Comparison of transthoracic echocardiography and right heart catheterization for assessing pulmonary arterial pressure in patients with congenital or valvular heart defects

Mojgan Parsaee MD[1], Ahmad Amin MD[2], Mahmood Reza Nematollahi MD[3], Azerakhsh Babolian MD[4]

Abstract

Background- Although right heart catheterization (RHC) has acceptable accuracy for the measurement of pulmonary arterial pressure (PAP), significant risks and cost issues are worrisome. Thus, a non-invasive technique such as echocardiography for assessing PAP would clearly be of great clinical value. We aimed to compare estimated systolic PAP (SPAP) by echocardiogram with the actual RHC measurements in the two groups of congenital and valvular heart diseases (CHD and VHD, respectively), in whom pulmonary hypertension (PHT) was clinically suspected.

Methods- A total of 103 consecutive patients with confirmed CHD or VHD referred to our center between January and December 2009 were studied. Participants underwent transthoracic echocardiography and RHC within 4 hours of each other.

Results- The mean SPAP in the CHD group was no different measured by RHC or echo (46.49 ± 29.04 vs. 46.45 ± 23 mmHg, p = 0.541). The mean SPAP in the VHD group measured by RHC was significantly higher than that measured by echo (48.70 ± 14.50 vs. 44.90 ± 11.0 mmHg, p = 0.041). Fifty-one (49.5%) patients were found to have PHT at RHC. Echocardiography correctly identified 48 of these patients (sensitivity= 94.1%). Nineteen of the 52 patients without PHT on RHC were correctly identified by echocardiography (specificity= 36.5%). The positive and negative predictive values for echocardiography in assessing the presence or absence of PHT were 59.3% and 86.4%, respectively.

Conclusion- Integration of hemodynamic data with the echo examination can appropriately provide comprehensive assessment of PHT with high sensitivity in individual patients with congenital or valvular heart defects***(Iranian Heart Journal 2011; 12 (4):54-61*)**.

Keywords: Pulmonary hypertension■ Echocardiography■Pulmonary artery pressure■ Right heart catheterization

Pulmonary hypertension (PHT) has been known as a severe and threatening disorder with a markedly decreased exercise tolerance and even heart failure. Because PHT has various major types, a series of diagnostic tests should be performed to distinguish it from some hypoxic, thromboembolic, or miscellaneous varieties.[1]

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[1]Department of Echocardiography, Rajaie Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran
[2]Department of Heart Failure and Transplantation, Rajaie Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran
[3]Department of Cardiology, Rajaie Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran
[4]Department of Cardiology, Rajaie Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran
corresponding author:Ahmad Amin MD, Cardiologist, Fellowship in Heart Failure and Transplantation

In addition, it has been confirmed that improving the survival of PHT patients correlates with its earlier diagnosis.[2,3]

Right heart catheterization (RHC) remains the "gold standard" for the measurement of pulmonary arterial pressure (PAP) and PHT confirmation.

PH has been defined as an increase in mean pulmonary arterial pressure (PAP) > 25 mmHg at rest as assessed by RHC; this value has been used for selecting patients in all RCTs and registries of PHT.[25]

Despite the fact this procedure has acceptable accuracy for the measurement of PAP, significant risks and cost issues are associated with this procedure. Moreover, the technique does not lend itself well to the repeated and temporally close measurements required in clinical trials of interventions in secondary PHT.[5] Thus, a reliable, reproducible, non-invasive technique for the assessment of PAP would clearly be of great clinical value.

Nowadays, echocardiography, particularly when employing the Doppler technique, has increasingly been used for the non-invasive measurement of PHT[6,7], and is currently integrated as an early step in diagnostic algorithms.[8,9] Reliability of this diagnostic approaches has been proven in some practices[10] and has been used for providing an estimate of SPAP and facilitating the identification of PHT in a variety of cardiopulmonary disorders.[11,12]

However, there are only a few studies specifically addressing the accuracy and predictive power of echocardiography for measuring SPAP, especially in conditions suspicious of PHT including congenital or valvular heart diseases. We undertook this study to compare estimated SPAP by echocardiogram with the actual measurements obtained at RHC in the two groups of congenital or valvular heart disease patients in whom PHT was clinically suspected.

