

Original Article

The Accuracy of Determining Pulmonary Artery Pressure by PR and TR Velocities Compared With Right Heart Catheterization in Patients With Right Ventricular Dysfunction

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ABSTRACT

Background: The gold standard for the diagnosis of pulmonary arterial hypertension (PAH) is right heart catheterization (RHC). The use of noninvasive echocardiographic methods to assess the pulmonary artery pressure (PAP) has been debated, and the role of echocardiography has been proposed to be more of estimating the probability of PAH rather than assessing the pressure. In this study, we assessed the accuracy of the use of pulmonary regurgitant (PR) and tricuspid regurgitant (TR) velocities in estimating PAH by comparison with RHC in patients with right ventricular dysfunction.

Methods: This cross-sectional study was performed in Rajaie Cardiovascular Medical and Research Center from 2015 through 2016. We selected patients with right ventricular dysfunction who were candidates for RHC. Echocardiography was performed within 24 hours before catheterization. PAH was estimated by using PR and TR velocities. The correlation between echocardiography and catheterization-derived PAH was tested by using the Pearson correlation test.

Results: There was significant accordance between the 2 tools in terms of the measurement of the systolic PAP ($r = 0.860$, $P < 0.001$), the diastolic PAP ($r = 0.793$, $P < 0.001$), and the mean PAP ($r = 0.739$, $P < 0.001$) in the diagnosis of PAH ($K = 0.964$, $P < 0.001$). Based on the receiver operating characteristic curve analysis, the measurement of the TR velocity had a moderate value in predicting PAH (the area under the curve = 0.622). The best cutoff value for the TR velocity in predicting PAH was 3.27, yielding a sensitivity of 72.1% and a specificity of 50.0%.

Conclusions: Echocardiography-derived measurements were in good correlation with RHC in the assessment of PAH in our patients with right ventricular dysfunction. (*Iranian Heart Journal 2021; 22(3): 81-87*)

KEYWORDS: Right heart catheterization, Echocardiography, RV failure, TRG, PR velocity

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Pulmonary arterial hypertension (PAH) is a pathological condition defined as an increase in the mean PAH to at least 25 mm Hg in right heart catheterization (RHC).¹ A wide range of underlying etiologic factors is associated with pathological changes in the pulmonary arteries, leading to an increase in PAH. Progress in the management and treatment of patients with PAH and the availability of specific treatments for the disease have significantly reduced the morbidity of the patients and improved their survival in recent years.² However, PAH remains a disabling disease with severe symptoms and ultimately death in patients. Right ventricular (RV) dysfunction remains an important cause of mortality in patients with PAH. The ability of the RV to adapt to the progressive increase in PAH is associated with significant changes in pulmonary adenoma in PAH, which is a predictive factor for patients' functional capacity and survival.³ The critical role of RV function in PAH has been confirmed in other studies.^{4,5} Therefore, an accurate measurement of the pulmonary artery pressure (PAP) in the setting of RV dysfunction can predict patients' severe clinical condition in the future.

According to the European Society of Cardiology (ESC)/the European Respiratory Society (ERS) guideline in 2015, RHC is required for the accurate determination of PAP.⁶ Although RHC is considered the gold standard for determining PAP and PAH diagnosis, this procedure is regarded as a relatively invasive procedure. Consequently, noninvasive, but precise methods have always been considered for assessing PAH in patients with right heart failure. Echocardiography is an available and commonly used tool for evaluating ventricular function and noninvasive hemodynamic assessment. The estimation of

the systolic PAP through the maximum velocity of the tricuspid regurgitant (TR) jet and the measurement of the mean PAP and the mean diastolic PAP through early diastolic and end-diastolic pulmonary valve regurgitant jet has been previously studied.^{7,8}

Furthermore, the mean PAH can also be estimated by determining the ratio of the acceleration time of the pulmonary insufficiency to the ejection time (or AT/ET).⁹ The cutoff point is considered 3 m/s for the tricuspid insufficiency velocity, 2 m/s for the pulmonary insufficiency velocity, and 0.3 for the ET/AT ratio, with higher values indicating pathological conditions.¹⁰ In this study, we sought to evaluate the efficacy of determining PAH by the pulmonary regurgitant (PR) velocity and the TR velocity by comparison with RHC in patients with RV dysfunction.

