Journal of the Saudi Heart Association

Volume 32 | Issue 4 Article 11

2020

# Right Atrial Volume Index as a Predictor of Persistent Right Ventricular Dysfunction in Patients with Acute Inferior Myocardial Infarction and Proximal Right Coronary Artery Occlusion Treated with Primary Percutaneous Coronary Intervention

Follow this and additional works at: https://www.j-saudi-heart.com/jsha

Part of the Cardiology Commons

This work is licensed under a Creative Commons Attribution-Noncommercial-No Derivative Works 4.0 License.

Recommended Citation

Naseem, Mohamed and Samir, Sameh (2020) "Right Atrial Volume Index as a Predictor of Persistent Right Ventricular Dysfunction in Patients with Acute Inferior Myocardial Infarction and Proximal Right Coronary Artery Occlusion Treated with Primary Percutaneous Coronary Intervention," *Journal of the Saudi Heart Association*: Vol. 32 : Iss. 4 , Article 11.

Available at: https://doi.org/10.37616/2212-5043.1208

This Original Article is brought to you for free and open access by Journal of the Saudi Heart Association. It has been accepted for inclusion in Journal of the Saudi Heart Association by an authorized editor of Journal of the Saudi Heart Association.

ORIGINAL ARTICLE

# Right Atrial Volume Index as a Predictor of Persistent Right Ventricular Dysfunction in Patients with Acute Inferior Myocardial Infarction and Proximal Right Coronary Artery Occlusion Treated with Primary Percutaneous Coronary Intervention

Mohamed Naseem*, Sameh Samir

Cardiovascular Medicine Department, Tanta Faculty of Medicine, Egypt

**Abstract**

*Objectives*: **Patients with right ventricular (RV) infarctions associated with inferior infarctions have higher rates of adverse events than isolated inferior infarctions. Right atrial volume index (RAVI) has recently been described as a predictor of clinical outcome in patients with chronic systolic heart failure and pulmonary hypertension. The aim of this study is to assess the ability of RAVI to predict the persistent RV dysfunction after acute inferior STEMI due to occlusion of proximal RCA. To the best of our knowledge, this is the first study to investigate the relation between RAVI and persistent RV dysfunction in such group of patients.**

*Patients and methods*: **Sixty-five consecutive patients with recent first acute inferior STEMI who underwent primary percutaneous coronary intervention (PPCI) were prospectively included in the study. Echocardiographic evaluation was performed at the time of discharge and at 3 months. All the patients underwent standard echocardiographic assessment using conventional 2D and tissue Doppler imaging (TDI).**

*Results*: **Patients were divided into two groups according to right ventricular function (RVF) 3 months after acute myocardial infarction (AMI). The normal RVF group included 41 (63%) patients and the impaired RVF group included 24 (37%) patients. RAVI was significantly higher in patients with impaired RVF (p=<0.001). RAVI was a predictor of persistently impaired RV function (odds ratio = 1.786, 95% confidence interval, 1.367−2.335, p value= <0.001) and (odds ratio = 1.829, 95% confidence interval, 1.358 −2.462, p value= <0.001) in univariate and multivariable logistic regression analyses respectively. In receiving operator characteristics (ROC) curve analysis, RAVI with a cutoff value ≥ 30 ml/m$^2$ had a 87.5% sensitivity, a 92.24% specificity area under Receiving operator characteristics (ROC) curve = 0.964 for predicting persistently impaired RVF.**

*Conclusion*: **In patients with inferior STEMI with proximal RCA occlusion, RAVI is an independent predictor of persistently impaired RVF with a cut-off value ≥ 30 ml/m$^2$.**

*Keywords:* Right atrial volume index, inferior STEMI, RV function

## 1. Introduction

In nearly half of acute inferior STEMI patients, the right ventricle is affected by acute myocardial infarction (AMI) mostly due to occlusion of the right coronary artery (RCA) proximal to the right ventricular (RV) branch[1]. Due to multiple factors the right ventricle is less affected by ischemia than the left ventricle (LV) [2], however when acute RV ischemia complicates

Received 4 September 2020; revised 23 November 2020; accepted 3 December 2020.
Available online 8 January 2021

* Corresponding author.
E-mail address: mohamednasim2011@gmail.com (M. Naseem).

https://doi.org/10.37616/2212-5043.1208
2212-5043/© 2020 Saudi Heart Association. This is an open access article under the CC-BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

acute inferior STEMI, it leads to poor clinical outcome especially short term one due to increased risk of arrhythmias and hemodynamic instability [3].

Successful revascularization by primary percutaneous coronary intervention (PPCI) usually improve the RV systolic function [4]. Patients who could pass the acute phase of ischemic RV dysfunction usually regain normal RV function over a period of weeks or months. Echocardiography remains the most commonly used technique to assess RV function due to its availability and its nature as non-invasive real time method. Multiple 2D parameters and tissue Doppler imaging (TDI) are used to assess RV function. However, the complex shape of the RV makes these measurements a real challenge in routine clinical practice. In contrast, the right atrium (RA) can be visualized clearly using 2D echocardiography allowing accurate and reproducible measurement of the RA volume. RA volume indexed to body surface area (RAVI) has recently been described as an echocardiographic parameter that is linked to adverse outcome in patients with heart failure (HF)[5,6] and pulmonary hypertension[7]. In contrast, no studies have explored relationship between RAVI and RV dysfunction in patients with acute inferior STEMI due to occlusion of proximal RCA. We hypothesized that RAVI can serve as a marker of RV dysfunction to predict the persistent of RV dysfunction after acute inferior STEMI due to occlusion of proximal RCA.

