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ORIGINAL ARTICLE

Evaluation of right ventricular function in patients undergoing coronary intervention and presenting with non-ST-segment elevation myocardial infarction (NSTEMI)

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ABSTRACT

Background: Following an acute myocardial infarction (MI), right ventricular (RV) affection had been linked to increased morbidity and death. In individuals with acute myocardial ischemia, RV dysfunction has been identified as a predictor of death. The aim of this work is to evaluate the right ventricular function in patients undergoing coronary intervention and presenting with non-ST-segment elevation myocardial infarction (NSTEMI).

Methods: The study was a case control study that included 44 individuals over the age of 18 who were hospitalized or referred to Zagazig University Hospital and National Heart Institute with manifestations of NSTEMI between 2019 and 2020. Patients were split into two groups based on their RV function: Group I (n = 22 patients) had normal RV function, and Group II (n = 22 patients) had impaired RV function. All patients were subjected to complete history, full clinical examination, ECG, echocardiography to assess RV function, laboratory investigations and PCI then follow up for three months to evaluate improvement in RV function.

Results: There was a statistically significant negative correlation between baseline TAPSE and peak Tpn and RVEDD. There was also a statistically high significant positive correlation between baseline TAPSE and RVFAC. In addition, there was a statistically high significant negative correlation between RVFAC and RVEDD with p value ≤ 0.001 .

Conclusion: Patients with NSTEMI and poor RV function should be treated by PCI revascularization of the culprit lesion to enhance RV function, and it should be assessed by ECG and 2D echocardiography.

Key words: PCI, NSTEMI, CAD.

INTRODUCTION

Increased levels of cardiac enzymes and indicators of myocyte necrosis differentiate non-ST-segment elevation myocardial infarction (NSTEMI) from unstable angina. Following an acute myocardial infarction (MI),

right ventricular (RV) affection had been linked to increased morbidity and death. [1]

In postmortem and animal investigations, the frequency of RV involvement in acute MI had been reported to vary from fifty to eighty percent, although it is usually overestimated in

clinical settings due to the diagnostic difficulties of the electrocardiogram (ECG) and also the echocardiography. [2]

In individuals with acute myocardial ischemia, RV dysfunction has been identified as a predictor of death. Furthermore, severe RV dysfunction was associated with substantial Right Coronary Artery (RCA) stenosis proximal to the main blood supplying RV branches. [3]

In addition, RV involvement can be detected in heart failure caused by chronic myocardial ischemia, and Venner and colleagues revealed that RV dysfunction is more apparent in cases of chronic myocardial ischemia using the TDI technique to examine RV function on the long axis.[4]

The aim of this work is to evaluate the right ventricular function in patients undergoing coronary intervention and presenting with non-ST-segment elevation myocardial infarction (NSTEMI).

METHODS

Technical design: The study was a case control study that included 44 patients over the age of 18 who were hospitalized or referred to Zagazig University Hospital and National Heart Institute with manifestations of NSTEMI between 2019 and 2020. Patients were split into two groups based on their RV function: Group I (n = 22 patients) had normal RV function, and Group II (n = 22 patients) had impaired RV function. Patients with ST segment elevation myocardial infarction, prior coronary revascularization (PCI or CABG), or a concomitant clinical condition that might impair RV function, such as pericardial illness, chronic lung disease, pulmonary hypertension, or connective tissue problem, were excluded. The participants in our study were also excluded if they had moderate or severe valvular heart disease or atrial fibrillation. After excluding non-responders, dropouts, and those who met exclusion criteria, the study was completed by 44 patients (this number was considered suitable enough sample for statistical analysis with significant results and correlations).

Methods: All patients underwent a thorough medical history, physical examination, ECG, and echocardiography to assess RV function, as well as laboratory tests such as CBC, coagulation profile, kidney function tests, liver enzymes, HbA1C, CKMB, and high sensitivity troponin, as well as PCI. They were then followed for three months to see if their RV function had improved.

Administrative considerations: Written informed consent was obtained from all participants after clear explanation of the study and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University (Institutional Research Board IRB). The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

STATISTICAL ANALYSIS

Statistical analysis was performed using Statistical Package for The Social Sciences Version 22 (IBM Corp., Armonk, NY, USA). Quantitative data are expressed as means and standard deviations. P-Value ≤ 0.05 was considered to indicate significance. Correlation analysis assesses the strength of association between two variables. Qualitative data were described using number and percent. Association between categorical variables was tested using Chi-square test. Continuous variables were presented as mean \pm SD (standard deviation) for normally distributed data and median (min-max) for non-normal data. The two groups were compared with Student t test for normal data and Mann Whitney test for non-normal data. Fischer exact test and Monte Carlo tests were also used. Pearson correlation (parametric) and Spearman correlation (non-parametric) was used to correlate continuous data.

