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Right ventricular functional involvement and recovery in patients with a first acute myocardial infarction treated by primary PCI. Does essential hypertension play any role?

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Abstract

The aim of this study was to assess right ventricular (RV) involvement in patients with acute ST-segment elevation myocardial infarction before undergoing primary PCI and to evaluate the changes in RV function throughout hospitalization. Considering that patients with essential hypertension are majority, we also thought to assess if there are specific changes in RV function during acute ST elevation myocardial infarction in this category. 53 patients with a first acute myocardial infarction (MI) referred for primary PCI were included and prospectively analyzed. 32 of them (60.4%) had anterior and 21 (39.6%) had non-anterior MI, while 11 (20.8%) patients had signs of RV necrosis on the surface ECG. Serial echocardiograms were performed before PCI, 24 hours afterwards and at discharge. In order to accurately quantify RV function, we used a multi-parametric approach, with conventional as well as novel parameters derived from 2D strain echocardiography. Right ventricular myocardial performance index (RV MPI) was high from admission in both patients with and without RV infarction and this parameter remained high at 24 hours and at discharge. RV systolic dysfunction (assessed by TAPSE, RV longitudinal strain and RV fractional area change) was present on admission in patients with RV infarction, but not in patients without RV infarction. RV systolic function gradually improved throughout hospitalization and became normal at discharge. Patients with essential hypertension didn't show any specific changes in RV functional parameters. RV global dysfunction is found in the setting of an acute MI irrespective of the culprit coronary artery and it persists at discharge. On the other hand, RV systolic function, which is altered on admission in the subgroup with RV MI, normalizes over the course of hospitalization. Hypertensive patients didn't show any specific changes in RV function in the setting of AMI.

Keywords: RV function, STEMI, Myocardial performance index, essential hypertension

Introduction

In the setting of an acute ST-elevation myocardial infarction (STEMI), attention has been mainly focused on the left ventricle (LV), with LV dysfunction being

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one of the most important, well-known prognostic parameters [1]. Transthoracic echocardiogram is an indispensable tool for LV assessment in patients with acute myocardial infarction (MI) because it is a widely available investigation, easy to perform at bedside, which can help in making therapeutic choices.

On the other hand, the occurrence and clinical significance of right ventricular (RV) involvement in the setting of an acute MI is less clear and far less studied, different studies offering conflicting results. In addition , RV myocardial dysfunction in acute inferior myocardial infarction can result in hemodynamic instability, atrioventricular conduction delay , ventricular arrhythmias and therefore increased in-hospital mortality and poor long-term outcome [2]. Several studies using different diagnostic approaches have addressed the issue, concluding that early post-infarction RV injury is common irrespective of the localization of the MI [3,4]. Furthermore, the extent of RV dysfunction is an independent predictor of outcomes [5] in acute MI patients treated with different reperfusion strategies [6,7]. In patients with reperfused MI and RV ischemic injury, RV function recovers more rapidly and to a greater extent than LV function [8], suggesting that RV myocardial stunning might be involved in the pathophysiology of RV dysfunction [9] and that acute post-MI RV injury is the expression of viable, reversibly damaged myocardium.

Echocardiographic evaluation of RV function is more challenging than that of the LV mainly because of the complex anatomy and asymmetrical shape of the RV. Standard echocardiographic measurements allow morphological and functional assessment of the RV, but they have limited value because of the pyramidal shape of the RV which makes geometric assumptions difficult. Strain echocardiography is a reliable method of quantifying regional contractility and it can detect subclinical cardiac dysfunction. Initially used to measure LV deformation, strain analysis is also a useful tool for the assessment of RV function in a non-geometric manner and it was shown to provide strong prognostic information for patients with inferior STEMI treated with primary PCI [10]. On the other hand, little is known about the behavior of the right ventricle in essential hypertension, in this area being published few studies suggesting that systemic hypertension could be associated with right ventricular morphological and functional abnormalities , including reduced right ventricular internal dimensions and right ventricular diastolic dysfunction [11,12].

