Tricuspid annular plane systolic excursion/pulmonary arterial systolic pressure ratio as a predictor of outcome in acute heart failure

Le rapport excursion systolique du plan de l'anneau tricuspide/ la pression artérielle pulmonaire systolique comme prédicteur du pronostic dans l'insuffisance cardiaque aiguë

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Abstract

Introduction: Acute heart failure (AHF) is a life-threatening condition that requires swift diagnosis and tailored management to enhance patient outcomes. In the pursuit of more precise prognostic indicators, Tricuspid Annular Plane Systolic Excursion (TAPSE) and Pulmonary Arterial Systolic Pressure (PASP) have emerged as potential significant advancements. The TAPSE/PASP ratio, a novel parameter, has recently gained attention as a promising predictor of outcomes in acute heart failure.

Aim: This study delves into the significance of TAPSE/PASP as a predictive tool, shedding light on its potential to revolutionize the landscape of AHF management.

Methods: We included 152 patients with AHF. Echocardiographic evaluation for left ventricle systolic and diastolic function was performed at the time of admission. RV functions were evaluated by calculating the following (TAPSE, PASP, TAPSE/PASP ratio). Data were analyzed to find the predictors of mortality and/or rehospitalization.

Results: The TAPSE/PASP ratio emerged as a significant independent predictor of clinical outcomes in AHF patients (HR=2.6; 95%CI: 1.04-6.47; p=0.04). Furthermore, it was the sole predictor of rehospitalization for AHF (HR=3.97; 95%CI: 1.38-11.40; p=0.01). It also independently predicted all-cause mortality in AHF, with an HR of 2.73 (95% CI: 1.25-9.12; p=0.03). When evaluating its predictive accuracy, the TAPSE/PASP ratio with a cutoff value <0.35 mm/mmHg demonstrated a sensitivity of 65%, specificity of 70%, and an area under the receiver operating characteristic curve of 0.70 for forecasting adverse outcomes.

Conclusion: The non-invasive TAPSE/PASP ratio is an independent predictor of mortality and /or rehospitalization in patients with acute heart failure.

Key words: TAPSE/PASP ratio, mortality, rehospitalization, acute heart failure

Résumé

Introduction: L'insuffisance cardiaque aiguë (ICA) est une condition grave qui nécessite un diagnostic rapide et une prise en charge adaptée pour améliorer le pronostic des patients. Dans la recherche d'indicateurs pronostiques plus précis, l'excursion systolique du plan de l'anneau tricuspide (TAPSE) et la pression artérielle pulmonaire systolique (PASP) ont émergé comme des paramètres significatifs. Le rapport TAPSE/PASP, un nouvel indicateur, a récemment attiré l'attention en tant que prédicteur prometteur des issues cliniques dans l'ICA. Cette étude explore la pertinence du rapport TAPSE/PASP comme outil prédictif, en mettant en lumière son potentiel pour transformer la prise en charge de l'ICA.

Méthodes: Nous avons inclus 152 patients atteints d'ICA. Une évaluation échocardiographique de la fonction systolique et diastolique du ventricule gauche a été réalisée à l'admission. Les fonctions du ventricule droit ont été évaluées à l'aide des paramètres suivants : TAPSE, PASP et rapport TAPSE/PASP. Les données ont été analysées pour identifier les facteurs prédictifs de mortalité et/ou de réhospitalisation.

Résultats: Le rapport TAPSE/PASP s'est révélé être un prédicteur indépendant significatif des issues cliniques chez les patients atteints d'ICA (HR = 2,601 ; IC 95 % : 1,044-6,477 ; p = 0,04). De plus, il a été le seul prédicteur de la réhospitalisation pour ICA (HR = 3,975 ; IC 95 % : 1,386-11,401 ; p = 0,010). Il prédit également de manière indépendante la mortalité toutes causes confondues chez les patients avec un HR de 2,735 (IC 95 % : 1,250-9,124 ; p = 0,031). En termes de précision prédictive, le rapport TAPSE/PASP avec une valeur seuil < 0,35 mm/mmHg a montré une sensibilité de 65 %, une spécificité de 70 %, et une aire sous la courbe ROC de 0,701 pour prédire des issues défavorables.

