

Time Dependence of the Effect of Right Ventricular Dysfunction on Clinical Outcomes After Myocardial Infarction: Role of Pulmonary Hypertension

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Background—The clinical importance of right ventricular (RV) function in acute myocardial infarction is well recognized, but the impact of concomitant pulmonary hypertension (PH) has not been studied.

Methods and Results—We studied 1044 patients with acute myocardial infarction. Patients were classified into 4 groups according to the presence or absence of RV dysfunction and PH, defined as pulmonary artery systolic pressure >35 mm Hg: normal right ventricle without PH (n=509), normal right ventricle and PH (n=373), RV dysfunction without PH (n=64), and RV dysfunction and PH (n=98). A landmark analysis of early (admission to 30 days) and late (31 days to 8 years) mortality and readmission for heart failure was performed. In the first 30 days, RV dysfunction without PH was associated with a high mortality risk (adjusted hazard ratio 5.56, 95% CI 2.05–15.09, $P<0.0001$ compared with normal RV and no PH). In contrast, after 30 days, mortality rates among patients with RV dysfunction were increased only when PH was also present. Compared with patients having neither RV dysfunction nor PH, the adjusted hazard ratio for mortality was 1.44 (95% CI 0.68–3.04, $P=0.34$) in RV dysfunction without PH and 2.52 (95% CI 1.64–3.87, $P<0.0001$) in RV dysfunction with PH. PH with or without RV dysfunction was associated with increased risk for heart failure.

Conclusion—In the absence of elevated pulmonary pressures, the risk associated with RV dysfunction after acute myocardial infarction is entirely confined to the first 30 days. Beyond 30 days, PH is the stronger risk factor for long-term mortality and readmission for heart failure. (*J Am Heart Assoc.* 2016;5:e003606 doi: 10.1161/JAHA.116.003606)

Key Words: heart failure • myocardial infarction • pulmonary hypertension • right ventricle infarction

The clinical importance of right ventricular (RV) function in heart failure (HF), pulmonary hypertension (PH), and acute myocardial infarction (AMI) is increasingly being recognized.^{1,2} Recent studies have shown that RV function is a significant independent predictor of a broad spectrum of cardiovascular outcomes in patients with left ventricular (LV) dysfunction and/or HF complicating AMI.^{1,2} An involvement of the right ventricle during inferior AMI is a strong predictor of major complications and in-hospital mortality.³ RV

dysfunction, however, is not uncommon in patients with anterior infarction, especially when moderate to severe LV dysfunction is present.^{2,4}

The right ventricle can accommodate large changes in volume loading but has limited contractile reserve to compensate for increased afterload.⁵ The ability of the right ventricle to adapt to the increase in pulmonary pressures secondary to changes in LV function in the setting of AMI may be an important determinant of clinical outcome. Despite the inextricable relationship of the right ventricle with the pulmonary circulation, previous studies in AMI have analyzed the impact of RV dysfunction in isolation.^{1,2} We sought to study the interrelationship between RV function and pulmonary artery pressure in patients with AMI.

Methods

The study cohort consisted of patients enrolled in a prospective, single-center, longitudinal observational study designed to determine predictors of postinfarction HF, with data collection starting in 2001.^{6,7} Consecutive patients were

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enrolled until the end of 2009. All patients presenting to the intensive coronary care unit with AMI were eligible for entry into the study. For the present analysis, exclusion criteria included patients with previous HF and patients in whom tricuspid regurgitation jets were not analyzable. The institutional human research committee approved the study protocol. The need to obtain written informed consent was specifically waived.

RV infarction was diagnosed in patients with acute inferior myocardial infarction (ST-segment elevation of ≥ 1 mV in ≥ 2 of leads II, III, and aVF) with ST-segment elevation of ≥ 0.1 mV in lead V4R at admission.⁸

Echocardiography

All echocardiographic studies were performed during hospital stay by an experienced, certified sonographer. The studies were performed after a median of 2 days from admission (interquartile range 1–3 days). Analysis of estimated pulmonary artery systolic pressure (PASP), LV function, and presence and degree of mitral regurgitation was carried out by one of 6 experienced noninvasive cardiologists (Y.A., D.M., J.L., D.A., S.R., and S.Y.) without knowledge of the patient outcome.

