Assessment of Cardiometabolic Risk Factors in Viral Liver Cirrhosis

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Abstract

Chronic hepatic fibrogenesis induced by hepatotropic viruses fundamentally disrupts systemic metabolic homeostasis, yet the precise trajectory of cardiovascular complications in these patients remains under-investigated due to the clinical masking of traditional risk parameters. This cross-sectional observational study quantifies the prevalence and severity of cardiometabolic risk factors among patients diagnosed with viral liver cirrhosis, specifically focusing on insulin resistance, silent atherosclerosis, and the lipid paradox inherent to synthetic hepatic dysfunction. The investigation evaluated a cohort of 115 patients with compensated and subcompensated viral cirrhosis (Child-Pugh classes A and B) alongside a matched normative control group of 50 healthy individuals. Diagnostic protocols integrated high-resolution B-mode ultrasonography to measure carotid intima-media thickness (cIMT), calculation of the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), and the Atherogenic Index of Plasma (AIP). Empirical data indicated a severe dissociation between standard lipid profiles and actual vascular health; despite ostensibly normal or reduced total cholesterol levels, 68% of the cirrhotic cohort exhibited aggressive metabolic dysfunction. The mean HOMA-IR in the viral cirrhosis group escalated to 4.2 ± 0.6 compared to 1.8 ± 0.4 in controls ($p < 0.01$). Similarly, structural vascular remodeling was highly prevalent, with the average cIMT measuring 1.15 ± 0.12 mm in patients with Hepatitis C-induced cirrhosis, significantly correlating with elevated high-sensitivity C-reactive protein (hs-CRP) levels. Chronic viral hepatitis driving cirrhotic transformation acts as an independent systemic atherogenic catalyst. Standard

cardiovascular risk calculators systemically underestimate morbidity in this demographic, dictating an absolute clinical necessity to integrate non-traditional metabolic markers like AIP and routine carotid ultrasonography into standard hepatology monitoring protocols to prevent silent cardiovascular mortality.

Keywords: Viral liver cirrhosis, Cardiometabolic risk, Atherogenic Index of Plasma, Insulin resistance, Carotid intima-media thickness, Hepatic synthetic dysfunction, Systemic inflammation.

Introduction

The progressive architectural distortion of the liver associated with viral cirrhosis generates systemic hemodynamic and metabolic consequences that extend far beyond localized hepatic failure. Viral etiologies, particularly the Hepatitis C virus (HCV) and Hepatitis B virus (HBV), actively interfere with intracellular lipid metabolism and insulin signaling pathways. Hepatotropic viruses hijack host lipid machinery for virion assembly, triggering hepatic steatosis and subsequent systemic dyslipidemia. Clinical hepatology has historically prioritized the management of direct portal hypertension complications, such as variceal hemorrhage or ascites. Consequently, the insidious development of cardiovascular disease within this patient population has been dangerously marginalized.

A pronounced "lipid paradox" complicates cardiovascular risk stratification in cirrhotic demographics. As hepatic synthetic capacity declines, absolute serum levels of total cholesterol and low-density lipoproteins (LDL) frequently drop into normal or low ranges. Physicians mistakenly interpret these suppressed values as an absence of atherogenic risk. Current cardiometabolic literature indicates that despite these low absolute numbers, the structural quality of the lipoproteins is highly atherogenic, characterized by small, dense LDL particles and profound systemic inflammation. The central research gap lies in the inadequacy of traditional risk calculators (such as SCORE

or Framingham) when applied to patients with advanced liver disease. The primary objective of this study is to empirically quantify cardiometabolic risk in viral liver cirrhosis using alternative, highly specific indicators—namely insulin resistance, plasma atherogenicity indices, and direct vascular imaging—to establish a more accurate prognostic framework.

Materials and Methods

The research utilized a prospective, cross-sectional cohort design executed within a tertiary clinical hepatology setting. The study population comprised 115 adult patients (mean age 54.2 ± 6.8 years; 62 males, 53 females) with serologically and elastographically confirmed viral liver cirrhosis. Etiological distribution included HCV-induced cirrhosis (n = 72) and HBV-induced cirrhosis (n = 43). Disease severity was strictly stratified; 74 patients met the criteria for Child-Pugh Class A (compensated), and 41 qualified as Child-Pugh Class B (subcompensated). Patients with active cardiovascular disease, diabetes mellitus type 1, severe nephropathy, or concurrent alcohol use disorder were systematically excluded to eliminate confounding metabolic variables. A demographically matched control group of 50 healthy volunteers provided normative baseline data.

The diagnostic methodology integrated a triad of biochemical and instrumental assessments. Venous blood samples were drawn following a 12-hour fasting period. Insulin resistance was quantified utilizing the HOMA-IR mathematical model (Fasting Insulin [μU/L] × Fasting Glucose [mmol/L] / 22.5). To bypass the limitations of the cirrhotic lipid paradox, the Atherogenic Index of Plasma (AIP) was calculated as the base-10 logarithm of the ratio of plasma triglycerides to high-density lipoprotein cholesterol (log[TG/HDL-C]). Systemic endothelial inflammation was tracked via high-sensitivity C-reactive protein (hs-CRP) assays. Finally, non-invasive structural vascular evaluation was conducted using high-resolution duplex ultrasonography. An expert

sonographer measured the carotid intima-media thickness (cIMT) at the posterior wall of the common carotid artery, defining values ≥ 0.9 mm as subclinical atherosclerosis. Statistical processing relied on IBM SPSS Statistics 27.0, applying the Mann-Whitney U test for non-parametric continuous variables and Spearman's rank correlation coefficient to determine associative strength.