This descriptive study assessed the pattern of RV functional change in a set of patients who underwent primary PCI for acute MI, before reperfusion therapy, 24 hours after PCI and at discharge. We also intended to determine whether the changes in RV function are different depending on the site of myocardial necrosis. In the subset of hypertensive patients with acute myocardial infarction, which actually represent majority, we thought to assess if hypertensive state induces specific changes in RV function compared to their nonhypertensive counterparts. While several studies have assessed RV function immediately after reperfusion therapy (whether primary PCI or thrombolysis) and during short- or long-term follow-up, this is the first study so far to evaluate RV involvement before undergoing primary PCI.

Methods

Study population

From March 2015 to April 2016, 53 consecutive patients presenting with a first acute STEMI and treated with primary PCI within the first 12 hours were included and screened prospectively. STEMI was diagnosed according to the universal definition of MI: rise and/or fall of cardiac biomarkers in addition to new or presumed new significant ST segment elevation or new left bundle branch block (LBBB) on ECG, with or without symptoms of ischemia [13]. Basic demographic data, past medical history and ECG traces were collected from their medical records by 3 investigators. Patients were included regardless of the site of myocardial necrosis; patients with previous history of cardiac disease or patients with conditions that could lead to right heart dysfunction such as chronic pulmonary diseases, as well as patients with poor echocardiographic images or atrial fibrillation on ECG tracings represented exclusion criteria.

All patients were treated according to current guidelines regarding management of patients with acute myocardial ST elevation myocardial infarction [14]. Coronary angiograms were analyzed to assess the severity of the coronary lesions and the distal coronary flow was graded before and after the procedure using the TIMI classification (TIMI flow grade 0 to 3). ECG

tracings were obtained at admission in all patients using classical 12 lead recordings. In addition, for the diagnostic of RV necrosis, right precordial leads have been used, RV infarction being defined by the occurrence of ST elevation of least 1 mm in leads V3R and V4R. All patients underwent serial echocardiographic examinations using conventional and special echocardiographic techniques on admission, 24 hours after PCI and at discharge. Regarding hypertensive disease, diagnostic of hypertension was made according to the current guidelines [15], but subjects with significant hypertensive heart disease were excluded.

Patients were followed for any major adverse cardiovascular events (MACE) - defined as recurrent ischemia , need for revascularization, heart failure or death - during hospitalization; this study is part of a larger, ongoing one which includes patients' follow-up at 1 month, 6 months and 12 months respectively.

The study was approved by the human research committee of our hospital and informed consent was obtained from each patient.

Echocardiographic assessment

Comprehensive echocardiographic examinations were performed using Vivid Q in the acute state and Vivid E9 machines with a 3.5 MHz transducer, according to current international recommendations [16]. Serial recordings were performed on admission, 24 hours after PCI and at discharge. All echocardiographic images were digitally stored and data analysis and measurements were performed offline by a well-trained echocardiographer using dedicated software (GE EchoPAC BT 12). The reference limits for all echocardiographic parameters were chosen according to the current guidelines [17].

LV functional assessment was made by measuring the LV ejection fraction (LVEF) via biplane Simpson method using the apical 4 chamber and 2 chamber views, as well as by measuring the LV myocardial performance index (LV MPI) and the global longitudinal strain of the LV (GLS-LV) assessed by speckle tracking echocardiography.

RV functional assessment was made using the multiparametric approach as recommended by the current guidelines [16, 17]. RV global function was assessed by RV myocardial performance index (RV MPI), which was determined using the pulsed Doppler (PW) method as well as tissue Doppler imaging (TDI). An average of 5 measurements was used for all RV MPI analyses. RV MPI represents the ratio between the difference tricuspid valve closure-opening time and the ejection time divided by the ejection time.

RV systolic function was assessed by conventional echocardiography by parameters such as tricuspid annular plane systolic excursion (TAPSE),TDI pulsed tissue Doppler S wave velocity, RV fractional area change (RV FAC) and by parameters derived from 2D strain echocardiography such as RV global longitudinal strain and strain of the right ventricular free wall.

