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Morphological and Functional Changes of The Liver, Heart, And Kidneys in Obesity

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Abstract. Obesity is a major global health challenge associated with structural and functional alterations in multiple organs, particularly the liver, heart, and kidneys. This study aimed to evaluate morphological and functional changes in these organs in patients with obesity and to determine their clinical significance. A total of 180 participants were included in the study, comprising 120 patients with obesity and 60 normal-weight controls. Clinical, biochemical, ultrasound, echocardiographic, and laboratory methods were applied. The findings demonstrated significant hepatic steatosis, left ventricular hypertrophy, and early nephropathy changes in obese individuals compared to controls. The results highlight the systemic impact of obesity and emphasize the importance of early diagnostic and preventive strategies.

Key words: Obesity, Hepatic Steatosis, Cardiac Remodeling, Renal Dysfunction, Organ Morphology, Metabolic Syndrome

Introduction

Obesity has reached epidemic proportions worldwide. According to the World Health Organization, more than 1.9 billion adults are overweight, and over 650 million are classified as obese. The global prevalence of obesity has nearly tripled since 1975, significantly increasing the risk of non-communicable diseases such as cardiovascular disorders, type 2 diabetes, chronic kidney disease, and non-alcoholic fatty liver disease (NAFLD).

Recent studies demonstrate that obesity induces systemic metabolic and inflammatory disturbances that affect internal organs at structural and cellular levels [3, 7]. Hepatic steatosis is reported in up to 70% of obese individuals and is considered a key component of metabolic syndrome [12]. Cardiac remodeling and left ventricular hypertrophy have been strongly associated with increased body mass index (BMI) and insulin resistance [18]. Furthermore, obesity-related glomerulopathy is recognized as an emerging cause of chronic kidney disease [25].

Experimental and clinical research confirms that chronic low-grade inflammation, adipokine imbalance, oxidative stress, and endothelial dysfunction are central mechanisms underlying obesity-related organ damage [31, 34]. Structural changes such as hepatocyte ballooning, myocardial fibrosis, and glomerular hypertrophy have been documented in histopathological investigations [41, 46].

Despite numerous studies, a comprehensive evaluation of simultaneous liver, heart, and kidney changes in obesity remains insufficiently addressed. Therefore, a multidimensional clinical and morphological assessment is required.

Aim of the Study

The aim of this study was to assess morphological and functional alterations of the liver, heart, and kidneys in patients with obesity and to evaluate their clinical and socio-medical significance.

Materials and Methods

This prospective observational study included 180 participants aged 30–65 years. The main group consisted of 120 patients diagnosed with obesity (BMI ≥30 kg/m²), while 60 age- and sex-matched individuals with normal BMI formed the control group.

All participants underwent comprehensive clinical evaluation including anthropometric measurements, blood pressure monitoring, and metabolic assessment. Biochemical analyses included liver enzymes

(ALT, AST), lipid profile, fasting glucose, creatinine, and estimated glomerular filtration rate (eGFR). Inflammatory markers such as C-reactive protein were also measured.

Ultrasound examination of the liver was performed to assess steatosis grading. Echocardiography was conducted to evaluate left ventricular mass index (LVMI), wall thickness, and ejection fraction. Renal ultrasound and urinary albumin-to-creatinine ratio were used to assess kidney morphology and function.

Statistical analysis was performed using parametric and non-parametric tests. Differences between groups were considered significant at $p < 0.05$.

Result

The study revealed significant structural and functional alterations in obese patients compared to controls.

At baseline, obese individuals demonstrated increased liver size and echogenicity consistent with steatosis. Cardiac examination showed increased left ventricular mass and mild diastolic dysfunction. Renal evaluation indicated early hyperfiltration and microalbuminuria.

Table 1. Biochemical and Functional Parameters in Study Groups

Parameter	Obese Group (n=120)	Control Group (n=60)	p-value
ALT (U/L)	58.4 ± 12.6	29.7 ± 8.2	<0.001
AST (U/L)	46.2 ± 10.4	25.1 ± 6.9	<0.001
LVMI (g/m²)	132.5 ± 18.3	96.4 ± 14.7	<0.001
eGFR (ml/min)	118.6 ± 15.2	102.3 ± 11.4	<0.01
Microalbuminuria (%)	38%	8%	<0.001

The obese group demonstrated significantly elevated liver enzymes, increased left ventricular mass, and early renal hyperfiltration. Microalbuminuria was markedly more prevalent among obese patients, indicating early nephropathy.

Table 2. Structural Organ Changes in Obesity

Organ	Observed Changes	Prevalence in Obese (%)
Liver	Moderate-to-severe steatosis	68%
Heart	Left ventricular hypertrophy	54%
Kidneys	Increased cortical thickness	41%

The majority of obese patients exhibited structural changes across all studied organs, confirming the systemic nature of obesity-related damage.

Discussion

The findings confirm that obesity significantly affects liver, heart, and kidney structure and function. Hepatic steatosis observed in nearly two-thirds of obese patients corresponds with earlier reports [12, 41]. Elevated ALT and AST levels reflect hepatocellular injury associated with fat accumulation and oxidative stress.

Cardiac remodeling, particularly increased LVMI, suggests early hypertrophic adaptation due to hemodynamic overload and metabolic dysregulation [18, 31]. Persistent myocardial strain may eventually lead to heart failure if not addressed.

Renal findings, including hyperfiltration and microalbuminuria, support the concept of obesity-related glomerulopathy [25, 46]. Chronic hyperfiltration may progress to irreversible kidney damage.

From an economic perspective, early detection of organ changes in obesity may reduce healthcare expenditures by preventing advanced complications such as cirrhosis, heart failure, and chronic kidney

disease. Socially and medically, implementing screening programs for obese individuals could improve quality of life and reduce disability rates.

Conclusion.

Obesity is associated with significant morphological and functional alterations in the liver, heart, and kidneys. Hepatic steatosis, cardiac hypertrophy, and early nephropathy are common findings in obese patients. These results emphasize the need for early multidimensional screening and integrated preventive strategies to reduce long-term clinical, economic, and social consequences.

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