Right Ventricular Dysfunction in Right Coronary Artery Infarction: A Primary PCI Registry Analysis

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ABSTRACT

Right ventricular involvement in inferior myocardial infarction (MI) was historically associated with a poor prognosis. However, few studies addressed the impact of right ventricular (RV) dysfunction in the primary percutaneous intervention (pPCI) era. Our aim was to assess the prognostic significance of RV dysfunction in right coronary artery (RCA) related MI treated with pPCI.

Methods: A total of 298 patients with a RCA related MI undergone pPCI between January 2011 and June 2015 were included. RV dysfunction was defined by a RV-FAC <35% at echocardiographic examination and further divided into mild (RV-FAC between 35 and 25%) and moderate-severe (RV-FAC <25%). RV function before discharge was reassessed in 95% of the study cohort. The primary endpoint was overall mortality. Median follow-up was 29 months.

Results: In RCA related MI, moderate-severe (HR 5.882, $p = 0.002$, 95% CI 1.882–18.385) but not mild RV dysfunction independently predicted lower survival at follow-up along with age (HR 1.104, $p < 0.001$, CI 1.045–1.167). Importantly, patients recovering RV function at discharge showed a lower mortality ($p = 0.001$) vs patients with persistent moderate-severe RV dysfunction) that approached the risk of patients without RV dysfunction at presentation.

Conclusion: In RCA related MI treated with pPCI, RV dysfunction was one of the strongest independent predictor of lower overall survival. However, patients with only transient RV dysfunction showed a better prognosis compared to patients who had persistent RV dysfunction. The focus on intensive support management of the RV in the first hours after pPCI may be important to overcome the acute phase and to promote RV recovery.

1. Introduction

The incidence of right ventricular (RV) myocardial infarction (MI) in patients presenting with acute inferior MI varies, depending on the diagnostic criteria applied, with estimates ranging from 20% to 50% [1,2]. RV MI is associated with an increased risk of death, cardiogenic shock and arrhythmias. This identifies a high-risk subset of patients with a mortality rate as high as 25% to 30%, compared to an overall mortality rate of approximately 6% of patients with inferior MI without RV involvement [3]. Proximal right coronary artery (RCA) occlusion is the most common anatomic substrate for RV dysfunction [4–6]. Whether the adverse prognosis in patients with RVMI is simply a consequence of a larger LV infarction or whether it may be due to the presence of RV myocardial involvement itself is still debated.

Primary percutaneous coronary intervention (pPCI) leads to lower mortality rates compared to thrombolytic therapy in patients with RVMI, with a higher benefit on mortality in patients with right ventricular failure and cardiogenic shock [7].

The right ventricle is more resistant to ischemia than the left ventricle and has a tendency to improve its function, but patients with persistent RV dysfunction after MI are more prone to develop heart failure and to die in late follow-up [8].

To our knowledge, in the primary percutaneous coronary intervention (pPCI) era, only few studies have assessed the value of RV dysfunction as a prognostic factor [9,10], and even fewer studies present serial evaluations of right ventricular dysfunction.

Therefore, the aims of the present study were to assess the prognostic significance of RV dysfunction in right coronary artery related myocardial infarction, specifically addressing the role of transitory

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[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