TAPSE was recorded in the apical 4 chamber view with M-mode echocardiography parallel to the RV free wall and across the tricuspid annulus, and measured as the systolic displacement of the tricuspid annular plane. S wave velocity was measured in the apical 4 chamber view, with the TDI pulsed Doppler sample placed on the tricuspid annulus or in the middle of the basal segment of the RV free wall. RV FAC was measured in the apical RV focused view, by manually tracking the endocardial border and calculated as the percentage change in areas of the end-diastolic and end-systolic areas of the RV. Peak longitudinal strain of the RV was measured in the RV focused 4-chamber view, using speckle tracking analysis with software dedicated for the LV, but applied to RV. Strain analysis was performed offline by an experienced observer using Q-Analysis package. Briefly, the endocardium was manually traced at end-systole and the region of interest (ROI) was adjusted to include the whole myocardial wall but to exclude the pericardium. The reference point of each cardiac cycle was placed at the beginning of the QRS complex. Strain analysis was feasible in all segments obtained from the 53 patients included in the study. Global longitudinal strain of the RV (GLS-RV) was calculated offline by the mean of 6 segmental values of the lateral wall and interventricular septum. Longitudinal strain of the RV free wall (GLS-RVFW) was measured by the average of 3 segmental values (basal, medial and apical).

For the analysis of RV diastolic function we used tricuspid E/A ratio and tricuspid E/E'ratio, measured by placing the PW sample in the four apical chamber view at the tips of the tricuspid valve in diastole assessing early and late diastolic velocity; for measuring E', pulsed TDI was used placed at the base of the RV free lateral wall recording early diastolic myocardial velocity.

An average of 5 measurement was used for reporting final results.

Statistical analysis

Normal distribution of variables was assessed by Kolmogorov-Smirnov test. Accordingly, continuous variables were summarized as mean ± SD if normally distributed and as median (interquartile range [IQR]) otherwise, scalar variables were reported as frequencies and percentages. The primary aim was to evaluate and compare the RV function in patients with and without right ventricular MI. Differences between the two populations mentioned above were assessed using non-parametric test (Mann-Whitney U test), as we did not find a normal distribution of variable for the population with right ventricular MI.

All analyses were performed using SPSS version 20.0 (SPSS, Inc, Chicago, I l). P-values <0.05 were considered significant.

Results

Clinical characteristics

A total of 53 patients were included, most of which were men (62.26%), with a mean age of 62 ± 13 years and an age range of 37 to 93 years. 32 patients (60.37%) had anterior MI with the culprit lesion on the left anterior descending artery, while the 21 remaining patients had non-anterior MI with either right coronary artery or circumflex artery as the culprit vessel. The patients' baseline characteristics depending on the

Table 1. Baseline characteristics (n=53).

	NoRV MI (n=42)	RV MI (n=11)	p
Male	27 (64.28%)	6 (54.54%)	.54
Age	61 ±13	64 ±10	.43
Cardiovascular risk factors			
• Current/ Ex-smoker	29 (69.04%)	5(45.45%)	.33
• Diabetes	11 (26.19%)	2 (18.18 %)	.59
• Dyslipidemia	27 (64.28%)	10 (90.90%)	.05
• Hypertension	27 (64.28%)	6 (54.54%)	.56
Symptom-to-balloon time (min)	852 ± 1659	471 ± 465	.43
Door-to-balloon time (min)	97 ± 28	42 ± 25	.45
Clinical presentation			
• Systolic BP (mm Hg)	133 ± 23	135 ± 17	.73
• Diastolic BP (mm Hg)	78 ± 12	72 ± 11	.15
• Heart rate (beats/min)	81 ± 13	74 ± 7	.09
MI localization			
• Topol 1		1 (1.88%)	
• Topol 2		19 (35.84%)	
• Topol 3		12 (22.64%)	
• Topol 4		18 (33.96%)	
• Topol 5		3 (5.66%)	
• RV MI		11 (20.75%)	
Multivessel coronary artery disease		29 (54.71%)	

Values are number (%) or mean ± SD
BP= blood pressure; RV= right ventricle; MI= myocardial infarction

Table 2. Echocardiographic findings regarding LV functional parameters throughout hospitalization.

