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EVALUATION OF ATRIAL FIBRILLATION RISK IN PATIENTS WITH METABOLIC SYNDROME: AN ORIGINAL STUDY

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Abstract. *Metabolic syndrome is a major risk factor for cardiovascular disease and atrial fibrillation. This original study included 40 patients with metabolic syndrome complicated by atrial fibrillation and aimed to assess clinical, metabolic, and hemostatic risk factors. All patients underwent evaluation of body mass index, blood pressure, glucose tolerance, and electrocardiography. Laboratory assessment included lipid profile analysis, 24-hour urinary adrenaline and noradrenaline excretion, and evaluation of hemostatic parameters. Risk stratification was performed using the CHA_2DS_2-VASc, CHARGE-AF, and HAS-BLED scores.*

High thromboembolic risk (CHA_2DS_2-VASc ≥2) was identified in 80% of patients, high atrial fibrillation risk according to CHARGE-AF in 70%, and increased bleeding risk in 15%. Significant associations were observed between metabolic syndrome components and atrial fibrillation risk. Comprehensive assessment allows individualized management and prevention of complications.

Keywords: *Metabolic syndrome; Atrial fibrillation; Risk assessment; CHA_2DS_2-VASc; CHARGE-AF; HAS-BLED.*

Introduction. Metabolic syndrome (MetS) represents a complex of interrelated metabolic disorders, including central obesity, arterial hypertension, impaired glucose metabolism, and dyslipidemia, which collectively increase the risk of cardiovascular diseases. The prevalence of MetS continues to rise worldwide, making it a major public health concern. One of the most clinically significant cardiovascular complications associated with MetS is atrial fibrillation (AF), the most common sustained cardiac arrhythmia encountered in clinical practice. Atrial fibrillation is associated with an increased risk of thromboembolic events, heart failure progression, reduced quality of life, and mortality [1, 2]. The pathophysiological mechanisms linking MetS and AF are multifactorial and include structural and electrical remodeling of the atria, chronic inflammation, autonomic nervous system imbalance, endothelial dysfunction, and activation of prothrombotic pathways. Obesity, insulin resistance, dyslipidemia, and hypertension contribute to atrial enlargement, fibrosis, and altered electrophysiological properties, thereby creating a substrate for AF development [3].

Early identification of AF risk in patients with MetS is essential for effective prevention and timely management. Risk stratification tools such as CHA2DS2-VASc, CHARGE-AF, and HAS-BLED scores allow clinicians to estimate thromboembolic and bleeding risks and to individualize therapeutic strategies. However, comprehensive evaluation that integrates clinical, biochemical, neurohormonal, and hemostatic parameters remains insufficiently studied in patients with MetS complicated by AF.

Therefore, the present study aimed to assess clinical and biochemical risk factors for atrial fibrillation in patients with metabolic syndrome and to evaluate their association with established AF risk scoring systems in order to improve risk stratification and management approaches.

Materials and methods. This original observational study included 40 patients diagnosed with metabolic syndrome and atrial fibrillation. The diagnosis of metabolic syndrome was established based on standard clinical criteria, while atrial fibrillation was confirmed by electrocardiographic examination. All patients underwent comprehensive clinical evaluation, including measurement of body mass index (BMI), systolic and diastolic blood pressure, and assessment of glucose tolerance. Electrocardiography was performed in all participants to document atrial fibrillation.

Laboratory investigations included determination of the plasma lipid profile, comprising total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides. Lipid parameters were measured using standard biochemical methods. Additionally, 24-hour urinary excretion of adrenaline and noradrenaline was assessed using enzyme-linked immunosorbent assay, reflecting sympathetic nervous system activity. Hemostatic evaluation included assessment of prothrombin time (PT), international normalized ratio (INR), and fibrinolytic system activity using standard biochemical techniques. These parameters were analyzed to identify potential prothrombotic or bleeding tendencies.

Risk stratification was performed using validated clinical scoring systems, including CHA2DS2-VASc for thromboembolic risk, CHARGE-AF for atrial fibrillation risk assessment, and HAS-BLED for bleeding risk evaluation. Correlation analysis was conducted to identify relationships between metabolic syndrome components, biochemical mediators, and AF risk scores. Statistical significance was considered at $p<0.05$.

Results. A total of 40 patients with metabolic syndrome and confirmed atrial fibrillation were included in the analysis. The study population consisted of 22 men (55%) and 18 women (45%), with a mean age of 56.3 ± 8.5 years. The majority of patients belonged to the middle-aged and older age groups, which is consistent with the known epidemiology of metabolic syndrome and atrial fibrillation.

The mean body mass index (BMI) of the study population was 32.7 ± 3.5 kg/m², indicating a predominance of obesity. Obesity (BMI ≥30 kg/m²) was observed in most patients, confirming its central role as a key component of metabolic syndrome. Mean systolic blood pressure was 138 ± 12 mmHg, and mean diastolic blood pressure was 86 ± 8 mmHg, reflecting a high prevalence of arterial hypertension among the participants. Impaired glucose tolerance was detected in 28 patients (70%), indicating significant disturbances in carbohydrate metabolism. These findings suggest that insulin resistance and dysglycemia are common metabolic abnormalities in patients with metabolic syndrome complicated by atrial fibrillation.

Lipid Profile

Analysis of the lipid spectrum revealed a high prevalence of atherogenic dyslipidemia.

Elevated total cholesterol levels were observed in 26 patients (65%), while increased low-density lipoprotein (LDL) cholesterol was detected in 24 patients (60%). Hypertriglyceridemia was the most frequent lipid abnormality, present in 30 patients (75%). Reduced high-density lipoprotein (HDL) cholesterol levels were identified in 22 patients (55%).