Echocardiograms were performed in multiple views to obtain the optimally appearing tricuspid regurgitation jet. The estimated PASP was calculated as the sum of the peak systolic pressure gradient across the tricuspid valve (approximated by the modified Bernoulli equation) and the right atrial pressure.⁹ Right atrial pressure was estimated according to the size and respiratory variation of the inferior vena cava diameter in the subcostal view using established criteria.⁹ PH was defined as an estimated PASP ≥ 35 mm Hg.⁷ LV wall-motion score index was calculated according to a 16-segment model.¹⁰

RV systolic function was assessed qualitatively by integrating visual assessment of the contractility of the RV walls from different views and classified on an ordinal scale (normal or mildly, moderately, or severely reduced, as described previously)^{11–13} and quantitatively by calculating the RV fractional area change, with a value $< 35\%$ defined as abnormal.^{9,12,13}

The interobserver variability in the assessment of RV function was evaluated by comparing the primary reader assessment with that of a second experienced physician for 150 randomly selected patients. Both readers had no knowledge of the outcome data. The agreement in assessment of RV function (normal or reduced) between echo readers was high (Cohen's κ 0.80, 95% CI 0.64–0.95).

Study End Points

The primary end point of the study was all-cause mortality. The secondary end point was readmission for HF. Following hospital discharge, clinical end point information was acquired

by reviewing the national death registry and by reviewing the hospital records for major clinical events if the patient had been rehospitalized.

Statistical Analysis

Continuous variables are presented as mean (SD) or median (interquartile range), and categorical variables are presented as numbers and percentages. The baseline characteristics of the groups were compared using ANOVA for continuous variables and by χ^2 statistic for categorical variables. LV wall-motion score index values of patients with RV dysfunction due to acute RV infarction and other causes were compared using Wilcoxon rank-sum tests.

Follow-up began on the date of admission and ended on the date of death or after 8 years (to ensure that at least 10% of the study sample remained at risk), whichever came first, with minimum follow-up of 5 years for all patients. To provide insight into differences in the early and late mortality rates in patients with normal and reduced RV function, we performed a landmark survival analysis¹⁴ with a landmark set at 30 days. The landmark analysis was preferable because of the observation that there was evidence of nonproportional hazards in the effect of RV function in the proportional hazards model ($P=0.0025$), based on the test using Schoenfeld residuals. With the landmark approach, the follow-up time is divided into periods of interest. Patients whose survival is shorter than the landmark point (in our case, a period of 30 days) are excluded from subsequent analysis. Surviving patients are then followed to establish whether the prognostic factor (eg, RV function, PH) had a significant effect on survival. Patient survival is then described with standard techniques conditional on the patient being alive at the start of the period.

The rationale for selection of the 30-day cut point for the landmark analyses was to separate early events that could be attributed to an acute severe ischemic RV injury and the generally favorable outcomes of patients surviving the early high-risk period.^{4,15,16} The 30-day threshold is frequently used to assess short-term outcome measures in patients with ST-segment elevation AMI.¹⁷

Survival curves were constructed using the Kaplan–Meier method, and comparisons were made using the log-rank test. Stepwise Cox proportional hazards models with backward selection were used to calculate hazard ratios and 95% CIs for PH and other risk variables. The following variables were considered in the multivariable procedure: age, sex, history of prior infarction, history of diabetes, history of hypertension, serum creatinine, Killip class on admission, ST-segment elevation infarction, anterior location of infarction, coronary revascularization during hospital course, and LV ejection fraction.

Because an assessment of the true reduction in HF hospitalization rates must consider the competing risk of death, survival free of HF was assessed treating death as a competing risk, using the Fine and Gray regression model (proportional subhazard model).¹⁸ The cumulative incidence function was used to display the proportion of patients with readmission for HF as time progressed.

The relation between the estimated PASP as a continuous variable and HF was also assessed with cubic splines. The restricted cubic spline function allowed us to explore nonlinear relationships between estimated PASP and HF. Differences were considered statistically significant at the 2-sided $P<0.05$ level. Statistical analyses were performed using Stata version 13.1 (StataCorp).