Conclusion: Le rapport TAPSE/PASP, un paramètre non invasif, constitue un prédicteur indépendant de mortalité et/ou de réhospitalisation chez les patients atteints d'insuffisance cardiaque aiguë.

Mots clés: rapport TAPSE/PASP, mortalité, réhospitalisation, insuffisance cardiaque aiguë

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Introduction

Acute heart failure (AHF) is associated with a grim prognosis, characterized by a significant increase in both mortality and rehospitalization (1, 2). Pulmonary hypertension emerges as the primary factor in the occurrence of right ventricular failure (RVF) in patients with heart failure. Both pulmonary hypertension and RVF contribute to worsening clinical outcomes in HF patients (3, 4).

Among the echocardiographic variables used for assessing RV systolic function, tricuspid annular plane systolic excursion (TAPSE) is readily obtainable, reproducible, and highly predictive of poor outcomes in a wide range of HF patients (5- 8).

Direct measurement of pulmonary arterial systolic pressure (PASP) is the gold standard for confirming a diagnosis of pulmonary hypertension (9), but right heart catheterization may not be the initial approach for assessing pulmonary pressure (10). In contrast, Doppler estimation of PASP is feasible, measurable in a high percentage of cases (11, 12), and clinically relevant in community and population-based studies (13, 14). The term "right ventricular-pulmonary artery coupling" relates to the ability of right ventricular contractility to counteract an elevated afterload (15). Echocardiographic measurement of the ratio between TAPSE and pulmonary artery systolic pressure (PASP) is a simple, noninvasive parameter that has demonstrated a strong correlation with invasively estimated RV-PA coupling (16). A reduced TAPSE/PASP ratio, indicating poor RV-PA coupling, has been linked to unfavorable outcomes in patients with cardiovascular conditions (16-24).

The present study aims to establish the predictive value of the TAPSE/PASP ratio in determining all-cause mortality and/or rehospitalization in patients dealing with acute heart failure.

Methods

Study population

This was a prospective, monocentric, observational study, conducted from December 2020 to December 2022 in the Cardiology Department of the Internal Security Forces Hospital of Marsa, Tunisia. The hospital's ethics committee approved the study (ISFHEC-2020-03). All patients obtained informed verbal consent before participating in the study. We included all patients hospitalized for AHF regardless of the etiology. We excluded patients lost to follow-up upon hospital discharge, as well as patients who died before any laboratory testing or echocardiographic examination.

Clinical evaluation

AHF is defined in alignment with the heart failure guidelines delineated by the European Society of Cardiology (ESC) in both 2016 and 2021 (25) by a "rapid or gradual onset of symptoms and/or signs of HF, severe enough for the patient to seek urgent medical attention, leading to an unplanned hospital admission or an emergency department visit" (26).

The study encompassed the collection of patient demographic and anthropometric information, cardiovascular risk factors, and comorbidities. Patients were categorized based on their clinical AHF presentations, which included acute pulmonary edema, isolated right ventricular failure, or global decompensation. Measurements were taken for systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR). Additionally, a 12-lead electrocardiogram was conducted for each patient to record heart rate and rhythm.

Laboratory evaluation

All patients had a measurement of B-type natriuretic peptide (BNP), hemoglobin level, serum sodium, serum potassium, C-Reactive Protein (CRP) and creatinine.

Echocardiographic evaluation

Two dimensional transthoracic echocardiographic and Doppler studies were performed at the time of admission of all patients using a Philips EPIQ 7C echocardiography with simultaneous and continuous electrocardiographic tracing.