These lipid abnormalities reflect a pronounced proatherogenic profile and may contribute to atrial structural remodeling, endothelial dysfunction, and progression of atrial fibrillation in patients with metabolic syndrome.

Neurohormonal Activity

Assessment of 24-hour urinary catecholamine excretion demonstrated increased sympathetic nervous system activity in a substantial proportion of patients. Elevated urinary adrenaline excretion was detected in 18 patients (45%), while increased noradrenaline levels were observed in 21 patients (52.5%). These findings indicate autonomic imbalance with sympathetic predominance, which is known to play an important role in atrial electrical instability and arrhythmogenesis.

Hemostatic and Fibrinolytic Parameters

Evaluation of hemostatic parameters revealed abnormalities in both coagulation and fibrinolytic systems. Prolonged prothrombin time was identified in 5 patients (12.5%), and elevated international normalized ratio values were observed in 3 patients (7.5%). In addition, mild dysregulation of the fibrinolytic system was detected in 10 patients (25%).

These changes indicate alterations in hemostatic balance, which may increase the risk of thromboembolic complications in patients with atrial fibrillation and metabolic syndrome.

Risk Stratification Scores

Risk assessment using validated clinical scoring systems demonstrated a high burden of cardiovascular risk. According to the CHA2DS2-VASc score, high thromboembolic risk (score ≥2) was identified in 32 patients (80%). Evaluation using the CHARGE-AF score showed elevated atrial fibrillation risk in 28 patients (70%). Increased bleeding risk, defined as HAS-BLED score ≥3, was observed in 6 patients (15%).

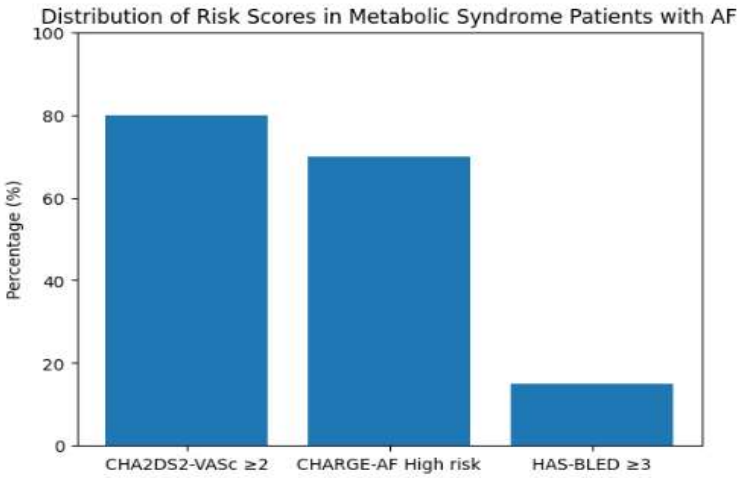

Figure 1. Distribution of risk scores in patients with metabolic syndrome and atrial fibrillation

As shown in Figure 1, the majority of patients demonstrated a high thromboembolic and arrhythmic risk according to CHA_2DS_2-VASc and CHARGE-AF scores.

These findings emphasize the importance of balanced risk assessment when selecting preventive and therapeutic strategies, particularly anticoagulation therapy.

Correlation Analysis

Correlation analysis revealed statistically significant associations between several components of metabolic syndrome and atrial fibrillation risk scores. Body mass index and triglyceride levels showed positive correlations with CHA2DS2-VASc and CHARGE-AF scores

($p < 0.05$). Additionally, elevated urinary catecholamine excretion was significantly associated with higher AF risk scores, suggesting a link between sympathetic overactivity and increased arrhythmic risk.

These results highlight the complex interaction between metabolic, neurohormonal, and hemostatic factors in the development and progression of atrial fibrillation in patients with metabolic syndrome.

Discussion. The results of this study demonstrate that patients with metabolic syndrome exhibit a high burden of interconnected risk factors that contribute to the development and progression of atrial fibrillation. Obesity, hypertension, impaired glucose tolerance, and dyslipidemia were highly prevalent in the studied cohort, consistent with existing evidence linking MetS to atrial structural and electrical remodeling [4]. Elevated urinary catecholamine levels observed in a substantial proportion of patients suggest increased sympathetic nervous system activity, which plays a significant role in AF initiation and maintenance. Sympathetic overactivation may enhance atrial automaticity, promote triggered activity, and facilitate reentry mechanisms, thereby increasing arrhythmogenic potential. The application of CHA2DS2-VASc, CHARGE-AF, and HAS-BLED scores enabled effective stratification of thromboembolic and bleeding risks. The high proportion of patients with elevated CHA2DS2-VASc scores underscores the necessity of careful anticoagulation management in this population. At the same time, assessment of bleeding risk using HAS-BLED allows identification of patients requiring closer monitoring.

The observed correlations between metabolic parameters, catecholamine excretion, and AF risk scores highlight the multifactorial nature of AF development in MetS. These findings emphasize the importance of comprehensive evaluation that goes beyond traditional clinical assessment and incorporates biochemical, neurohormonal, and hemostatic markers.

Conclusion. Patients with metabolic syndrome are at increased risk of atrial fibrillation due to the combined effects of metabolic, neurohormonal, and hemostatic disturbances. Comprehensive clinical and biochemical assessment, including lipid profile, catecholamine excretion, and hemostatic parameters, together with validated risk scoring systems, enables effective evaluation of AF risk. The results of this study involving 40 patients demonstrate significant associations between components of metabolic syndrome and atrial fibrillation risk, providing a basis for individualized management strategies. Targeted interventions addressing metabolic abnormalities and sympathetic overactivity may help prevent AF development and improve clinical outcomes in patients with metabolic syndrome.

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