Assessment of right ventricular function after successful revascularization for acute anterior myocardial infarction without right ventricular infarction by echocardiography

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Background: Right ventricular (RV) involvement in acute left ventricular (LV) myocardial infarction (MI) is frequently underestimated in the clinical setting owing to the diagnostic limitations of the electrocardiogram and echocardiography.

Objective: To assess RV function in patients presented with first acute anterior ST elevation myocardial infarction (STEMI) who underwent successful primary percutaneous coronary intervention (PCI) and factors affecting it.

Methods: Forty consecutive patients with anterior STEMI who underwent successful primary PCI were enrolled in the study. Presence of a coexisting clinical condition that might affect RV function, patients with RV infarction or those having significant stenosis (>50%) affecting RV branch or right coronary artery proximal to RV branch were excluded. Echocardiography was performed during the hospital stay to assess the LV and RV systolic and diastolic function with special focus on tricuspid annular plane systolic excursion, RV end-diastolic dimension, right atrial area, RV fractional area change, and tissue Doppler-derived myocardial performance index.

Results and Conclusion: RV dysfunction according to our definition in the first anterior MI occurred in (55%) of the study population. Independent predictors for abnormal RV function were left circumflex artery mid or proximal affection, eventful procedure, occurrence of no reflow, glucose level, LV end-systolic dimension, LV end-diastolic dimension, and LV ejection fraction.

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1. Introduction

Right ventricular (RV) involvement after an acute left ventricular (LV) myocardial infarction (MI) has been shown to be associated with higher morbidity and mortality [1]. The prevalence of RV involvement in acute LV MI reportedly ranges from 50% to 80% in postmortem and animal studies but is frequently underestimated in the clinical setting owing to the diagnostic limitations of the electrocardiogram (ECG) and echocardiography [2].

Quantitative assessment of RV function is often difficult using the various noninvasive imaging modalities owing to the inherently complex geometry of the right ventricle [3]. Echocardiography remains the most commonly used technique for RV function assessment in clinical practice because of its widespread availability. The myocardial performance index (MPI) of RV based on conventional Doppler echocardiography has been proven to be useful in the evaluation of RV function and recommended as one of the initial quantitative measurements of RV diastolic function and may be a sensitive tool for detecting ''occult'' RV dysfunction in acute LV MI [4].

Nevertheless, the conventional Doppler-derived MPI has one important limitation, namely, that the interval between the end and onset of tricuspid inflow and the ejection time are measured sequentially and not on the same cardiac cycle. By contrast, tissue Doppler imaging (TDI) can simultaneously measure these important time intervals on the same cardiac cycle, making the TDI-derived MPI superior in the estimation of global RV function [5].

The aim of the study was to assess RV function in patients with successful primary percutaneous coronary intervention (PCI) for acute anterior ST elevation myocardial infarction (STEMI) without RV infarction and determine factors affecting it.

2. Patients and methods

2.1. Patient eligibility

The current study enrolled 40 patients who underwent successful primary PCI in the Cath labs of Ain Shams University Hospital, Cairo, Egypt for first acute anterior STEMI without RV infarction from December 2013 to June 2014.

2.2. Inclusion criteria

All patients with the definite diagnosis of anterior STEMI as per the universal definition [6] were enrolled.

Abbreviations	
CABG	Coronary Artery bypass graft
CHF	Congestive heart failure
DTB	Door to balloon time
FAC	Fractional area Change
IRA	Infarct related artery
LVEDD	Left ventricular end diastolic dimension
LVEF	Left ventricular ejection fraction
LVESD	Left ventricular end systolic dimension
MPI	Myocardial Performance Index
PCI	Percutaneous coronary intervention
PTD	Pain to Door
RAA	Right atrial Area
RV	Right ventricle
RVEDD	Right Ventricular End Diastolic Dimension
RVRAC	Right Ventricular Fractional Area Change
ST resolution	ST segment elevation resolution
STEMI	ST. Elevation myocardial infarction
TAPSE	Tricuspid Annular Plane Systolic Excursion
TDI	Tissue Doppler Imaging
WMSI	Wall motion Score Index

To be eligible, patients had to undergo angiographically successful primary PCI defined as successful deployment of stent in culprit infarct-related artery (IRA), no residual dissection, and less than 20% residual stenosis in IRA [7].

