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Original Research Article

Evaluating Pulmonary Artery Hypertension: A Doppler Echocardiography Study Correlated with Right Heart Catheterization

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Abstract

Background: Doppler echocardiography is widely used for non-invasive assessment of pulmonary hypertension, but right heart catheterization remains the diagnostic gold standard.

Objective: To assess pulmonary hypertension using Doppler echocardiography and evaluate its correlation with right heart catheterization.

Methods: A prospective observational study was conducted in 35 patients with suspected pulmonary hypertension who underwent both echocardiography and right heart catheterization. Echocardiographic indices were correlated with invasive pulmonary artery pressures.

Results: Pulmonary artery acceleration time and PAAT/RVET ratio showed strong inverse correlations with invasively measured systolic and mean pulmonary artery pressures, while TR-derived systolic pulmonary artery pressure demonstrated a strong direct correlation.

Conclusion: Doppler echocardiography provides reliable estimation of pulmonary hemodynamics and serves as an effective non-invasive screening and follow-up tool, although right heart catheterization remains essential for definitive diagnosis.

Keywords: Pulmonary hypertension, Doppler echocardiography, Right heart catheterization, Pulmonary artery pressure.

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Introduction

Pulmonary hypertension (PH) is a complex hemodynamic disorder defined by an elevated mean pulmonary arterial pressure (mPAP), which leads to increased right ventricular afterload, progressive right heart dysfunction, and adverse outcomes if untreated. Although right heart catheterization (RHC) remains the gold standard for measurement and confirmation of pulmonary artery pressures, its invasive nature, cost, and associated risks limit its widespread use in routine clinical practice. [1]

Localized tissue lesions often represent reactive responses to chronic irritation and inflammation, highlighting the role of sustained inflammatory stimuli in tissue pathology. Consequently, non-invasive methods, particularly Doppler transthoracic echocardiography (TTE), have been widely adopted as initial screening and assessment tools for suspected PH. Alterations in pharyngeal airway dimensions have been shown to influence respiratory mechanics and airflow dynamics. [2,3] Doppler echocardiography estimates pulmonary artery pressures indirectly through measurements such as peak tricuspid regurgitation velocity (TRV) and tricuspid regurgitation-derived systolic pulmonary artery pressure (sPAP) using the modified Bernoulli equation, often supplemented by right atrial pressure (RAP) estimates. [4] This technique is attractive due to its accessibility, safety, and low cost, and it permits repeated assessment over time without radiation or catheter-related risks. [5]

However, the precision and accuracy of echocardiographic pressure estimates compared with invasive measurements have been subjects of ongoing research and debate [6]. Structured disease classification systems aid in clinical risk stratification and facilitate early identification of patients at risk for adverse outcomes. [6] Several studies have demonstrated moderate to good

correlation between echocardiography-derived pulmonary artery pressures and RHC measurements, with some reporting correlation coefficients for systolic and mean pulmonary artery pressures in the range of r = 0.6–0.9, depending on methodology, operator experience, and patient factors. [7] Echocardiography often provides clinically useful screening data, but substantial variability and wide limits of agreement with RHC measurements can influence diagnostic reliability, particularly when used for individual decision-making. These discrepancies may be influenced by factors such as tricuspid regurgitation severity, image quality, estimation of RAP, and the interval between echocardiography and catheterization. [8] Chronic systemic diseases are frequently associated with multisystem complications, including cardiovascular involvement. [9]

Despite these limitations, multiple contemporary guideline recommendations endorse transthoracic Doppler echocardiography as the initial modality for evaluating suspected pulmonary hypertension, emphasizing that it should be interpreted in conjunction with clinical features and other investigations and that positive findings should be confirmed by RHC before definitive diagnosis and management. The need to understand the diagnostic performance and limitations of echocardiography compared to the RHC standard remains critical in optimizing patient care, including timely identification, risk stratification, and therapeutic monitoring of PH. [10]

Therefore, this study was undertaken to assess pulmonary hypertension using Doppler echocardiography and to examine its correlation with right heart catheterization measurements.