Parameter	Before PCI			24 hours after PCI			Discharge		
	Non RV MI	RV MI	p	Non RV MI	RV MI	p	Non RV MI	RV MI	p
LVEDV (ml)	102± 30	100± 37	.87	94± 25	85 ± 27	.39	101 ± 27	96 ± 24	.59
LV ESV (ml)	61± 21	61± 25	.98	53 ± 21	47 ± 19	.43	57 ± 20	51 ± 15	.36
LVEF (%)	42 ± 8	41± 9	.88	43 ± 9	47 ± 6	.28	43.8 ± 8	47 ± 5	.29
GLS-LV (%)	-11.6± 4.5	-12.6 ± 4.3	.56	-11.5± 3.5	-12.2 ± 2.5	.60	-12 ± 3.4	-14 ± 2.5	.12
LAV (ml)	54 ± 16	65 ± 19	.79	55 ± 20	69 ± 28	.09	57 ± 21	59 ± 12	.88

Values are number (%) or mean ± SD

LV=left ventricle; EDV=end-diastolic volume; ESV=end-systolic volume; LVEF=left ventricular ejection fraction; GLS-LV=global longitudinal strain of the LV; LAV=left atrial volume

right ventricle involvement on the surface ECG are listed in Table 1.

Echocardiographic findings

Patients' LV and RV functional parameters were assessed with serial echocardiograms on admission, immediately before coronary angiograms, 24 hours after PCI and at discharge (~after 7 days). Parameters assessing RV systolic (RV FAC, GLS-RV, TAPSE,) and global function (RV MPI) were altered on admission in the group of patients with RV involvement based on electrical criteria , except for S wave velocity measured by tissue Doppler imaging, which was within normal range from the very beginning. The evolution of RV's systolic function throughout hospitalization appears to be consistent with gradual improvement and complete recovery, as shown by normalization of the TAPSE, RV FAC and GLS-RV at discharge. The RV-MPI, however, remained high at discharge (using either one of the 2 methods for measuring it), indicating that global RV dysfunction persists despite normalization or near normalization of RV systolic function.

We sought to assess and compare differences in patterns of RV dysfunction and/or RV function recov-

Table 3. RV function in patients with and without RV infarction before PCI.

Parameter	RV MI (n=11)	No RV MI (n=42)	p value
RV FAC	23.1% ± 10.1%	40.8% ± 8.7%	<0.001
GLS-RV	-14.07 ± 4.67	-19.47 ± 7.16	0.035
GLS-RVFW	-14.49 ± 7.50	-23.48 ± 8.40	0.008
RV MPI (PW)	0.59 ± 0.20	0.53 ± 0.16	0.440
RV MPI (TDI)	0.67 ± 0.24	0.55 ± 0.14	0.189
TAPSE (mm)	18.6 ± 4.3	21.2 ± 4.0	0.03
S' (m/s)	0.10 ± 0.02	0.12 ± 0.02	0.118

Values are number (%) or mean ± SD.

Table 4. RV function in patients with and without RV infarction 24 hours after PCI.

Parameter	RV MI (n=11)	No RV MI (n=42)	p value
RV FAC	29.9% ± 14.3%	40.6% ± 7.9%	0.035
GLS-RV	-12.72 ± 3.21	-17.53 ± 5.07	0.004
GLS-RVFW	-14.77 ± 6.10	-22.26 ± 6.16	0.005
RV MPI (PW)	0.55 ± 0.18	0.54 ± 0.19	0.941
RV MPI (TDI)	0.69 ± 0.15	0.61 ± 0.19	0.151
TAPSE (mm)	18.3 ± 4.7	20.3 ± 3.7	0.24
S' (m/s)	0.12 ± 0.03	0.13 ± 0.03	0.679

Values are number (%) or mean ± SD

ery between patients with and without RV myocardial infarction, defined by electrical criteria. Regarding LV functional and structural parameters, there were no statistically significant differencies between patients with or without RV infarction . Results are depicted in table 2.