2.3. Exclusion criteria

Patients with any of the following criteria were excluded from the study: (1) presence of RV infarction defined by an ST-segment elevation of 0.1 mV or greater in lead V4R on ECG at presentation and/or any RV wall motion abnormality detect by echocardiography; (2) previous history of MI or coronary revascularization; (3) persistent hemodynamic instability necessity use of positive inotropes; (4) atrial fibrillation; (5) moderate or severe valvular heart disease; (6) presence of a coexisting clinical condition that might affect RV function, including pericardial disease, chronic lung disease, pulmonary hypertension, or connective tissue disorder; (7) patient with contraindication for coronary angiography such as severe renal impairment, coagulopathy, etc.; (8) patient with significant stenosis (>50%) affecting RV branch or right coronary artery proximal to RV branch; and (9) consent refusal.

Patients who met the inclusion criteria were subjected to the following. (1) Thorough history taking. (2) Full clinical examination: targeted physical examination data were recorded including general and local examination with special attention to vital data and signs suggestive of risk factor for coronary artery disease (CAD), mechanical complications, signs of heart failure, and Killip classification. (3) Twelve-lead surface ECG was

performed on admission, 90 minutes after reperfusion, then every 8 hours in the first 24 hours, then daily thereafter to confirm the diagnosis of STEMI and to exclude the presence of RV infarction, and to follow up the ST segment resolution. (4) Laboratory data: all labs including troponin, cardiac enzymes [creatine kinase (CK) total and CK muscle/brain] every 8 hours for first 24 hours, then daily for 3 days, partial thromboplastin time, complete blood count, and renal and hepatic profile. (5) Primary PCI: after diagnostic coronary angiography, stenting with or without balloon predilatation, then assessment of post procedure flow using thrombolysis in MI, i.e., TIMI grading [8]. (6) Echocardiography: performed within 72 hours of successful reperfusion using Philips SONOS 7500 (Agilent Technologies, Andover, MA, USA) phased-array system equipped with TDI technology; we conducted a comprehensive 2D Doppler echocardiography and pulsed-wave TDI while the patients were lying in the partial left lateral decubitus position during either shallow respiration or a breath hold.

All the image acquirement and analyses were performed by echocardiography expert who was blinded to the patients' clinical data. (1) The LV end-systolic dimension (LVESD) and end-diastolic dimension (LVEDD) were obtained from M-mode recording, from the parasternal short-axis view. The LV ejection fraction (LVEF) was estimated from the apical four-chamber view using the modified Simpson method. (2) The 16-segment model for LV segmentation was used to evaluate regional wall motion abnormalities as recommended by the American Society of Echocardiography [9]. Each segment was assessed individually for systolic thickening and endocardial excursion. Each segment was given a score from 1 (normal), 2 (hypokinesia), 3 (akinesia), and 4 (dyskinetic), and the global wall motion score index (WMSI) was calculated as a sum of all the scores divided by the number of segments visualized. (3) The RV end-diastolic dimension was assessed at the mid-cavity of the right ventricle in the apical four-chamber view. (4) The RV fractional area change (RVFAC) was calculated as the RV end-diastolic area – the end-systolic area/the RV end-diastolic area. (5) The right atrial area (RAA) was estimated by planimetry at the end of ventricular systole. (6) The tricuspid annular plane systolic excursion (TAPSE) was measured from the apical four-chamber view at the RV free wall level by placing an M-mode cursor passed through the tricuspid lateral annulus in apical four-chamber view, and measuring the amount of longitudinal

displacement of the annulus at peak-systole. (7) The trans-mitral and -tricuspid Doppler flow velocities were recorded from the apical four-chamber view with the sample volume placed between the tips of the mitral and tricuspid valves, respectively, and the peak early filling velocity (E), peak atrial velocity (A) were measured, and E/A ratio was calculated. (8) Pulsed-wave TDI images were acquired from the standard apical four-chamber view, a 5.2-mm sample volume was placed at the lateral tricuspid annulus, mitral septal, and mitral lateral annular sites to obtain the spectral pulsed tissue Doppler data. Three cardiac cycles were averaged for each TDI measurement.

Peak systolic annular velocity (Str), early diastolic annular velocity (Etr), and late diastolic annular velocity (Atr) of the right ventricle were measured offline; and the Etr/Atr and E/Etr ratios were calculated. Isovolumic relaxation time (IVRT) was measured as the time interval from the end of Str to the onset of Etr, isovolumic contraction time (measured as the time interval from the end of the Atr to the beginning of the Str), and ejection time (measured from the onset to the end of Str) of the right ventricle were calculated to obtain the TDI-derived RV MPI with the formula: IVRT + isovolumic contraction time/ejection time. Abnormal RV MPI was defined as an MPI >0.55 [10].