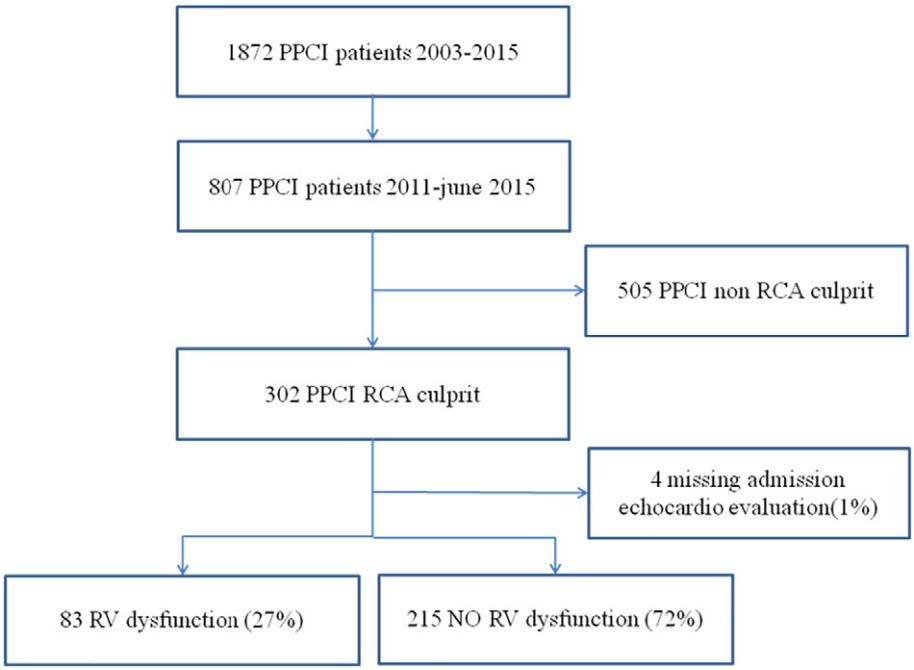

Fig. 1. Study design.

acute RV dysfunction versus persisting RV dysfunction, using a large database from a tertiary centre, representing real life patients with acute MI treated with primary PCI.

2. Methods

2.1. Study design and study population

We retrospectively analyzed all patients with ST-Segment Elevation Myocardial Infarction (STEMI) undergoing pPCI consecutively listed in the Trieste pPCI-Registry; an ongoing research database prospectively including all patients undergoing pPCI for STEMI at our Institution. Further description of the pPCI registry of Trieste has been previously published [11].

For the analysis we recruited patients from January 2011 to June 2015 (median follow up 29 months, interquartile range 17–45 months) in order to obtain a homogeneous population with regard to technical and pharmacological peri-procedural strategies. In order to assess the specific role of acute ischemic RV dysfunction we selected 302 consecutive patients with a right coronary culprit artery. Thus, we minimized the potential secondary RV dysfunction due to large LV STEMI. Among these patients, 298 (99%) had a complete echocardiographic exam at admission (Fig. 1) and were considered for the analysis. Acute STEMI was defined by: 1) presentation <12 h of typical symptoms onset; 2) an electrocardiogram (ECG) with ≥1 mm ST-segment elevation in two contiguous leads (≥2 mm in precordial leads); and 3) angiographically confirmed acute coronary artery occlusion or subocclusion (Thrombolysis In Myocardial Infarction flow grade 0 to 1).

From the apical 4-chamber, long axis parasternal, and subcostal views, RV performance was assessed by measuring ventricular size at end diastole and end systole and evaluating overall RV free wall motion. Furthermore, according to the institutional protocol, a complete echocardiographic evaluation was largely encouraged prior to discharge.

2.2. Primary endpoint

The primary outcome was overall mortality at follow-up. In-hospital mortality was considered as a secondary endpoint. Data on clinical endpoints were obtained from the regional database and from regional health-care system cardiology software. Informed consent was obtained under the institutional review board policies of hospital administration.

2.3. Echocardiographic study

Echocardiograms were recorded on digital media storage (EchoPAC, GE, Horton) at the echocardiographic core laboratory of our Institution and analyzed offline. For the present study, images were specifically reviewed by two independent observers with expertise in RV assessment, blinded to the status of the patients. In case of discordance (>5% units) a third operator was involved. Ventricular dimensions, systolic and diastolic function were assessed according to international guidelines [12]. Specifically, RV area fractional area change, tricuspid annular plane systolic movement (TAPSE), tricuspid regurgitation, pulmonary artery systolic pressure (PAPs), inferior vena cava (IVC) collapsibility were assessed at admission and discharge. RV systolic dysfunction was defined as RV fractional area change (FAC) <35%; moderate-severe RV dysfunction was defined as FAC <25%.

Mitral regurgitation (MR) and tricuspid regurgitation (TR) were assessed using a multiparametric approach [13]. Left ventricular systolic dysfunction was defined as left ventricular ejection fraction <45%.