**List of abbreviations**

| | |
|---|---|
| RV | Right ventricular |
| RAVI | Right atrial volume index |
| STEMI | ST segment elevation myocardial infarction |
| RCA | Right coronary artery |
| PPCI | Primary percutaneous coronary intervention |
| NYHA | New York Heart Association |
| TDI | Tissue Doppler imaging |
| RVF | Right ventricular function |
| AMI | Acute myocardial infarction |
| ROC | Receiving operator characteristics |
| LV | Left ventricle |
| RA | Right atrium |
| HF | Heart failure |
| RV-MI | Right ventricular myocardial infarction |
| COPD | Chronic obstructive pulmonary disease |
| TIMI | Thrombolysis in Myocardial Infarction flow |
| MBG | Myocardial blush grade |
| EF | Ejection fractions |
| LAVI | Left atrial volume index |
| TAPSE | Tricuspid annular plane systolic excursion |
| RVFAC | Right ventricular fractional area change |
| MPI | Myocardial performance index |
| IVRT | Isovolumetric relaxation time |
| IVCT | Isovolumetric contraction time |
| PCI | Percutaneous coronary intervention |
| LVESV | LV end-systolic volume |
| LVEDV | LV end diastolic volume |
| PASP | pulmonary artery systolic pressure |
| IVC | inferior vena cava |
| IWMI | Inferior wall myocardial infarction |
| CMR | Cardiac magnetic resonance |

## 2. Materials and Methods

### 2.1. Study population

Sixty-five consecutive patients with recent first acute inferior STEMI who underwent PPCI in Tanta University, Cardiology department were prospectively included in the study during the period from July 2018 till April 2019.

Informed consent was taken from all patients and the study was approved by the local ethical committee.

During the enrollment period, 80 consecutive patients screened for admission to the study.

#### 2.1.1. Inclusion criteria

(1) Confirmed first inferior STEMI based on the presence of typical angina pain more than 20 minutes, new ST-segment elevation at J point $\geq$ 0.1 mv in two or more inferior leads. The diagnosis was confirmed by the elevation in troponin levels. The presence of associated right ventricular myocardial infarction (RV-MI) was defined as ST-segment elevation $\geq$0.1 mv in lead V4R [8].
(2) Onset of symptoms <12 h before hospital admission.
(3) Patients with culprit proximal right coronary artery (RCA) at coronary angiography.

#### 2.1.2. Exclusion criteria

(1) Patients with poor echogenic window.
(2) Prior STMI.
(3) Documented pulmonary hypertension.
(4) Documented LV dysfunction.
(5) Atrial fibrillation.
(6) Significant valvular regurgitation or stenosis (moderate or severe).
(7) Chronic obstructive pulmonary disease (COPD).
(8) History of coronary artery bypass grafting.

For various reasons, 15 were not considered eligible: 5 patients had poor echocardiographic views, 2 patients had prior STEMI, 1 patient had atrial fibrillation, 2 patients had history of coronary artery bypass grafting, 1 patient had (COPD) and 4 others refused to take part in the research (Fig. 1).

### 2.2. Angiographic Procedure

Coronary angiography and percutaneous coronary intervention were done through the femoral or radial approach. All patients received the following regimen: (1) Ticagrelor 180 mg initial dose followed by a maintenance dose of 90 mg twice daily or clopidogrel 600 mg loading dose orally followed by maintenance dose of 75 mg/day if ticagrelor is contraindicated; (2) Aspirin 300 mg followed by 75-100 mg/day and (3)During the procedure patients received unfractionated heparin (100 IU/kg), the dose was reduced to (70 IU/kg) in case of administration of glycoprotein IIb/IIIa inhibitor (eptifibatide).

Thrombolysis in Myocardial Infarction flow (TIMI) rate [9] was assessed before and at the end PPCI also myocardial blush grade (MBG) [10] was assessed at the end of the procedure.

The use of manual thrombus aspiration was left upon the operator discretion.

On coronary angiography the site of occlusion of the RCA was defined based on the origin of first major (>1 mm) RV branch [11].

### 2.3. Echocardiographic evaluation

All patients underwent two dimensional transthoracic echocardiographic and Doppler studies using the commercially available GE Vivid 7 echocardiograph with 2.5 MHz transducer. Echocardiographic evaluation was performed at the time of discharge and at 3 months interval. LV ejection fractions (EF), LV end-systolic volume (LVESV), LV end diastolic volume (LVEDV) were evaluated (using biplane method of discs) [12].

Right atrial volume indexed to body surface area (RAVI) using the 4-chamber single-plane Simpson

