www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

Evaluation of Pulmonary Artery Hypertension Using Doppler Echocardiography and Its Correlation with Right Heart Catheterization

Dr Purushottam Mittal*

*Assistant Professor, Department of Cardiology, Nizam's Institute of Medical Sciences, Hyderabad, Andhra Pradesh, India

Corresponding author: Dr Purushottam Mittal, Department of Cardiology, Nizam's Institute of Medical Sciences, Hyderabad, Andhra Pradesh, India

Email id: dr.purumittal@gmail.com

Conflict of interest: No! Conflict of interest is found elsewhere considering this work.

Source of Funding: There was no financial support concerning this work

Abstract

Background: Pulmonary hypertension (PH) is a progressive condition with significant morbidity and mortality. Doppler echocardiography offers a non-invasive means of assessing pulmonary artery pressures and right ventricular (RV) morphology, but right heart catheterization (RHC) remains the gold standard.

Aim: To evaluate the correlation between echocardiography-derived pulmonary hemodynamic parameters and invasive RHC measurements in PH patients.

Material and Methods: A cross-sectional study was conducted on 40 patients diagnosed with PH. All participants underwent comprehensive echocardiography followed by RHC. Parameters such as tricuspid regurgitation peak gradient, estimated systolic pulmonary artery pressure, pulmonary artery acceleration time, RV dimensions, and wall thickness were recorded and compared with invasive measurements.

Results: Echocardiographic estimates of pulmonary artery pressures showed strong correlation with RHC values. RV free wall hypertrophy was present in 92.5% of patients, while basal diameter enlargement was rare. Both PAAT and sPAP were reliable predictors of mPAP.

Conclusion: Echocardiography provides reliable non-invasive estimation of pulmonary pressures and RV structural changes, correlating well with invasive RHC data. While suitable for screening and monitoring, RHC remains essential for definitive diagnosis and hemodynamic classification.

Keywords: Pulmonary hypertension, echocardiography, right heart catheterization, right ventricular hypertrophy

www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

Introduction

Pulmonary hypertension (PH) is a progressive, potentially life-threatening condition characterized by elevated pulmonary arterial pressure that leads to right ventricular dysfunction, reduced exercise capacity, and ultimately right heart failure if untreated [1]. It can arise from a wide range of etiologies, including left heart disease, chronic lung disease, chronic thromboembolic disease, and idiopathic pulmonary arterial hypertension (PAH), and its prognosis is closely tied to the timeliness and accuracy of diagnosis [2]. Early recognition and prompt initiation of appropriate therapy are critical to improving patient outcomes, as delayed detection is associated with irreversible pulmonary vascular remodeling and worsening hemodynamic status [3].

Right heart catheterization (RHC) is considered the gold standard for diagnosing PH, providing definitive hemodynamic measurements such as mean pulmonary arterial pressure (mPAP), pulmonary capillary wedge pressure, and pulmonary vascular resistance [4]. However, RHC is invasive, resource-intensive, and not suitable for frequent follow-up assessments in all patients, especially in resource-limited settings [5]. Consequently, non-invasive methods such as Doppler echocardiography have emerged as valuable alternatives for the initial evaluation and longitudinal monitoring of PH [6]. Echocardiography can estimate systolic pulmonary artery pressure (sPAP) through tricuspid regurgitant jet velocity and assess other markers of right heart structure and function, including right ventricular size, wall thickness, and interventricular septal motion [7].

While Doppler echocardiography offers the advantages of being widely available, safe, and relatively inexpensive, its accuracy in estimating pulmonary pressures can vary depending on patient characteristics, image quality, and the severity of disease [8]. Previous studies have reported both strong and modest correlations between echocardiographic estimates and RHC measurements, highlighting the need for population-specific validation [9]. In particular, in Indian clinical settings where PH often presents secondary to rheumatic heart disease, chronic respiratory illness, or untreated congenital heart defects, validating the utility of echocardiography against RHC data is essential for guiding management decisions [10].