Results

Between July 2001 and July 2009, a total of 1593 patients fulfilled the study entry criteria. Of these, PASP could be

estimated in 1044 patients (66%) who composed the study population. Compared with participants for whom PASP was available, patients with missing PASP data were younger (aged 60 ± 12 versus 64 ± 13 years; $P<0.0001$), were less likely to have reduced LV ejection fraction ($P<0.05$), and were more likely to undergo percutaneous revascularization ($P<0.001$). Mortality was lower among patients with missing PASP (log-rank $P<0.0001$).

Median estimated PASP in the overall study population was 35 mm Hg (interquartile range 29–43 mm Hg). The characteristics of the study participants according to the presence or absence of RV dysfunction and PH are summarized in Table 1. Patients with PH, with or without RV dysfunction, were more likely to be older and male, had worse renal function, and were more likely to have had a previous myocardial infarction and a history of diabetes and hypertension; they presented with higher Killip class. Patients with PH also had worse LV systolic function.

Table 1. Baseline Clinical Characteristics According to Pulmonary Hypertension and RV Dysfunction Category

Characteristic	Normal PASP/Normal Right Ventricle (n=509)	Elevated PASP/Normal Right Ventricle (n=373)	Normal PASP/RV Dysfunction (n=64)	Elevated PASP/RV Dysfunction (n=98)	P Value
Age, y	60±12	69±11	61±12	69±12	<0.0001
Female sex	117 (23)	138 (37)	15 (23)	31 (32)	<0.0001
History of hypertension	243 (48)	236 (63)	33 (51)	52 (53)	<0.0001
Diabetes mellitus	122 (24)	122 (33)	13 (20)	38 (39)	0.001
Current smoker	101 (20)	61 (16)	12 (19)	19 (19)	0.61
Previous infarction	96 (19)	95 (25)	8 (13)	21 (21)	0.03
Creatinine, mg/dL	1.0±0.5	1.2±0.7	1.1±0.4	1.3±0.8	<0.0001
eGFR, mL·min ⁻¹ ·1.73 m ⁻²	91±38	72±31	80±29	66±26	<0.0001
ST-segment elevation myocardial infarction	431 (85)	298 (80)	61 (95)	80 (81)	0.01
Coronary revascularization	289 (57)	163 (44)	27 (42)	36 (37)	<0.0001
Killip class >I	59 (12)	150 (40)	8 (13)	45 (46)	<0.0001
LV wall motion score index	1.6±0.4	1.8±0.4	1.7±0.4	1.9±0.4	<0.0001
LVEF <45%	144 (28)	223 (60)	26 (41)	70 (71)	<0.0001
PASP, mm Hg	29±4	46±9	28±5	48±11	<0.0001
RV infarction	33 (6)	10 (3)	50 (78)	47 (48)	<0.0001
Medications					
Beta blockers	453 (89)	323 (87)	55 (86)	79 (81)	0.14
ACEIs/ARBs	441 (87)	320 (86)	56 (88)	70 (71)	0.001
Antiplatelet agents	504 (99)	360 (97)	63 (98)	91 (93)	0.002
Statins	444 (83)	275 (74)	52 (81)	66 (67)	<0.0001

Values are expressed as number (%) of patients or mean value±SD. ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; eGFR, estimated glomerular filtration rate; LV, left ventricular; LVEF, left ventricular ejection fraction; PASP, pulmonary artery systolic pressure; RV, right ventricular.

