



ORIGINAL ARTICLE / *Cardiovascular*

Relationships between left atrial pericardial fat and permanent atrial fibrillation: Results of a case-control study



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KEYWORDS

Atrial fibrillation;
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Abstract

Purpose: The goal of this study was to retrospectively investigate the relationships between pericardial fat, left atrium volume (LAV) as measured on multidetector row computed tomography (MDCT) and persistent atrial fibrillation (AF) using a case-control study.

Materials and methods: The study population consisted of 58 patients (19 men, 39 women; mean age, 67.8 ± 10 [SD] years) with persistent AF and 74 control subjects (30 men, 44 women; mean age, 67.8 ± 10.9 [SD] years). The associations between the presence of persistent AF and periatrial pericardial fat volume (PAFV), periatrial pericardial fat thickness (PAFT), and LAV as measured on MDCT were searched for using univariate and multiple linear regression analysis.

Results: On univariate analysis, significant differences were found between patients with AF and control subjects for mean PAFV ($54.33 \text{ cm}^3 \pm 23.43$ [SD]; range: 12.2–111.1 cm^3 vs $42.99 \text{ cm}^3 \pm 20.76$ [SD]; range: 7.4–103.9 cm^3 , respectively) ($P=0.01$), PAFT at the esophagus

Abbreviations: AF, Atrial fibrillation; LA, Left atrium; PAFV, Pericardial fat volume surrounding the LA; PAFT, Periatrial fat thickness; ECG, Electrocardiography; LA-ESO, The shortest fat thickness between the LA and the esophagus; LA-PA, The shortest fat thickness between the LA and the pulmonary artery; LA-DA, The shortest fat thickness between the LA and the descending aorta; BMI, Body mass index.

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(1.87 mm \pm 1.65 [SD]; range: 0.1–9.5 mm vs 1.12 mm \pm 0.77 [SD]; range: 0.1–3.6 mm, respectively) ($P < 0.001$) and normalized LAV (78.3 cm³/m² \pm 48.84 [SD]; range: 32.1–319.6 cm³/m² vs 42.1 cm³/m² \pm 25.43 [SD]; range: 15.7–191.4 cm³/m², respectively) ($P < 0.001$). Multiple linear regression analysis revealed that only LAV was an independent predictor ($P < 0.001$) of persistent AF. Also PAFV was significantly associated with LAV ($P = 0.01$).

Conclusion: LAV is greater in patients with AF than in control subjects and PAFV is strongly associated with LAV. PAFV and PAFT are not independently associated with AF.

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Atrial fibrillation (AF) is a common arrhythmia with high morbidity and mortality outcomes. AF frequency in the general population is 1% and its incidence rises with age [1–4]. The pathogenesis of AF is clearly multifactorial. AF is commonly accompanied by central obesity, hypertension, heart valve diseases, coronary artery diseases, heart failure and hyperthyroidism [4–6]. The pericardial fat is a special, metabolically active tissue with the same embryological origin as mesenteric and omental fat [7,8]. Pericardial fat affects the myocardium and coronary arteries by releasing many inflammatory mediators, leading to structural changes caused by inflammation [9,10]. Furthermore, mediators in pericardial fat may increase the predisposition to AF by increasing in vagal tonus through a direct local effect [10–12].

AF generally originates from the ectopic foci in the muscular sleeves reaching the pulmonary vein ostia in the left atrium (LA) [13]. It is believed that inflammation plays a significant role in AF etiopathogenesis [14]. Biopsies of the LA in patients with AF have shown a significant amount of inflammatory infiltrate in atrial tissue [1,15]. Inflammatory pathways may influence structural changes within LA and lead to the development of AF. Persistent AF is a chronic type of AF and several studies have reported the relationships between persistent AF and pericardial fat [16–20]. However, a relationship between the local pericardial fat deposits surrounding left atrium, persistent AF, and a potential effect of left atrium enlargement has not yet been evaluated clearly.

The goal of this study was to retrospectively investigate the relationships between pericardial fat, left atrium volume (LAV) as measured on multidetector row computed tomography (MDCT) and persistent AF using a case-control study.

Materials and methods

Study population

This prospective study was conducted between May 2012 and September 2012. Fifty-eight patients with persistent AF (19 men, 39 women) with a mean age of 67.8 years \pm 10 (SD) (range, 48–90 years) were included. This group was compared to 74 age- and gender-matched controls without AF

(30 men, 44 women) with a mean age of 67.8 years \pm 10.9 (SD) (range, 34–86 years). The participants in this study were assessed by clinicians as having a low estimated likelihood of coronary artery disease. Coronary artery calcium scoring examination was performed to determine if additional cardiac testing was indicated. All participants fulfilled the inclusion criteria that consisted of atypical chest pain in combination with negative biomarkers and inconclusive electrocardiography. Exclusion criterion was having prosthetic heart valve that induced image artifacts.

The diagnosis of persistent AF was made when the patient had a type of arrhythmia lasting over one year, and when cardioversion has failed or not been tested. Hypertension was defined as a blood pressure \geq 140/90 mmHg or when the patient was receiving a treatment with an antihypertensive drug. Diabetes was defined as fasting plasma glucose level \geq 126 mg/dL or treatment with insulin or a hypoglycemic drug. Clinically significant valvular disease was defined as a systolic murmur grade \geq 3/6 or any diastolic murmur noted on examination by the clinic physician. Patients with heart failure and myocardial infarction event at any point before MDCT assessment were deemed to have heart failure or myocardial infarct, respectively. The local ethical committee approved the study, written informed consent for the research procedures was obtained.

Image acquisition and analysis

All participants underwent coronary artery calcium scoring evaluation using 16-slice MDCT scanner (Aquilion®, Toshiba Medical Systems Corporation, Otawara-shi, Japan). MDCT examinations were performed without intravenous administration of iodinated contrast material. No additional beta-blockers were given for heart rate control before the MDCT examination. The heart was scanned from the level of carina to the end of the left ventricular apex with 3-mm slice thickness and 300 mA, 120 kV, 0.25 ms scan time. In order to reduce stair-step artifacts caused by imaging in different cardiac cycle phases, data acquisition was performed using prospective ECG gating of the R–R interval. The obtained images were evaluated using a commercially available workstation (Vitrea, Minnetonka, MN, USA).

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