Patients were assigned as having abnormal RV function if they had a combination of at least two of: RVFAC <35%, RV MPI using TDI >0.55, TAPSE <16 mm [10,11].

2.4. Statistical analysis

Data was collected, verified, revised, and then edited. Categorical variables were expressed as an absolute and relative frequency (percentage), while continuous variables were presented as mean values ± standard variation. Comparisons were made between the two groups using t test for continuous variables and Chi-square test or Fisher exact test when the expected count was less than five for the qualitative data and Pearson correlation coefficient for categorical variables. Statistical analysis was performed using SPSS version 20 (SPSS Inc., Chicago, IL, USA). Difference was considered statistically significant at $p < 0.05$ and highly significant at $p < 0.01$.

3. Results

3.1. Descriptive patient data

The mean age was 45.9 ± 7.6 years, 22 (55%) were males, 27 (67.5%) were smokers, 35 (87.5%)

were diabetic, 30 (75%) were hypertensive, 17 (42.5%) were dyslipidemic, and 15 (37.5%) had positive family history premature CAD. With regard to Killip classification, 19 (47.5%) presented with Killip class 1, 15 (37.5%) with Killip class 2, six (15%) with Killip class 3, and none with Killip class 4.

The mean pain-to-door time was (9.7 ± 4.0) hours and the mean door-to-balloon time was (1.1 ± 0.49) hours. During primary PCI predilatation in 18 patients (45%), single stent was used in 13 (75%), and two stents were used in 10 (25%) patients.

Echocardiography showed that the mean EF was (50.5 ± 9.1%), LVESD was (35.2 ± 4.8) mm, LVEDD was (52 ± 4.3) mm, whereas the mean WMSI was (1.9 ± 0.2). Mitral E/A was (1.10 ± 0.11), whereas tricuspid E/A was (1.09 ± 0.1).

With regard to RV function, the mean RVEDD was (26.8 ± 5.6) mm, RVFAC was (37.5 ± 7.5%), RAA was (16.8 ± 5.5) cm^2, TAPSE was (14.1 ± 4.3) mm, MPI was (0.44 ± 0.18), E_{tr}/A_{tr} ratio was (0.76 ± 0.51), and E/E_{tr} ratio was (5.8 ± 1.7).

3.2. Comparative analysis

According to RV function assessment based on RVFAC, RV MPI, and TAPSE, patients were classified into two groups. Group 1 ($n = 22$): patients were assigned as having abnormal RV function if they had a combination of at least two of TAPSE, RVFAC <35%, and RV MPI using TDI >0.55. Group 2 ($n = 18$): patients with normal RV function having one criterion or none of the criteria.

Comparing patients with normal and abnormal RV function (according to the abovementioned method), abnormal RV function was significantly correlated with older age of patients (42.78 ± 7.6 vs. 48.45 ± 6.47); however, there was no statistically significant difference between groups with regard to sex or predisposing risk factors (Tables 1 and 2).

Patients with proximal or mid left circumflex artery (LCX) lesions as well as patients with no reflow and worse procedural events were correlated with RV dysfunction (Table 3). Patients with higher blood glucose levels on admission had worse RV function as well (Table 4). RV dysfunction was significantly correlated with poor echocardiographic LV data, larger LVESD and LVEDD, lower LVEF, and higher WMSI (Table 5).

3.3. Multivariate analysis

On logistic multi-regression analysis for abnormal RV function and other variables that showed statistically significance, LCX mid or proximal affection, eventful procedure, occurrence of no reflow, glucose level, LVESD, LVEDD, and LVEF were independently significantly correlated to RV dysfunction (Table 6).

Table 1. Comparison between normal and abnormal RV function according to demographic data.

Demographic data	RV function			t/χ^2	p
	Normal ($n = 18$)	Abnormal ($n = 22$)	Total ($n = 40$)		
Sex, n (%)					
Female	6 (33.3)	12 (54.5)	18 (45.0)	1.800	0.180*
Male	12 (66.7)	10 (45.5)	22 (55.0)		
Age (y)					
Mean ± SD	42.78 ± 7.60	48.45 ± 6.74	45.90 ± 7.60	6.267	0.017**
Range	30–61	33–63	30–63		

RV = right ventricular.
* $p < 0.05$, significant.
** $p > 0.05$, non-significant.