Measurements were obtained from the mean of 3 beats (patients in-sinus rhythm) or 5 beats (patients in atrial fibrillation). Inter- and intra-observer variability regarding chamber quantification was previously reported by our group [14].

2.4. Statistical analysis

Summary statistics of clinical and instrumental variables are expressed as mean ± SD (standard deviation), medians and interquartile ranges (IQRs), or counts and percentage, as appropriate. Comparisons between groups were made by analysis of variance for continuous variables, using the Brown-Forsythe statistic when the assumption of equal variances did not hold, or by the nonparametric median test; chi-square statistics were calculated for discrete variables. Survival Kaplan-Meier curves according to baseline RV function and RV evolution between admission and discharge were calculated and subsequently compared with the log-rank test. Univariate Cox regression was then carried out to investigate the association of each of the

Table 1
Characteristics of the right coronary MI according to culprit vessel.

	Total population ($n = 807$)	Other vessel ($n = 505$)	Cdx ($n = 302$)	P
Age (years)	65 ± 12	65 ± 13	65 ± 12	0.804
Male gender (%)	76	75	77	0.337
Previous MI (%)	10	11	10	0.440
Previous revascularization (%)	9	9	10	0.463
Hypertension (%)	58	56	63	0.399
Diabetes (%)	19	18	20	0.090
Dyslipidemia (%)	59	59	58	0.449
Smoke (%)	55	52	60	0.016
Cardiac arrest (%)	11	11	11	0.452
OTI (%)	7	8	5	0.135
Killip class 3–4 (%)	11	12	10	0.178
TIMI index	26 ± 15	27 ± 15	25 ± 15	0.158
LVEF<45% b (%)	28	36	13	<0.001
Troponin peak (ng/L)	80 ± 108	96 ± 123	53 ± 67	<0.001
Glycemia adm (mg/dl)	163 ± 68	165 ± 72	161 ± 63	0.523
Renal dysfunction adm (%)	19	21	17	0.145
Multivessel disease (%)	42	42	42	0.507
Radial access (%)	79	81	76	0.087
GpIIbIIIa inhibitors (%)	25	26	23	0.196
Thrombectomy (%)	74	73	75	0.232
IABP (%)	5	6	3	0.036
TIMI flow 2-3basal (%)	25	28	21	0.021
TIMI flow 2-3final (%)	96	96	96	0.523
Ischemia time (min)*	197(145–327)	196 (142–316)	197 (147–341)	0.097

Values are expressed by mean ± SD or (*)median with inter-quartile range and by percentage (%).

observed variables with mortality. Starting from the list of statistically significant and clinically relevant variables, a multivariate Cox model was built, using the backward-conditional stepwise algorithm as a tool for covariate selection. Receiver-operating characteristic (ROC) curve analysis was used to evaluate the global accuracy of the multivariate Cox model with or without right ventricular dysfunction, and corresponding areas under the curve (AUC) were computed and compared using Delong's method. The entire analysis was performed using the SPSS version 19 (SPSS, Inc., Chicago, Illinois) and R version 2.14.1 (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Study population

Among 807 consecutive STEMI patients treated with pPCI in our department during the study period, a total of 302 (37%) patients had a culprit right coronary artery (RCA) at presentation. A complete echocardiographic examination at admission was available for 298 patients (99%) that were considered eligible for the study. With respect to non RCA culprit there was no major difference in Killip class at presentation, TIMI index, door to balloon (DTB) time, in-hospital and follow-up mortality (Table 1).

3.2. RV impairment in culprit RCA

Main characteristics of the study population divided according to the RV function are summarized in Table 2. In particular patients with severe RV dysfunction presented higher Killip class (III or IV) ($p < 0.001$), worse renal dysfunction ($p < 0.001$). Proximal right coronary artery was more frequently involved ($p < 0.001$), TIMI 2–3 flow in culprit artery was less frequent at presentation. Patients more often developed heart failure during hospital stay ($p < 0.001$) and required inotropic

Table 2
Characteristics of the right coronary MI according to RV dysfunction.