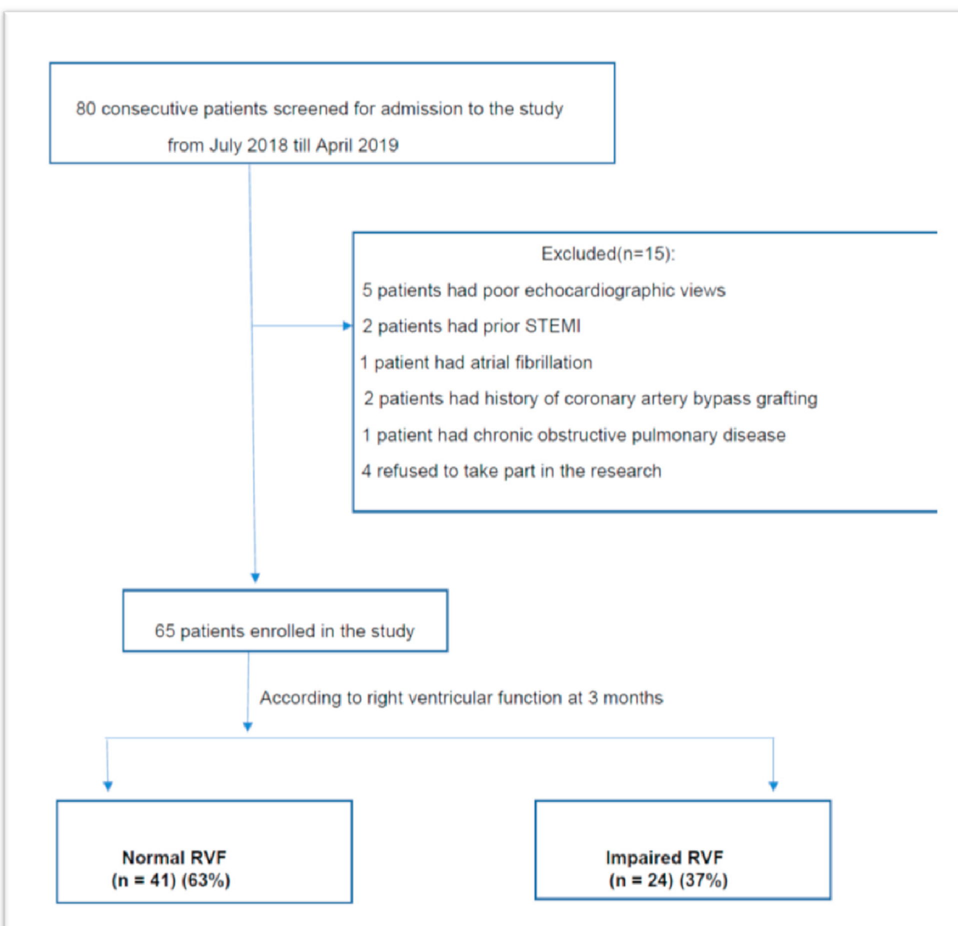


*Fig. 1. Flow chart of patient selection.*

method averaged over 5 consecutive cardiac cycles. An Apical 4 chamber view that includes the entire RA was used. The RA contours were traced on sequential images at end-ventricular systole. Foreshortening of the RA was carefully avoided. The RA appendage, coronary sinus, and the confluence of inferior vena cava were excluded [13] (Fig. 2).

From the trans-mitral flow profile, the E and A waves peak velocities were calculated. TDI of the mitral annulus was performed in the apical 4 chamber view using 1- to 2- mm sample volume placed in the sepal mitral valve annulus. The value of è was measured and E/è was obtained [12].

### 2.4. Evaluation of RV function

Tricuspid annular plane systolic excursion (TAPSE) was measured in the apical 4-chamber view by M-mode echocardiography with the cursor placed through the tricuspid lateral annulus and measuring the amount of longitudinal motion of the annulus at peak systole [12].

Right ventricular fractional area change (RVFAC) was defined as (RV end-diastolic area-RV end-systolic area)/RV end-diastolic area. The RV endocardium was traced both in systole and diastole along the free wall to the apex and then back to annulus along the inter-ventricular septum in the apical 4-chamber view [12].

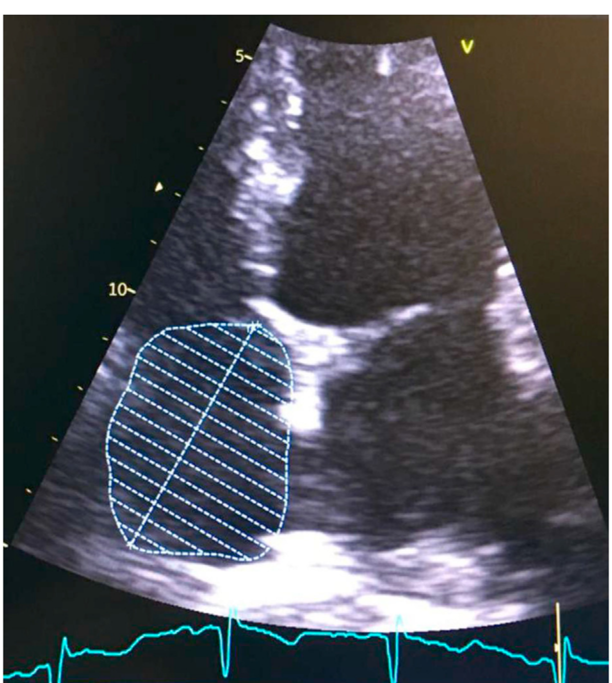


*Fig. 2. Measurement of right atrial volume in apical 4 chamber view.*

TDI derived RV- myocardial performance index (MPI) was calculated. Pulsed TDI analysis was obtained from apical 4-chamber view with TDI cursor placed at the level of tricuspid annulus. One major positive velocity (S′) was recorded with movement of the annulus towards the apex during systole. Two major negative waves were recorded, one during early diastole (E′) and one during late diastole (A′). (S′) duration was measured as ejection time. The time between the end of (S′) and beginning of (E′) was measured as isovolumetric relaxation time (IVRT). The time between the end of (A′) and beginning of (S′) was measured as isovolumetric contraction time (IVCT). MPI was calculated as (IVRT + IVCT)/ET [12].

Impaired right ventricular systolic function defined as presence of these three parameters together FAC <35%, TAPSE <17 mm and TDI derived RV- (MPI) > 0.54 [12].