Table 2. Comparison between normal and abnormal RV function according to risk factors.

Risk factors, n (%)	RV function			χ^2	p*
	Normal ($n = 18$)	Abnormal ($n = 22$)	Total ($n = 40$)		
Smoker	13 (72.2)	14 (63.6)	27 (67.5)	0.333	0.564
Diabetes mellitus	15 (83.3)	20 (90.9)	35 (87.5)	0.519	0.471
Hypertension	14 (77.8)	16 (72.7)	30 (75.0)	0.135	0.714
Dyslpid obese	7 (38.9)	10 (45.5)	17 (42.5)	0.175	0.676
Familial hypercholesterolemia	4 (22.2)	11 (50.0)	15 (37.5)	3.259	0.071

RV = right ventricular.
* $p > 0.05$, non-significant.

Table 3. Comparison between normal and abnormal RV function according to catheter data.

Catheter	RV function, n (%)			χ^2	p
	Normal (n = 18)	Abnormal (n = 22)	Total (n = 40)		
Infarction					
Ant	11 (61.1)	15 (68.2)	26 (65.0)	4.074	0.130
Antseptal	7 (38.9)	4 (18.2)	11 (27.5)		
Exten ant	0 (0.0)	3 (13.6)	3 (7.5)		
LAD					
Mid total	6 (33.3)	5 (22.7)	11 (27.5)	0.623	0.732
Ostal total	1 (5.6)	1 (4.5)	2 (5.0)		
Px total	11 (61.1)	16 (72.7)	27 (67.5)		
LCX					
Mid	1 (5.6)	7 (31.8)	8 (20.0)	8.234	0.016*
NSL	16 (88.9)	10 (45.5)	26 (65.0)		
Proximal	1 (5.6)	5 (22.7)	6 (15.0)		
TIMI					
1	0 (0.0)	4 (18.2)	4 (10.0)	26.777	<0.001**
2	0 (0.0)	14 (63.6)	14 (35.0)		
3	18 (100.0)	4 (18.2)	22 (55.0)		

LCX, left circumflex artery; RV = right ventricular; TIMI = thrombolysis in myocardial function, LAD = left anterior descending; Px = proximal, NSL = non significant lesion.
* $p < 0.05$, significant.
** $p < 0.001$, highly significant.

Table 4. Comparison between normal and abnormal RV function according to laboratory data.

Laboratory data	RV function			t test	p
	Normal (n = 18)	Abnormal (n = 22)	Total (n = 40)		
Glucose level					
Mean ± SD	122.44 ± 12.63	172.55 ± 47.53	150.00 ± 43.85	18.828	<0.001**
Range	100–156	101–244	100–244		
CK total					
Mean ± SD	185.39 ± 52.03	209.64 ± 61.06	198.73 ± 57.76	1.779	0.190
Range	101–308	112–394	101–394		

CK = creatine kinase; RV = right ventricular.
** $p < 0.001$, highly significant.

Table 5. Comparison between normal and abnormal RV function according to echo data.

Echo data	RV function			t test	p
	Normal (n = 18)	Abnormal (n = 22)	Total (n = 40)		
LVESD					
Mean ± SD	33.56 ± 3.78	36.64 ± 5.29	35.25 ± 4.87	4.304	0.045*
Range	29–46	28–49	28–49		
LVEDD					
Mean ± SD	50.72 ± 2.40	53.14 ± 5.28	52.05 ± 4.36	3.214	0.044*
Range	47–57	41–60	41–60		
EF%					
Mean ± SD	54.00 ± 8.20	47.68 ± 9.05	50.53 ± 9.14	5.242	0.028*
Range	37–67	30–67	30–67		
WMSI					
Mean ± SD	1.86 ± 0.24	2.08 ± 0.11	1.98 ± 0.21	16.220	<0.001**
Range	1.1–2.3	1.8–2.3	1.1–2.3		

EF = ejection fraction; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; RV = right ventricular; WMSI = wall motion score index.
* $p < 0.05$, significant.
** $p < 0.001$, highly significant.

Table 6. Logistic regression multivariate analysis of factors affecting abnormal RV function.