Patients And Methods

Study population

The study population consisted of 103 consecutive adult patients with confirmed congenital or valvular heart diseases as defined according to the recently released American College of Cardiology/American Heart Association (ACC/AHA) guidelines criteria. Patients were referred to Rajaie Cardiovascular, Medical and Research center between January and December 2009. Those with a history of coronary artery disease, asthma or obstructive pulmonary disease, and current cigarette smokers were excluded. The patients with poor view in echocardiography or with pulmonary stenosis in transthoracic echocardiography were also excluded. The study protocol was approved by the Ethic Committee on Human Study of Tehran University of Medical Sciences, and written informed consent was obtained from all the patients.

Study measurements

The participants underwent transthoracic echocardiography and RHC within 4 hours of each other.

RHC was performed in the standard manner, and pulmonary artery pressure was measured by a fluid-filled system applying Multi-Purpose A1 catheter, which was introduced under fluoroscopy through the right femoral vein passing through the inferior vena cava to the right ventricle and pulmonary artery. Systolic Pulmonary Arterial Pressure (SPAP) was measured at end-expiration after proper irrigation of the system and confirming the catheter position in fluoroscopy.

Echocardiography was also performed applying a Vivid 3 echo machine (GE) and a 3.5-MHz phase array probe. The IVC size and degree of its collapse were obtained at subcostal view, and a range of right atrial pressure (RAP) was estimated as follows: IVC diameter ≤ 2.1 cm that would collapse >50% with a sniff was taken as normal RA pressure of 3 mmHg (range, 0-5 mm Hg), whereas IVC diameter > 2.1 cm that would collapse < 50% with a sniff was assumed high RA pressure of 15 mmHg (range: 10-20 mm Hg). In scenarios in which the IVC diameter and collapse did not fit this paradigm, an intermediate value of 8 mm Hg (range: 5-10 mmHg) was used.[13, 26] RVSP was then determined from peak tricuspid regurgitation (TR) jet velocity, using the simplified Bernoulli equation and combining this value with an estimate of the RA pressure: RVSP = $4(V)^2$ + RA pressure, where V is the peak velocity (in meters per second) of the tricuspid valve regurgitant jet. In the absence of a pressure gradient across the pulmonic valve or right ventricular outflow tract, SPAP is equal to RVSP.

TR signals were gathered from several views at Doppler sweep speeds of 100 mm/s and technically adequate signals of well-defined borders with the highest velocity were used and averaged in three consecutive beats at end expiration. [Fig. 1] TR signals were enhanced with blood saline contrast in the case of weak signals.

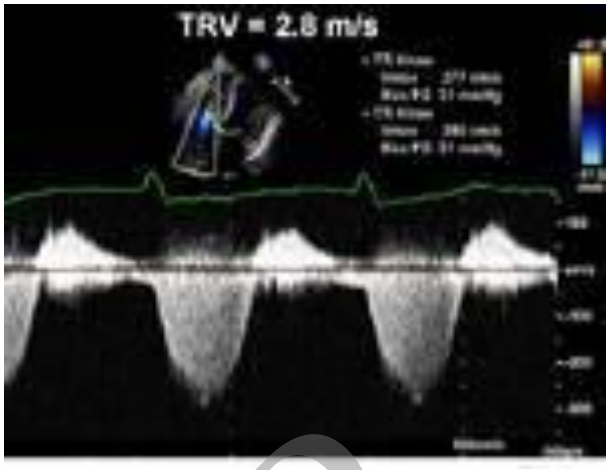

Fig I. Doppler echocardiographic determination of systolic pulmonary artery pressure (SPAP). Spectral continuous-wave Doppler signal of tricuspid regurgitation corresponding to the right ventricular (RV)–right atrial (RA) pressure gradient. SPAP was calculated as the sum of the estimated RA pressure (RAP) and the peak pressure gradient between the peak right ventricle and the right atrium, as estimated by application of the modified Bernoulli equation to peak velocity represented by the tricuspid regurgitation Doppler signal. In this example, SPAP is estimated at 31 + central venous pressure, or 34mmHg, if RAP is assumed to be 3 mm Hg. Adapted from J Am Soc Echocardiogr [26]

Statistical analysis

Data are presented as mean ± SD for the continuous variables and percentages for the categorical variables. Comparisons of the categorical variables across the groups were performed using an overall chi-square test or Fisher exact test if required, while comparisons of the continuous variables were performed using an independent *t*-test or Mann-Whitney U test. RHC diagnosis was considered the standard of reference, and descriptive statistics including sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of echocardiography in determining the presence of pulmonary hypertension (PHT) were calculated. P values of 0.05 or less were considered statistically significant. All the statistical analyses were performed using SPSS version 13 (SPSS Inc.,

Chicago, IL, USA) and the STATA statistical package (version 10.0; College Station, TX, USA).