The pulmonary artery systolic pressure (PSAP) was assessed by measuring the difference in pressures between the right ventricle and the right atrium using the peak velocity (Vmax) of the tricuspid regurgitation Continuous wave Doppler trace. The simplified Bernoulli equation (PSAP = 4(Vmax)2 + right atrial pressure) was used [14]. Right atrial pressure (RAP) is assumed by the size and distensibility of inferior vena cava (IVC) during respiration. IVC diameter 2.1 cm that collapsed >50% with a sniff indicated normal RA pressure of 3 mmHg, whereas IVC diameter >2.1 cm that collapsed <50% with a sniff suggested high RA pressure of 15 mmHg. If IVC diameter and collapse did not fit this paradigm, an intermediate value of 8 mmHg was used. Pulsed wave Doppler recordings of flow velocities of the hepatic vein flow was done using a sample volume placed in the hepatic vein 1 cm proximal to junction of IVC and hepatic veins and hepatic vein systolic/diastolic (S/D) ratio was calculated [5].

### 2.5. Reproducibility

All measurements were performed by an experienced echo cardiographer. Intraobserver and interobserver variability was assessed using intraclass correlation coefficients in 15 randomly selected patients by repeated analysis on the same cine loop by the same investigator (S.S) or independently by two separate investigators (M.N and S.S).

#### 2.5.1. Study endpoint definition

The primary end point was persistent of RV dysfunction (defined as presence of the following parameters together FAC < 35%, TAPSE < 17 mm and TDI derived RV- (MPI) > 0.54) after acute inferior STEMI due to occlusion of proximal RCA at 3- month.

### 2.6. Statistical study

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). The Kolmogorov- Smirnov test was used to verify the normality of distribution of variables; Comparisons between groups for categorical variables were assessed using Chi-square test (Fisher). Student t-test was used to compare two groups for normally distributed quantitative variables. Mann Whitney test was used to compare between two groups for not normally distributed quantitative variables. Univariate and multivariable logistic regression analyses were performed to identify predictors for persistently impaired RV function. Receiving operator characteristics (ROC) curve is used to detect optimal cut-off values of RAVI in predicting persistently impaired RV function. A p value < 0.05 is considered as statistically significant. In addition, power of the sample size was calculated by G Power tool (Franz Faul, University of Kiel, Germany, version 3.1.9.4) with 0.05 alpha and 0.8 effect size. The calculated power value was 0.86 according to post hoc-type power analysis.

## 3. Results

Sixty-five patients with first acute inferior wall STEMI, who were treated with PPCI, were included in the study.

In the 3 months after AMI, 41 of the 65 patients (63%) had normal right ventricular function (RVF) and were included in the normal RVF group. The remaining 24 (37%) patients had persistent RV dysfunction and were assigned to the impaired RVF group.

### 3.1. Baseline clinical characteristics

There were no statistically significant differences between both groups in respect to age, sex, hypertension, dyslipidemia, diabetes mellitus, smoking status, family history of premature coronary artery disease, HF history, New York Heart Association (NYHA) functional class, creatinine level, heart rate, hypotension at presentation, troponin level nor number of patients with ST-segment elevation in V4R. Both groups did not differ regarding major medications prescribed at discharge (Table 1).

*Table 1. Comparison between the studied groups according to baseline clinical characteristics.*

| | Normal RVF (n = 41) (63%) | Impaired RVF (n = 24) (37%) | p |
|---|---|---|---|
| Age (years) | 57.8 ± 8.7 | 54.6 ± 5.7 | 0.077 |
| Male | 30 (73.2%) | 16 (66.7%) | 0.578 |
| Hypertension | 21 (51.2%) | 13 (54.2%) | 0.818 |
| Dyslipidemia | 22 (53.7%) | 11 (45.8%) | 0.543 |
| Diabetes mellitus | 10 (24.4%) | 6 (25%) | 0.956 |
| Smoker | 24 (58.5%) | 15 (62.5%) | 0.753 |
| Family history of premature CAD | 8 (19.5%) | 5 (20.8%) | 1.000 |
| NYHA functional class | | | |
| NYHA I | 6 (14.6%) | 4 (16.7%) | 0.951 |
| NYHA II | 26 (63.4%) | 14 (58.3%) | |
| NYHA III | 5 (12.2%) | 4 (16.7%) | |
| NYHA IV | 4 (9.8%) | 2 (8.3%) | |
| Creatinine (mg/dl) | 1.1 ± 0.2 | 1.1 ± 0.2 | 0.502 |
| Heart rate (beat/min) | 77.2 ± 8.3 | 74.7 ± 9.6 | 0.266 |
| Hypotension—baseline (n %) | 9 (22%) | 6 (25%) | 0.778 |
| Peak troponin (ng/ml) | 3.5 ± 2.5 | 4 ± 2.5 | 0.138 |
| ST elevation in V4R | 26 (63.4%) | 14 (58.3%) | 0.684 |
| Major medications | | | |
| Beta- blocker | 31 (75.6%) | 20 (83.3%) | 0.465 |
| ACEI or ARB | 23 (56.1%) | 13 (54.2%) | 0.880 |
| Statin | 38 (92.7%) | 22 (91.7%) | 1.000 |
| Diuretics | 4 (9.8%) | 2 (8.3%) | 1.000 |
| Mineralocorticoid-receptor antagonist | 2 (4.9%) | 1 (4.2%) | 1.000 |

RVF = Right ventricular failure. ACEI = angiotensin-converting enzyme inhibitor. ARB = Angiotensin II receptor blocker.

### 3.2. Angiographic characteristics

There were no significant differences between both groups regarding number of diseased vessels, TIMI flow before percutaneous coronary intervention (PCI), TIMI flow after (PCI), reference vessel diameter, stent diameter, stent length, the time from symptom onset to PCI, the rate of use of thrombus aspiration device or rate of use of Glycoprotein IIb/IIIa inhibitors (Table 2).