Parameters	B	Sig.	Odds ratio	95% Confidence interval	
				Lower	Upper
Age (y)	0.146	0.108	1.158	0.968	1.384
LCX: Mid	−1.147	0.026	0.439	0.175	1.101
LCX: NSL	0.705	0.133	1.137	0.179	2.875
LCX: Px	−0.551	0.018	0.73	0.482	1.106
No reflow and low TIMI grade	0.08	0.007	1.252	1.19	1.318
Glucose level	0.068	0.009	1.070	1.017	1.127
LVESD	−0.981	0.037	0.375	0.149	0.941
LVEDD	0.602	0.047	1.826	1.007	3.312
EF%	−0.471	0.026	0.624	0.412	0.946
WMSI	0.262	0.280	0.483	0.192	1.212

EF = ejection fraction; LCX = left circumflex artery; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; RV = right ventricular; TIMI = thrombolysis in myocardial infarction; WMSI = wall motion score index.

4. Discussion

The current study aimed to assess the RV function for 40 patients who underwent primary PCI in the Catheter Laboratory of Ain Shams University Hospital for first acute anterior STEMI without RV infarction. We found that 55% of patients had RV dysfunction using the definition of a combination of at least two of: TAPSE, RVFAC <35%, RV MPI using TDI >0.55.

Hsu et al. [4] studied a total of 102 consecutive patients admitted with the first episode of acute MI and found that 17% of the study population who presented with anterior STEMI and underwent primary PCI had RV dysfunction, yet inferior wall MI was correlated with more impairment of RV function.

Also, Karakurt and Akdemir [12] reported that 22% of their study population had abnormal MPI.

The lower incidence of RV involvement in the studies by Hsu et al. [4] and Karakurt and Akdemir [12] than in our study could be explained by higher risk profile of our patients such as diabetes mellitus (DM; 87.5%) in our study, 28.5% in Hsu et al. study [4], and 25.9% in Karakurt and Akdemir study [12], which may affect the microvascular integrity leading to higher incidence of RV involvement.

Higher incidence of DM could be correlated to the tertiary nature of our center and high incidence of DM in Egypt (15.6% for type 2) [13].

Kidawa et al. [14] found that 64% of their patients had RV dysfunction measured by TAPSE; this higher incidence may be explained by the fact that they included all STEMI patients with possible occurrence of RV infarction in addition to the different assessment parameters.

Elserafy et al. [15] found RV dysfunction assessed by TAPSE to occur in 61.3% of patients presenting with non-STEMI managed by invasive strategy. Significant improvement occurred to TAPSE after 3 months compared with TAPSE at baseline.

The incidence of abnormal MPI was 55% in our study population compared with 19% in the study by Hus et al; this could also be explained by our higher risk profile [4].

Møller et al. [16] found that 33% of the study population who presented with STEMI had abnormal conventional Doppler-derived RV MPI; this could be explained by the lower accuracy of conventional Doppler than tissue Doppler-derived MPI used in our study.

In our study, we found that older patients had significantly more RV dysfunction, whereas Hsu et al. [4] found no association between increasing age and the depressed RV function and abnormal MPI. This can be explained by the fact that our study population included rather younger patients with the mean age of 45.9 ± 7.6 years versus 59 ± 12 years in the study by Hsu et al; therefore, on comparing both groups normal and abnormal RV function, the impact of the age appear comparable with our population.

We also found patients with proximal or mid LCX lesions as well as patients with no reflow and worse procedural events to be correlated with RV dysfunction. Kidawa et al. [14] found results similar to those of our study.

Our study showed admission glucose level to be correlated with RV dysfunction, whereas Hsu et al. [4] found no relationship; however, the prevalence of DM in their population was much lower than that in our patients (28.5% vs. 87.5%).

We also found that total CK was higher in patients with abnormal MPI, yet not correlated with RV dysfunction according to our definition; MPI finding is in agreement with Hsu et al. [4], which could be explained by the larger infarction associated with higher total CK more frequently affecting RV function.

Echocardiographic assessment showed RV dysfunction to be significantly correlated with poor echocardiographic LV data, larger LVESD and LVEDD, lower LVEF, and higher WMSI. Results of Hsu et al. [4] study were in agreement with those of our study.

Similar to our data, Karakurt and Akdemir [12] reported that the RV global function assessed by RV TDI was depressed in anterior STEMI, but not in inferior STEMI regardless of the reperfusion strategy.