Left heart

The LV diastolic diameter was measured in parasternal long-axis view either by M-mode tracing or bi-dimensional guided method. The biplane method of discs was used to measure LVEF. In each apical view (4, 3, and 2 chambers), the endocardial border was traced on the end-systolic frame. Left ventricular global longitudinal strain (LV-GLS) was calculated as the mean value of 6 segments of each apical view and was measured as the average of GLS values from the three apical views. GLS was analyzed on three cardiac cycles in the patients with sinus rhythm and on the average of 5 cardiac cycles in the patients with atrial fibrillation.

From the trans-mitral flow profile, trans-mitral pulsed-wave Doppler was obtained, and the peaks of both early diastolic filling (E) and late diastolic filling (A) were measured. In the apical four-chamber view tissue Doppler imaging (TDI) of the mitral annulus was performed by placing the sample volume over the septal mitral valve annulus. The value of e' was measured and E/e' was calculated.

The Maximal LA Volume was measured in the apical 4-chamber view at the ventricular end-systolic frame. LA volumes were indexed to the body surface area (LAVI).

Right heart

The RV end-diastolic diameter (RVEDD) was measured in apical 4-chamber axis at basal level under the tricuspid annular site. The RV peak systolic wave velocity (RV S') was calculated in DTI-mode echocardiograms from the 4-chamber view by positioning the cursor on the lateral tricuspid annulus and calculating the peak systolic

velocity. TAPSE was calculated in 2-dimensional M-mode echocardiograms from the 4-chamber view by positioning the M-mode cursor on the lateral tricuspid annulus and calculating the amount of longitudinal displacement of the annulus at peak systole (27).

Using the peak velocity (Vmax) of the tricuspid regurgitation continuous-wave Doppler tracing, the pulmonary artery systolic pressure (PASP) was determined as the difference in pressures between the right ventricle and the right atrium. The simplified Bernoulli equation (PASP=4(Vmax) 2+right atrial pressure) was used (28). Right atrial pressure (RAP) was derived based on the size and distensibility of the inferior vena cava (IVC) during respiration. RV-PA coupling was calculated using the ratio between TAPSE and PASP (TAPSE/PASP) (29).

Right Ventricle Fraction area change (RVFAC) was obtained by tracing the RV end-diastolic area (RVEDA) and end-systolic area (RVESA) in the apical 4-chamber view using the formula (RVEDA−RVESA)/ RVEDA×100 (27).

RV- free wall strain was measured only in the apical 4-chamber or focused RV view. We evaluated the average value of the longitudinal peak systolic strain only from the free wall. RV free wall strain was analyzed on three cardiac cycles in the patients with sinus rhythm and on the average of 5 cardiac cycles in the patients with atrial fibrillation. The RA Volume was measured using the 4-chamber at the ventricular end-systolic frame. The severity of tricuspid regurgitation was also evaluated in from the apical four-chamber view and assessed semi quantitatively from the Color Doppler Flow. All the valvular heart diseases were classified according the ESC valvular heart disease guidelines published in 2021 (30).

All clinical, demographic, echocardiographic data and laboratory investigations were evaluated at the first 72 hours of admission.

Follow-Up

Mean follow up was 18±6 months Patients were evaluated according to their medical records or by phone calls. Primary endpoint was all-cause mortality or rehospitalization for AHF. Secondary endpoints were all-cause mortality and rehospitalization for AHF.

Statistical analysis

Data was gathered and analyzed using IBM SPSS Statistics 23 software. For comparability between groups, the Chi-square test and Fisher's exact test were used for categorical variables, Student's t-test for independent series and Kruskal-Walli's test were used for continuous variables. Multinomial logistic regression was then performed for multivariate analysis. All ultrasound parameters for which the univariate analysis had p-values < 0.05 were entered in the multivariate study. Receiver operating characteristic (ROC) curves were used to assess sensitivity and specificity, and to identify threshold values for the parameters studied. In all statistical tests, the significance threshold (p) was set at 0.05.

Results

The study cohort consisted of 152 consecutive patients (Figure 1), with patients' characteristics summarized in Table 1. The average age was 65 years with a standard deviation of 10, and the majority (70%) were men.

