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INFLAMMATORY AND ELECTROPHYSIOLOGICAL PREDICTORS OF ATRIAL FIBRILLATION PROGRESSION IN PATIENTS WITH CARDIOVASCULAR DISEASE

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Abstract: Atrial fibrillation (AF) is one of the most common cardiac arrhythmias associated with increased morbidity, thromboembolic complications, and mortality. Recent evidence indicates that inflammatory activation and structural myocardial remodeling play a central role in AF progression. The present study aimed to evaluate inflammatory and electrophysiological predictors of atrial fibrillation progression in patients with cardiovascular disease. The study included 52 patients with documented AF who underwent electrocardiographic, echocardiographic, and biochemical evaluation. Elevated C-reactive protein (CRP), left atrial enlargement, and prolonged P-wave duration were significantly associated with recurrent arrhythmic episodes and worsening clinical status. Statistical analysis demonstrated a strong positive correlation between CRP levels and AF recurrence frequency ($r = 0.71$; $p < 0.001$), while left atrial diameter negatively correlated with sinus rhythm maintenance ($r = -0.64$; $p < 0.01$). The findings confirm the prognostic importance of inflammatory and electrophysiological abnormalities in atrial fibrillation progression.

Keywords: atrial fibrillation, arrhythmia, inflammation, CRP, electrophysiology, cardiovascular disease

Introduction

Atrial fibrillation is the most frequently encountered sustained cardiac arrhythmia in clinical practice and represents a major global health problem. According to international epidemiological data, AF affects more than 37 million people worldwide, and its prevalence continues to increase due to population aging and the rising incidence of cardiovascular diseases [1]. The condition is associated with a fivefold increase in stroke risk, a threefold increase in heart failure incidence, and significantly elevated cardiovascular mortality. The pathogenesis of atrial fibrillation is multifactorial and involves electrical, structural, and inflammatory remodeling of atrial myocardium. Persistent inflammation contributes to fibrosis, conduction abnormalities, and heterogeneous electrical impulse propagation, thereby creating a substrate for arrhythmogenesis. Elevated inflammatory biomarkers, particularly C-reactive protein and interleukin-6, have been identified as independent predictors of AF recurrence and progression [2]. Structural changes such as left atrial enlargement and myocardial fibrosis further contribute to electrophysiological instability. In addition, metabolic disturbances including obesity,

hypertension, and diabetes mellitus increase atrial pressure and promote atrial remodeling. The interaction between inflammation and electrophysiological abnormalities accelerates the transition from paroxysmal to persistent forms of atrial fibrillation. Early identification of predictors associated with AF progression is essential for improving therapeutic strategies and preventing severe complications such as stroke and chronic heart failure.

Materials and Methods

This study was conducted in the cardiology department of a multidisciplinary clinical center and included 52 patients diagnosed with atrial fibrillation according to ESC diagnostic criteria. The mean age of the patients was 63.2 ± 7.1 years. Men accounted for 31 patients (59.6%), while women represented 21 patients (40.4%). Patients older than 65 years comprised 46.1% of the study population. All participants underwent clinical examination, 12-lead electrocardiography, Holter monitoring, echocardiography, and laboratory testing. The evaluated parameters included heart rate variability, P-wave duration, left atrial diameter, left ventricular ejection fraction, CRP levels, lipid profile, fasting glucose, and electrolyte balance.

Paroxysmal AF was diagnosed in 57.7% of patients, while persistent AF was observed in 42.3%. Statistical analysis was performed using Pearson correlation analysis and variation statistics. Quantitative variables were expressed as mean ± standard deviation (M±SD). Statistical significance was accepted at $p < 0.05$.

Results

The study demonstrated significant inflammatory and electrophysiological abnormalities in patients with atrial fibrillation. The mean CRP level was 7.9 ± 2.1 mg/L, exceeding normal values approximately threefold. Elevated CRP concentrations were more pronounced in patients with persistent AF compared to paroxysmal AF (9.1 ± 2.3 mg/L vs 6.4 ± 1.8 mg/L; $p < 0.01$). Electrocardiographic analysis revealed prolonged P-wave duration averaging 128 ± 14 ms, while atrial conduction abnormalities were observed in 69.2% of patients. Left atrial enlargement was identified in 61.5% of cases, with a mean left atrial diameter of 4.7 ± 0.5 cm. A strong positive correlation was identified between CRP levels and AF recurrence frequency ($r = 0.71$; $p < 0.001$). Additionally, prolonged P-wave duration showed a significant association with progression to persistent AF ($r = 0.66$; $p < 0.01$). Left atrial diameter negatively correlated with maintenance of sinus rhythm after therapy ($r = –0.64$; $p < 0.01$).

Metabolic abnormalities were highly prevalent. Hypertension was present in 71.2% of patients, obesity (BMI ≥30 kg/m²) in 44.2%, and diabetes mellitus in 30.8%. Elevated LDL cholesterol levels (>3.0 mmol/L) were observed in 65.4% of patients. Men demonstrated more pronounced electrophysiological disturbances, whereas women showed higher rates of metabolic abnormalities and obesity. In elderly patients older than 65 years, AF recurrence rates were approximately 32% higher than in younger individuals. Patients with combined inflammatory and structural abnormalities exhibited significantly worse outcomes. Hospitalization frequency due to arrhythmic episodes increased by 38%, while thromboembolic complications were identified in 13.5% of high-risk patients.

Discussion

The findings of this study confirm the important role of inflammation and myocardial remodeling in the progression of atrial fibrillation. Elevated CRP levels reflect chronic inflammatory activation, which contributes to atrial fibrosis and electrical instability. Previous studies have demonstrated that inflammatory cytokines alter atrial conduction and promote arrhythmogenic substrate formation [3].

Structural remodeling, particularly left atrial enlargement, significantly impairs maintenance of sinus rhythm and increases AF recurrence risk. The strong relationship between

prolonged P-wave duration and arrhythmia persistence confirms the prognostic value of electrophysiological markers.

Metabolic disorders such as obesity and diabetes mellitus additionally aggravate atrial remodeling through oxidative stress, endothelial dysfunction, and neurohormonal activation. Elderly patients demonstrated more severe arrhythmic progression due to cumulative structural and inflammatory changes. The integration of inflammatory biomarkers and electrophysiological assessment may improve risk stratification and facilitate early therapeutic intervention in patients with atrial fibrillation.

Conclusion

Atrial fibrillation progression is strongly associated with inflammatory activation, structural remodeling, and electrophysiological abnormalities. Elevated CRP levels, prolonged P-wave duration, and left atrial enlargement significantly correlate with recurrent arrhythmic episodes and poor clinical outcomes. The findings of this study demonstrate that inflammatory and electrophysiological predictors can serve as valuable markers for risk assessment and prognosis in patients with atrial fibrillation. Early identification and correction of these abnormalities may improve rhythm control and reduce cardiovascular complications.

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