	Total population ($n = 298$)	FAC > 35 ($n = 215$)	35 ≤ FAC > 25 ($n = 47$)	FAC ≤ 25 ($n = 36$)	P
Age (years)	65 ± 12	64 ± 11	65 ± 12	68 ± 12	0.178
Male gender (%)	77	77	77	75	0.77
Previous MI (%)◇	10	10	4	20	0.065
Hypertension (%)	63	67	54	53	0.084
Diabetes (%)	19	19	15	26	0.503
Dyslipidemia (%)*,◇	58	59	46	69	0.098
Smoke (%)	61	61	63	54	0.702
Cardiac arrest (%)*	10	7	17	19	0.016
OTI (%)*	5	4	4	14	0.034
Killip Class 3–4 (%)*,◇	9	4	13	34	<0.001
TIMI index*	25 ± 15	24 ± 15	25 ± 12	33 ± 17	0.007
LVEF<45% adm (%)*	13	8	18	36	<0.001
Troponin peak ng/L*,#	54 ± 67	45 ± 66	71 ± 63	86 ± 72	0.001
Glycemia adm (mg/dl)	160 ± 61	155 ± 58	170 ± 65	196 ± 85	0.028
Creatinine adm (mg/dl)*	1.03 ± 0.59	1.01 ± 0.61	1,01 ± 0,31	1.44 ± 0.65	0.027
Renal dysf adm (%)◇,*	17	12	19	57	<0.001
Multivessel disease (%)	42	42	37	53	0.333
Proximal RCA (%)◇,#,*	36	26	51	72	<0.001
Radial access (%)*	76	81	72	53	0.001
GpIIbIIIa inhibitors (%)#	24	19	45	25	0.001
Thrombectomy (%)#	75	72	85	80	0.135
IABP (%)◇,*	3	1	0	14	0.001
TIMI 2-3basal (%)◇,*	21	25	19	3	0.013
TIMI 2-3final (%)*	96	98	94	89	0.012
Ischemia time (min)♣*	200 (146–340)	188 (144-318)	266 (150–397)	269 (199–436)	0.008
RV dilatation adm (%)#,*	6	1	13	27	<0.001
TR adm (%)#,*	24	12	58	57	<0.001
Increased PAPs a (%)◇,#,*	13	4	30	59	<0.001
IVC dilatation a (%)◇,#,*	20	4	52	79	<0.001
MR a (%)	5	3	6	11	0.1
FAC ≤ 35% dis (%)#,*	16	0	42	61	<0.001
FAC < 25% dis (%)#,*	11	0	8	25	<0.001
RV dilatation dis (%)#,*	5	0.6	11	19	<0.001
TR dis (%)#,*	15	5	42	36	<0.001
Increased PAPs dis (%)#,*	10	4	27	26	<0.001
IVC dilatation dis (%)#,*	12	5	26	33	<0.001
MR dis (%)	5	3	9	9	0.161
LVEF<45% dis (%)#,*	12	6	20	24	0.005
Heart failure (%)◇,#,*	24	13	36	72	<0.001
Bradyarrhythmia (%)#,*	24	19	32	44	0.001
PM temp(%)*	6	4	9	17	0.015
VT/VF (acute phase) (%)*	12	10	15	26	0.007
AF (acute phase) (%)*	16	13	23	28	0.024
Inotropes (%)◇,#,*	14	5	17	61	<0.001
Betablock d (%)*	78	81	74	61	0.058
ACEi d (%)*	73	77	57	50	0.011
In hospital mortality (%)◇,#,*	4	0.9	2.1	27.8	<0.001
Follow up mortality (%)◇,*	11.7	5.6	14.9	44.4	<0.001

Values are expressed by mean ± SD or (♣)median with inter-quartile range and by percentage (%).
= p value <0.05 between FAC > 35% vs FAC ≤ 35%, >25%.
* = p value <0.05 between FAC > 35% vs FAC < 25%.
◇ = p value <0.05 between FAC ≤ 35%, >25% vs FAC <25%.

