

ON THE ROLE OF LIPID METABOLISM DISORDERS IN THE DEVELOPMENT OF KIDNEY DAMAGE IN METABOLIC SYNDROME

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Abstract. *Metabolic syndrome is one of the most significant medical and social problems of modern healthcare due to its high prevalence and association with cardiovascular diseases and chronic kidney disease. Disorders of lipid metabolism play a key role in the pathogenesis of renal damage in patients with metabolic syndrome, contributing to glomerular hyperfiltration, endothelial dysfunction, and progressive decline of renal function. The aim of this study was to assess the role of lipid metabolism disorders in the development of kidney damage in patients with metabolic syndrome. The study included 95 patients aged 19 to 77 years who underwent anthropometric, biochemical, and functional assessment of renal function. Lipid profile parameters and glomerular filtration rate calculated by the MDRD formula were analyzed depending on body mass index. The results demonstrated that dyslipidemia in patients with metabolic syndrome is characterized by elevated levels of triglycerides, total cholesterol, and low-density lipoproteins, accompanied by a decrease in high-density lipoproteins. A significant correlation was identified between lipid spectrum disturbances and markers of renal dysfunction, including elevated serum creatinine levels and reduced glomerular filtration rate.*

These findings indicate that lipid metabolism disorders play an important role in the development of renal impairment in metabolic syndrome and highlight the necessity of early detection of kidney dysfunction using estimated glomerular filtration rate even in patients with normal creatinine levels.

Keywords: *Metabolic syndrome; dyslipidemia; chronic kidney disease; glomerular filtration rate; obesity.*

Introduction

Over the past several decades, the prevalence of overweight and obesity has increased dramatically worldwide, reaching epidemic proportions. According to epidemiological data, more than 40% of the adult population is expected to be overweight or obese in the coming decade. This trend represents a major global health challenge due to the close association of obesity with metabolic syndrome, cardiovascular diseases, and chronic kidney disease.

Metabolic syndrome is a complex of interrelated metabolic disorders, including abdominal obesity, insulin resistance, dyslipidemia, and arterial hypertension. Among these components, lipid metabolism disorders play a central role in the development and progression of organ damage, particularly affecting the cardiovascular system and kidneys. Numerous population-based studies have demonstrated a strong association between increased body mass index and the risk of chronic kidney disease, independent of other risk factors.

Pathophysiological mechanisms linking dyslipidemia to renal damage include lipid accumulation in renal tissues, oxidative stress, endothelial dysfunction, inflammation, and activation of profibrotic pathways. Elevated levels of triglycerides and low-density lipoproteins contribute to glomerular sclerosis and tubular interstitial damage, leading to a gradual decline in renal function. Despite the growing evidence of the role of lipid metabolism disorders in kidney damage, early stages of renal dysfunction in patients with metabolic syndrome often remain undiagnosed due to normal serum creatinine levels.

Therefore, assessment of glomerular filtration rate using estimating formulas such as MDRD is of particular importance for early detection of subclinical kidney impairment. In this context, the present study aims to evaluate the contribution of lipid metabolism disorders to kidney damage in patients with metabolic syndrome and to identify early markers of renal dysfunction.

Materials and Methods

The present study was conducted as an observational clinical investigation aimed at assessing lipid metabolism disorders and their association with renal function impairment in patients with metabolic syndrome. A total of 95 patients aged from 19 to 77 years were included in the study. Among them, 33 patients (35%) were men and 62 patients (65%) were women. All patients were examined on an outpatient and inpatient basis during the period from 2021 to 2024.

Inclusion criteria were the presence of metabolic syndrome diagnosed according to generally accepted clinical and laboratory criteria. Patients with acute inflammatory diseases, malignant neoplasms, and terminal stages of chronic kidney disease were excluded from the study. All patients underwent anthropometric measurements, including body weight and height determination, followed by calculation of the body mass index (BMI) using the Quetelet formula ($BMI = \text{weight}/\text{height}^2$). Based on BMI values, the degree of obesity was determined according to the World Health Organization (WHO) classification (1997).

According to BMI, patients were divided into four groups:

- Group 1 (control group): BMI 18.5–24.9 kg/m² (n = 20)
- Group 2: BMI 25.0–29.9 kg/m² (n = 22)
- Group 3: BMI 30.0–34.9 kg/m² (n = 25)
- Group 4: BMI 35.0–39.9 kg/m² (n = 28)

All patients underwent laboratory testing to assess lipid metabolism parameters. The lipid profile included determination of:

- total cholesterol,
- triglycerides,
- low-density lipoprotein cholesterol (LDL-C),
- high-density lipoprotein cholesterol (HDL-C).