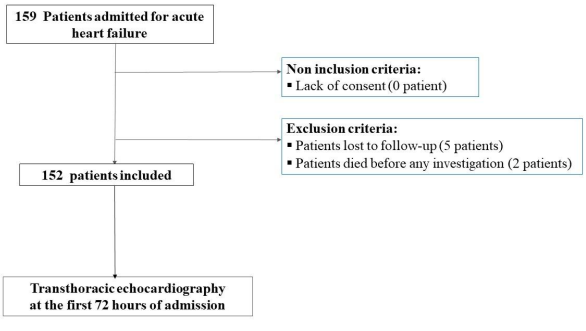

Figure 1. Study Flow chart

Among the cardiovascular risk factors, diabetes and smoking were prevalent, being present in 66% and 60% of the patients, respectively. Ischemic heart disease was the leading cause of heart failure, accounting for 50% of all cases. Additionally, 67 patients (44%) were hospitalized due to a new onset of AHF. The primary factor contributing to decompensation was ischemic exacerbation, accounting for 30% of cases. The mean left ventricular ejection fraction (LVEF) was 38% with a standard deviation of 13.

In terms of clinical presentation, 48% of the patients exhibited global heart failure, 41% had acute pulmonary edema, and 11% had isolated right ventricular failure.

Clinical outcome

During follow-up, 69 patients died or were hospitalized for AHF (45%), 29 patients died (19%) within an average period of 3.7 months and 57 patients were hospitalized for AHF (37%).

Impact of clinical profile on primary and secondary endpoints

Age was found to be significantly correlated with all-cause mortality or rehospitalization for acute heart failure, with p-values of 0.002. Additionally, age and NT-ProBNP levels were significantly correlated with all-cause mortality, with p-values of 0.028 and 0.002, respectively. Furthermore, age and obesity showed significant correlations with rehospitalization for acute heart failure, with p-values of 0.007 and 0.021, respectively.

Correlation between echocardiographic parameters and endpoints

In multivariate analysis, two echocardiographic parameters independently predicted all-cause mortality or rehospitalization for AHF: LV-GLS (hazard ratio (HR) =0.88; 95% confidence interval (CI): 0.80-0.95; p=0.003) and the TAPSE/PASP ratio (hazard ratio (HR) =2.6; 95%CI:

1.04-6.47; p=0.040), as illustrated in Table 2.

Table 1. Baseline characteristics of the study population

	General population (n=152)
Age (years)	65±10
Gender (Male/Female)	55 (106)/27 (46)
Hypertension (n, %)	87 (57)
Diabetes (n, %)	101 (66)
Dyslipidemia (n, %)	73 (48)
Obesity (n, %)	64 (42)
smoking (n, %)	92 (60)
Etiology of HF	
Ischemic heart disease (n, %)	76 (50)
Valvular heart disease (n, %)	20 (13)
Rhythmic heart disease (n, %)	14 (9)
Amyloidosis (n, %)	6 (4)
Clinical presentation	
Left Heart Failure (n, %)	62 (41)
Right Heart Failure (n, %)	17 (11)
Global Heart Failure (n, %)	73 (48)
New onset Heart Failure (n, %)	67 (44)
Biological parameters	
NT-Pro BNP (ng/l)	4129 [2498-9205]
C-reactive protein (mg/l)	12 [6-30]
Echocardiographic parameters	
Parameter	Value
Left ventricular end-diastolic diameter (mm)	57±9
Indexed left ventricular end-diastolic volume (ml/m²)	72±29
Indexed left ventricular end-systolic volume (ml/m²)	47±26
Left ventricular ejection fraction (%)	38 [25-55]
LVEF (n, %)	82 (54)
LVEFmr (n, %)	15 (10)
LVEFgp (n, %)	55 (36)
Global longitudinal strain of the LV (absolute value)	11±5
Left ventricular hypertrophy (n, %)	66 (43)
Indexed LV mass (gr/m²)	122±38
Left atrial area (cm²)	30±9
Left atrial Indexed volume (ml/m²)	59±33
E wave (cm/sec)	96±27
A wave (cm/sec)	60±20
Deceleration time (ms)	152±48
Average E/e' ratio	15 [14-18]
Right ventricular basal diameter (mm)	47±14
TAPSE (mm)	17±4
SRV (cm/s)	10±3
Fractional shortening (%)	39±10
RV free wall strain (absolute value)	11 [8-13]
PASP (mmHg)	53±16
Severe aortic stenosis (n, %)	8 (5.3)
Severe mitral stenosis (n, %)	4 (2.6)
Severe aortic regurgitation (n, %)	2 (1.3)
Severe mitral regurgitation (n, %)	8 (5.3)
Severe tricuspid regurgitation (n, %)	3 (2)
Pulmonary B lines (n, %)	125 (82)
Pleural effusion (n, %)	43 (28)
Pericardial effusion (n, %)	23 (15)

