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CLINICAL TRIAL

Study of Aldosterone and Brain Natriuretic Peptide Levels in Patients with Paroxysmal Atrial Fibrillation

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Abstract

Objective: The study examined blood levels of aldosterone and brain natriuretic peptide in patients with paroxysmal atrial fibrillation. The research included 28 patients with chronic coronary syndrome and NYHA class II-III chronic heart failure (mean age 58.5 ± 2.4 years). Among participants, 19 patients had atrial fibrillation (AF) (Group 1), and 9 had sinus rhythm (Group 2).

Results: Study findings showed that aldosterone levels in AF patients were significantly higher compared to non-AF patients (245.4 ± 72.5 pg/ml vs 151.1 ± 62.1 pg/ml, $p<0.01$). Simultaneously, brain natriuretic peptide levels were assessed in both groups. NT-proBNP concentrations in Group 1 (AF patients) were significantly higher than in Group 2 (non-AF) (9811.5 ± 38 pg/ml vs 766.5 ± 29 pg/ml; $p<0.001$).

Conclusions: Elevated aldosterone levels in AF patients confirm more extensive myocardial fibrosis coverage. Higher NT-proBNP levels indicate more severe atrial remodeling. These results suggest that elevated aldosterone serves both as a myocardial fibrosis biomarker and a prognostic indicator of AF development.

Keywords: Atrial Fibrillation, Aldosterone, NT-proBNP

Introduction

Atrial fibrillation (AF) is one of the most common cardiac arrhythmias encountered in cardiovascular diseases. This, in turn, causes a 5-fold increase in stroke risk and is a leading cause of mortality [1, 2]. Studies show that AF prevalence in individuals over 20 years exceeds 3%, with wider dissemination in comorbid conditions as age increases [3, 4].

Chronic coronary syndrome (CCS) plays a special role in AF pathogenesis, closely linked to chronic ischemia of atrial cardiomyocytes [5].

Recent extensive clinical and experimental studies demonstrate that left atrial fibrosis plays a decisive role in both AF initiation and maintenance [6]. Fibrosis, wall thickening, muscle fiber destruction, anisotropy, and electrical remodeling create synergy, leading to specific atrial tissue changes and AF substrate formation [7]. Despite this, atrial fibrosis remains the key pathological substrate for AF development, carrying fundamental pathoanatomic significance [8].

Currently, it is known that renin-angiotensin-aldosterone system (RAAS) activation occurs during ischemic heart disease. RAAS main components—angiotensin II and aldosterone—play a special role in target organ damage. Recently, aldosterone has been shown not only to participate in sodium-water metabolism but also to exert profibrotic effects, causing vascular wall and myocardial fibrosis development. A close relationship between elevated aldosterone levels and AF occurrence has been proven. AF patients exhibit higher plasma aldosterone levels and increased atrial aldosterone receptor gene expression [9].

However, the exact mechanisms by which aldosterone triggers fibrosis development remain unclear. Experimentally, galectin-3 is suggested to play a key role in realizing aldosterone's proinflammatory and profibrotic effects [10]. Experimental studies confirm that galectin-3 inhibitor application reduces myocardial fibrosis induced by aldosterone [11]. Results also show aldosterone receptor antagonist spironolactone prevents fibrosis development and reduces AF occurrence risk.

Thus, if aldosterone positively contributes to atrial myocardial fibrosis development, and fibrosis represents AF's main pathoanatomic substrate, then elevated blood

Table 1: Blood levels of aldosterone and NT-proBNP in patients with ischemic chronic heart failure

Indicators	*Group 1 (AF, n=19)*	*Group 2 (No AF, n=9)*	*p-value*
Aldosterone (pg/ml)	245.4 ± 72.5	151.1 ± 62.1	<0.01
NT-proBNP (pg/ml)	9811.5 ± 38	766.5 ± 29	<0.001

aldosterone levels can be evaluated as biomarkers for both fibrosis and AF. This creates grounds for timely identification of AF risk patients and implementation of preventive measures against this arrhythmia.