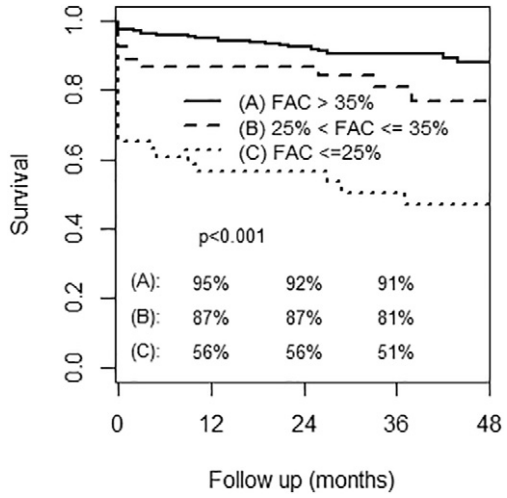

Fig. 2. Kaplan Meier survival curves according to RV dysfunction at admission.

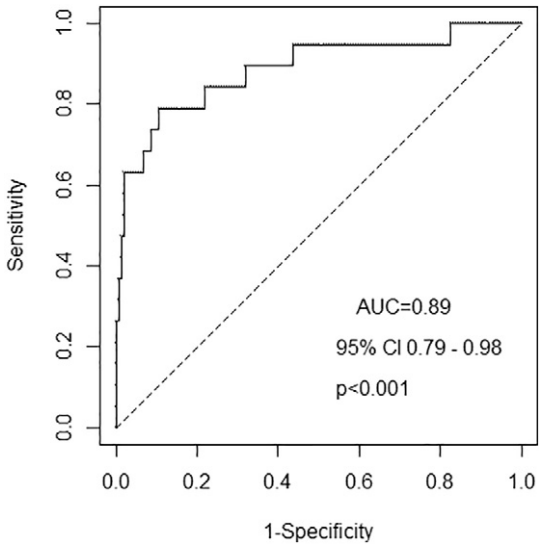

Fig. 3. ROC Curve of the multivariate model.

support ($p < 0.001$). Right ventricular dilatation ($p < 0.001$), inferior vena cava dilatation ($p < 0.001$) and a higher systolic pulmonary artery pressure ($p < 0.001$) were more frequent. In hospital mortality in the RCA culprit population was 4.4% (13 patients). Patients with moderate-severe RV dysfunction experienced a higher in-hospital mortality rate compared to patients without or with only mild RV dysfunction (27.8% vs 0.9 and 2.1%, respectively, $p < 0.001$). Overall, at 29 months follow-up, 35 patients died (11.7%), and a lower survival was observed in the group with moderate-severe RV dysfunction compared to patients without RV dysfunction (44.4% vs 5.6%, $p < 0.01$, respectively) and compared with patients with mild RV dysfunction (44% vs 14.9%, $p > 0.01$, respectively) (Fig. 2). At multivariable analysis, predictors of mortality at follow-up were moderate-severe RV dysfunction at admission (HR 4.189, $p = 0.011$, 1.393–12.597) and age (HR 1.122, $p < 0.001$, CI 1.063–1.185) (Table 3). The global model including moderate-severe RV (FAC < 25%) at admission and age showed a good accuracy for outcome prediction (AUC at ROC curve = 0.89, $p < 0.001$, 95%CI 0.79–0.98, Fig. 3).

3.3. Transient vs persistent RV impairment

Complete echocardiographic evaluation was performed <24 h prior to discharge (median time 6 days, IQR 4–9 days) in 291 patients (95%).

83 patients presented RV dysfunction at admission: 36 (43%) had a moderate-severe RV dysfunction, the remaining 47 (58%) had mild RV dysfunction (Fig. 4).

Among patients with moderate-severe RV dysfunction at presentation 8 (22.2%) died in hospital, 21 (58.3%) improved to mild or absent RV dysfunction while the RV remained severely impaired in 7 (19.5%) patients. Notably, patients that improved RV function at discharge had a survival similar to patients without RV dysfunction at presentation and significantly better compared to patients with persistent moderate-severe RV dysfunction (Fig. 5).

Table 3
Multivariate predictors of death at 29 months follow up.

	HR	P	C.I.
Age	1.122	<0.001	1.063–1.185
FAC < 25%	4.189	0.011	1.393–12.597
CKD-EPI GFR (mg/dl/1.73 m2)	0.989	0.278	0.971–1.009
LVEF<45%	0.678	0.617	0.148–3.110

HR = Hazard Ratio; C.I. = Confidence Interval.