LV: Left Ventricular,**EF**: Ejection Fraction,**LVEF**: Left Ventricular Ejection Fraction, **LVEFmr**: Left Ventricular Ejection Fraction midrangeLVEFgp: Left Ventricular Ejection Fraction preserved,**TAPSE**: Tricuspid Annular Plane Systolic Excursion,**SRV**: S of the right venricule,RV: Right Ventricular,**PASP**: Pulmonary Artery Systolic Pressure

Table 2. Multivariate analysis for primary endpoint

All-cause mortality or rehospitalization for AHF	P	HR	CI 95 %	
			IB	SB
LVEF	0.208	1.020	0.989	1.051
LV-GLS	**0.003**	0.880	0.809	0.957
TAPSE	0.859	0.988	0.865	1.129
RV S'	0.432	0.926	0.763	1.123
TAPSE/PASP ‹ 0,35	**0.040**	2.601	1.044	6.477

LVEF: Left ventricular ejection fraction, **LV-GLS**: left ventricular Global longitudinal strain, **TAPSE**: Tricuspid annular plane systolic excursion, **RVS'**: Peak systolic velocity of tricuspid annulus, **PASP**: Pulmonary arterial systolic pressure

A cutoff value of 0.35 mm/mmHg was associated with the worst outcome, with sensitivity (Se) =65% and specificity (Sp) =70% (OR =3.88; 95% CI: 1.97-7.64; p=0.001) (Figure 2).

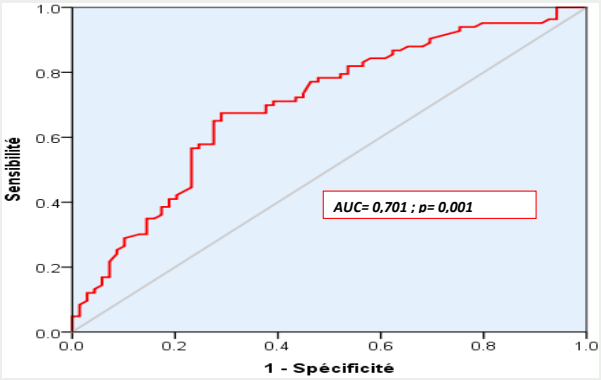

Figure 2. ROC curve for TAPSE/PASP ratio as a predictor of mortality or rehospitalization for acute heart failure

PASP (HR=1.04; 95%CI: 1.00-1.07; p=0.016) and TAPSE/PASP (HR=2.73; 95%CI: 1.25-9.12; p=0.031) were independent predictors of mortality in AHF (Table 3). A cutoff value of 0.32 mm/mmHg was associated with a higher risk of mortality, with Se =64%, Sp =70%, OR =3.53; 95% CI: 1.51-8.27; p = 0.003 (Figure 3).