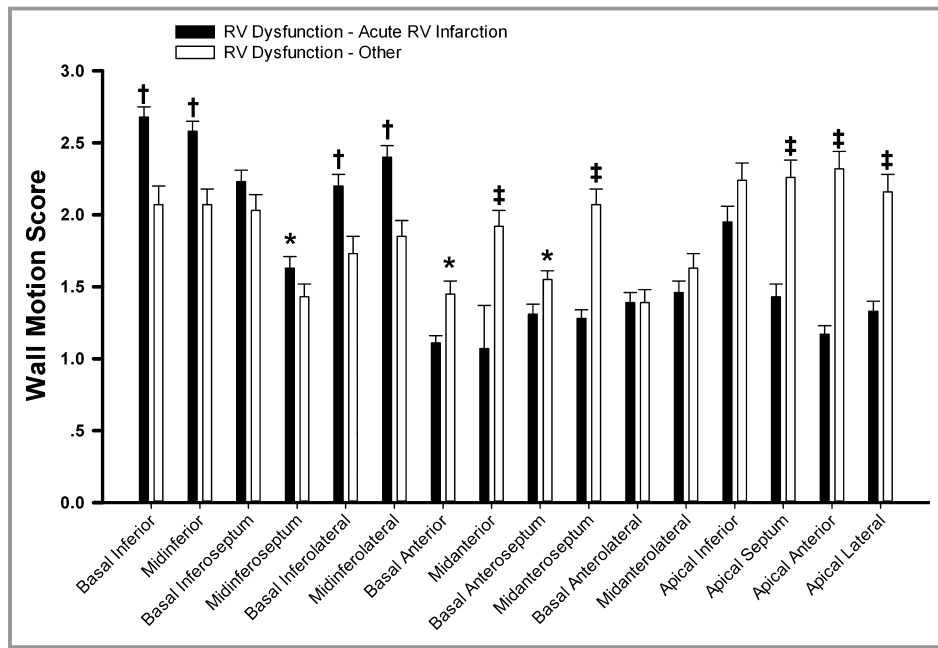


Figure 1. Comparison of regional left ventricular wall motion score (16-segment model)¹⁰ in patients with RV dysfunction in the setting of acute RV infarction and RV dysfunction from other causes. Each segment received a score of 1–5, and the bars represent the mean±SE of the score for each segment. **P*<0.01; †*P*<0.001; ‡*P*<0.0001. RV indicates right ventricular.

Of the 162 patients with RV dysfunction, 97 (59.9%) presented with acute RV infarction (Table 1). As expected, regional LV function in the inferior, inferolateral, and inferior

septum segments was decreased in patients with RV dysfunction in the setting of acute RV infarction (Figure 1), whereas the anterior, anteroseptal, and apical segments were

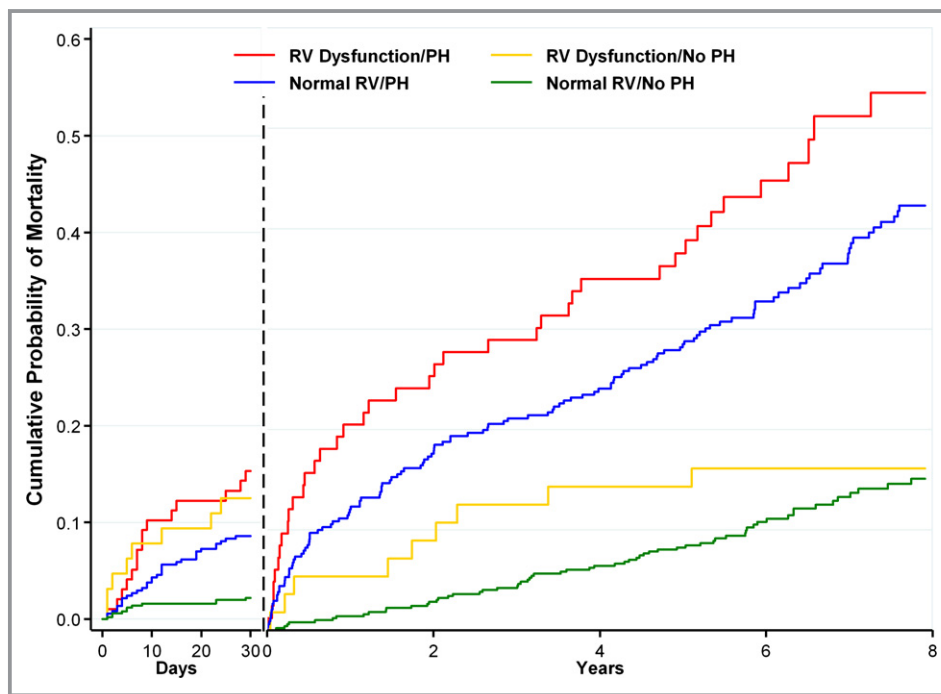


Figure 2. Landmark analyses (Kaplan–Meier estimates) of the cumulative probability of mortality during the first 30 days (left side panel) and beyond 30 days to 8 years (right side panel) for each of the 4 study groups. PH indicates pulmonary hypertension; RV, right ventricular.

reduced with RV dysfunction that was unrelated to acute RV infarction.

