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Original Article

# Association of serum nesfatin-1 concentrations with atrial fibrillation

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**KEYWORDS**
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**Abstract** Nesfatin-1, a newly discovered adipokine, inhibits inflammatory response. Inflammation is involved in the mechanism of atrial fibrillation (AF). We aim to determine the association between serum nesfatin-1 concentrations and AF. A population of 200 patients with AF and 108 patients without AF were enrolled in this study. These patients were divided into three subgroups of paroxysmal AF, persistent AF, and permanent AF. Serum nesfatin-1 concentrations were lower in AF patients than in controls. Logistic regression analysis showed that serum nesfatin-1 concentrations were inversely associated with AF. Serum nesfatin-1 concentrations in permanent AF patients decreased compared with those in persistent and paroxysmal AF groups. In addition, persistent AF patients showed reduced serum nesfatin-1 concentrations compared with paroxysmal AF subjects. Serum nesfatin-1 concentrations were negatively correlated with left atrial diameter. In conclusion, serum nesfatin-1 concentrations were inversely correlated with AF development.


## Introduction

Atrial fibrillation (AF), one of the most common clinical arrhythmia, increases the risk of death, stroke, and heart failure [1]. Age, obesity, diabetes, hypertension, and cardiovascular diseases increase the risk of AF development [2]. Recent investigations have demonstrated that inflammation plays an important role in AF development and progression [3].

Nesfatin-1 is an 82-amino acid peptide highly expressed in several regions of the hypothalamus [4]. In rats, intracerebroventricular injection of nesfatin-1 suppresses appetite, whereas antibodies against nesfatin-1 contribute to weight gain [5]. Tang et al. have reported that nesfatin-1 down-regulated the expression of inflammatory genes including tumor necrosis factor-α (TNF-α), interleukin-1β

Conflicts of interest: All authors declare no conflicts of interests.

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(IL-1β) and IL-6 in traumatic rat brain tissues [6]. This finding indicates that nesfatin-1 inhibits inflammation. Considering the potential role of inflammation in AF mechanism, we hypothesized that nesfatin-1 plays a protective role in AF development.

This study aims to determine the correlation between serum nesfatin-1 concentrations and AF development.

## Materials and methods

### Patients

This cross-sectional study included a consecutive population of 200 AFF patients who went to the outpatient clinic of our hospital between 2015 year and 2016 year. AF was diagnosed by personal interview and reviewing the medical history and electrocardiogram data. Patients were excluded if they had dilated or hypertrophic cardiomyopathy, congenital heart disease, congestive heart failure or valvular heart diseases. Valvular heart diseases are defined by echocardiography as follows: aortic stenosis with a maximal jet velocity of $\geq$2.5 m/s, or mitral stenosis with a valve area of $\leq$2 $cm^2$, or mitral regurgitation grade of $\geq$2/4, or aortic regurgitation with a grade of $\geq$2/4 [7]. AF patients were divided into the following subgroups according to the guidelines prescribed by the American Heart Association guideline [8]: paroxysmal AF (n = 73), persistent AF (n = 72), and permanent AF (n = 55). The control group was recruited from subjects who received medical check-up in our hospital. They subjects did not suffered from any systemic disease.

This study was reviewed and approved by the Ethics Committee of our hospital. All individuals signed the informed consent forms prior to enrollment.

### Measurements

Peripheral blood samples from participants were collected in test tubes. Plasma was obtained by centrifugation at 3000 rpm for 10 min, repackaged, and stored at −80 °C. Then the blood was tested at the same time after all blood sample were collected. We collected the blood of some patients because our hospital has founded a biobank which included blood sample. The biobank was approved by the institutional review board (IRB) (NO: WFPH20140028). Serum triglycerides (TG), serum total cholesterol (TC), high-density lipoprotein cholesterol (LDL-C), low-density lipoprotein cholesterol (LDL-C), and C-reactive protein (CRP) were measured by auto biochemistry instrument (Hitachi 7170, Tokyo, Japan). Serum nesfatin-1 concentrations were measured using an enzyme-linked immunosorbent assay kit (Phoenix Pharmaceuticals, Inc, USA) according to the manufacturer's instructions. In addition, each assay was repeated to confirm the consistency and accuracy of the measurements. Two-dimensional and Doppler echocardiography was performed by an experienced sonographer by using a Vivid4 System (GE Healthcare Systems, Piscataway, NJ, USA). Left atrial diameter (LAD) was evaluated by an experienced sonographer in parasternal long axis view at end systole.

### Statistical analysis

The data were presented as means ± standard errors. Unpaired t test or Chi-square tests was used to determine the parameters differences between case and control groups. Univariate analysis was performed with all variables and the variables with a $P < 0.10$ were then entered into a backward stepwise multivariate logistic regression model to calculate the Odds ratio values (OR) and 95% confidence intervals (CI) for the presence of AF. The characteristics between the three AF subgroups were compared by Chi-square tests or one-way ANOVA. The correlation of serum nesfatin-1 concentrations with left atrial diameter (LAD) were analyzed by Spearman correlation analysis. *P* less than 0.05 was considered statistically significant.

## Results

### Baseline clinical characteristics

AF patients showed higher levels of systolic blood pressure (SBP), diastolic blood pressure (DBP), TC, LDL-C, CRP, and LAD compared with healthy controls (Table 1).

### Serum nesfatin-1 concentrations in AF patients

AF patients showed significantly decreased serum nesfatin-1 concentration and elevated CRP concentration compared with healthy controls (Table 1). Simple and multivariate logistic regression analysis both indicated that serum nesfatin-1 and CRP concentrations were correlated with AF development (Table 2). In AF subgroups, permanent AF patients showed reduced serum nesfatin-1 concentration and increased CRP concentration compared with the paroxysmal and persistent AF groups (Table 3). Furthermore, persistent AF patients showed a significantly lower serum nesfatin-1 concentration and significantly increased CRP compared with the paroxysmal AF subjects (Table 3). Spearman correlation analysis revealed that serum nesfatin-1 and CRP were correlated with AF type ($r = -0.336$, $P < 0.001$ and $r = 0.419$, $P < 0.001$, respectively).

**Table 1** Clinical and biochemical characteristics of the case and control groups.

| | The controls | AF patients | *P* value |
|---|---|---|---|
| N | 108 | 200 | |
| Age (years) | 63.12 ± 9.75 | 62.10 ± 9.88 | 0.383 |
| Gender (M/F) | 54/54 | 111/89 | 0.356 |
| BMI ($Kg/m^2$) | 25.15 ± 2.63 | 25.31 ± 3.09 | 0.642 |
| SBP (mmHg) | 124.58 ± 8.80 | 133.82 ± 12.12 | <0.001 |
| DBP (mmHg) | 77.92 ± 6.84 | 82.60 ± 8.64 | <0.001 |
| TC (mmol/L) | 4.79 ± 0.82 | 5.02 ± 1.00 | 0.042 |
| TG (mmol/L) | 1.54 ± 0.53 | 1.58 ± 0.80 | 0.605 |
| LDL-C (mmol/L) | 3.07 ± 0.50 | 3.42 ± 0.75 | <0.001 |
| HDL-C (mmol/L) | 1.15 ± 0.22 | 1.12 ± 0.21 | 0.343 |
| LAD (mm) | 29.12 ± 3.31 | 41.35 ± 4.30 | <0.001 |
| Nesfatin-1 (ng/mL) | 1.19 ± 0.31 | 0.90 ± 0.18 | <0.001 |
| CRP (mg/L) | 1.49 ± 0.66 | 2.91 ± 1.64 | <0.001 |

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