METHODS

The present cross-sectional study was performed on patients referred to Rajaie Cardiovascular Medical and Research between 2015 and 2016 for RHC. Written informed consent was obtained from all the participants, and the study was approved by the ethics committee of the hospital. Detailed demographic information, past medical history, and drug history were collected through interviews and medical record reviews.

Echocardiographic Assessment

An echocardiography examination was performed with GE Vivid 7 (GE Healthcare, Milwaukee, WI, USA) for all the participants within 24 hours before clinically indicated RHC. All the measurements were performed by a single level III cardiologist/echocardiographer in keeping with the guidelines of the American Society

of Echocardiography. RV function was assessed using global eye assessment in addition to tricuspid annular plane systolic excursion in the 4-chamber view and the RV pulsed tissue Doppler S wave (S') taken at the lateral tricuspid annulus in systole. The RA pressure was estimated according to the diameter of the inferior vena cava (IVC) and the percentage of respiratory collapsibility (the IVC diameter <2.1 cm with collapses >50%). The RA pressure was considered 3 mm Hg. If the IVC diameter was greater than 2.1 cm and collapses were below 50% with a sniff, the RA pressure was considered 15 mm Hg. If the IVC diameter and collapses failed to fit this paradigm, an intermediate value of 8 mm Hg was considered. PAH was assessed based on the TR velocity via the following formula:

$$\text{systolic PAP} = 4(\text{TR velocity})^2 + \text{RA pressure}$$

The PR velocity was assessed through the following formula:

$$\text{mean PAP} = 4(\text{PRVBD})^2 + \text{RA pressure}$$

where PRVBD stands for the pulmonary regurgitant jet velocity taken at the beginning of diastole.

The TR jet continuous-wave Doppler was acquired in the best accessible view (either the RV inflow or the modified 4-chamber view.) The PR jet continuous Doppler was obtained in the high parasternal short-axis view. The application of the agitated saline contrast was performed in weak TR signal waves.

Pulmonary vascular resistance (PVR) was calculated via the following formula:

$$\text{TR velocity} / \text{RVOT VTI} \times 10 + 0.16$$

where RVOT stands for the right ventricular outflow tract and VTI stands for the velocity-time integral.

Cardiac output was obtained by the following formula:

$$(\text{LVOT diameter}^2) \times 0.785 \times \text{LVOT VTI} \times \text{HR}$$

where LVOT stands for the left ventricular outflow tract and HR stands for heart rate.

RHC

Hemodynamic measurements were performed by using the Swan–Ganz catheter via the femoral access. Pressures were obtained in the supine position and at end-expiration. The systolic PAP, the mean PAP, the diastolic PAP, and the pulmonary artery wedge pressure with an average of 3 measurements over 3 consecutive heart cycles were recorded at the end of the expiratory phase. The pressure transducer was zeroed using the reference point of the mid-thoracic line halfway between the anterior sternum and the bed surface. Cardiac output was determined by the indirect Fick method.

Statistical Analysis

Descriptive analysis was applied to describe the data, including the mean \pm the standard deviation (SD) for quantitative variables and frequencies (percentages) for categorical variables. The χ^2 test, the independent *t* test, and the Mann–Whitney *U* test were utilized for the comparison of the variables. The correlation between the quantitative variables was tested by using the Pearson correlation test. For the statistical analyses, the statistical software IBM SPSS Statistics for Windows, version 22.0, (IBM Corp, Released 2013, Armonk, New York) was used. A *P*-value of less than 0.05 was considered statistically significant.

RESULTS

Forty-five patients completed the study protocol undergoing echocardiography and RHC. The mean age of the patients was 45.31 ± 17.83 years (range =21–85 y). In terms of sex distribution, 18 cases (40%) were men, and 27 cases (60%) were women. The mean body mass index of the patients was 25.97 ± 5.71 kg/m², and their mean

body surface area was $1.85 \pm 0.51 \text{ c/m}^2$. Vis-à-vis the primary diagnosis, left ventricular dysfunction was diagnosed in 23 cases (51.1%), idiopathic PAH in 12 cases (26.7%), valvular heart diseases in 6 cases (13.3%), chronic thromboembolic pulmonary hypertension in 3 cases (6.7%), and collagen vascular disease in 1 case (2.2%). The mean heart rate of the patients was $94.22 \pm 11.29 \pm 1$ beats/min. The parameters measured by echocardiography are summarized in Table 1.