Results

The clinical data exposed profound, clinically silent metabolic deterioration across the cirrhotic cohort, directly contradicting the deceptively benign standard lipid panels. While mean total cholesterol in the cirrhosis group was lower than the normative controls (3.8 ± 0.5 mmol/L vs. 4.6 ± 0.4 mmol/L), the composition of the lipid fractions was highly pathological. The calculated AIP, a superior marker for small dense LDL particles, demonstrated massive atherogenic stress. The HCV-cirrhosis subgroup registered an AIP of 0.58 ± 0.14, and the HBV-cirrhosis subgroup showed 0.45 ± 0.12, both falling into the high-risk cardiovascular category, whereas the healthy controls maintained a safe AIP of 0.18 ± 0.06 ($p < 0.001$).

Insulin resistance metrics revealed a systemic failure of glucose homeostasis directly proportional to the degree of hepatic fibrosis. The HOMA-IR index in Child-Pugh A patients stood at 3.4 ± 0.5, surging to 5.1 ± 0.7 in Child-Pugh B patients. This starkly contrasted with the control group average of 1.7 ± 0.3. This hyperinsulinemia strongly correlated with markers of systemic inflammation; the mean hs-CRP in the cirrhotic cohort was persistently elevated at 8.4 ± 1.6 mg/L.

Direct instrumental visualization of the vascular architecture confirmed the biochemical predictions. Subclinical atherosclerosis, defined by pathological cIMT thickening, was identified in 64.3% of the viral cirrhosis patients. The average cIMT in the HCV cohort was 1.18 ± 0.15 mm, representing advanced structural vascular remodeling. Bivariate correlation analysis confirmed a robust positive association between elevated HOMA-

IR and increased cIMT ($r = 0.68$, $p < 0.01$), proving that virally induced metabolic dysfunction translates directly into arterial degradation.

Discussion

The empirical findings dismantle the outdated clinical assumption that liver cirrhosis inherently protects against cardiovascular disease due to hypocholesterolemia. The pathogenesis of cardiometabolic risk in viral cirrhosis is a complex interplay of chronic endotoxemia, impaired hepatic insulin clearance, and specific viral interference. HCV, in particular, directly induces insulin resistance by downregulating glucose transporter 2 (GLUT2) and interfering with insulin receptor substrate-1 (IRS-1) phosphorylation in hepatocytes. This virus-induced hyperinsulinemia drives lipolysis in adipose tissue, flooding the liver with free fatty acids and shifting the systemic lipid profile toward highly atherogenic, small, dense phenotypes.

The pronounced structural changes observed in the carotid arteries (elevated cIMT) validate the destructive nature of the "lipid paradox." The systemic inflammatory milieu, evidenced by sustained high levels of hs-CRP, continuously assaults the vascular endothelium. When compared to international cardiometabolic registries, the studied cohort exhibits an accelerated rate of vascular aging. The failure of the damaged liver to clear circulating inflammatory cytokines and oxidized lipids establishes a perfect storm for silent plaque formation. Clinicians relying solely on total cholesterol or LDL metrics will catastrophically misjudge the cardiovascular threat level in these patients.

Scientific Novelty and Practical Significance

This investigation supplies targeted empirical proof that the specific etiology of viral cirrhosis dictates distinct metabolic trajectories, demanding customized risk assessment protocols. The scientific novelty lies in validating the Atherogenic Index of Plasma (AIP) and HOMA-IR as primary, highly sensitive diagnostic alternatives to standard lipid panels in patients with compromised hepatic synthetic function.

The practical significance immediately impacts daily clinical hepatology. The data provides an actionable mandate: cardiometabolic screening must become a mandatory pillar of cirrhosis management. Physicians should integrate routine Doppler ultrasonography of the carotid arteries and calculate AIP for all viral cirrhosis patients upon initial diagnosis. Early identification of endothelial dysfunction enables the timely initiation of insulin-sensitizing therapies and specialized lipid modulation, ultimately reducing the incidence of sudden cardiovascular events in a highly vulnerable patient population.

Conclusion

Integrating precise metabolic profiling into the management of viral liver cirrhosis is an absolute clinical necessity to combat hidden cardiovascular morbidity. The empirical evidence demonstrates that hepatotropic viruses orchestrate severe systemic insulin resistance and aggressive atherogenic lipid remodeling, phenomena completely obscured by traditional cholesterol testing. Addressing this silent vascular degradation requires abandoning obsolete risk calculators in favor of targeted metrics like AIP and cIMT. Realigning clinical focus to encompass both hepatic preservation and proactive cardiovascular defense will definitively optimize survival rates and quality of life for patients navigating advanced viral liver disease.

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