Hepatic alterations in patients with type 2 diabetes mellitus and arterial hypertension: a comparative clinical study

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Abstract: Background: Type 2 diabetes mellitus (T2DM) and arterial hypertension (AH) frequently coexist and are strongly associated with metabolic dysfunction-related liver disease, particularly non-alcoholic fatty liver disease (NAFLD). The combined impact of these conditions on hepatic structure and function remains insufficiently characterized in clinical settings. Objective: To evaluate liver condition and degree of hepatic damage in patients with T2DM and AH compared to healthy controls, and to investigate underlying pathophysiological mechanisms. Methods: A comparative clinical study was conducted including 72 patients with T2DM and AH (36 males, 36 females) and 36 healthy controls (18 males, 18 females). All participants underwent biochemical testing (ALT, AST, GGT), abdominal ultrasound, and transient elastography. Statistical analysis included independent t-tests or Mann-Whitney U tests, correlation analysis, and multivariate regression modeling. Results: Patients with T2DM and AH demonstrated significantly elevated levels of ALT, AST ($p=0.002$), and GGT compared to controls. Ultrasound revealed a markedly higher prevalence of hepatic steatosis. Elastography indicated increased liver stiffness values suggestive of fibrosis. Positive correlations were observed between liver enzymes and body mass index, fasting glucose, and systolic blood pressure. Regression analysis identified insulin resistance and systolic blood pressure as independent predictors of liver stiffness. Conclusion: The coexistence of T2DM and AH significantly contributes to hepatic injury, characterized by steatosis and early fibrosis. These findings underscore the importance of early screening and integrated management strategies targeting metabolic and vascular risk factors.

Keywords: type 2 diabetes mellitus; arterial hypertension; non-alcoholic fatty liver disease; liver fibrosis; insulin resistance; oxidative stress; elastography

INTRODUCTION

Type 2 diabetes mellitus (T2DM) and arterial hypertension (AH) are among the most prevalent chronic diseases worldwide and frequently coexist as components of metabolic syndrome. Their combined presence significantly increases the risk of cardiovascular complications and has been increasingly linked to chronic liver disease, particularly non-alcoholic fatty liver disease (NAFLD).

NAFLD represents a spectrum of liver disorders ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, and cirrhosis. Epidemiological studies suggest that up to 70% of patients with T2DM have NAFLD, and the presence of hypertension further accelerates disease progression. Despite its high prevalence, NAFLD often remains underdiagnosed due to its asymptomatic course.

The pathogenesis of liver injury in T2DM and AH is multifactorial, involving insulin resistance, chronic inflammation, oxidative stress, and endothelial dysfunction. These mechanisms act synergistically to promote hepatocellular injury and fibrogenesis.

The aim of this study was to assess liver condition in patients with T2DM and AH compared to healthy individuals, using biochemical and imaging methods, and to analyze the relationship between metabolic and hemodynamic factors and hepatic damage.

MATERIALS AND METHODS

Study Design and Population

This comparative clinical study included a total of 108 participants divided into two groups:

- Main group: 72 patients diagnosed with T2DM and AH
  - 36 males (50%)
  - 36 females (50%)
- Control group: 36 healthy individuals without T2DM or AH
  - 18 males (50%)
  - 18 females (50%)

Participants were recruited from outpatient and inpatient departments. Inclusion criteria for the main group were confirmed diagnosis of T2DM (according to ADA criteria) and essential hypertension. Exclusion criteria included viral hepatitis, alcohol abuse, autoimmune liver disease, and use of hepatotoxic drugs.

Clinical and Laboratory Assessment

All participants underwent:

- Anthropometric measurements (BMI)
- Blood pressure evaluation
- Fasting blood glucose

Biochemical analysis included:

- Alanine aminotransferase (ALT)
- Aspartate aminotransferase (AST)
- Gamma-glutamyl transferase (GGT)

Imaging Studies

- Ultrasound examination: to detect hepatic steatosis
- Transient elastography: to assess liver stiffness and estimate fibrosis

Statistical Analysis

Data were analyzed using standard statistical software. Continuous variables were expressed as mean ± standard deviation.