Table 1. Parameters measured by echocardiography

Mean LVEF	$35.56 \pm 1.54\%$	Percent
LV Systolic Function		
Normal	2	4.4
Mild dysfunction	21	46.7
Moderate dysfunction	5	11.1
Severe dysfunction	17	37.8
Mean RV size	4.23 ± 0.59	
RV Function		
Normal	3	6.7
Mild dysfunction	5	11.1
Moderate dysfunction	21	46.7
Severe dysfunction	16	35.6
TR Severity		
Mild dysfunction	1	2.2
Moderate dysfunction	21	46.7
Severe dysfunction	23	51.1
Mean LVOT VTI	12.69 ± 3.54	
Mean RVOT VTI	16.36 ± 30.00	
Mean TRG	62.49 ± 29.95	
IVC size		
Normal	13	28.9
Increased	32	71.1
IVC Collapse		
Normal	17	37.8
Decreased	28	62.2
Mean TR velocity	3.80 ± 0.83	
Mean PV	3.72 ± 2.65	
Mean PVR	3.66 ± 1.66	
Mean CO	3.59 ± 1.09	
Mean TAPSE	8.97 ± 3.25	

LVEF, Left ventricular ejection fraction; RV, Right ventricle; TR, Tricuspid regurgitation; LVOT, Left ventricular outflow tract; VTI, Velocity-time integral; RVOT, Right ventricular outflow tract; TRG, Tricuspid regurgitant gradient; IVC, Inferior vena cava; PVR, Pulmonary vascular resistance; CO, Cardiac output; TAPSE, Tricuspid annular plane systolic excursion

Overall, in echocardiographic assessment, RV function was normal in 3 cases (7.7%), while mild, moderate, and severe dysfunction was revealed in 11.1%, 46.7%, and 35.6%, respectively. Mild, moderate, and severe TR was present in 2.2%, 46.7%, and 51.1%, respectively. The mean TR velocity in these patients was 3.80 ± 0.83 m/s. As is shown in Table 2, 84.4% and 95.6% of the patients suffered from PAH based on echocardiography and catheterization, respectively. There was significant accordance between the 2 tools concerning the measurements of the systolic PAP ($r = 0.860$, $P < 0.001$), the diastolic PAP ($r = 0.793$, $P < 0.001$), and the mean PAP ($r = 0.739$, $P < 0.001$). Additionally, a high agreement was revealed between echocardiography and catheterization in the diagnosis of PAH ($K = 0.964$, $P < 0.001$). The results also revealed a strong correlation between the CO parameter assessed by left heart catheterization and echocardiography ($r = 0.754$, $P < 0.001$). A similar result was also obtained in the measurement of the PVR value between the 2 procedures ($r = 0.675$, $P < 0.001$). Further, a significant correlation was detected between the mean PAP value and the TR velocity ($r = 0.789$, $P < 0.001$) and the PR velocity ($r = 0.659$, $P < 0.001$). Based on the receiver operating characteristic curve analysis (Fig. 1), the measurement of the TR velocity had a moderate value in predicting PAH (the area under the curve = 0.622). The best cutoff value for the TR velocity in predicting PAH was 3.27, yielding a sensitivity of 72.1% and a specificity of 50.0%.