Blood samples were collected in the morning after overnight fasting. Biochemical analyses were performed using standardized enzymatic methods. Renal function was assessed by measuring serum creatinine levels and calculating the estimated glomerular filtration rate (eGFR) using the MDRD formula, expressed in ml/min/1.73 m². This method was chosen due to its clinical relevance and widespread use for early detection of chronic kidney disease. Statistical data processing was performed using standard statistical methods. Quantitative variables were expressed as mean values with standard deviations (M±SD). Comparative analysis between groups was carried out using Student's t-test. Correlation analysis was performed to evaluate the relationship between lipid metabolism parameters and markers of renal dysfunction, including serum creatinine levels and eGFR. A p-value of <0.05 was considered statistically significant.

Results

The analysis of clinical and laboratory data demonstrated that lipid metabolism disorders were present in the majority of patients with metabolic syndrome and their severity increased in parallel with body mass index growth. Patients with metabolic syndrome exhibited a typical atherogenic lipid profile.

Mean serum triglyceride levels were significantly elevated and reached 2.01 ± 0.12 mmol/L, while total cholesterol concentration averaged 7.5 ± 0.42 mmol/L. Low-density lipoprotein cholesterol levels were also increased (3.94 ± 0.20 mmol/L), whereas high-density lipoprotein cholesterol levels were reduced (1.12 ± 0.06 mmol/L). The degree of dyslipidemia was directly related to the severity of obesity. Patients in groups with higher BMI values demonstrated more pronounced lipid profile disturbances compared with the control group with normal body weight. Evaluation of renal function using the MDRD formula revealed a gradual decline in glomerular filtration rate as BMI increased. In the control group (BMI 18.5–24.9 kg/m²), the mean estimated glomerular filtration rate (eGFR) remained within normal limits.

However, patients with overweight and obesity demonstrated a progressive reduction in eGFR values, even in the absence of clinically significant elevation of serum creatinine levels.

This finding indicates the presence of early, subclinical renal dysfunction in patients with metabolic syndrome, which may not be detected by routine creatinine measurement alone.

Correlation analysis revealed a statistically significant positive relationship between lipid metabolism disorders and markers of renal damage. In particular, a direct correlation was identified between serum lipid parameters and serum creatinine levels ($r = 0.210$, $p = 0.010$). In addition, worsening lipid profile parameters were associated with a decrease in eGFR values, reflecting impaired filtration capacity of the kidneys. These results confirm that dyslipidemia contributes to renal dysfunction in patients with metabolic syndrome and plays an important role in the early stages of chronic kidney disease development.

Discussion

The results of the present study confirm the significant role of lipid metabolism disorders in the development of renal impairment in patients with metabolic syndrome. Dyslipidemia, characterized by elevated levels of triglycerides, total cholesterol, and low-density lipoproteins along with reduced high-density lipoproteins, was observed in the majority of examined patients and became more pronounced with increasing body mass index. These findings are consistent with data from numerous population-based and clinical studies demonstrating that obesity and metabolic syndrome are independent risk factors for chronic kidney disease. Excess circulating lipids contribute to lipid accumulation in renal tissues, leading to glomerular and tubular damage.

Lipotoxicity induces oxidative stress, endothelial dysfunction, and activation of inflammatory and profibrotic pathways, which ultimately result in a progressive decline in renal function. An important observation of this study is the detection of reduced glomerular filtration rate in patients with metabolic syndrome despite normal or near-normal serum creatinine levels.

This emphasizes the limited diagnostic value of serum creatinine as an isolated marker of renal function, particularly in the early stages of kidney damage. Calculation of eGFR using the MDRD formula allows for more accurate identification of subclinical renal dysfunction and provides an opportunity for early intervention. The identified correlation between lipid profile parameters and serum creatinine levels supports the concept that dyslipidemia directly contributes to renal impairment. Even a moderate increase in atherogenic lipid fractions was associated with a decrease in filtration capacity, highlighting the importance of comprehensive metabolic control in patients with metabolic syndrome. From a clinical perspective, the obtained results underline the necessity of regular assessment of lipid metabolism and renal function in patients with metabolic syndrome. Early detection of renal involvement enables timely implementation of preventive and therapeutic measures aimed at slowing the progression of chronic kidney disease.

Conclusion

The study demonstrates that disorders of lipid metabolism play a significant role in the development of kidney damage in patients with metabolic syndrome. Progression of dyslipidemia is associated with a gradual decline in glomerular filtration rate, even in patients with normal serum creatinine levels. Calculation of estimated glomerular filtration rate using the MDRD formula allows for the identification of early, preclinical stages of renal dysfunction.

These findings confirm the necessity of comprehensive evaluation of lipid profile and renal function in all patients with metabolic syndrome in order to improve early diagnosis, risk stratification, and prevention of chronic kidney disease progression.

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