### 3.3. Echocardiographic characteristics

#### 3.3.1. Acute phase (Table 3)

a. Left ventricle function: both groups did not differ with respect to LVESV, LVEDV, EF%, peak mitral E wave velocity, peak mitral A wave velocity, mitral E/e′ ratio and left atrial volume index (LAVI).

b. RV function: there were no significant differences between both groups regarding (PASP), TAPSE, MPI-TDI, RVFAC, peak tricuspid E wave velocity and peak tricuspid A wave velocity, Tricuspid E/è, RV- isovolumic relaxation time (IVRT), deceleration time, Hepatic vein systolic/diastolic ratio.

*Table 2. Comparison between the studied groups according to angiographic characteristics.*

| | Normal RVF (n = 41) (63%) | Impaired RVF (n = 24) (37%) | P |
|---|---|---|---|
| Number of diseased vessels | | | 0.869 |
| 1 | 9 (22%) | 4 (16.7%) | |
| 2 | 17 (41.5%) | 11 (45.8%) | |
| 3 | 15 (36.6%) | 9 (37.5%) | |
| TIMI flow before PCI | | | 0.778 |
| 0 | 32 (78%) | 18 (75%) | |
| 1 | 9 (22%) | 6 (25%) | |
| TIMI flow after PCI | | | 1.000 |
| 2 | 4 (9.8%) | 2 (8.3%) | |
| 3 | 37 (90.2%) | 22 (91.7%) | |
| RD | 3.1 ± 0.4 | 3.2 ± 0.3 | 0.392 |
| Stent diameter | 21.2 ± 5.4 | 22.2 ± 5.2 | 0.505 |
| Stent length | 3.1 ± 0.4 | 3.2 ± 0.3 | 0.681 |
| Time from symptom onset to PCI (mins) | 290.1 ± 121.5 | 240.6 ± 126.2 | 0.093 |
| Thrombus aspiration | 5 (12.2%) | 3 (12.5%) | 1.000 |
| GPIIb/IIIa | 19 (46%) | 10 (42%) | 0.134 |

RVF = Right ventricular failure. TIMI = Thrombolysis in Myocardial Infarction. PCI = percutaneous coronary intervention. RD = reference diameter. G IIb/IIIa inhibitors = glycoprotein IIb/IIIa inhibitor.

c. RAVI: patients in the impaired RV function group had higher RAVI (p=<0.001).

*3.3.2. 3-months follow up (Table 4)*

a) Left ventricle function: both groups did not differ with respect to LVESV, LVEDV, EF%, peak mitral E wave velocity, peak mitral A wave velocity, mitral E/e′ ratio and LAVI.

*Table 4. Comparison between the studied groups according to echocardiographic characteristics at 3-month follow up.*

| | Normal RVF (n = 41) (63%) | Impaired RVF (n = 24) (37%) | p |
|---|---|---|---|
| LVESV (ml) | 50.4 ± 15.5 | 44.5 ± 13.7 | 0.073 |
| LVEDV (ml) | 125.4 ± 24 | 115.7 ± 19.8 | 0.051 |
| EF % | 59.2 ± 5.4 | 61.2 ± 6.4 | 0.192 |
| LAVI | 28.3 ± 3.1 | 29.6 ± 2.9 | 0.100 |
| Peak E (m/s) | 0.7 ± 0.1 | 0.7 ± 0.1 | 0.665 |
| Peak A (m/s) | 0.8 ± 0.1 | 0.8 ± 0.1 | 0.628 |
| E/e′ ratio | 11.9 ± 1.8 | 11.3 ± 1.6 | 0.163 |
| PASP (mmhg) | 26.4 ± 6.6 | 29.1 ± 5.2 | 0.093 |
| TAPSE (mm) | 22 ± 4.7 | 14.8 ± 2.8 | <0.001* |
| MPI-TDI | 0.4 ± 0.1 | 0.6 ± 0.1 | <0.001* |
| RVFAC% | 38 ± 2.5 | 30 ± 3.1 | <0.001* |
| Tricuspid E (m/s) | 0.5 ± 0.1 | 0.5 ± 0.1 | 0.933 |
| Tricuspid A (m/s) | 0.4 ± 0.1 | 0.4 ± 0.1 | 0.190 |
| Tricuspid E/è | 4.2 ± 0.8 | 4.3 ± 0.7 | 0.704 |
| RV-IVRT (ms) | 82.99 ± 7.32 | 86.01 ± 3.47 | 0.063 |
| Deceleration time (ms) | 197.96 ± 5.85 | 200.47 ± 10.59 | 0.222 |
| Hepatic vein systolic/diastolic ratio | 1.3 ± 0.5 | 1.55 ± 0.6 | 0.076 |

RVF = Right ventricular failure. LVESV = Left ventricular end-systolic volume. LVEDV = Left ventricular end-diastolic volume. EF%, Ejection fraction; E: peak flow velocity during the early rapid filling phase; A: peak flow velocity during atrial contraction. E/è, the ratio of early flow velocity to the early annular velocity. PASP = pulmonary artery systolic pressure. TAPSE = Tricuspid annular plane systolic excursion. MPI = Myocardial performance index. TDI = Tissue Doppler imaging. RVFAC = Right ventricular fractional area change. IVRT= Isovolumetric relaxation time.

b) RV function: there was no significant difference between both groups regarding PASP, peak tricuspid E wave velocity, peak tricuspid A wave velocity and RV- isovolumic relaxation time (IVRT),