Table 3. Multivariate analysis for secondary endpoint: all-cause mortality

DCD	P	HR	CI 95 %	
			IB	SB
RV S'	0.825	0.973	0.766	1.237
PASP	**0.016**	1.041	1.007	1.077
TAPSE	0.204	0.885	0.732	1.069
TAPSE/PASP<0.32	**0.031**	2.735	1.250	9.124
AS	0.059	0.174	0.028	1.067
TR	0.246	0.495	0.151	1.625

TAPSE: Tricuspid annular plane systolic excursion **RVS'**: Peak systolic velocity of tricuspid annulus, **PASP**: Pulmonary arterial systolic pressure, **AS**: aortic stenosis, **TR**: tricuspid regurgitation

Only the TAPSE/PASP ratio was an independent echocardiographic parameter predicting rehospitalization for AHF (HR = 3.97; 95% CI: 1.38-11.40; p=0.010), as shown in Table 4.

The cutoff value of 0.36 mm/mmHg was predictive of a higher risk of rehospitalization, with Se =70%, Sp = 68%, OR =4.47; 95% CI: 2.17-9.17; p < 0.001 (Figure 4).

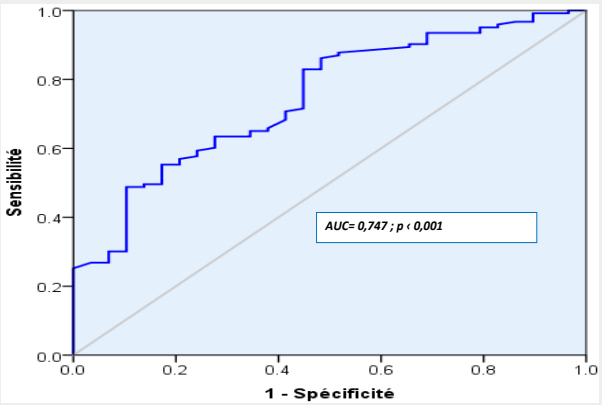

Figure 3. ROC curve for TAPSE/PASP ratio as a predictor of mortality

Table 4. Multivariate analysis for secondary endpoint: rehospitalization

Rehospitalization	P	HR	CI 95 %	
			IB	SB
RV S'	0.819	0.975	0.784	1.212
TAPSE	0.798	0.981	0.847	1.136
LVEF	0.969	1.001	0.944	1.061
LV-GLS	0.089	1.110	0.984	1.252
TAPSE/PASP<0.36	**0.010**	3.975	1.386	11.401

RVS': Peak systolic velocity of tricuspid annulus, **TAPSE**: Tricuspid annular plane systolic excursion **LVEF**: Left ventricular ejection fraction, **LV-GLS**: left ventricular Global longitudinal strain, PASP: Pulmonary arterial systolic pressure

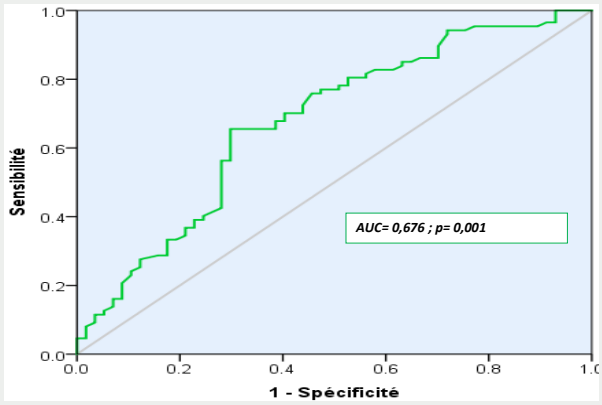

Figure 4. ROC curve for TAPSE/PASP ratio as a predictor of rehospitalization

Discussion

In this prospective study conducted of 152 patients with AHF, TAPSE/PASP ratio was predictive of AHF clinical outcome (HR=2.6; 95%CI: 1.04-6.47; p=0.04). It was the only independent echocardiographic parameter predicting rehospitalization for AHF (HR=3.97; 95%CI: 1.38-11.40; p=0.010) and mortality (HR=2.73; 95%CI: 1.25-9.12; p=0.03). TAPSE/PASP with a cut-of value <0.35 mm/mmHg had a sensitivity of 65%, a specificity of 70%, and an area under ROC curve=0.701 for predicting the worst outcome.