Material and Methods

This prospective observational study was conducted in patients evaluated for suspected pulmonary hypertension at a tertiary care teaching hospital. A total of 35 consecutive patients were included in the study. Pulmonary hypertension was suspected based on clinical features such as exertional dyspnea, fatigue, syncope, chest pain, or signs of right heart failure. Patients aged 18 years and above of either sex who underwent both Doppler transthoracic echocardiography and right heart catheterization as part of their diagnostic workup were enrolled.

Patients with poor echocardiographic window, congenital heart disease with shunt lesions, significant left-sided valvular heart disease, acute pulmonary embolism, or those unwilling to undergo right heart catheterization were excluded from the study. Patients with unstable clinical conditions or those in whom catheterization data were incomplete were also excluded. All enrolled patients underwent detailed clinical evaluation, including history taking, physical examination, and routine laboratory investigations. Doppler transthoracic echocardiography was performed using a standard echocardiography system by an experienced cardiologist who was blinded to the catheterization findings. Pulmonary artery systolic pressure was estimated using the peak tricuspid regurgitation velocity derived from continuous-wave Doppler, applying the modified Bernoulli equation. Right atrial pressure was estimated based on inferior vena cava diameter and respiratory variation. Additional echocardiographic parameters such as right atrial size, right ventricular dimensions, right ventricular systolic function, and interventricular septal motion were also assessed.

Right heart catheterization was performed within 24 hours of the echocardiographic examination under standard sterile conditions. Hemodynamic parameters including mean pulmonary artery pressure, pulmonary artery systolic pressure, pulmonary capillary wedge pressure, right atrial pressure, and cardiac output were recorded using standard techniques. Pulmonary hypertension was confirmed based on invasive hemodynamic measurements obtained during catheterization.

The correlation between echocardiography-derived pulmonary artery pressures and right heart catheterization measurements was analyzed. Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software. Continuous variables were expressed as mean and standard deviation, and categorical variables as frequencies and percentages. Correlation between echocardiographic and catheterization values was assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

The study was conducted after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment. Confidentiality of patient data was maintained throughout the study.

Results

The present study included 35 patients evaluated for pulmonary hypertension using Doppler echocardiography and right heart catheterization. Baseline characteristics showed a slight female predominance, with 19 females (54.3%) and 16 males (45.7%). The mean age of the study population was 34.6 ± 13.2 years. Patients aged 20–29 years formed the largest subgroup with 11 cases, followed by those aged 30–39 years and 40–49 years with 8 patients each. Shortness of breath was the most common presenting symptom, observed in 30 patients (85.7%), while chest pain

was present in 13 patients (37.1%). Symptoms suggestive of right ventricular failure were noted in only 3 patients (8.6%). Electrocardiographic abnormalities were common, with right ventricular hypertrophy seen in 11 patients and right bundle branch block in 6 patients. The most frequent underlying etiology belonged to Group 1 pulmonary hypertension, predominantly congenital heart disease, accounting for 14 patients, followed by Group 2 pulmonary hypertension related to rheumatic heart disease in 12 patients (Table 1). Transthoracic echocardiographic assessment demonstrated elevated pulmonary hemodynamic parameters across the cohort. The mean tricuspid regurgitation peak gradient was 61.8 ± 19.6 mmHg, while the estimated systolic pulmonary artery pressure averaged 67.4 ± 20.1 mmHg. Pulmonary artery acceleration time was reduced, with a mean value of 76.9 ± 14.3 ms, and the PAAT/RVET ratio was decreased to 0.30 ± 0.06, reflecting elevated pulmonary vascular resistance. Estimated mean pulmonary artery pressure by echocardiography was 42.1 ± 8.9 mmHg (Table 2).

