International Journal of Cardiology xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Right ventricular function measured by TAPSE in obese subjects at the time of acute myocardial infarction and 2 year outcomes

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ARTICLE INFO

Article history:
Received 12 August 2016
Received in revised form 2 January 2017
Accepted 4 January 2017
Available online xxxx

Keywords:
TAPSE
Obesity paradox
RV function

ABSTRACT

Introduction: Obesity is associated with significantly better outcome after acute myocardial infarction (AMI), a phenomenon known as 'obesity paradox'. Tricuspid annular plane systolic excursion (TAPSE) is an echocardiographic measurement of right ventricular (RV) function and has prognostic implications at the time of AMI.
Methods: We examined the difference in RV function among patients admitted with AMI according to obesity status. In a single center cohort analysis of 105 patients admitted between 2010 and 2011 with the diagnosis of AMI. Demographic, anthropometric data and cardiovascular risk factors were prospectively collected. All subjects had echocardiogram within 48 h of AMI diagnosis for TAPSE calculations. Subjects were divided into two groups based on their obesity status.
Results: Obese subjects had better RV function compared to non-obese, TAPSE: 19 ± 6.6 vs. 16 ± 4.9 mm; p 0.02 at the time of AMI. There was no significant difference in TAPSE between OSA and non-OSA subjects, 19 ± 6.3 vs. 17 ± 6.2 mm; p 0.21. After 2 years of follow up, patients with obesity and better RV function were less likely to develop new onset heart failure (HF) with OR 0.30 (95% CI 0.09–0.93; p 0.03) and OR 0.31 (95% CI 0.11–0.76; p 0.007) respectively.
Conclusion: Obese patients had better RV function measured by TAPSE at the time AMI when compared non-obese patients. Patients with better RV function at the time of AMI were less likely to develop new-onset HF and there was a trend in the obese group to less likely develop new-onset HF after 2 year follow up.
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1. Introduction

Obesity is an independent risk factor for developing coronary artery disease (CAD) and acute myocardial infarction (AMI) [1,2]. Despite obesity being a risk factor for AMI, obese patients who experienced an AMI have a better prognosis and outcomes compared to non-obese patients, a phenomenon called the 'obesity paradox' [3–7]. The epidemiological observation of the obesity paradox cannot be fully explained by confounding factors or the lack of discrimination power to differentiate between body fat and lean mass of body mass index (BMI) as data has shown that obesity paradox was present even when identifying obesity using body fat composition [8,9]. The obesity paradox has been observed in numerous disease states other than CAD, including heart failure (HF), chronic obstructive pulmonary disease and end-stage renal disease [10–13]. Recent studies have suggested that obesity is associated with a smaller infarct size and less complex CAD lesion on presentation with AMI which could help to understand the obesity paradox [14–18]. Right ventricle (RV) function is an important prognostic factor in the setting AMI [19]. Deferential RV function between obese and non-obese patients at the time of AMI is unknown and thus, it was the purpose of our study to test whether differences in RV function exist between obese and non-obese patients at the time AMI.

2. Methods

Our study was a single center cohort study of 105 consecutive patients admitted to the coronary care unit from March 2010 to March 2011 with a diagnosis of AMI who underwent coronary angiography. All subjects had echocardiogram within 48 h of the diagnosis of AMI. We excluded subjects with recent cocaine use, ejection fraction <20%, those on hemodialysis and those who have right coronary artery culprit lesions. Subjects with right coronary artery culprit lesion were excluded to avoid the confounding effect of the ischemic lesion on the right ventricular function. Subjects were followed for 2 years after AMI to assess adverse cardiovascular outcomes including mortality.

Standardized forms were used at the time of the coronary care unit admission to collect subjects' demographics, presence or absence of cardiovascular risk factors including hypertension, diabetes mellitus, dyslipidemia, smoking (current/former), history of CAD, heart failure (HF), the likelihood of having obstructive sleep apnea (OSA) according to a prefilled modified Berlin questionnaire [20], family history of premature CAD, and medication history. Echocardiographic data, including evaluation of left ventricular function, was

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http://dx.doi.org/10.1016/j.ijcard.2017.01.033
0167-5273/Published by Elsevier Ireland Ltd.

obtained. Anthropometric data were obtained and obesity was defined as a BMI >30 kg/m^2 and/or WC >88 cm in women and >102 cm in men [15]. Subjects were divided into two groups based on the obesity status. The diagnosis of AMI, including non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI), was made according to American Heart Association and American College of Cardiology Foundation recommendations [21]. Coronary angiography at the time of AMI was reviewed and culprit lesions were identified in all cases.