Landmark Analysis of the Relationship Between RV Dysfunction, PH, and Mortality

During a mean follow-up of 77±35 months, 305 patients died (29.2%), with 66 deaths occurring in the first 30 days. The landmark analysis for mortality from study entry to 30 days is shown in the left panel of Figure 2. Mortality was low among patients with both normal RV function and normal PASP (2.2%). Mortality rates in patients with RV dysfunction and normal PASP were particularly high in the first 30 days (12.5%) and similar to the mortality rates of patients with RV dysfunction and PH (15.3%), whereas patients with PH and normal RV function were at intermediate risk (8.6%).

Model 1 in Table 2 displays the results of the Cox proportional hazards model examining the relationship

between RV dysfunction and PH categories with the risk of 30-day mortality. The association between PH (with and without RV dysfunction) and mortality was greatly attenuated after adjustments for the differences in baseline characteristics such that only RV dysfunction with normal PASP remained a strong independent predictor of 30-day mortality.

In contrast, landmark analysis performed after the first 30 days demonstrated that the association between RV dysfunction without PH and mortality was weaker (Figure 2, right panel). A test for interaction between the effect of isolated RV dysfunction and time (0–30 days and 31 days to 8 years) was statically significant ($P=0.01$) such that beyond 30 days, there was no significant effect of RV dysfunction on mortality (model 2 in Table 2). PH patients displayed a progressive increase in mortality, with the highest mortality risk in patients with both PH and RV dysfunction (Figure 2, right panel; model 2 in Table 2).

Because the dichotomization of PASP to define PH is somewhat arbitrary, PASP was also used as a continuous

Table 2. Unadjusted and Adjusted Cox Proportional Hazards Model for 30-Day All-Cause Mortality

Characteristic	Unadjusted		Adjusted	
	HR (95% CI)	P Value	HR (95% CI)	P Value
Model 1: entry to 30 days				
RV function and PH category				
Normal right ventricle, no PH	1.0 (Referent)	—	1.0 (Referent)	—
Normal right ventricle, PH	4.84 (2.24–10.47)	<0.0001	1.43 (0.64–3.22)	0.39
RV dysfunction, no PH	8.23 (3.09–21.92)	<0.0001	5.56 (2.05–15.09)	<0.0001
RV dysfunction, PH	9.26 (3.93–21.83)	<0.0001	1.44 (0.55–3.77)	0.46
Age (per 10-year increase)	2.28 (1.79–2.88)	<0.0001	1.88 (1.45–2.42)	<0.0001
Diabetes mellitus	1.91 (1.17–3.11)	0.01	1.68 (1.00–2.80)	0.048
Primary PCI	0.34 (0.18–0.62)	<0.0001	0.37 (0.19–0.69)	0.002
Killip class >I	2.48 (2.04–3.01)	<0.0001	2.26 (1.77–2.88)	<0.0001
LVEF <45%	4.09 (2.33–7.18)	<0.0001	2.08 (1.13–3.81)	0.018
Model 2: 31 days to 8 years				
RV function and PH category				
Normal right ventricle, no PH	1.0 (Referent)	—	1.0 (Referent)	—
Normal right ventricle, PH	3.24 (2.37–4.43)	<0.0001	1.89 (1.37–2.60)	<0.0001
RV dysfunction, no PH	1.32 (0.63–2.77)	0.46	1.44 (0.68–3.04)	0.34
RV dysfunction, PH	4.91 (3.27–7.37)	<0.0001	2.52 (1.64–3.87)	<0.0001
Age (per 10-year increase)	2.03 (1.81–2.28)	<0.0001	1.73 (1.52–1.96)	<0.0001
Diabetes mellitus	2.00 (1.55–2.60)	<0.0001	1.68 (1.29–2.19)	<0.0001
eGFR <60 mL·min ⁻¹ ·1.73 m ⁻²	2.79 (2.16–3.61)	<0.0001	1.36 (1.03–1.79)	0.03
Coronary revascularization	0.35 (0.26–0.46)	<0.0001	0.43 (0.32–0.58)	<0.0001
LVEF <45%	2.22 (1.72–2.88)	<0.0001	1.61 (1.23–2.10)	<0.0001

eGFR indicates estimated glomerular filtration rate; HR, hazard ratio; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; PH, pulmonary hypertension; RV, right ventricular.