Results

One hundred and three eligible patients (mean age= 41.0 ± 15.8 years, 44.0% male) were enrolled. Among them, 52 (50.5%) patients suffered VHD (male to female ratio of 19/33) and others had CHD (male to female ratio of 27/24). Distribution of the different types of VHD is presented in Fig. II.

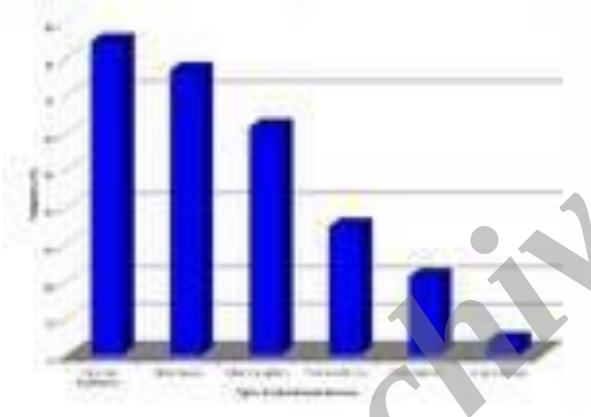

Fig II. Different types of valvular heart diseases in study patients suspected to pulmonary hypertension

The most frequent VHD in the study participants was pulmonary insufficiency (84.6%), followed by mitral stenosis (76.9%) and mitral regurgitation (61.5%).

The mean SPAP in the CHD group was 46.49±29.04 mmHg measured by RHC and 46.45±23.50 mmHg measured by echocardiography, which was not different between the two diagnostic procedures (p= 0.541). The mean SPAP in the VHD group was 48.70 ± 14.50 mmHg measured by RHC and 44.90 ± 11.0 mmHg measured by echocardiography, which was significantly higher in the former group (p = 0.041).

In the VHD group, the mean difference between measured SPAP by RHC and echocardiography in the men was 4.8 mmHg and in the women was 3.3 mmHg (Figure 3). In the CHD group, this mean difference between measured SPAP by the two diagnostic procedures in the men was 2.5 mmHg and in the women was 2.9 mmHg (Figure 3).

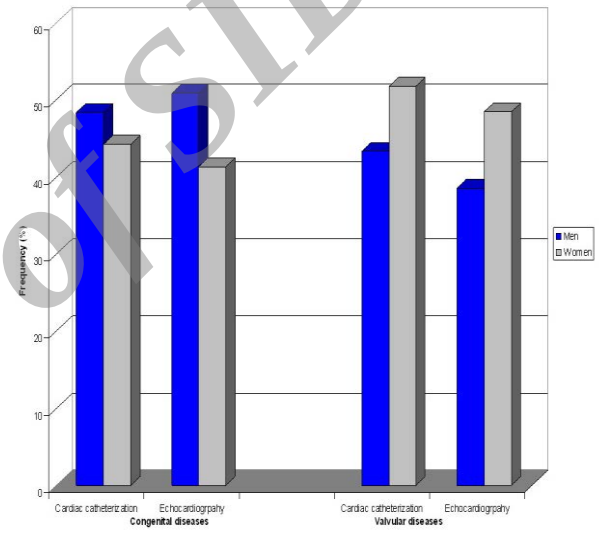

Fig. III. Pulmonary artery pressure measured by right cardiac catheterization and echocardiography in patients with congenital or valvular heart diseases

Fifty-one (49.5%) patients were found to have PHT at RHC. Echocardiography correctly identified 48 of these patients, giving sensitivity for the diagnosis of PHT of 94.1% (Table 1). Nineteen of the 52 patients without PHT on RHC were correctly identified by echocardiography, giving specificity for the diagnosis of 36.5% (Table 1). The positive and negative predictive values for echocardiography in assessing the presence or

absence of PHT were 59.3% and 86.4%, respectively.

