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EARLY STRUCTURAL AND FUNCTIONAL CHANGES OF THE MYOCARDIUM IN PATIENTS WITH METABOLIC SYNDROME: A CLINICO-MORPHOLOGICAL ANALYSIS

Madraximova Nigoraxon Ravshanbek kizi
Fergana medical institute of public health
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Abstract. This study aimed to evaluate early structural and functional myocardial changes in patients with metabolic syndrome. A clinico-morphological approach was applied, including anthropometric, biochemical, and echocardiographic assessments[1,2,3]. Insulin resistance was evaluated using the HOMA-IR index. The results demonstrated early diastolic dysfunction, mitochondrial impairment, and microvascular disturbances, contributing to fibrotic changes in the myocardium. These findings highlight the importance of early diagnosis and pathogenetic treatment to prevent progression to heart failure.

Keywords: metabolic syndrome, myocardium, insulin resistance, diastolic dysfunction, lipotoxicity, oxidative stress, cardiomyopathy

Introduction

Metabolic syndrome is a complex and multifactorial pathological condition characterized by the coexistence of abdominal obesity, arterial hypertension, dyslipidemia, and insulin resistance, and it represents one of the most significant risk factors for the development of cardiovascular diseases worldwide[4,5,6]. The central pathogenic component—insulin resistance—leads to persistent hyperglycemia, altered lipid metabolism, and increased circulating free fatty acids, which exert toxic effects on cardiomyocytes. These metabolic disturbances induce myocardial injury through lipotoxicity, characterized by intracellular lipid accumulation, and oxidative stress, associated with excessive production of reactive oxygen species and impaired antioxidant defense systems[7,8,9].

At the molecular and cellular levels, these processes activate key pathogenic pathways, including mitochondrial dysfunction, endoplasmic reticulum stress, and chronic low-grade inflammation. Dysregulation of adipokines (e.g., leptin, adiponectin), activation of the renin–angiotensin–aldosterone system (RAAS), and endothelial dysfunction further contribute to myocardial injury. These mechanisms promote cardiomyocyte hypertrophy, apoptosis, and extracellular matrix remodeling via increased collagen synthesis and reduced degradation, leading to interstitial fibrosis and increased myocardial stiffness. In parallel, microvascular rarefaction and impaired nitric oxide bioavailability reduce coronary microcirculatory reserve[10,11,12].

Importantly, these structural alterations begin at early stages of metabolic syndrome and often precede clinically detectable cardiovascular disease and systolic dysfunction. Functionally, early myocardial impairment is primarily manifested as diastolic dysfunction, reflecting disturbances in myocardial relaxation and ventricular filling dynamics. This stage represents a critical preclinical phase in the development of heart failure with preserved ejection fraction (HFpEF), a phenotype strongly linked to metabolic and inflammatory burden[13,14,15].

From a clinical and imaging perspective, early changes may be detected not only by conventional echocardiographic parameters (E/A, E/e) but also by advanced techniques such as tissue Doppler imaging, speckle-tracking echocardiography (global longitudinal strain), and assessment of left atrial function. Circulating biomarkers, including natriuretic peptides, inflammatory cytokines, and markers of oxidative stress, may provide additional insight into subclinical myocardial involvement[16,17,18].

Despite the growing body of evidence, early morphofunctional alterations of the myocardium in metabolic syndrome remain incompletely understood, particularly regarding their

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clinico-morphological correlations and temporal progression[19,20,21]. Therefore, a comprehensive approach integrating clinical, biochemical, imaging, and pathogenetic analyses is essential for early detection, accurate risk stratification, and the development of targeted, mechanism-based therapeutic strategies[22,23,24].

Materials and methods. The study included patients diagnosed with metabolic syndrome according to internationally accepted diagnostic criteria. Inclusion criteria comprised patients aged 30–65 years with confirmed metabolic syndrome, while exclusion criteria included known cardiovascular diseases, diabetes mellitus requiring insulin therapy, chronic inflammatory diseases, and severe renal or hepatic impairment.

Clinical evaluation involved anthropometric measurements, including body mass index (BMI) and waist circumference. Biochemical analysis included fasting blood glucose, insulin levels, and lipid profile parameters. Insulin resistance was calculated using the HOMA-IR index.

Transthoracic echocardiography was performed using standardized protocols to assess cardiac structure and function. Diastolic function was evaluated using transmitral flow velocity (E/A ratio) and tissue Doppler imaging (E/e' ratio). Left ventricular mass and wall thickness were also assessed.