Table 2. PAP index values based on echocardiography and catheterization

Based on Echocardiography	
Mean SPAP	73.60 ± 30.14
Mean DPAP	28.40 ± 13.23
Mean MPAP	41.07 ± 15.79
Mean PAH	38 (84.4)

Based on Catheterization	
Mean SPAP	68.18 ± 29.44
Mean DPAP	34.76 ± 17.32
Mean MPAP	45.71 ± 18.95
Mean PAH	43 (95.6)
Mean PVR	9.18 ± 10.01
Mean CO	3.70 ± 1.43

SPAP, Systolic pulmonary artery pressure; DPAP, Diastolic pulmonary artery pressure; MPAP, Mean pulmonary artery pressure; PAH, Pulmonary arterial hypertension; PVR, Pulmonary vascular resistance

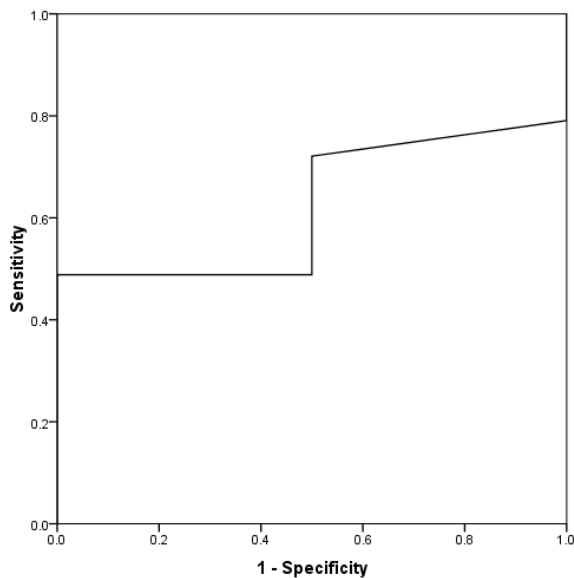


Figure 1. The figure depicts the area under the receiver operating characteristic curve in determining the value of the tricuspid regurgitant velocity in the detection of pulmonary arterial hypertension (area under the curve = 0.622, 95% CI: 0.394 to 0.850).

DISCUSSION

The evaluation of PAH can be done with high accuracy and sensitivity through RHC. Nevertheless, given its invasive nature, the need for short-term admission to the hospital, pre and post-care, and costs, this practice mainly affects patient satisfaction, and it is sometimes accompanied by annoying complications. The use of noninvasive methods such as color Doppler ultrasound has allowed the evaluation of ventricular function and noninvasive hemodynamic assessment at the same time. However, the use of echocardiography to

assess PAP has been debated, and the role of echocardiography has been proposed to be more of estimating the probability of PAH rather than assessing the pressure.⁶ In this study, in the group of patients with PAH and RV dysfunction, we used PAP estimates in echocardiography with TR and PR velocity indices and compared them with RHC-derived measures. We showed a high agreement between the 2 methods in assessing PAP and the detection of PAH. According to our findings, PAH was detected in RHC and echocardiography in 95.6% and 84.4% of the study population, respectively. We found underestimation in 10.2% of our cases undergoing echocardiography, which did not seem to have affected the diagnosis of the disease significantly.

The findings in this study are consistent with most studies. Raffoul et al¹² reported a strong correlation between PAH based on echocardiography and angiography (correlation coefficient = 0.97). Masuyama et al¹³ reported that a rise in PAH was RHC consistent with an increased PAP. Additionally, PAH recorded on the PR velocity was in accordance with the value determined by RV catheterization (correlation coefficient = 0.92). Abbas et al¹⁴ showed that early and end-diastolic PR values were significantly correlated with the mean PAH and the end-diastolic PAP.

Therefore, the use of both TR and PR velocity indices in echocardiography can allow a relatively accurate estimation of PAH and, thus, rule in or rule out PAH.

Our study has some limitations. Firstly, the small sample size had a significant adverse effect on some correlations. Secondly, our study was conducted in a single tertiary center. Still, the advantage is quality consistency in echocardiography and RHC. In the current investigation, we did not perform echocardiography and RHC simultaneously; however, we performed

echocardiography less than 24 hours prior to catheterization to lessen possible errors. Lastly, the effects of confounding factors correlated with PAH or RV function (eg, the New York Heart Association functional class, RV and right atrial size, and ventricular hypertrophy) were not adjusted in this study.

CONCLUSIONS

As a general conclusion, echocardiography and the indices of PR and TR velocities can be used to assess PAH accurately and, thereby, rule in or rule out the diagnosis of PAH. Therefore, echocardiography is an acceptable noninvasive alternative to the RV catheterization modality in the evaluation of PAH in patients with right heart systolic dysfunction.

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