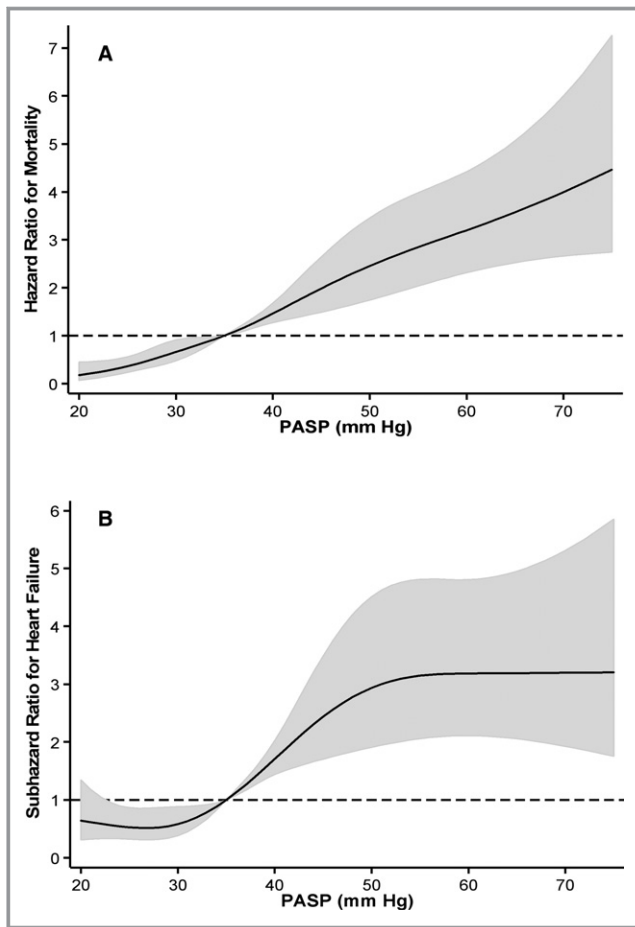


Figure 3. Cubic spline analysis showing the change in (A) the hazard ratio for mortality and (B) the subhazard ratio for readmission for heart failure with increasing estimated PASP. PASP indicates pulmonary artery systolic pressure.

variable in a cubic spline analysis. Figure 3A demonstrates that the relationship between PASP and the hazard ratio of mortality was nearly linear (Figure 3A).

Effect of RV Dysfunction and PH on Readmission for HF

During the follow-up period, 199 patients were readmitted for HF (19.1%). The cumulative incidence of readmission for HF over the follow-up period is shown in Figure 4. RV dysfunction without PH was not associated with readmission for HF, whereas PH was associated with increased risk of HF regardless of RV function. Similar results were obtained after adjustments for potential confounders in a multivariable model (Table 3). Using PASP as a continuous variable demonstrated that the subhazard ratio of readmission for HF during follow-up increased sharply, with estimated PASP >30 mm Hg reaching a plateau at \approx 50 mm Hg (Figure 3B).

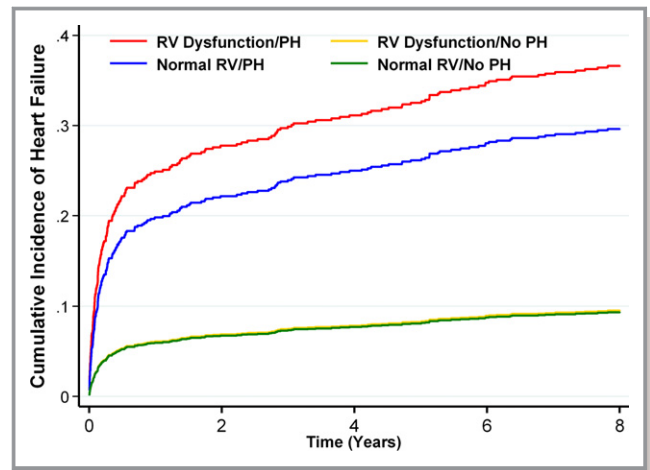


Figure 4. Cumulative incidence of readmissions due to heart failure in the 4 study groups. The cumulative incidence function indicates the probability of observing a heart failure event before a given time. PH indicates pulmonary hypertension; RV, right ventricular.