4. Discussion

This study evaluated the prognostic significance of the persistent and transient RV dysfunction in RCA related MI. In the current era of pPCI, we found that in RCA MI, moderate-severe RV dysfunction is the strongest independent predictor of mortality. However, patients with only transient moderate-severe RV dysfunction show a better prognosis compared to patients who had persistent moderate-severe RV dysfunction. Since RV dysfunction may recovery after pPCI the focus on acute phase support is important in order to promote the recovery of RV function and improve long-term mortality.

4.1. Right ventricular dysfunction in right coronary artery myocardial infarction

RV involvement during acute inferior MI is associated with increased early morbidity and mortality [1]. According to Mehta et al., right ventricular MI seems to have an intermediate risk between anterior myocardial infarction and inferior infarction [1], while Assali et al. reported a mortality in RVMI even higher than anterior myocardial infarction [9]. However the evolution of RV function after STEMI, in the current era of new-generation stents and newer pharmacological strategies, has not been well established. Recently Smarz et al. correlated late right ventricular function (evaluated by echo) and prognosis (OR 0.56, 95% CI 0.34–0.92, $p = 0.02$) [10].

In our population of patients with a culprit RCA the presence of moderate-severe RV dysfunction produced a significant increase in incidence of in-hospital and long term mortality.

Many factors may contribute to low cardiac output in patients with RV failure such as RV systolic dysfunction, tricuspid regurgitation, ventricular interdependence, brady or tachyarrhythmias, or suboptimal preload. Hypotension may further aggravate RV dysfunction leading to RV ischemia [15]. In a previous meta-analysis, the augmented risk of RV MI was related to the presence of RV myocardial involvement itself rather than the extent of LV myocardial damage [1]. This was confirmed in the work of Assali et al. [9] as the increased risk was not related to LV infarct size and function. Indeed, in our study, using multivariable analysis, moderate-severe right ventricular dysfunction was the single strongest predictor of 2.5 years mortality in patients with RCA MI.

RV myocardial involvement was associated with an augmented incidence of life-threatening ventricular arrhythmias and severe bradyarrhythmias, as previously described [16]. Patients were more prone to acute atrial fibrillation, which in the setting of acute RV failure

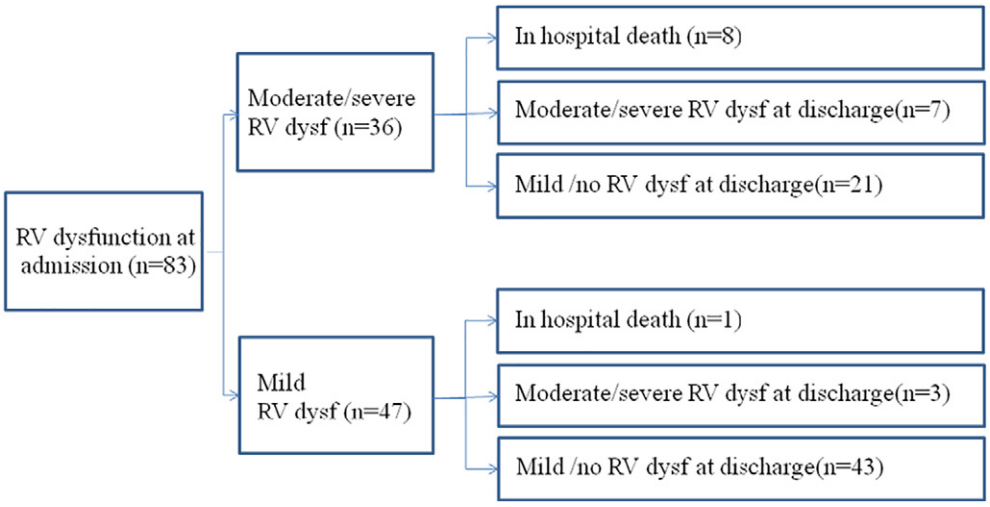

Fig. 4. Evolution of RV function in RCA MI with RV dysfunction at admission.

may lead to hemodynamic instability and an increased risk of morbidity or mortality [17].