RESULTS

Table (1) reveals the medical history among Normal and Depressed RV function groups. There was a statistically non-significant difference regarding HF, PVD and stroke between both groups p values were 1 for all. The clinical data of the two groups was showed

in **Table (2)**. Regarding the clinical data, weight, height, BMI and SBP, there was a statistically non-significant difference between both groups with p values 0.811, 0.385, 0.457 and 0.843 respectively. There was a statistically non-significant increase in DBP in group I compared to group II with p values 0.379. Regarding the ECG data, there was a non-significant increase in heart rate in group I compared to group II, p values 0.169 as demonstrated in **Table (3)**. There was a statistically non-significant difference in ST segment abnormalities and T wave abnormalities between both groups p value 0.629 and 0.248 respectively. **Table (4)** shows that there is a statistically non-significant difference in number of stents and predilation in group I as compared to group II. There is a statistically non-significant difference in medications in group I as compared to group II as demonstrated in **Table (5)**. As regarding patients' outcome, **Table (6)** revealed that there was a statistically significant difference in

cardiogenic shock between both groups. There was also a statistically non-significant decrease in stroke, TIA, mechanical support, and death in group I as compared to group II.

Table (7,8) demonstrate the there was a statistically significant negative correlation between baseline TAPSE and peak Tpn and RVEDD with p values 0.017 and 0.001 respectively. There was also a statistically high significant positive correlation between baseline TAPSE and RVFAC with p value ≤ 0.001 . In addition, there was a statistically high significant negative correlation between RVFAC and RVEDD with p value ≤ 0.001 Mean while for LVEF, it has non-significant difference between the two groups with p value 0.616.

Table (9) shows the laboratory investigations among the two groups; HTC, RBCs, WBCs, Platelets, HB and serum creatinine there was non-significant difference with p values 0.824, 0.082, 0.628, 0.201, 0.833 and 0.690.

Table (1): Medical history among both groups.

Medical history	Group 1 (n=22)	Group 2 (n=22)	P value
HF	3 (13.6%)	4 (18.2%)	1.00
PVD	3 (13.6%)	3 (13.6%)	1.00
Stroke	3 (13.6%)	4 (18.2%)	1.00

HF heart failure, PVD peripheral vascular disease,

Table (2): Clinical data both groups.

Index events	Group 1 (n=22)	Group 2 (n=22)	Test of significance	P value
Height/m	1.67±0.08	1.68±0.08	t=-0.241	0.811
Weight/kg	90.86±13.24	94.36±13.19	t=-0.878	0.385
BMI	32.37±4.43	33.48±5.36	t=-0.750	0.457
SBP	122.45±18.43	123.50±16.32	t=-0.199	0.843
DBP	84.09±13.45	81.04±8.80	t=0.888	0.379

BMI body mass index, DBP diastolic blood pressure SBP systolic blood pressure

Table (3): ECG among both groups.

ECG	Group 1 (n=22)	Group 2 (n=22)	Test of significance	P value
Rate:	111.14±19.48	102.59±20.97	t=1.40	0.169
ST depression.:			MC	0.629

Isoelectric	7 (31.8%)	9 (40.9%)		
Anterior	7 (31.8%)	5 (22.7%)		
Inferior	3 (13.6%)	3 (13.6%)		
Anterolateral.	2 (9.1%)	2 (9.1%)		
Anteroseptal	1 (4.5%)	1 (4.5%)		
Septal	2 (9.1%)	1 (4.5%)		
v1-v6	0 (0%)	1 (4.5%)		
T wave inversion:				
NAD	9 (40.9%)	9 (40.9%)		
Anterior	5 (22.7%)	4 (18.2%)		
Inferior	3 (13.6%)	2 (9.1%)		
Lateral	3 (13.6%)	1 (4.5%)		
Septal	2 (9.1%)	1 (4.5%)		
v1-v6	0 (0%)	5 (22.7%)	MC	0.248

Table (4): PCI strategy among both groups.