Independent predictors of in-hospital RV dysfunction according to our definition included LCX mid or proximal affection, eventful procedure, occurrence of no reflow, glucose level, LVESD, LVEDD, and LVEF.

RV systolic function has multitude of factors and is strongly dependent on LV function and shape and septal motion; additionally, territories of coronary arteries are overlapping with very variable overlap patterns between different patients, which can explain the correlations found.

5. Conclusion, recommendations, and study limitations

We conclude that affection of RV function as assessed by echocardiography is commonly associated with anterior STEMI.

Age, pain to door, predilatation, admission glucose level, WMSI, mitral E/A, tricuspid E/A, and MPI were the independent predictors of abnormal RV function.

Thus, we recommend that RV function must be assessed by echocardiography using either MPI or by the RV function assessment parameters in all patients presenting with STEMI. Educational campaigns must be launched to increase patient awareness regarding the importance of seeking medical advice promptly if chest pain occurs.

Limitations of our study include small study population and many exclusion criteria. Furthermore, RV functional recovery may occur over time after acute MI, so we could not exclude the possibility that different timing of the assessment would have resulted in different findings. Finally, we were not able to follow-up patients after discharge to assess the long-term effects and improvement.

Conflicts of interest

Authors declare no conflict of interest and no special funding was acquired.

References

[1] Zehender M, Kasper W, Kauder E, Schonthaler M, Geibel A, Olschewski M, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 1993;328:981–8.

[2] Lopez-Sendon J, Coma-Canella I, Alcasena S, Seoane J, Gamallo C. Electrocardiographic findings in acute right ventricular infarction: sensitivity and specificity of electrocardiographic alterations in right precordial leads V4R, V3R, V1, V2, and V3. J Am Coll Cardiol 1985;6:1273–9.

[3] Jensen CJ, Jochims M, Hunold P, Sabin GV, Schlosser T, Bruder O. Right ventricular involvement in acute left ventricular myocardial infarction: prognostic implications of MRI findings. AJR Am J Roentgenol 2010;194:592–8.

[4] Hsu SY, Chang SH, Liu CJ, Lin JF, Ko YL, Cheng ST, et al. Correlates of impaired global right ventricular function in patients with a reperfused acute myocardial infarction with and without right ventricular infarction. J Investig Med 2013;61:715–21.

[5] Sade LE, Gülmez Ö, Özyer U, Özgül E, Ağıldere M, Müderrisoğlu H. Tissue Doppler study of the right ventricle with a multisegmental approach: comparison with cardiac magnetic resonance imaging. J Am Soc Echocardiogr 2009;22:361–8.

[6] Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD. Third universal definition of myocardial infarction. Circulation 2012;126:2020–35.

[7] van 't Hof AW, Liem A, Suryapranata H, Hoorntje JC, de Boer MJ, Zijlstra F. Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Zwolle Myocardial Infarction Study Group. Circulation 1998;97:2302–6.

[8] Webb I, Redwood S. Measuring reperfusion in the catheter laboratory. Heart Metab 2007;37:9–13.

[9] Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63.

[10] Harada K, Tamura M, Toyono M, Yasuoka K. Comparison of the right ventricular Tei index by tissue Doppler imaging to that obtained by pulsed Doppler in children without heart disease. Am J Cardiol 2002;90:566–9.

[11] Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23:685–713. quiz 786–8.

[12] Karakurt O, Akdemir R. Right ventricular function in ST elevation myocardial infarction: effect of reperfusion. Clin Invest Med 2009;32:E285–92.

[13] Hegazi R, El-Gamal M, Abdel-Hady N, Hamdy O. Epidemiology of and risk factors for type 2 diabetes in Egypt. Ann Glob Health 2015;81:814–20.

[14] Kidawa M, Kasprzak JD, Wierzchowski T, Krzeminska-Pakula M. Right ventricular function suffers from reperfusion delay: tissue Doppler study. Clin Cardiol 2010;33:E43–8.

[15] Elserafy AS, Nabil A, Ramzy AA, Abdelmenem M. Right ventricular function in patients presenting with non-ST-segment elevation myocardial infarction undergoing an invasive approach. Egypt Heart J 2018;70:149–53.

[16] Møller JE, Søndergaard E, Poulsen SH, Appleton CP, Egstrup K. Serial Doppler echocardiographic assessment of left and right ventricular performance after a first myocardial infarction. J Am Soc Echocardiogr 2001;14:249–55.