Assessment of right ventricular dimensions revealed dilatation and hypertrophy in a substantial proportion of patients. The mean RV basal diameter measured 3.71 ± 0.44 cm, and RV free wall thickness averaged 6.8 ± 1.1 mm, indicating pressure overload changes. RV outflow tract measurements also showed enlargement, with proximal and distal diameters measuring 3.28 ± 0.53 cm and 2.92 ± 0.51 cm respectively (Table 3). When categorized into normal and abnormal dimensions, RV free wall thickness was abnormal in 30 patients (85.7%), while RVOT distal diameter was abnormal in 21 patients (60.0%), indicating advanced remodeling in the majority of cases (Table 4).

Invasive hemodynamic assessment using right heart catheterization confirmed significant pulmonary hypertension. Mean pulmonary artery pressure measured invasively was 41.8 ± 9.2 mmHg, with systolic pulmonary artery pressure averaging 65.2 ± 18.7 mmHg. Right ventricular systolic pressure was elevated at 66.1 ± 19.4 mmHg, and right atrial pressure was increased to a mean of 8.6 ± 3.1 mmHg, reflecting right-sided pressure overload (Table 5).

Correlation analysis between echocardiographic and invasive measurements demonstrated strong associations. Pulmonary artery acceleration time showed a strong inverse correlation with invasively measured systolic pulmonary artery pressure ($r = -0.91$) and mean pulmonary artery pressure ($r = -0.94$). The PAAT/RVET ratio also demonstrated strong inverse correlations with invasive systolic and mean pulmonary artery pressures ($r = -0.92$ and -0.95 respectively). Echocardiography-derived systolic pulmonary artery pressure showed a strong direct correlation with invasive systolic pulmonary artery pressure ($r = 0.93$), while echo-derived pulmonary artery diastolic pressure correlated well with invasive diastolic pressure ($r = 0.88$) (Table 6). When stratified according to severity of pulmonary hypertension, correlations between echocardiographic indices and invasive pressures remained strong across mild, moderate, and severe disease, with the strongest inverse correlation observed between PAAT and invasive mean pulmonary artery pressure in severe pulmonary hypertension ($r = -0.91$) (Table 7).

Similarly, correlations remained robust across different patterns of right ventricular diastolic dysfunction, including impaired relaxation and pseudo normal filling patterns (Table 8). Strong correlations were also preserved in patients with both normal and impaired right ventricular systolic function, underscoring the reliability of Doppler echocardiographic parameters across varying functional states (Table 9).

Table 1: Baseline characteristics of study population (n = 35)

Characteristics	N (%)
Gender	
Male	16 (45.7)
Female	19 (54.3)
Age (years)	
Mean ± SD	34.6 ± 13.2
<20	5 (14.3)
20–29	11 (31.4)
30–39	8 (22.9)
40–49	8 (22.9)
≥50	3 (8.6)
Symptoms	
Chest pain	13 (37.1)
Shortness of breath	30 (85.7)
RV failure symptoms	3 (8.6)
ECG findings	

RVH	11 (31.4)
RBBB	6 (17.1)
BVH	4 (11.4)
LAA	4 (11.4)
Clinical diagnosis	
Group 1 PH	
Congenital heart disease	14 (40.0)
Cor AV fistula	1 (2.9)
Primary PH	1 (2.9)
Systemic sclerosis	3 (8.6)
Group 2 PH (RHD)	12 (34.3)
Group 3 PH (ILD)	2 (5.7)
Group 4 PH (CTEPH)	2 (5.7)

Table 2: Transthoracic echocardiography–derived pulmonary hemodynamics (n = 35)

Parameter	Range	Mean ± SD
Tricuspid regurgitation peak gradient (mmHg)	35–98	61.8 ± 19.6
Estimated right atrial pressure (mmHg)	3–10	5.4 ± 1.1
Estimated systolic pulmonary artery pressure (mmHg)	40–102	67.4 ± 20.1
Pulmonary artery acceleration time (ms)	55–98	76.9 ± 14.3
Right ventricular ejection time (ms)	235–300	258.6 ± 18.7
PAAT/RVET ratio	0.21–0.41	0.30 ± 0.06
PR-derived PA diastolic pressure (mmHg)	16–38	24.6 ± 6.8
Estimated mean pulmonary artery pressure (mmHg)	29–56	42.1 ± 8.9