PCI strategy	Group 1 (n=22)	Group 2 (n=22)	Test of significance	P value
No of Stents				
1	15 (68.2%)	14 (63.6%)		
2	7 (31.8%)	7 (31.8%)	MC	0.596
3	0 (0%)	1 (4.5%)		
Predilation	6 (27.3%)	6 (27.3%)	$\chi^2=0$	1.000

Table (5): Pre-study medications among both groups.

Medications	Group 1 (n=22)	Group 2 (n=22)	P value
No medications	5 (22.7%)	8 (36.4%)	
BB	8 (36.4%)	7 (31.5%)	
ACEi	5 (22.7%)	3 (13.5%)	0.636
CCB	3 (13.5%)	2 (9%)	
Metformin	3 (13.5%)	2 (9%)	
Insulin	4 (18%)	5 (22.5%)	
ARBs	2 (9.1%)	1 (4.5%)	
Statin	4 (18%)	2 (9.1%)	
Diuretic	1 (4.5%)	0 (0.0%)	

BB beta blocker, ACEI angiotensin-converting enzyme inhibitor, CCB calcium channel blocker, ARBs Angiotensin II receptor blocker **Table (6):** Outcome among both groups.

Outcome	Group 1 (n=22)	Group 2 (n=22)	P value
Cardiogenic shock	2 (9.1%)	8 (36.4%)	0.031*
Stroke	0 (0%)	3 (13.6%)	0.233
Bleeding	0 (0%)	0 (0%)	-
TIA	0 (0%)	1 (4.5%)	1.00
Mechanical support	1 (4.5%)	3(13.6%)	0.607
Outcome			0.132

Improved	20 (90.9%)	15 (68.2%)	
	Died	2 (9.1%)	7 (31.8%)

TIA transient ischaemic attack

Table (7): Correlation between baseline TAPSE, RVFAC and other variables.

	Baseline TAPSE		RVFAC	
	r	p	r	p
Peak Tpn	-0.359	0.017*	-0.098	0.529
Rate	0.152	0.324	0.264	0.083
LVEF	0.048	0.756	-0.146	0.345
RVEDD	-0.479	0.001*	-0.552	≤0.001*
RVFAC	0.580	≤0.001*	-	-
# of Stents	-0.196	0.203	0.090	0.561

Tpn troponin LVED left ventricular ejection fraction, RVEDD right ventricular end-diastolic dimension

RVFAC Right ventricular fractional area change TAPSE tricuspid annular plane systolic excursion

Table (8): ECHO findings among both groups

ECHO	Group 1 (n=22)	Group 2 (n=22)	Test of significance	P value
LVEF	53.77±6.78	52.72±6.94	t=0.505	0.616
EDD	24.81±4.54	29.31±4.46	t=3.31	0.002*
RVFAC	40.45±5.38	30.59±6.16	t=5.65	≤0.001*

LVEF left ventricular ejection fraction, RVFAC Right ventricular fractional area change, EDD End diastolic dimension, ECHO Echocardiogram

Table (9): Laboratory investigations among both groups

Laboratory investigations	Group 1 (n=22)	Group 2 (n=22)	Test of significance	P value
HCT	31.36±2.61	31.18±2.78	t=0.223	0.824
RBCs	4.67±0.44	4.38±0.62	t=1.782	0.082
WBCs	6.99±1.57	7.28±2.24	t=0.487	0.628
PLT	333.32±69.10	306.77±66.49	t=1.298	0.201
HB	11.90±2.08	11.77±2.02	t=0.212	0.833
Creatinine Median (Min-Max)	1.09 (0.47-5.32)	1.12 (0.62-5.22)	Z=0.399	0.690
Peak Tpn	2.20±0.97	3.01±0.87	t=2.930	0.005*

WBC, White blood cell,. Tpn troponin, RBCs red blood cells PLT platelets, HB haemoglobin,

DISCUSSION

In ST segment elevation myocardial infarction (STEMI), right ventricular affection increases morbidity and death. However, there is very little information on its effects in non-ST

segment elevation myocardial infarction (NSTEMI). [5]

Right Ventricular (RV) dysfunction is considered as a predictor for mortality in acute myocardial ischemia patients. Additionally, significant Right Coronary Artery (RCA)

stenosis proximal to the main blood supplying RV branches was associated by severe RV dysfunction. [6]

Our study aimed at evaluation the right ventricular function in patients undergoing coronary intervention and presenting with non-ST-segment elevation myocardial infarction (NSTEMI).