An experienced echocardiographer, who was blinded to subjects' data performed tricuspid annular plane systolic excursion (TAPSE) measurements. Echocardiography was done within two days of AMI. TAPSE is a well-known, established tool to assess RV systolic function. TAPSE was measured using the trans-apical four chambers view on 2D echocardiography. The end systolic distance between the tricuspid annulus and a fixed point of reference was subtracted from the end diastolic distance in millimeters. Measurements were performed according to the American Society of Echocardiography guidelines [22].

Subjects were followed up for 2 years after the AMI. Adverse cardiovascular events such as re-infarction, new onset HF, new onset arrhythmias, stroke, re-intervention and death were collected. Eight subjects (9.8%) were lost to follow up.

2.1. Statistical analysis

Data were summarized as mean and standard deviation for continuous variables and as numbers and percentages for categorical variables. To assess baseline differences between obese and non-obese subjects we used independent *t*-test for continuous variables and ×2 or Fisher exact test for categorical variables. To assess the independent contribution of obesity and RV systolic function on outcomes at two years–including re-infarction, new onset HF, new onset arrhythmias, stroke, re-intervention and death we performed logistic regression analyses after controlling for known cardiovascular risk factors. In addition, to assess the effect of obesity on RV systolic function independent of OSA, we preformed stratified analyses by the presence or absence of high likelihood of OSA. A 2-tail *p*-value <0.05 was considered significant in advance. All statistical tests were performed using JMP software (SAS 12.2.0, NC).

3. Results

Out of 105 subjects, 81 were included as 24 subjects were excluded for having significant RCA culprit lesions. Out of 81 subjects included in the analysis, 67 subjects were found to have significant coronary artery disease and 14 subjects were not found to have significant coronary artery disease. Out of the 67 subjects who had significant coronary artery disease, 65 subjects underwent percutaneous coronary intervention, 2 underwent coronary artery bypass grafting. Table 1 shows the baselines characteristics based on their obesity status. There were no significant differences in age, sex or race between the two study groups. The obese group, when compared to the non-obese group, had a higher prevalence of hypertension: 98 vs 85%; p 0.04, diabetes: 54 vs 22%; p 0.007 and high likelihood of OSA: 72 vs 15%; p 0.0001. There were no significant differences in other risk factors including hyperlipidemia, smoking status, history of CAD, stroke, HF or the number of diseased vessels. The obese group, when compared to the non-obese group, had comparable percentages of both STEMI: 33 vs 30% and NSTEMI: 67 vs 70%; p 0.73. There was no significant difference in the complexity of coronary lesions as measured by SYNTAX score between the obese and the non-obese groups: 14.8 ± 10.2 vs 18 ± 9.5; p 0.18.

Fig. 1 shows that the obese group had better RV function when compared to the non-obese group, as determined by the TAPSE measurements at the time of AMI. TAPSE in obese was 19 ± 6.6 mm, while in non-obese it was 16 ± 4.9 mm; p 0.02. There was no statistically significant difference in LV function in both groups as EF in the obese group was 46 ± 6% and the non-obese group was 40 ± 14%; p 0.1.

Given the higher prevalence of OSA likelihood in the obese group, we tested the independence of obesity effect on RV function. Subjects with high OSA likelihood, when compared to subjects with low OSA likelihood, demonstrated no significant difference in RV function as determined by TAPSE: 19 ± 6.3 mm vs 17 ± 6.2 mm respectively; p 0.21 as shown in Fig. 2.

Table 2 shows the major adverse cardiovascular outcomes in both study groups. There were no significant differences in the incidence of re-infarction, stroke, re-intervention, arrhythmia or mortality between the two study groups. Subjects in the obese group were less likely to develop HF when compared to the non-obese group 20 vs. 41%; p 0.05. Moreover, in logistic regression analysis adjusting for age, race, sex, HTN, diabetes and smoking history, subjects with obesity were less likely to develop new-onset HF when compared to the non-obese subjects with OR 0.30 (95% CI 0.09–0.93; p 0.03) as well as those with better RV function as measured by TAPSE with OR 0.31 (95% CI 0.11–0.76; p 0.007). When fully adjusted model including both TAPSE and obesity was used, subjects with better RV function as measured by TAPSE were still less likely to develop new-onset HF with OR 0.35 (95% CI 0.13–0.90; p 0.02) and there was a trend in the obese group when compared to the non-obese group to less likely develop a new-onset HF with OR 0.34 (95% CI 0.09–1.13; p 0.07).