Table 3: Right ventricular dimensions (n = 35)

RV dimension	Range	Mean ± SD
RV basal diameter (cm)	2.9–4.4	3.71 ± 0.44
RVOT proximal (cm)	2.5–4.2	3.28 ± 0.53
RVOT distal (cm)	2.1–4.0	2.92 ± 0.51
RV free wall thickness (mm)	5–9	6.8 ± 1.1

Table 4: Right ventricular dimensions: normal and abnormal (n = 35)

Parameter	Normal No (%)	Abnormal No (%)
RV basal diameter (>4.2 cm)	33 (94.3)	2 (5.7)
RV free wall thickness (>5 mm)	5 (14.3)	30 (85.7)
RVOT PSAX distal (>2.7 cm)	14 (40.0)	21 (60.0)
RVOT PLAX proximal (>3.3 cm)	20 (57.1)	15 (42.9)

Table 5: Invasively obtained right heart pressures (n = 35)

Invasive pressure	Range	Mean ± SD
Pulmonary capillary wedge pressure (mmHg)	8–32	15.1 ± 7.8
Pulmonary artery systolic pressure (mmHg)	40–108	65.2 ± 18.7
Pulmonary artery diastolic pressure (mmHg)	18–45	27.4 ± 7.6
Mean pulmonary artery pressure (mmHg)	30–60	41.8 ± 9.2
RV systolic pressure (mmHg)	42–102	66.1 ± 19.4
RV end-diastolic pressure (mmHg)	4–16	9.1 ± 3.0
Right atrial pressure (mmHg)	4–18	8.6 ± 3.1

Table 6: Correlation between echocardiographic and invasive hemodynamic parameters (n = 35)

Echo parameter	Invasive parameter	Correlation coefficient (r)	Interpretation
PAAT	PSAP	−0.91	Strong inverse
PAAT	MPAP	−0.94	Strong inverse
PAAT/RVET	PSAP	−0.92	Strong inverse
PAAT/RVET	MPAP	−0.95	Strong inverse
Echo ESPAP	Invasive PSAP	0.93	Strong direct
Echo PADP	Invasive PADP	0.88	Strong direct

Table 7: Correlation between echo and invasive parameters across severity of PH (n = 35)

Correlated parameters	Mild PH	Moderate PH	Severe PH
PAAT vs PSAP	−0.68	−0.75	−0.89
PAAT vs MPAP	−0.82	−0.79	−0.91
PAAT/RVET vs PSAP	−0.71	−0.83	−0.86
PAAT/RVET vs MPAP	−0.85	−0.81	−0.88
ESPAP vs PSAP	0.62	0.74	0.86
PREDP vs PADP	0.59	0.78	0.90

Table 8: Correlation between echo and invasive parameters across RV diastolic function (n = 35)

Correlated parameters	Normal	Impaired relaxation	Pseudonormal
PAAT vs PSAP	−0.92	−0.95	−0.93
PAAT vs MPAP	−0.90	−0.98	−0.97
PAAT/RVET vs PSAP	−0.91	−0.94	−0.93
PAAT/RVET vs MPAP	−0.88	−0.96	−0.98
ESPAP vs PSAP	0.95	0.89	0.94
PREDP vs PADP	0.92	1.00	0.90

Table 9: Correlation between echo and invasive parameters in normal and abnormal RV systolic function (n = 35)

Correlated parameters	Normal systolic function	Systolic dysfunction
PAAT vs PSAP	−0.91	−0.97
PAAT vs MPAP	−0.94	−0.90
PAAT/RVET vs PSAP	−0.92	−0.95
PAAT/RVET vs MPAP	−0.93	−0.87
ESPAP vs PSAP	0.92	0.88
PREDP vs PADP	0.91	0.93