Analysing by multimodality approach RV functional changes we noticed that on admission (Table 3), TAPSE, RV FAC, GLS-RV and GLS-RVFW (all of them assessing RV systolic dysfunction) were all altered in the subgroup of patients with RV necrosis (18.6±4.3, 23.1% ± 10.1%, -14.07 ± 4.67 and -14.49 ± 7.50, respectively), but normal in the larger subgroup of patients without RV infarction (21.2±4,40.8% ± 8.7%, -19.47 ± 7.16 and -23.48 ± 8.40, respectively), and the difference was statistically significant (p=0.03, p<0.001, p=0.035, p=0.008, respectively).

At 24 hours (Table 4), the same difference in pattern was observed between the two subgroups also statistically significant,excepting TAPSE which the first parameter that recovers at 24 hours. At discharge (Table 5), RV FAC, GLS-RV and GLS-RVFW regained to normal values in the subgroup of patients with RV infarction.

The only parameter which was altered in both subgroups from the beginning was the RV MPI. We meas-

Table 5. RV function in patients with and without RV infarction at discharge.

Parameter	RV MI (n=11)	No RV MI (n=42)	p value
RV FAC	40.4% ± 14.2%	42% ± 7.3%	0.664
GLS-RV	-18.88 ± 5.76	-20.89 ± 5.62	0.401
GLS-RVFW	-22.38 ± 10.39	-23.26 ± 8.63	0.864
RV MPI (PW)	0.62 ± 0.20	0.54 ± 0.18	0.337
RV MPI (TDI)	0.57 ± 0.19	0.58 ± 0.19	0.834
TAPSE (mm)	19 ± 3.1	20 ± 4.3	0.5
S' (m/s)	0.12 ± 0.02	0.13 ± 0.02	0.41

Values are number (%) or mean ± SD.

Table 6. Comparison of RV function in patients without RV infarction with normal values from literature.

Parameter	Before PCI	p value	24 hours after PCI	p value	at discharge	p value	Normal values
RV FAC %	40.8± 8.7	.22	40.6 ± 7.9	.52	42% ± 7.3%	.000	35
GLS-RVFW	-23 ± 8.4	.33	-22.26 ± 6	.82	-23.2 ± 8.63	.23	-20
RVMPI (PW)	0.53± 0.16	.00	0.54 ± 0.19	.00	0.54 ± 0.18	.00	0.43
RVMPI (TDI)	0.58±0.14	.15	0.61 ± 0.19	.005	0.58 ± 0.19	.01	0.54

Values are number (%) or mean ± SD
Normal values- according to Cardiac Chamber Quantification 2015 [14].

ured this parameter using two different methods, the pulsed Doppler (PW) and the tissue Doppler (TDI) method, with similar results: the RV MPI is abnormal both in the infarcted RV subgroup (0.59 ± 0.20 for PW method, 0.67 ± 0.24 for TDI method) and in the non-infarcted RV subgroup (0.54 ± 0.19 for PW method, 0.61 ± 0.19 for TDI method), indicating that RV global dysfunction in the setting of an acute STEMI is present irrespective of the site of necrosis and irrespective of the electrical involvement of the RV in the acute ischemic process. The RV MPI remains high at 24 hours in both subgroups and also at discharge.

Another purpose of our study was to assess wether there are significant changes in RV functional parameters during in hospital serial echocardiographic assessment in patients without RV necrosis based on ECG criteria. In this regard we compared RV systolic global (RV FAC) or regional (GLS-RVFW) and RV global functional parameters (RV-MPI) obtained in our study population with normal values described in the recent guidelines [17]. Results , depicted in Table 6, show that there is a significant impairement in global RV function expressed through the RV myocardial performance index also in patients without electrical signs of RV necrosis, impairement that remains until discharge. (0.53+/-0.16 at admission, 0.54 +/-0.19 at 24 hours and 0.54 +/-0.18 at discharge respectively by the PW method and 0.55± 0.14 at admission, 0.61 ± 0.19 at 24 hours and 0.61 ± 0.19 at discharge respectively by the TDI method .