*Table 3. Comparison between the studied groups according to echocardiographic characteristics acute phase.*

| | Normal RVF (n = 41) (63%) | Impaired RVF (n = 24) (37%) | p |
|---|---|---|---|
| LVESV (ml) | 47.4 ± 11.2 | 42.5 ± 10 | 0.078 |
| LVEDV (ml) | 120.9 ± 18.6 | 113.5 ± 12.3 | 0.086 |
| EF % | 60.8 ± 6.2 | 62 ± 6.4 | 0.467 |
| LAVI | 26.8 ± 2.1 | 27.9 ± 2.7 | 0.072 |
| Peak E (m/s) | 0.7 ± 0.1 | 0.7 ± 0.1 | 0.928 |
| Peak A (m/s) | 0.8 ± 0.1 | 0.8 ± 0.1 | 0.821 |
| E/e′ ratio | 11.4 ± 1.6 | 12.2 ± 2.2 | 0.078 |
| PASP (mmhg) | 29.7 ± 5.3 | 27.3 ± 5.2 | 0.078 |
| TAPSE (mm) | 18 ± 3.5 | 17 ± 3.5 | 0.291 |
| MPI-TDI | 0.5 ± 0.1 | 0.5 ± 0.2 | 0.995 |
| RVFAC % | 35.3 ± 5.1 | 34.2 ± 4.5 | 0.385 |
| Tricuspid E (m/s) | 0.5 ± 0.1 | 0.5 ± 0.1 | 0.845 |
| Tricuspid A (m/s) | 0.4 ± 0.1 | 0.4 ± 0.1 | 0.694 |
| Tricuspid E/è | 4.5 ± 0.8 | 4.1 ± 0.9 | 0.055 |
| RV-IVRT (ms) | 95.25 ± 19.22 | 97.31 ± 4.37 | 0.608 |
| Deceleration time (ms) | 191.89 ± 8.95 | 196.22 ± 10.9 | 0.088 |
| Hepatic vein systolic/diastolic ratio | 1.15 ± 0.5 | 1.4 ± 0.7 | 0.099 |
| RAVI (ml/m$^2$) | 25.6 ± 3.2 | 37 ± 2.2 | <0.001* |

RVF = Right ventricular failure. LVESV = Left ventricular end-systolic volume. LVEDV = Left ventricular end-diastolic volume. EF%, Ejection fraction; E: peak flow velocity during the early rapid filling phase; A: peak flow velocity during atrial contraction. E/è, the ratio of early flow velocity to the early annular velocity. PASP = pulmonary artery systolic pressure. TAPSE = Tricuspid annular plane systolic excursion. MPI = Myocardial performance index. TDI = Tissue Doppler imaging. RVFAC = Right ventricular fractional area change. IVRT= Isovolumetric relaxation time. RAVI = Right atrial volume index.

*Table 5. Univariate and multivariate logistic regression analysis for predicting impaired RV function.*

| | Univariate | | Multivariate | |
|---|---|---|---|---|
| | OR (95%C.I) | p | OR (95%C.I) | p |
| Age (years) | 0.113 | 0.946 (0.883 − 1.013) | 0.367 | 0.931 (0.798 − 1.087) |
| Male | 0.656 | 0.889 (0.485 − 1.112) | 0.789 | 0.863 (0.474 − 1.101) |
| Time from symptom onset to PCI (mins) | 0.126 | 0.997 (0.992 − 1.001) | 0.191 | 0.993 (0.982 − 1.004) |
| EF % | 0.461 | 1.031 (0.951 − 1.118) | 0.444 | 0.933 (0.782 − 1.114) |
| RAVI (ml/m$^2$) | <0.001 | 1.786 (1.367 − 2.335) | <0.001 | 1.829 (1.358 −2.462) |

C.I, Confidence interval. OR: Odd's ratio. EF%, Ejection fraction. RAVI = Right atrial volume index.

deceleration time, Hepatic vein systolic/diastolic ratio. TAPSE, MPI-TDI and RVFAC were significantly better in normal RVF group. P value= (<0.001).

Univariate and multivariable logistic regression analyses to assess predictor of persistently impaired RV function included age, sex, time from symptom onset to PCI, EF% and RAVI. The results showed that RAVI was the only factor that predicted persistently impaired RV function (odds ratio = 1.786, 95% confidence interval, 1.367−2.335, p value= <0.001) and (odds ratio = 1.829, 95% confidence interval, 1.358 −2.462, p value= <0.001) in univariate and multivariable logistic regression analyses respectively (Table 5).

In ROC curve analysis, RAVI with a cutoff value ≥ 30 ml/m$^2$ had a 87.5% sensitivity, a 92.24% specificity area under ROC curve = 0.964 for predicting persistently impaired RV function (Fig. 3).

### *3.4. Reproducibility*

Intraobserver and interobserver variability for conventional two-dimensional/Doppler measurements and TDI-derived parameters ranged from 0.95 and 0.98 and 0.92 and 0.95 respectively.

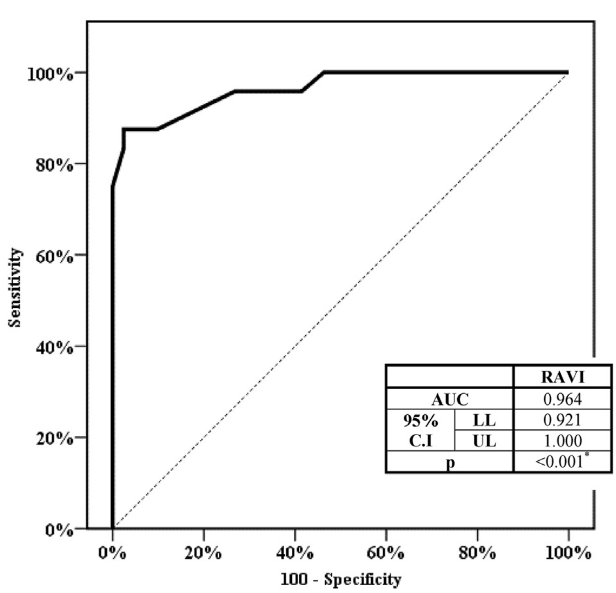


*Fig. 3. Receiver operating curve characteristic (Roc) curve analyses for RAVI as predictor for impaired RVF.*

## 4. Discussion

The present study evaluated the ability of RAVI to predict persistent RV dysfunction in patients with inferior STEMI with proximal RCA occlusion.