Acute heart failure (AHF) stands as a primary reason for hospital admissions and is associated with a significant rise in morbidity and fatal outcomes (31). Even with advancements in available treatments, the outlook for AHF remains grim, with in-hospital mortality rates ranging from 4 to 7% (32).

Left-sided heart disease stands out as the predominant cause of pulmonary hypertension, responsible for 65-80% of all cases (33). The chronic elevation of left-sided filling pressure, whether due to systolic or diastolic dysfunction in heart failure (HF), is transmitted backward passively. Elevated left atrial pressure is transmitted upstream to the pulmonary vasculature, triggering pulmonary vascular remodeling (34). This, in turn, transfers pulmonary hypertension to the thin-walled flow-generator right ventricle (RV), which is ill-equipped to handle rapid increases in pressure (35).

In the initial phases of pulmonary hypertension, the RV adapts by increasing its contractility to match the excess afterload and maintain pulmonary circulation. However, as the disease advances, the compensatory phase concludes, resulting in RV failure and a disruption of the normal RV-PA coupling.

In healthy hearts, the synchronization between the right ventricle (RV) and the pulmonary circulation is of paramount importance. This synchronization allows them to function as a cohesive cardiopulmonary unit, maintaining a harmonious balance between contractility and afterload—a phenomenon known as "RV-PA coupling" (36). In patients with pulmonary hypertension, however, there is an increase in pulmonary vascular resistance and a decrease in vascular bed compliance. This leads to an elevated afterload and a disruption in RV-PA coupling, ultimately resulting in adverse outcomes for individuals with pulmonary hypertension (37).

RV-PA uncoupling signifies an advanced stage of disease progression in left-sided heart disease, where attempts to alter the disease course with advanced therapies may prove futile. Identifying such high-risk patients easily in clinical practice is crucial for risk stratification and avoiding unnecessary procedures. The echocardiographic TAPSE/PASP ratio has emerged as an alternative predictor of invasive pressure-volume loop-derived end-systolic/arterial elastance, considered the gold standard measure of RV-PA coupling (38).

This index is the most extensively established surrogate for RV-PA coupling in HF (39, 40), with fewer studies investigating alternative indices such as longitudinal strain of RV free wall/PASP (41, 42). There is ample evidence that the TAPSE/PASP ratio provides prognostic information beyond that offered by TAPSE or PASP separately in HF. Worse TAPSE/PASP values have been consistently associated with adverse long-term outcomes in various HF cohorts, including outpatients with HF (43), patients in the acute phase of decompensation (20), subjects with HF with preserved (44) or reduced (45) ejection fraction, as well as recipients of cardiac resynchronization therapy (CRT) (39) or transcatheter valve procedures (46).

Tello et al. demonstrated the reliability of the TAPSE/PASP ratio as a method for assessing RV-PA coupling in patients with severe idiopathic and thromboembolic pulmonary hypertension. They indicated that a ratio less than 0.31 mm/mm could serve as a predictive marker for RV-PA uncoupling (37). The TAPSE/PASP ratio is indicative of the right ventricle's response to changes in afterload, with higher values associated with better RV function (15). In

the early stages of chronic pulmonary hypertension, as RV-PA coupling remains intact, the right ventricle adapts by increasing its contractility to counteract the elevated afterload, often resulting in RV hypertrophy and dilation. However, as chronic pulmonary hypertension progresses to RV failure, the uncoupling of RV-PA leads to a decline in RV systolic function (47).

This study contributes to the existing body of knowledge regarding the importance of non-invasive echocardiographic assessment of RV-PA coupling for risk stratification in critically ill patients and those with heart failure. In a cohort study, Jentzer et al. observed that the loss of RV-PA coupling was associated with an elevated risk of mortality, both during hospitalization and after discharge, among patients admitted to the cardiac intensive care unit (48).