Table 7. Comparison of RV function in patients without RV MI with or without hypertension.

Parameter	Before PCI			24 hours after PCI			Discharge		
	Non HTA	HTA	p	Non HTA	HTA	p	Non HTA	HTA	p
RVFAC %	40± 8.8	41± 8.8	.74	40± 6.7	41± 8.7	.55	41 ± 6	42 ± 8	.81
GLS-RV	-20± 6	-19± 6	.81	-19± 4	-19± 5	.65	-19.9± 5	-21 ± 5	.26
GLS-RVFW	-23± 7	-23± 6	.61	-22± 5	-22± 6	.81	-22± 8	-23± 7	.95
RV MPI (PW)	0.58± 0.13	0.51± 0.15	.26	0.52 ± 0.18	0.56 ± 0.21	.58	0.55± 0.17	0.54 ± 0.19	.96
RV MPI (TDI)	0.53± 0.13	0.55 ± 0.15	.64	0.57 ± 0.15	0.63 ± 0.22	.42	0.55± 0.15	0.61 ± 0.21	.37
E/A	1.08± 0.35	1.02± 0.30	.58	1.1± 0.4	1.08± 0.27	.44	1.1± 0.22	0.9 ± 0.23	.01
E/E′	5.4±1.8	4.9±1.7	.64	5.2±2.1	4.6±1.9	.35	5.1±2.0	5.8±1.6	.48

Values are number (%) or mean ± SD

Our final purpose was to assess if special cathegories of patients like hypertensive patients exhibit specific changes in RV function during acute STEMI , given the fact the in our study population hypertensive patients represent the majority (69.8 %). In this regard we compared the previously mentioned systolic and global RV functional parameters together with the diastolic RV functional parameters like tricuspid E/A ratio and E/E' ratio in hypertensive and non hypertensive patients during the serial echocardiograms. Table 7 is showing the results with no statistical differencies regarding also systolic and also diastolic function, findings that were contrary to our expectations.

Discussion

Acute myocardial infarction is associated with compensatory mechanisms involving both the left and also the right ventricle . Myocardial dysfunction in the setting of an acute MI is the result of a complex interplay of hemodynamic load and neurohormonal modulation, causing ventricular remodeling as a response to altered wall stress. Hypertrophy and apoptosis of myocites at the site of AMI and at remote sites result in biventricular remodeling [18]. RV injury following a MI appears to be common irrespective of the culprit coronary artery [19-21]. In our study, RV MPI was high on admission regardless of the site of myocardial necrosis. We measured MPI as the ratio between the sum of the isovolumic contraction and relaxation time and the ejection time, using two different methods (pulsed Doppler method and tissue Doppler method), with similar results: the RV MPI was high on admission for both patients with and without right ventricular MI, defined by electrical criteria [14]. The same results were found at 24 hours after PCI and at discharge, with both subgroups having a persistently high RV MPI. In a study conducted by Hsu and colleagues , in patients with inferior necrosis and without RV involvement only diastolic dysfunction was observed , whereas global RV dysfunction as expressed through RV MPI was more pronounced in anterior myocardial infarction [22].

This can be explained by the well-known interaction of the two ventricles, which can lead to secondary impairment of the RV in the setting of LV dysfunction. The two ventricles share common muscle fibers and contraction of the interventricular septum contributes to RV ejection; therefore, RV injury is partially explained by the infarcted myocardium in the septal segments and by myofibril interaction [23]. The two ventricles are functionally and structurally interdependent and RV function is highly influenced by changes in afterload, which also depends on left heart's hemodynamics [23]. Another explanation for RV involvement in anterior STEMI is that left anterior descending (LAD) coronary artery occlusion puts a large area of the anterior RV wall at risk [24], due to its dual coronary blood supply (RV branches from the LAD and the conus branch of the RCA).