The main findings of the present study were: (1) Persistent RV dysfunction occurred in (37%) patients. (2) RAVI is an independent predictor of persistent RV dysfunction. The cutoff value of RAVI ≥30 ml/m$^2$ had a 87.5% sensitivity, a 92.24% specificity for predicting persistently impaired RV function.

The importance of predicting persistence RV dysfunction came from the fact that persistent RV dysfunction is associated with long term poor prognosis irrespective of left ventricle (LV) systolic function[15] [16]. Liao H et al, compared in-hospital outcomes between left ventricular myocardial infarction patients with and without right ventricular myocardial infarction in 458 patients with acute (STEMI) undergoing (PPCI). They reported that patients with concomitant RVMI have higher of in-hospital complications, particularly all-cause mortality and new onset acute HF. [17] .

The occlusion of proximal RCA in patients with IWMI is associated with more worse RV function indices compared to patients without proximal RCA occlusion[1]. This raises the importance of precisely assessing the RV function in such patients. For long time the assessment of the cardiac function was all about the left ventricle and the right ventricle was the forgotten chamber. Nowadays the importance of the right ventricle as a prognostic marker in different cardiovascular diseases is well accepted [18].

Although different imaging modalities are available nowadays to assess both RV function and structure, still echocardiography is the most used imaging technique due to its wide availability and cost issues especially in the developing countries. The standard techniques of echocardiography which are recommended by the current guidelines to evaluate RV function, such as 2D, Doppler and M mode, are faced by the complex geometry of the right ventricle and difficulty in obtaining accurate and clear images in some patients[19]. The new techniques such as speckle tracking and 3D are still limited by many concerns making them difficult to use in every day practice[20]. In comparison to the right ventricle, the right atrium is easy to visualize and assess allowing quantitative and highly reproducible calculation of RAVI.

Sallach et al. evaluated the relationship between (RAVI) and (RV) systolic and diastolic function, as well as long-term prognosis in patients with chronic systolic (HF). RAVI was an independent predictor of long-term adverse clinical events. RAVI $\geq$ 30.6 ml/m2 (optimal ROC cutoff) had a 78% sensitivity and a 77% specificity ($p < 0.0001$) for predicting RV systolic dysfunction stage $\geq$ 3. This cutoff is similar to that found in the present study, however we used RAVI as a marker of persistently impaired of RV dysfunction at 3 months follow up and in different patients population which are acute inferior STEMI and RV involvement based on coronary angiographic findings[6].

Ivanov A et al, evaluate the predictive value of RAVI assessed by cardiac magnetic resonance (CMR) for all-cause mortality in 243 patients with HFrEF (LVEF< 35% measured by CMR). They concluded that RAVI measured by CMR imaging is an independent predictor of mortality in patients with heart failure with reduced ejection fraction [21]. Alexandre Altes et al examined the relation between RAVI and long term mortality of patient with systolic HF who received cardiac resynchronization therapy, they included 172 patients and they found that for every 1 mL/m2 increase in RAVI the risk of death was increasing $p = (0.042)$ and patients in the highest tertile (RAVI >29 mL/m2) had significantly higher risk of death compared with those with RAVI $\leq$29 mL/$m^2$ $p = (0.014)$ [22]. The RAVI value in the last 2 studies which predict poor outcome is close to our finding that RAVI$\geq$30 ml/$m^2$ predicts persistent RV dysfunction after acute IWMI due to proximal RCA occlusion.

## 5. Conclusion

The findings of the present study suggest that RAVI may be a useful predictor of persistently impaired RVF in patients with inferior STEMI with proximal RCA occlusion with a cut-off value $\geq$ 30 ml/$m^2$. The clinical application of these findings requires larger studies to test drugs that target the RV with the aim of preventing RV dysfunction. Also, further studies are required to determine the impact of these findings on the clinical outcome e.g.: re-hospitalization and or death.

## Author contributions

Conception and design of Study: Mohamed Naseem, Sameh Samir. Literature review: Mohamed Naseem. Acquisition of data: Sameh Samir. Analysis and interpretation of data: Mohamed Naseem, Sameh Samir. Drafting of manuscript: Mohamed Naseem, Sameh Samir. Revising and editing the manuscript critically for important intellectual contents: Mohamed Naseem, Sameh Samir.

## Disclosure

Authors have nothing to disclose with regard to commercial support

## Conflict of interest

The authors declare that there is no conflict of interest.