Discussion

The results of the present study demonstrate a complex and time-dependent relationship among RV function, PH, and clinical outcomes after AMI. The impact of RV dysfunction was derived mainly from the events occurring within the first 30 days after the infarct, without a significant effect on long-term mortality. Beyond the 30-day landmark, PH was the dominant risk factor for long-term mortality. In addition, PH, a surrogate of increased left atrial pressures, was a strong independent predictor of readmission for HF, whereas RV dysfunction without PH was not.

RV Dysfunction and Short-Term Outcome After AMI

Previous studies in patients with LV dysfunction complicating AMI have shown that RV systolic dysfunction is an independent predictor of death and the development HF, but the impact of PH was not considered.^{1,2} The present study reveals a complex relationship between RV function and clinical outcomes that is both time dependent and modified by the presence of concomitant PH. The impact of RV dysfunction without PH on mortality was restricted to the first 30 days after the infarct, without any effect on long-term mortality or the risk of readmission for HF.

Within the early 30-day period, absolute mortality was high among patients with RV dysfunction, regardless of the presence of PH; however, after multivariate adjustments, the increased crude mortality of patients with RV dysfunction and PH was fully explained by other influential confounders,

Table 3. Unadjusted and Adjusted Competing Risk Regression Analysis for Heart Failure

Characteristic	Unadjusted		Adjusted	
	sHR (95% CI)	P Value	sHR (95% CI)	P Value
RV function and PH category				
Normal right ventricle, no PH	1.0 (Referent)	—	1.0 (Referent)	—
Normal right ventricle, PH	3.36 (2.38–4.75)	<0.0001	2.10 (1.44–3.06)	<0.0001
RV dysfunction, no PH	1.12 (0.47–2.67)	0.79	1.03 (0.42–2.48)	0.96
RV dysfunction, PH	4.25 (2.69–6.70)	<0.0001	2.32 (1.38–3.88)	0.002
Age (per 10-year increase)	1.55 (1.38–1.75)	<0.0001	1.30 (1.13–1.50)	<0.0001
Previous infarction	2.02 (1.51–2.71)	<0.0001	1.64 (1.20–2.22)	0.002
Diabetes mellitus	1.73 (1.30–2.29)	<0.0001	1.38 (1.03–1.85)	0.03
eGFR <60 mL·min ⁻¹ ·1.73 m ⁻²	2.31 (1.74–3.06)	<0.0001	—	—
Coronary revascularization	0.62 (0.47–0.82)	0.001	—	—
Killip class >I at admission	2.59 (1.95–3.42)	<0.0001	1.43 (1.03–2.00)	0.04
LVEF <45%	2.69 (2.00–3.60)	<0.0001	1.61 (1.16–2.56)	0.005

eGFR indicates estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; PH, pulmonary hypertension; RV, right ventricular; sHR, hazard ratio of the subdistribution hazard function.

including older age, worse renal function and Killip class, and LV systolic dysfunction, whereas the relative risk of mortality was highest among patients with RV dysfunction without PH. These patients had an unexpectedly high relative risk of mortality, given their more favorable risk profile.

The finding of higher early relative risk of mortality in patients with RV dysfunction without PH compared with patients with RV dysfunction and PH may seem counterintuitive; however, patients with RV dysfunction and normal PASP may be at high risk from a predominantly severe irreversible RV impairment secondary to RV infarction,¹⁵ as 78% of patients in this group presented with acute RV infarction. In severe RV dysfunction, a decrease in pulmonary arterial pressure may occur as a consequence of low cardiac output due to RV failure.⁵ The absence of PH may represent severe RV dysfunction with elevated early mortality risk.