Table I. Comparison of right heart catheter and echocardiogram results for all 103 patients studied

	Right cardiac catheterization		
Echo	**PHT**	**Normal**	**Total**
PHT	48	33	**81**
Normal	3	19	**22**
Total	**51**	**52**	**103**

Discussion

Transthoracic echocardiography typically provides several variables in correlation with right heart hemodynamics and should always be performed in the case of suspected PH.[24] Unfortunately, Doppler-derived pressure estimation may be inaccurate in the individual patient. Besides, use of the simplified form of the Bernoulli equation, which was applied in this study, may lead to under or overestimation of SPAP by 10 mmHg. Therefore, PH cannot be reliably defined by a cut-off value of Doppler-derived PA systolic pressure, especially not suitable for screening for mild, asymptomatic PHT.[24] Moreover, the accuracy of transthoracic echocardiography has been investigated in many studies and still remains in question in many cases like HIV, scleroderma, and chronic lung disease in children.[28, 29, 30]

In this study, we focused on the usefulness of echocardiography in identifying the presence or absence of PHT in those with congenital or valvular heart diseases among Iranian population and showed that echocardiography is highly sensitive, but not sufficiently specific for a tool for the diagnosis of PHT in patients with these two types of diseases and clinical suspicion of PHT. Furthermore, our results revealed a significant similarity between the pressure measures obtained at RHC and echocardiography: The difference between the two ranged between 3.3 and 4.8 mmHg in the valvular heart disease group and between 2.5 and 2.9 mmHg in the congenital heart disease group. The clinical usefulness of transthoracic echocardiography as a non-invasive means of estimating SPAP has been reported by several previous investigators.[14-16] In addition, although in some studies the systolic arterial pressure in the pulmonary artery determined on the basis of echocardiography showed a high agreement with the pressure recorded during cardiac catheterization in both human[17] and animal[18] studies, a wide spectrum of its accuracy and predictive power was reported. Some researchers were able to confirm high sensitivity and specificity of this tool[11, 19, 20]; while contrary to our study, some of them revealed its only good and acceptable specificity. As was shown in the Hsu et al. study, in the evaluation of those with suspected PHT, echo appeared to be the most useful of the non-invasive tests, mainly due to the high specificity and high positive predictive value. However the authors reconsidered RHC as the gold standard due to the low sensitivity of the echocardiography testing.[21]

According to our findings, echocardiography offers a reliable and accurate method for the

non-invasive assessment of SPAP with high sensitivity in patients with congenital or valvular defects. In fact, echocardiography may be particularly useful for the early detection and monitoring of potentially fatal complications of these defects with high sensitivity. Be that as it may, because of its low specificity, the use of RHC in suspected patients is strongly recommended. It seems that the diagnostic power of echocardiography can be mainly dependant on the underlying pathophysiology and hemodynamic status of the patient. Campos Filho et al. showed that echo was a useful and non-invasive method for an indirect evaluation of SPAP in adults, especially during stable sinus rhythm, in heart rate ranging from 60 to 115 bpm.[22] Furthermore, Attaran et al. obtained a poor correlation between echocardiography and RHC in both ischemic and non-ischemic cardiomyopathy among patients referred for heart transplant evaluation.[23] Therefore, understanding the hemodynamical profile of the right ventricle and pulmonary circulation can be critical to not only the initial evaluation of, but also the continued management of PHT measured by the two diagnostic tools.[24]

Overall, although RHC remains the gold standard for the diagnosis of PHT and its various causes in most clinical centers, integration of hemodynamic data with the echo examination can appropriately provide a comprehensive assessment of the pathophysiology of PHT with high sensitivity in the individual patients with congenital or valvular heart defects.

Limitations: This study was conducted on a relatively small number of cases and indeed lacks homogeneity for the types of valvular and congenital heart disease. RHC-derived PAP reading is subject to certain errors like whipping artifact, leading to certain overestimation of systolic pulmonary arterial pressure, and also to heart rate and loading status at the time of catheterization and as a result might not be 100% reliable for detecting true pulmonary hypertension. The simplified Bernoulli equation, which was used to estimate the SPAP in echo, may occasionally underestimate the right ventricle-right atrium gradient because of its neglect of the inertial component of the complete Bernoulli equation. Moreover, dependence of echo-derived SPAP on the square of tricuspid regurgitant jet velocity significantly affects the accuracy in cases where there is inadequate spectral Doppler envelope.[27] Much as we tried to make the time between RHC and echo as short as possible, the patients surely had different hemodynamic states during the measurement. Still, these differences might have been too small to consider. What is more, echo data were collected by one sonographer; consequently, whereas interobserver variability is not a problem, intraobserver variability can affect the results and should be considered.

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