As far as parameters of RV systolic function are concerned, there was a significant difference between patients with and patients without RV MI on admission. RV systolic function, assessed using regional parameters such as longitudinal strain of the RV free wall and global parameters such as GLS-RV and RV FAC, differed significantly between the two groups before PCI: all parameters were altered in the RV MI groups, but not in the non-RV MI group. Interestingly and unexpected , regional parameters of RV systolic function like S' velocity of the right ventricular wall were unaffected in both study groups, highlightening the fact that they are not independent parameters in describing RV function and reinforcing the need for a multiparametric approach in RV functional assessment .

Another purpose of our study was to assess the RV functional changes in patients with AMI but without RV involvement based on electrical criteria .Compared to normal values reported in the recent published guidelines [17], in patients without RV involvement only global RV function was affected but parameters reflecting systolic RV function didn't gain statistical significance. This finding can suggest that RV-MPI can became abnormal before an ejection phase measure like RV FAC indicates an abnormality, since it integrates both isoolumic and and ejection phase indices [25].

On the other hand, assessing the recovery potential of RV dysfunction in patients with AMI treated by primary PCI , GLS-RV considerably improved, while RV FAC and RV free wall strain normalized throughout hospitalization. This suggests an excellent recovery potential of RV systolic function at 7-10 days post-MI, irrespective of the electrical involvement of the RV in the acute ischemic process.

The RV has a good recovery potential after an ischemic insult [8] due to its favorable oxygen demand/supply profile (RV extracts less oxygen at rest and thereby has greater oxygen reserve during stress) and due to the low afterload , but also due to protective anatomic collaterals from the left coronary system. As a consequence, ischemic RV dysfunction normalizes rapidly [26]. Dysfunctional RV ischemic myocardium consists primarily of viable or only modestly necrotic tissue, thus indicating that the RV is more resistant to ischemic injury than the LV and that its function recovers to a greater extent than LV function.

Finally we thought to assess if hypertensive patients with AMI exhibit specific changes in RV functional parameters compared to their nonhypertensive counterparts with possible implications in the treatment of this cathegory which actually represents majority. Contrary to our expectations, in our sample size , global, systolic and diastolic parameters describing RV function didn't show statistical significance compared to nonhypertensive patients , maybe due to the fact that significant hypertensive cardiomiopathy including patients with left ventricular hypertrophy represented an exclusion criteria from the study .In addition, RV morphological and functional changes which can occur in systemic hypertension [11] may characterize later stages of hypertensive disease. To note that none of the patients included in the study developed significant pulmonary hypertension during follow up.

Study limitations

The main limitations reside in the fact that this ongoing study is a single-center experience with a small sample size and a short follow-up. Other diagnostic tools than echocardiography might be more sensitive in detecting RV subclinical dysfunction (for example, contrast-enhanced cardiac MRI, which depicts RV damage on a histological level). Future studies are needed to check if the pattern of RV functional change is the same on long-term follow-up and the prognostic significance of this findings.

Conclusions

This study reveals that RV global dysfunction is found after acute STEMI irrespective of the culprit coronary artery and of the electrical involvement of the RV in the ischemic process. As RV myocardial involvement induces an increased risk of shock, arrhythmias and in-hospital mortality [2], routine measurements of RV functional parameters should be considered in all patients with acute STEMI, regardless of the site of myocardial necrosis. Between the echocardiographic parameters, RV–MPI is a sensitive, nongeometric measurement to detect RV dysfunction to an early stage This could facilitate early identification of patients at high risk for life-threatening complications, who may need more careful monitoring and more aggressive treatment.

Our study also confirms the great recovery potential of the RV after an ischemic insult: its systolic function begins to improve 24 hours after reperfusion and normalizes 7 days after the acute ischemic event. Therefore, successful reperfusion in acute STEMI can result in quick improvement of RV performance and thus of clinical outcome. Hypertensive patients , a majority subgroup through AMI population, don't exhibit specific changes in RV function .

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Conflict of interest

The authors confirm that there are no conflicts of interest.

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