Our results regarding the clinical data among the two groups regarding height, weight, BMI, SBP and DBP there was non-significant difference with p values 0.811, 0.385, 0.457, 0.843 and 0.379. Assali and colleagues agreed with our results as regarding SBP which had no significant correlation between the two groups with p value (0.1). [7]

In the same line, Elserafy and colleagues, were concordant with our results and found no relation between the two groups as regarding obesity represented by BMI. [5]

Our study regarding medications between the two groups, 36.4% taking BB, 22.7% taking ACEi, 13.5% taking CCB, 13.5% taking metformin, 18% taking insulin, 9.1% taking ARBs, 18% taking statins and 4.5% taking diuretics in group I. While for group II, 31.5% taking BB, 13.5% taking ACEi, 9% taking CCB, 9% taking metformin, 22.5% taking insulin, 4.5% taking ARBs, 9.1% taking statins and 0% took diuretics, with non-significant difference between the two groups, p value 0.636. In a study by El-Adawy and colleagues, they stated that all patients were medicated on aspirin, 12 (24%) patients on beta-blockers, 23 (46%) on angiotensin converting enzyme inhibitors and 12 (24%) of them on statin therapy. [8]

In addition, Hoogslag and colleagues found a significant correlation between the two same groups regarding ACEi with p value 0.03 which was discordant with our results. [9]

Our study found regarding the ECG data among the two groups; rate had non-significant difference with p values 0.169. Also, for the ECG changes between the two groups as regarding ST segment abnormalities and T wave abnormalities there was non-significant difference with p value 0.629 and 0.248

respectively. According to Mehta and colleagues, in patients with RV myocardial involvement, the incidence of all serious arrhythmic complications was significantly higher than in patients without RV myocardial involvement, with a significant p value for ventricular fibrillation, sustained ventricular tachycardia, and AV lock, these results was discordant with ours as all our patients had sinus rhythm. [10]

Our results found as regarding coronary angiography data regarding number of stents between the two group 68.2% had one stent, 31.8% had two stents and 0% had three stents in normal RV function group, while 63.6% had one stent, 31.8% had two stents and 4.5% had three stents in depressed RV function group with no significant difference p value 0.596. Also, balloon predilatation was done in 27.3% in both groups non-significant between the two groups with p value 1.

Elserafy and colleagues stated that patients with normal TAPSE who underwent predilatation were twenty-four (41.4%), patients with abnormal TAPSE who underwent predilatation were nineteen (20.7%) which was discordant to our results. Meanwhile, 58.6% had one stent, 39.7% had two stents and 1.7% had three stents in normal RV function group, while 70.7% had one stent, 29.3% had two stents and 0% had three stents in depressed RV function group, this was in agreement with us. [5]

Our study stated that patients' outcome between the two groups, 2 had cardiogenic shock in group I and 8 in group II with statistically significant difference between them p value 0.031. Stroke, TIA, mechanical support, and death had non-significant correlation between the two groups with p values 0.233, 1, 0.607 and 0.132 respectively. Mehta and colleagues agreed with our results and stated that in individuals with RV myocardial involvement, there was also a trend toward greater incidence of cardiogenic shock when compared to those without it (OR 1.3, 95% CI 0.8 to 2.1) with significant p value. [10]

Also, in terms of cerebrovascular stroke, respiratory failure, and mortality, Assali and colleagues discovered no significant differences between the two groups. which agreed with our results, with only disagreement with us regarding cardiogenic shock with non-significant p value. [7]

Our results found that there is a statistically significant negative correlation between baseline TAPSE and RVEDD, while there is a positive correlation between it and RVFAC with p value ≤ 0.001 . Meanwhile for RVFAC, it had a negative correlation with RVEDD with p value ≤ 0.001 . A study by Hoogslag and colleagues, found a significant univariate correlates of RV dysfunction were multivessel coronary disease, peak cardiac troponin T level, LVEF, and TAPSE. In the multivariate model, the same parameters were independently associated with RV dysfunction at 6 months' follow-up. [9]

CONCLUSION

Patients with NSTEMI and poor RV function should be treated by PCI revascularization of the culprit lesion to enhance RV function, and it should be assessed by ECG and 2D echocardiography, which should be done and not overlooked. All of these individuals should be closely monitored for improvements in RV function.

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