## References

[1] Albulushi A, Giannopoulos A, Kafkas N, Dragasis S, Pavlides G, Chatzizisis YS. Acute right ventricular myocardial infarction. Expert Rev Cardiovasc Ther 2018;16:455–64. https://doi.org/10.1080/14779072.2018.1489234.
[2] Crystal GJ, Pagel PS. Right Ventricular Perfusion: Physiology and Clinical Implications. Anesthesiology 2018;128:202–18. https://doi.org/10.1097/ALN.0000000000001891.
[3] García-Niebla J, Pérez-Riera AR, Barbosa-Barros R, Díaz-Muñoz J, Daminello-Raimundo R, de Abreu LC, et al. Acute inferior myocardial infarction with right ventricular involvement and several clinical-electrocardiographic markers of poor prognosis. Ann Noninvasive Electrocardiol 2019;24:4–7. https://doi.org/10.1111/anec.12592.
[4] Gul I, Zungur M, Aykan AC, Gokdeniz T, Alkan MB, Sayin A, et al. The change in right ventricular systolic function according to the revascularisation method used. following acute ST-segment elevation myocardial infarction 2016;27:37–44. https://doi.org/10.5830/CVJA-2015-077.
[5] Darahim K. Usefulness of right atrial volume index in predicting outcome in chronic systolic heart failure. J Saudi Hear Assoc 2014;26:73–9. https://doi.org/10.1016/j.jsha.2013.09.002.
[6] Sallach JA, Tang WHW, Borowski AG, Tong W, Porter T, Martin MG, et al. Right Atrial Volume Index in Chronic Systolic Heart Failure and Prognosis. JACC Cardiovasc Imaging 2009;2:527–34. https://doi.org/10.1016/j.jcmg.2009.01.012.
[7] Article O. Right Atrium Volume Index in Patients with Secondary Pulmonary Hypertension Due to Chronic Obstructive. Pulmonary Disease 2015;I:325–36. https://doi.org/10.6515/ACS20150119A.
[8] Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol 2018;72:2231–64. https://doi.org/10.1016/j.jacc.2018.08.1038.
[9] Yildiz M, Henry TD. Preprocedure Thrombolysis In Myocardial Infarction (TIMI) flow grade: Has its time come

and gone? Catheter Cardiovasc Interv 2020;95:501−2. https://doi.org/10.1002/ccd.28770.

[10] Sharma V, Jolly SS, Hamid T, Sharma Di, Chiha J, Chan W, et al. Myocardial blush and microvascular reperfusion following manual thrombectomy during percutaneous coronary intervention for ST elevation myocardial infarction: Insights from the TOTAL trial. Eur Heart J 2016;37:1891−8. https://doi.org/10.1093/eurheartj/ehw157.

[11] Pourafkari L, Tajlil A, Mahmoudi SS, Ghaffari S. The Value of Lead aVR ST Segment Changes in Localizing Culprit Lesion in Acute Inferior Myocardial Infarction and Its Prognostic Impact. Ann Noninvasive Electrocardiol 2016;21: 389−96. https://doi.org/10.1111/anec.12324.

[12] Lang RM, Badano LP, Victor MA, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1−39. https://doi.org/10.1016/j.echo.2014.10.003. e14.

[13] Ebtia M, Murphy D, Gin K, Lee PK, Jue J, Tsang TSM. Best Method for Right Atrial Volume Assessment by Two-Dimensional Echocardiography: Validation with Magnetic Resonance Imaging Echocardiography. Epub 2014 Sep 18. PMID: 25231096. https://doi.org/10.1111/echo.12735 2015 May;32(5):734−9. https://doi.org/10.1111/echo.12735.

[14] Parasuraman S, Walker S, Loudon BL, Gollop ND, Wilson AM, Lowery C, et al. Assessment of pulmonary artery pressure by echocardiography-A comprehensive review. IJC Hear Vasc 2016;12:45−51. https://doi.org/10.1016/j.ijcha.2016.05.011.

[15] Monitillo F, Di Terlizzi V, Gioia MI, Barone R, Grande D, Parisi G, et al. Right Ventricular Function in Chronic Heart Failure: From the Diagnosis to the Therapeutic Approach. J Cardiovasc Dev Dis 2020;7:12. https://doi.org/10.3390/jcdd7020012.

[16] Shahar K, Darawsha W, Yalonetsky S, Lessick J, Kapeliovich M, Dragu R, et al. Time Dependence of the Effect of Right Ventricular Dysfunction on Clinical Outcomes After Myocardial Infarction: Role of Pulmonary Hypertension. J Am Heart Assoc 2016;5:1−9. https://doi.org/10.1161/JAHA.116.003606.

[17] Liao H, Chen Q, Liu L, Zhong S, Deng H, Xiao C. Impact of concurrent right ventricular myocardial infarction on outcomes among patients with left ventricular myocardial infarction. Sci Rep 2020;10:1−6. https://doi.org/10.1038/s41598-020-58713-0.

[18] Lee JH, Park JH. Strain analysis of the right ventricle using two-dimensional echocardiography. J Cardiovasc Imaging 2018;26:111−24. https://doi.org/10.4250/jcvi.2018.26.e11.

[19] Jones N, Burns AT, Prior DL. Echocardiographic Assessment of the Right Ventricle-State of the Art. 2019. https://doi.org/10.1016/j.hlc.2019.04.016.

[20] Longobardo L, Suma V, Jain R, Carerj S, Zito C, Zwicke DL, et al. Role of Two-Dimensional Speckle-Tracking Echocardiography Strain in the Assessment of Right Ventricular Systolic Function and Comparison with Conventional Parameters. J Am Soc Echocardiogr 2017;30:937−46. https://doi.org/10.1016/j.echo.2017.06.016. e6.

[21] Ivanov A, Mohamed A, Asfour A, Ho J, Khan SA, Chen O, et al. Right atrial volume by cardiovascular magnetic resonance predicts mortality in patients with heart failure with reduced ejection fraction. 2017. p. 1−13.

[22] Altes A, Appert L, Delelis F, Guyomar Y, Menet A, Ennezat PV, et al. Impact of Increased Right Atrial Size on Long-Term Mortality in Patients With Heart Failure Receiving Cardiac Resynchronization Therapy. Am J Cardiol 2019;123:936−41. https://doi.org/10.1016/j.amjcard.2018.12.015.