Table 1
Baseline characteristics by obesity status.

Variables	All patients, $n = 81$	Non-obese, $n = 27$	Obese, $n = 54$	*p*-value
Age, years	62.5 ± 12	64 ± 12	61 ± 12	0.37
Gender, Men	34 (42%)	8 (30%)	26 (48%)	0.11
Race,				0.08
AA	49 (61%)	14 (52%)	35 (65%)	
Caucasian	17 (21%)	7 (26%)	10 (18%)	
Hispanic	10 (12%)	2 (7%)	8 (15%)	
Other	5 (6%)	4 (15%)	1 (2%)	
Type of MI				0.73
STEMI	26 (32%)	8 (30%)	18 (33%)	
NSTEMI	55 (68%)	19 (70%)	36 (67%)	
SYNTAX score	15.9 ± 10	18 ± 9.5	14.8 ± 10.2	0.18
Number of diseased vessels				0.67
1 vessel disease	26 (39%)	8 (32%)	18 (43%)	
2 vessels disease	27 (40%)	11 (44%)	16 (38%)	
3 vessels disease	14 (21%)	6(24%)	8 (19%)	
Likelihood of OSA	43 (53%)	4 (15%)	39 (72%)	0.0001
Obese	54 (67%)			
Smokers	37 (46%)	14 (52%)	23 (43%)	0.43
HTN	76 (94%)	23 (85%)	53 (98%)	0.04
Diabetes	35 (43%)	6 (22%)	29 (54%)	0.007
HLD	61 (76%)	19 (73%)	42 (78%)	0.64
H/O CAD	27 (33%)	10 (37%)	17 (31%)	0.61
H/O CHF	6 (7%)	4 (15%)	2 (4%)	0.08
Stroke	6 (7%)	3 (11%)	3 (6%)	0.36
CKD	3 (4%)	0 (0%)	3 (6%)	0.21
PVD	3 (4%)	0 (0%)	3 (6%)	0.2

AA: African American; CAD: coronary artery disease; CKD: chronic kidney disease; CHF: congestive heart failure; HLD: hyperlipidemia; H/O: history of; HTN: hypertension; OSA: obstructive sleep apnea; PVD: peripheral vascular disease; STEMI: ST elevation myocardial infarction.

Furthermore, we stratified the outcomes according to the type of AMI (STEMI vs. NSTEMI) in subjects divided based on their obesity status. In our cohort, 26 subjects suffered STEMI. Among subjects who had STEMI, the non-obese group when compared to the obese developed more new onset arrhythmia (38% vs. 0%; $p < 0.001$), new onset HF (88% vs. 22%; $p < 0.001$) and strokes (25% vs. 6%; p 0.007) after adjusting for age, race, sex, HTN, diabetes and smoking history, Table 3. Among subjects admitted with NSTEMI, there was no significant difference in outcomes between the non-obese and the obese groups, Table 4.

4. Discussion

To the best of our knowledge, this is the first study to report better RV function, as measured by TAPSE, in obese patients when compared to non-obese patients in at the time of AMI. There was no statistical significant difference in the LVEF in both study groups. Hence OSA is more common in the obese population and it has been shown to alter myocardial performance [23,24], we examined the relation between the RV function and OSA likelihood at the time of AMI. We found that TAPSE was comparable between subjects with a high likelihood of OSA when compared with subjects with a low likelihood of OSA. Furthermore, after 2 years follow up, we found that subjects with better RV function as measured by TAPSE at the time of AMI were less likely to develop new-onset HF and there was a trend in the obese group to less

Please cite this article as: Y.S. Alhamshari, et al., Right ventricular function measured by TAPSE in obese subjects at the time of acute myocardial infarction and 2year outcomes, Int J Cardiol (2017), http://dx.doi.org/10.1016/j.ijcard.2017.01.033