In the present study, only 60% of patients with RV dysfunction had acute RV infarction in the setting of a typical acute inferior/posterior infarction, indicating that other mechanisms contribute to impaired RV function in patients with AMI. In AMI, RV ischemic injury can occur in patients with anterior LV infarcts⁴ because the RV free wall may be partially supplied by right-sided branches of the left anterior descending coronary artery or when the left anterior descending artery wraps around the apex and ascends into the posterior interventricular groove to supply part of the apical and inferior RV wall.^{4,19,20} RV function may also be impaired because of other mechanisms including ventricular

interdependence associated with septal dysfunction and limited pericardial flexibility.

RV Function and Long-Term Outcome After AMI

Beyond 30 days, PH determines the long-term clinical course of patients with impaired RV function, with regard to both mortality and HF events. Importantly, long-term mortality was highest when both RV dysfunction and PH were present. An increase in pulmonary pressures with RV dysfunction results in pressure overload on the RV, exacerbating the hemodynamic compromise associated with RV dysfunction and increasing myocardial oxygen consumption, leading to RV ischemia, which may further aggravate RV dysfunction.⁵ If persistent, PH may induce adverse RV remodeling and functional tricuspid regurgitation, further aggravating the initial ischemic injury. Consequently, ischemic RV dysfunction that occurs in patients without clinical evidence of hemodynamic RV compromise in the acute phase is important with respect to long-term clinical outcome when accompanied by PH. The increased risk of long-term mortality associated with RV dysfunction in the presence of PH underscores the profound clinical consequences of an acute pressure overload on the ischemic RV and thus the importance of assessment of both RV function and PASP in the setting of AMI.

The lack of association between isolated RV function and clinical events following the 30-day landmark may be related to the ability of RV function to recover after an initial ischemic injury.¹⁵ Alternatively, the dysfunctional right ventricle may

compensate successfully in the absence of increased afterload.¹³

PH in AMI

A considerable proportion of patients with elevated left-sided filling pressures develop PH.²¹ The prognostic importance of PH has been studied mainly in the setting of established HF or valvular diseases.^{13,22,23} Few data are available on the relationship between PH and clinical outcomes in patients with AMI.^{7,24}

There were marked differences in the clinical profiles of patients with and without PH. Regardless of RV function, PH patients were significantly older and had poorer renal function, higher Killip class, higher likelihood of prior MI, and higher rates of diabetes and hypertension. In addition, the presence of PH was associated with worse LV systolic function. These differences in baseline characteristics represent comorbidities predisposing to higher left-sided filling pressures, leading to secondary PH.⁷ Importantly, a single estimation of PASP shortly after admission was strongly associated with long-term clinical outcome. These results demonstrate the importance of PH as a hemodynamic risk factor after AMI and the long-term implications of increased afterload in the pulmonary vascular system when RV dysfunction is present.

Study Limitations

It is important to consider several limitations pertinent to the methods of this study. This was a single-center study, and the results cannot be generalized to a broader population without external validation. Assessment of RV function is complicated because of the complexity of the RV geometry. In the current study, we used qualitative estimation by visual assessment and calculated RV fractional area change to estimate RV systolic function and RV size. These techniques are accepted⁹ as proper estimations of RV function and have been used in previous studies^{1,2,12} but remain imprecise. Importantly, RV fractional area change as a measure of RV systolic function has been validated against magnetic resonance imaging²⁵ and is related to clinical outcomes in the setting of AMI.^{1,2}

PH has been estimated to be present in >40% of patients following AMI⁷; however, PH could be a result of separate etiologies such as sleep apnea,²⁶ pulmonary embolism, or chronic lung disease. Chronic lung disease was not associated with higher PASP in a large studies of patients with chronic HF.^{27,28} Given our study population, it is likely that the major reason for PH was elevated left-sided filling pressures.²⁹ Notwithstanding, the current study did not exclude or account for these variables because our goal was to examine the absolute prognostic value of PH (as measured in the setting of acute infarction) regardless of cause.

The right ventricle has an immense ability to adapt and remodel following ischemic insult; therefore, echocardiographic follow-up of RV several months after AMI would allow for assessment of RV remodeling and adaptation and thereby enable better understanding of the impact of PH on RV function.

Conclusion

In the absence of elevated pulmonary pressures, RV dysfunction is a risk factor for mortality mainly in the first 30 days after the index event. Beyond 30 days, PH is a determinant the long-term clinical course of patients with impaired RV function, both with regard to mortality and HF events.

Disclosures

None.

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