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Review Article

Left atrial volume index is the strongest predictor of development of persistent atrial fibrillation in obese non-hypertensives

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1. Introduction

The global nature of the obesity epidemic was formally recognized by a World Health Organization consultation in 1997.[1] Prior epidemiologic studies have yielded conflicting results regarding whether obesity is a risk factor for Atrial fibrillation (AF), but these studies were potentially limited by short-term follow up and lack of echocardiography data.[2] It is not sure if obesity itself predisposes to AF and so hypothesizing such a link by suggesting that adiposity influences atrial and ventricular structure,[3] autonomic tone[4] and ventricular diastolic function.[5] The actual mechanism by which obesity may increase AF risk is unknown, but several mechanisms have been suggested, including increased left atrial (LA) size, chronic inflammation, and development of other cardiovascular risk factors or cardiovascular disease.[6] AF is the most prevalent sustained cardiac arrhythmia, and its prevalence is increasing[7] specially with age,[8] diabetes, obesity, hypertension, left ventricular hypertrophy (LVH), coronary heart disease, congestive heart failure, Valvular heart disease, and increased left atrial size by echocardiography. Obesity occurs in

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association with most of these conditions.[9] AF is associated with a 4–5 fold increased risk of ischemic stroke[10] and with a nearly doubled cardiovascular mortality risk.[11] Because the onset of AF is associated with considerable morbidity and mortality despite contemporary therapies, the identification of potentially modifiable risk factors for AF is an important goal.[12] Prevention of AF is thus of great importance, and hypertension is currently the most prevalent, potentially modifiable risk factor, accounting for 14–22% of AF cases.[1]

2. Aim of the study

We aim to discover the predictors of persistent AF in obese non-hypertensives.

3. Patients and methods

Retrospective case control study included 110 non-hypertensives, obese patients (Body mass index "BMI" > 30Kg/m^2).[13] ***Left ventricular mass index by American society of echocardiography (LVMI ASE)*** was calculated. Formula is as follows: LVM = 0.8 × (1.04 × (LVEDD + PWTd + SWTd)^3 –(LVEDD)^3)) + 0.6. LVEDD = left ventricular end-diastolic diameter, PWTd = diastolic posterior wall thickness, and SWTd = diastolic septal wall thickness. All measures by M-mode.[14] Normalization of left ventricular mass by height has been shown to be more sensitive than normalization to body surface area to identify obesity-related left ventricular hypertrophy.[15]

3.1. Left ventricular stiffness (LVS)

pulsed wave was used to measure the velocity of early diastolic velocity (E) and pulsed wave TDI *"Tissue Doppler imaging"* was used to measured early diastolic annular velocities (e') at both septal and lateral annular sites from the apical four chamber view and then be averaged. LVS is then calculated from the equation: [(E/e'')/LVEDD].[16]

3.2. Left atrial volume index "LAVI"

using biplane area-length method. LA size should be measured at the end-ventricular systole (maximum LA size). Foreshortening should be avoided. When planimetry is performed, LA confluences of the pulmonary veins and the LA appendage should be excluded. The length, L, remains the LA long-axis length determined as the distance of the perpendicular line measured from the middle of the plane of the mitral annulus to the superior aspect of the LA. In the area-length formula the length is measured in both the 4- and 2-chamber views and the shortest of these 2 length measurements is used in the formula: ***LA Volume = (0.85) X (A1XA2/L) then LAVI = LA Volume/BSA.*** Although there are gender differences in LA size, these are completely accounted for once indexed to body size, such as body surface area (BSA).[17] by history, we collect the cases that get persistent AF during 1 year of follow up. Two operators, to avoid the intra-observer and inter-observer variabilities, did all echocardiography measures.

3.3. Exclusion criteria

Hypertension, Coronary artery disease "CAD" (*negative stress test and minority underwent coronary angiogram – for suspicion of CAD- and proved to be normal or non-significant lesions*), Diabetes mellitus "DM"; all have normal blood sugar and HbAIC, Valvular heart disease, Heart muscle disease, Pulmonary, renal diseases and HF were also excluded.

3.4. Statistical Analysis

All analyses were performed with "SPSS 20 for Windows" software package. Continuous variables were expressed as mean± standard deviation; categorical variables were expressed as percentages. Independent *T* test was used to compare means. Pearson's correlation coefficients were used to assess the strength of relationship between continuous variables. ROC curve was plotted to get cut off value, A p value of less than 0.05 was considered significant.

4. Results

4.1. Development of AF

(Table 1) in our study; 30 cases developed persistent AF during 1 year of follow up (27.3%), while 80 cases did not develop persistent AF (72.7%). Concerning age; it was 49.30 ± 5.66 years old in AF group while it was 47.85 ± 5.59 years old in Non-AF group. This difference was not significant at all (t = −1.21, p > 0.05). In the AF group, ***LVS*** was higher in AF group (0.12 ± 0.05) more than non-AF group (0.07 ± 0.04) and this difference was highly significant (t = −4.69, p < 0.001). Concerning ***BMI;*** it was 36.48 ± 3.15 Kg/m^2 in AF group while it was 31.57 ± 1.45 Kg/m^2 in non-AF group, highly significant difference (t = −11.18, p < 0.001). Lastly; ***LAVI*** was higher in AF group (36.88 ± 0.79 ml/m^2) when compared in non-AF group (32.54 ± 1.01 ml/m^2); this difference is highly significant (t = −21.19, p < 0.001).

4.2. According to gender

(Tables 2 & 3) we had 86 females, 20 of them developed persistent AF during 1 year of follow up (23.3%) while we had 24 males, of which 10 developed persistent AF during 1 year of follow up (41.6%). This difference was not significant (X = 3.21, p > 0.05).

4.3. Age

in females; it was 47.85 ± 5.60 years old in females who get AF while it was 47.42 ± 5.69 years old in non-AF females, this difference was not significant (t = −1.18, p > 0.05). The same was in males; age was 52.20 ± 4.80 years old in males who got AF while it was 49.86 ± 4.79 years old in males who did not get AF. This difference was not significant (t = −0.29, p > 0.05).

4.4. LVS

in females; it was 0.11 ± 0.06 in those who got AF while it was 0.07 ± 0.04 in non-AF females, this difference was highly significant (t = −3.39, p < 0.001.). The same was in males; MSI was 0.12 ± 0.05 in males who got AF while it was 0.07 ± 0.02 in males who did not get AF. This difference was significant (t = −3.56, p < 0.05).

Table 1
Comparison of the study variables between both groups.

Parameter	AF group (30 cases)	Non-AF group (80 cases)	(t)	(p)
Age	49.30 ± 5.66	47.85 ± 5.59	−1.21	>0.05
LVS*	0.12 ± 0.05	0.07 ± 0.04	−4.69	<0.001
BMI kg/m$^{2\times}$	36.48 ± 3.15	31.57 ± 1.45	−11.18	<0.001
LAVI ml/m$^{2¥}$	36.88 ± 0.79	32.54 ± 1.01	−21.19	<0.001

*Left Ventricular Stiffness, ×Body Mass index, ¥Left Atrial volume Index.

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