Notably, patients who regained normal RV function or only mild dysfunction at pre-discharge echocardiography have a long-term prognosis that is not significantly different from patients without RV failure at admission. In acute MI the RV shows a remarkable ability to regain systolic function, highlighting its resistance to irreversible ischemic injury, and the role of myocardial-stunning in the pathophysiology of RV dysfunction [15].

The need for a prompt and complete coronary reperfusion is the goal of treatment, as complete revascularization of RCA is independently associated with improved mortality and the best chances of recovery of RV function [4,18,19]. Our data also emphasize the importance of intensive care management of acute RV dysfunction to support RV recovery in the early phase post reperfusion.

Further characterization of this subgroup of patients may help define optimal management strategies of RV failure in this setting.

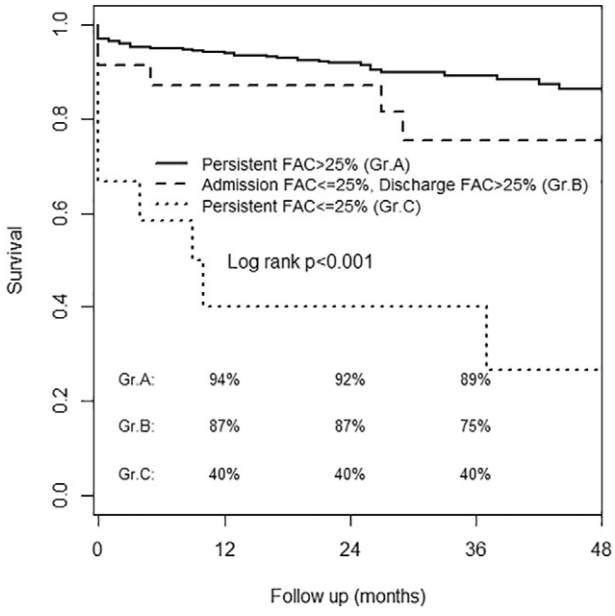

Fig. 5. Kaplan Meier survival curves in patients with transient vs persistent RV dysfunction.

The role of echocardiography is increasingly recognized in assessing RV function in intensive care unit (ICU) settings.

In this set of high risk patients, prompt echocardiographic evaluation is of definite value in RV failure diagnosis and in guiding therapy in the ICU [20,21].

The prognostic role of RV recovery versus persistent dysfunction at discharge needs to be validated in larger groups, as further characterization of the factors associated with early RV recovery may lead to new therapeutic goals in the acute management of RV dysfunction in STEMI.

4.2. Limitations

Limitations in the present study need to be acknowledged. Our findings should be interpreted in light of the common limitations of retrospective observational studies, The principal outcome of the study was total mortality, and follow up revascularization and repeated hospitalization were not considered. The assessment of RV function was obtained by 2D echocardiography evaluation of RV-FAC using a dichotomic cut-off point of 35%, according to current recommendations [12] However, despite the limitations related to this approach [12] it has been shown to correlate reasonably well with cardiac magnetic resonance [22]. Other echocardiographic indices of RV function similarly suffer from several limitations. Tricuspid annular plane systolic excursion (TAPSE) is limited to the assessment of longitudinal systolic function that is highly dependent on RV loading conditions and confounded by significant LV systolic impairment and tricuspid regurgitation [23]. Moreover, it disregards the contribution of the interventricular septum and the RV outflow tract to overall RV function. There is a growing body of evidence on emerging echocardiographic techniques, such as 3D echocardiography and RV strain, that should actually improve the assessment of RV function in the future.

5. Conclusion

In RCA MI treated with pPCI, moderate-severe RV dysfunction is the strongest independent predictor of in hospital and medium-term mortality. However, patients with only transient moderate-severe RV dysfunction show a better prognosis compared to patients who had persistent RV dysfunction. The focus on RV acute phase support management is important in order to facilitate RV recovery and improve patients' outcome. Furthermore, extensive characterization of these patients may lead to new therapeutic goals in the ICU management of RV failure.

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