- Independent t-test or Mann-Whitney U test was used for group comparisons
- Pearson or Spearman correlation analysis assessed relationships between variables
- Multiple linear regression identified independent predictors of liver stiffness

A p-value <0.05 was considered statistically significant.

RESULTS

Biochemical Findings

Patients with T2DM and AH showed significantly higher liver enzyme levels compared to controls:

- ALT: 52.4 ± 14.8 vs 26.3 ± 8.7 U/L ($p<0.001$)
- AST: 41.7 ± 12.1 vs 24.5 ± 7.9 U/L ($p=0.002$)
- GGT: 68.9 ± 20.3 vs 29.8 ± 10.4 U/L ($p<0.001$)

These findings indicate subclinical hepatic injury in the main group.

Imaging Findings

- Hepatic steatosis (ultrasound):
  - Main group: 68.1%
  - Control group: 11.1% ($p<0.001$)
- Liver stiffness (elastography):
  - Main group: significantly higher mean values (7.8 ± 2.1 kPa vs 4.9 ± 1.3 kPa, $p<0.01$)

Approximately 30% of patients in the main group showed values suggestive of early fibrosis.

Correlation Analysis

Significant positive correlations were observed:

- ALT with BMI ($r=0.52$, $p<0.01$)
- GGT with fasting glucose ($r=0.61$, $p<0.001$)
- Liver stiffness with systolic BP ($r=0.47$, $p<0.01$)

Regression Analysis

Multivariate regression identified:

- Insulin resistance ($\beta=0.39$, $p<0.01$)
- Systolic blood pressure ($\beta=0.31$, $p=0.02$)

as independent predictors of liver stiffness.

DISCUSSION

This study demonstrates that patients with T2DM and AH exhibit significant hepatic alterations compared to healthy controls, including elevated liver enzymes, increased prevalence of steatosis, and early signs of fibrosis. These findings support the hypothesis that metabolic and hemodynamic disturbances synergistically contribute to liver damage.

Insulin resistance plays a central role in the pathogenesis of NAFLD by promoting hepatic lipid accumulation and impairing mitochondrial function. Increased free fatty acid flux to the liver leads to steatosis, while oxidative stress and lipid peroxidation drive progression to NASH. In parallel, arterial hypertension contributes to endothelial dysfunction, reducing hepatic microcirculation and promoting hypoxia-induced fibrogenesis.

The observed elevation of ALT, AST, and GGT reflects hepatocellular injury and oxidative stress. GGT, in particular, has been associated with cardiometabolic risk and may serve as an early marker of hepatic involvement.

Our findings are consistent with recent studies (post-2020) demonstrating a high prevalence of NAFLD in patients with T2DM and an increased risk of fibrosis in those with coexisting hypertension. For instance, large cohort studies have shown that hypertension independently predicts advanced fibrosis in NAFLD populations. Similarly, recent meta-analyses highlight the role of systemic inflammation and endothelial dysfunction in disease progression.

The significant correlations between liver stiffness and both metabolic (BMI, glucose) and hemodynamic (blood pressure) parameters underscore the multifactorial nature of liver injury. Regression analysis further confirms the independent contribution of insulin resistance and hypertension.

Clinically, these findings emphasize the importance of routine liver screening in patients with T2DM and AH. Early identification of steatosis and fibrosis allows timely intervention, potentially preventing progression to cirrhosis.

CONCLUSION

Patients with type 2 diabetes mellitus and arterial hypertension are at significantly increased risk of liver damage, characterized by steatosis, elevated liver enzymes, and early fibrosis. The interplay between insulin resistance, oxidative stress, chronic inflammation, and endothelial dysfunction underlies disease progression.

Combined metabolic and hemodynamic disturbances act synergistically, accelerating hepatic injury. Non-invasive diagnostic tools such as ultrasound and elastography are effective for early detection.

These findings highlight the need for integrated management strategies targeting both metabolic control and blood pressure regulation to prevent progression of liver disease.

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