In their prospective cohort study, Naseem et al. demonstrated the significance of the TAPSE/PASP ratio as a predictor of in-hospital mortality among patients with acute heart failure, specifically those with heart failure with reduced ejection fraction (HFrEF) who were admitted with AHF. Notably, the majority of patients in the group that experienced in-hospital mortality exhibited a TAPSE/PASP ratio of less than 0.4 (49)

Additionally, Guazzi et al. observed an inverse relationship between TAPSE/PASP and the NYHA functional class. They concluded that this ratio, which combines the measurement of both longitudinal right ventricular (RV) fiber shortening (TAPSE), and the pressure developed in the pulmonary artery, serves as a valuable clinical index. It relates the length of RV contraction to the force generated, and the utilization of this ratio is superior to using each measurement separately. This observation held true for both heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF) patients (15).

In one of the largest series of patients with acute HF and preserved ejection fraction, Santas et al. disclosed that TAPSE/PASP <0.36 mm/mmHg could independently predict unplanned rehospitalization for HF (20). The risk was increased in a stepwise manner, with lower quintiles of TAPSE/PASP (20).

Contemporary evidence demonstrates than TAPSE/PASP can potentially identify non-responders to interventional HF therapies. Stassen et al. illustrated that among 807 CRT recipients TAPSE/PASP <0.45 mm/mmHg provided incremental prognostic information on top of impaired TAPSE (39). In addition, lack of improvement in TAPSE/PASP ratio after CRT was associated with worst survival. Those findings exemplify that when RV-PA uncoupling is established, the disease stage is so advanced that CRT implantation might come with little clinical benefit for the patient.

On the contrary, Braganca et al. failed to elicit prognostic information for HF patients with reduced ejection fraction undergoing CRT, but their study was small and underpowered; a total of 70 patients were included with only 15 events (17).

Karam et al. have demonstrated that patients with functional MR and pre-operative RV-PA uncoupling, defined as TAPSE/PASP <0.27 mm/mmHg, derived significantly less survival benefit from transcatheter mitral valve repair compared to their counterparts (46).

Substantial heterogeneity was observed in the definition of RV-PA uncoupling, with suggested TAPSE/PASP cutoff values varying from 0.27 to 0.58 mm/mmHg. Five studies (15,18, 41, 43,44) implemented the cutoff of 0.36 mm/mmHg, initially proposed by Guazzi et al. (15) as derived by receiver operating characteristic curve analysis from a mixed cohort of 293 HF patients. This cutoff value was strongly linked with rehospitalization in the current study.

The current study's findings, indicating a connection between reduced TAPSE/PASP and mortality in patients experiencing AHF, underscores the significance of right ventricular function in individuals with left ventricular failure and the necessity of optimizing both ventricles' performance within this patient group. It highlights the potential benefits of interventions designed to improve RV-PA coupling. These interventions could encompass treatments aimed at reducing right atrial and pulmonary artery pressures (50). Such therapeutic measures might involve the use of diuretics, medications affecting the pulmonary vasculature, fluid removal, and short-term inotropic agents. These options may lead to a temporary improvement in RV-PA coupling during the hospitalization period and facilitate recovery from the acute phase (51,52).

Limitations

It is essential to acknowledge that this study possesses significant limitations. Firstly, the most prominent constraint lies in the relatively small number of patients included. Future studies should encompass a larger and more diverse pool of participants, ideally from multiple medical centers.

Secondly, the patient cohort was exclusively drawn from those admitted to the cardiology department, which excludes heart failure patients treated in other units, such as the emergency department or internal medicine services.

Thirdly, echocardiograms were conducted by different operators, resulting in a diminished level of both intra- and inter-observer reproducibility.

Lastly, but equally important, our protocol is subject to limitations related to the timing of echocardiographic examinations. These were not performed upon admission but rather after several hours of intravenous diuretic use. This temporal gap may account for the underestimation of certain echocardiographic parameters, such as PASP.

Conclusion

The non-invasive TAPSE/PASP ratio may be used as an independent predictor of both mortality and rehospitalization in patients with AHF. We recommend conducting additional multicenter studies with a larger patient population to validate our findings.

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