Morphological analysis focused on identifying cardiomyocyte hypertrophy, interstitial fibrosis, and microvascular dysfunction based on pathogenetic interpretation. Statistical analysis was conducted using SPSS software. Continuous variables were expressed as mean ± standard deviation. Correlation analysis (Pearson or Spearman) was applied to evaluate relationships between metabolic and cardiac parameters, with statistical significance set at $p<0.05$.

Results and discussion. The study demonstrated that patients with metabolic syndrome exhibit early and clinically significant alterations in myocardial structure and function. The most prominent functional finding was the presence of diastolic dysfunction, identified through changes in transmitral flow parameters (E/A ratio) and elevated E/e' values, indicating impaired myocardial relaxation and increased filling pressures. Importantly, these abnormalities were observed in the absence of overt systolic dysfunction, supporting the concept of an early, subclinical stage of myocardial involvement.

A strong positive correlation was identified between insulin resistance (HOMA-IR index) and the severity of diastolic dysfunction, suggesting that metabolic imbalance plays a central role in myocardial impairment. Elevated levels of glucose and dyslipidemia were also associated with worsening cardiac parameters, reinforcing the systemic nature of metabolic syndrome.

From a pathogenetic perspective, the observed functional disturbances can be explained by underlying structural and cellular alterations. Lipotoxicity, resulting from excess free fatty acid accumulation in cardiomyocytes, leads to mitochondrial dysfunction and reduced ATP production, thereby impairing myocardial contractility and relaxation. Concurrently, oxidative stress promotes cellular damage through reactive oxygen species, contributing to apoptosis and further functional decline.

Morphologically, these processes manifest as cardiomyocyte hypertrophy, interstitial fibrosis, and microvascular dysfunction. Fibrotic remodeling increases myocardial stiffness, which directly contributes to diastolic dysfunction. Microvascular impairment reduces coronary perfusion, leading to chronic low-grade ischemia and insufficient trophic support of the myocardium. This creates a vicious cycle in which structural damage and functional impairment reinforce each other.

These findings are consistent with the contemporary concept of metabolic cardiomyopathy, in which metabolic disturbances precede and drive structural remodeling of the myocardium. The early presence of diastolic dysfunction highlights its value as a sensitive clinical marker for identifying patients at high risk of developing heart failure with preserved ejection fraction (HFpEF).

Furthermore, the integration of clinical, biochemical, and echocardiographic data provides a more comprehensive understanding of disease progression. The results emphasize that

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myocardial involvement in metabolic syndrome is not merely a late complication but begins at an early stage and progresses silently if not detected in time.

From a clinical standpoint, these findings underscore the importance of early screening using echocardiographic parameters and metabolic profiling. Identification of high-risk patients enables timely implementation of targeted therapeutic strategies aimed at improving insulin sensitivity, reducing oxidative stress, and preventing fibrotic remodeling.

Overall, the combined analysis of results and their pathogenetic interpretation confirms that metabolic syndrome induces early, progressive, and clinically relevant myocardial alterations that require proactive diagnostic and therapeutic approaches.

Conclusion. Metabolic syndrome induces early, progressive, and clinically relevant morphofunctional changes in the myocardium, even before overt cardiovascular disease becomes apparent. These alterations—characterized by diastolic dysfunction, cardiomyocyte hypertrophy, interstitial fibrosis, and microvascular impairment—constitute the pathogenetic substrate for the development of metabolic cardiomyopathy and subsequent heart failure, particularly heart failure with preserved ejection fraction (HFpEF). Importantly, the strong association between insulin resistance, dyslipidemia, and myocardial dysfunction underscores the central role of metabolic imbalance in cardiac remodeling.

Early detection of these subclinical changes, especially through sensitive echocardiographic parameters of diastolic function (E/A ratio, E/e') and complementary metabolic profiling, is crucial for timely risk stratification. The implementation of targeted pathogenetic therapy—aimed at improving insulin sensitivity, reducing oxidative stress, modulating RAAS activity, and preventing fibrotic remodeling—can significantly alter disease trajectory. In addition, lifestyle interventions, including weight reduction, dietary modification, and physical activity, remain fundamental components of comprehensive management.

Therefore, a multidisciplinary and proactive approach combining early диагностика, continuous monitoring, and mechanism-based therapeutic strategies is essential for improving clinical outcomes, preventing progression to advanced heart failure, and enhancing long-term prognosis in patients with metabolic syndrome.

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