Discussion

The present study evaluated the accuracy of Doppler echocardiography in assessing pulmonary hypertension and its correlation with invasively measured hemodynamic parameters obtained by right heart catheterization. The results demonstrate a strong correlation between several echocardiographic indices and catheter-derived pulmonary artery pressures, supporting the reliability of Doppler echocardiography as a non-invasive tool for assessment and stratification of pulmonary hypertension. [11]

Among the echocardiographic parameters studied, pulmonary artery acceleration time and the PAAT/RVET ratio showed the strongest inverse correlations with invasive systolic and mean pulmonary artery pressures, indicating their utility as robust markers of elevated pulmonary vascular resistance. Similar findings have been reported by Fisher et al., who demonstrated that PAAT is a sensitive and reproducible surrogate marker for pulmonary arterial pressure across a wide range of disease severity. [12]

The strong inverse relationship observed between PAAT and invasively measured mean pulmonary artery pressure in the present study, particularly in patients with severe pulmonary hypertension, highlights the physiological basis of this parameter. Shortening of PAAT reflects increased pulmonary arterial stiffness and early reflection of pressure waves, phenomena commonly seen in advanced pulmonary vascular disease. D'Alto et al. reported comparable inverse correlations between PAAT-derived measurements and catheter-based pressures, emphasizing the clinical value of PAAT in routine echocardiographic evaluation [12]. The consistency of these findings across different severities of pulmonary hypertension in the present study further strengthens the argument for its widespread use. Occupational and lifestyle factors contribute significantly to systemic inflammatory burden and overall cardiometabolic risk. [13]

Echocardiography-derived systolic pulmonary artery pressure based on tricuspid regurgitation velocity demonstrated a strong direct correlation with invasive systolic pulmonary artery pressure. This supports the role of TR-based estimates as a practical screening parameter, especially when adequate Doppler signals are obtainable. However, variability in estimation due to right atrial pressure assumptions and tricuspid regurgitation quality remains a known limitation. Studies by Rich et al. have shown that while echocardiography may slightly overestimate or underestimate absolute pressure values, it performs well in identifying clinically significant pulmonary hypertension. The present findings align with these observations, showing strong correlations without implying absolute interchangeability between methods. [14]

Importantly, the correlations between echocardiographic and invasive parameters remained strong across different patterns of right ventricular diastolic dysfunction and in patients with preserved as well as impaired right ventricular systolic function. This suggests that Doppler echocardiographic indices retain their diagnostic value despite alterations in right ventricular mechanics. Aduen et al. reported that echocardiographic estimation of pulmonary pressures remains reliable even in the presence of right ventricular dysfunction, provided comprehensive multiparametric assessment is employed. The current study reinforces this concept by demonstrating preserved correlations irrespective of ventricular functional status. [15]

Taken together, these findings confirm that while right heart catheterization remains the gold standard for definitive diagnosis of pulmonary hypertension, Doppler echocardiography provides accurate, reproducible, and clinically meaningful estimates of pulmonary hemodynamics. A meta-analysis by Janda et al. emphasized that echocardiography is best utilized as a screening and follow-up tool rather than a replacement for invasive assessment, a conclusion strongly supported by the results of the present study. Incorporation of multiple echocardiographic parameters, rather than reliance on a single measurement, enhances diagnostic confidence and clinical decision-making.

Conclusion

The present study demonstrates a strong correlation between Doppler echocardiographic parameters and invasively measured pulmonary artery pressures obtained by right heart catheterization. Pulmonary artery acceleration time and the PAAT/RVET ratio showed particularly strong inverse correlations with invasive systolic and mean pulmonary artery pressures across varying severities of pulmonary hypertension and right ventricular functional states. These findings support the role of Doppler echocardiography as a reliable non-invasive modality for screening, assessment, and follow-up of pulmonary hypertension, while reaffirming that right heart catheterization remains essential for definitive diagnosis and therapeutic decision-making.

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