Considering the above, the main objective of the study was to investigate the role of blood plasma aldosterone levels in the development of atrial fibrillation (AF) among patients with chronic coronary syndrome and atrial fibrillation.

Materials and Method

The study included 28 patients with chronic coronary syndrome and NYHA class II-III chronic heart failure. Patient ages ranged from 42 to 69 years (mean age 58.5 ± 2.4 years). Eight patients had a history of myocardial infarction. Among participants, 19 patients (mean age 59.4 ± 2.0) had atrial fibrillation (AF), and 9 patients (mean age 60.1 ± 3.5) had sinus rhythm. All patients underwent clinical examinations including ECG, echocardiography, and laboratory tests.

Blood plasma aldosterone levels were measured in the upright position using chemiluminescent immunoassay (CLIA) method on Beckman Coulter Access (Unicel Dxi) analyzer (USA).

To determine brain natriuretic peptide levels, NT-proBNP (N-terminal fragment) concentrations in blood were studied. For this purpose, fluorescent immunoassay (FIA) method was used on AFIAS-10 automatic analyzer (Sandwich-type immunoassay, Korea).

Statistical analysis of results was performed using IBM SPSS STATISTICS 22 software. Differences with $p<0.05$ were considered statistically significant.

Results and Discussion

Study participants were divided into 2 groups: Group 1 - patients with AF (19 patients), Group 2 - patients without AF (9 patients).

Both patient groups were age-matched. As shown in the table, aldosterone levels in Group 1 patients ranged from 71.7 to 353.5 pg/ml, with a mean of 245.4 ± 72.5 pg/ml. In Group 2 patients, aldosterone levels ranged from 47.5 to 187.1 pg/ml, averaging 151.1 ± 62.1 pg/ml. The difference between these indicators was statistically significant ($p<0.01$). This demonstrates that aldosterone levels were higher in patients with AF.

The exact mechanisms by which aldosterone causes fibrosis development remain unclear. Experimental studies have confirmed aldosterone's proinflammatory and profibrotic effects in myocardium. Galectin-3 plays a key role in implementing this effect [10]. It has been established that using galectin-3 inhibitors reduces myocardial fibrosis occurring under aldosterone influence [11]. Galectin-3 is a member of the lectin family and a potent stimulator of fibroblast and collagen synthesis activation [12]. As shown in the table, NT-proBNP levels (one of the main heart failure biomarkers) in AF patients ranged from 830.1 ± 45 to 18200 ± 46 pg/ml, averaging 9811.5 ± 38 pg/ml. In non-AF patients, this peptide level ranged from 551 ± 20 to 1526 ± 38 pg/ml, averaging 766.5 ± 29 pg/ml. The difference was statistically significant ($p<0.001$). This further confirms that both heart failure presence and AF can contribute to increased natriuretic peptide blood levels. NT-proBNP is known as one of the main chronic heart failure biomarkers.

Our findings prove that AF presence in chronic heart failure patients intensifies myocardial remodeling and leads to greater increases in natriuretic peptide blood levels.

One of our interesting findings is the parallelism between blood aldosterone levels and NT-proBNP concentrations. We determined that both groups showed a direct proportional relationship between aldosterone and NT-proBNP concentrations. In other words, a positive correlation exists between aldosterone and NT-proBNP levels.

In conclusion, our results provide grounds to hypothesize that elevated blood aldosterone levels in chronic heart failure patients with atrial fibrillation serve not only as myocardial fibrosis markers but also as prognostic predictors of atrial fibrillation development.

Conclusion

- In patients with chronic coronary syndrome who have both atrial fibrillation and heart failure, blood aldosterone levels were statistically significantly higher compared to patients without atrial fibrillation.
- A positive correlation exists between blood NT-proBNP levels (a biomarker of chronic heart failure) and aldosterone concentrations. Specifically, in both groups, as NT-proBNP blood levels increased, aldosterone levels also increased.
- Elevated aldosterone levels serve not only as myocardial fibrosis biomarkers but also as potential prognostic indicators of atrial fibrillation development.

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