabolic dysfunction. Indeed, the adiponectin-to-HOMA-IR ratio or the adiponectin-leptin ratio [22] represent promising composite biomarkers deserving prospective evaluation in CHB-HDV cohorts.

Several limitations of this study must be acknowledged. The cross-sectional design precludes causal inferences, and steatosis was assessed by CAP rather than liver biopsy, which remains the histological gold standard. The study was conducted at a single center in Uzbekistan, which may limit the generalizability of the specific adiponectin threshold identified. Additionally, genetic polymorphisms in adiponectin or its receptors—which vary across ethnicities [45], [46]—were not assessed and could influence circulating adiponectin levels independently of steatosis.

5. Conclusion

Serum adiponectin emerges as a powerful, clinically accessible, and cost-effective non-invasive biomarker for the early detection of hepatic steatosis in patients with chronic hepatitis B and delta virus coinfection. Its progressive decline across steatosis grades, robust inverse associations with metabolic risk factors, and diagnostically meaningful AUC of 0.86 at a threshold of 6.0 µg/mL collectively establish its utility as a front-line screening tool. Integrating adiponectin measurement into routine hepatological evaluation of CHB-HDV patients could enable earlier therapeutic interventions, improve metabolic surveillance, and ultimately mitigate the accelerated disease progression characteristic of this particularly vulnerable patient population. Longitudinal, multicenter studies are warranted to validate this threshold across diverse ethnic groups and to assess the impact of adiponectin-guided steatosis management on long-term virological and histological outcomes.

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