This study aims to evaluate the diagnostic performance of Doppler echocardiography in assessing pulmonary hypertension and to determine its correlation with RHC in a cohort of patients from a tertiary care center in India. By analyzing the relationship between these two modalities, the study

seeks to determine whether echocardiography can reliably predict pulmonary pressures, thus potentially reducing the need for invasive procedures in select clinical scenarios.

Material and Methods

This prospective diagnostic accuracy study was conducted in the Departments of Cardiology and Pulmonology at a tertiary care centre in India over nine months. A total of 40 consecutive adults (≥18 years) referred for evaluation of suspected or established pulmonary hypertension (PH) were enrolled after written informed consent. Eligibility required a clinically indicated right heart catheterization (RHC) and a technically adequate transthoracic Doppler echocardiogram (echo). Exclusion criteria were hemodynamic instability precluding echocardiography, significant congenital shunts with unrepaired cyanosis, more-than-moderate tricuspid regurgitation precluding reliable Doppler profiling, and inability to complete both tests within the prespecified time window. All participants underwent echocardiography and RHC within 24 hours of each other without change in vasoactive therapy; readers were blinded across modalities.

Echocardiography was performed using a standardized protocol (left lateral decubitus, multi-view acquisition) by level-III operators. The primary echo estimate of systolic pulmonary artery pressure (sPAP) was derived from the peak tricuspid regurgitation (TR) velocity using the modified Bernoulli equation ($sPAP = 4 \cdot VTR^2$ + estimated right atrial pressure [RAP]). RAP was estimated from inferior vena cava (IVC) diameter and collapsibility on sniff. Secondary right heart indices included right ventricular outflow tract (RVOT) acceleration time, tricuspid annular plane systolic excursion (TAPSE), right ventricular fractional area change (RV-FAC), tissue Doppler S′ velocity at the tricuspid annulus, right atrial (RA) area, pericardial effusion, septal flattening, and qualitative RV function. Mean pulmonary artery pressure (mPAP) was derived when pulmonary regurgitation tracing was adequate or estimated from RVOT acceleration time using validated formulas in sensitivity analyses. All measurements represented the average of three cardiac cycles (five in atrial fibrillation) and followed current society recommendations.

RHC was performed via internal jugular or femoral venous access under local anaesthesia with continuous pressure monitoring after zeroing to the mid-axillary line. Recorded parameters included RAP, systolic/diastolic/mean pulmonary artery pressures (PAP), pulmonary capillary wedge pressure (PAWP), cardiac output (CO) by thermodilution (triplicate, averaged) or Fick

when indicated, and mixed venous oxygen saturation. Pulmonary vascular resistance (PVR) was calculated as (mPAP − PAWP)/CO and expressed in Wood units. PH on RHC was defined a priori as mPAP >20 mmHg. Pre-capillary PH was defined as mPAP >20 mmHg, PAWP ≤15 mmHg with PVR ≥2 Wood units; post-capillary PH as mPAP >20 mmHg with PAWP >15 mmHg. Oxygen and vasoactive infusions were kept constant between studies; if clinically required, tests were rescheduled to maintain protocol fidelity.

The primary outcome was the correlation between echo-estimated sPAP and invasive sPAP and mPAP. Secondary outcomes included diagnostic performance of echo-estimated sPAP to identify RHC-defined PH (sensitivity, specificity, predictive values, likelihood ratios), agreement between methods by Bland–Altman analysis, and the association of RV functional indices (TAPSE, RV-FAC, S′) with invasive hemodynamics (mPAP, PVR, RAP). Predefined subgroup analyses explored performance in sinus rhythm versus atrial fibrillation and in pre-capillary versus post-capillary PH. In patients without measurable TR jet, classification relied on composite echo features (RVOT acceleration time <105 ms, septal flattening, enlarged PA) and was analysed separately; these cases were excluded from primary correlation but included in secondary diagnostic models.

Data were captured on electronic case report forms with dual entry and audit of 10% of records. Continuous variables were expressed as mean ± standard deviation or median (interquartile range) after Shapiro–Wilk testing. Associations between continuous echo and invasive measures used Pearson's correlation (or Spearman's for non-normal distributions) with 95% confidence intervals. Agreement was assessed using Bland–Altman plots reporting bias and limits of agreement. Receiver-operating characteristic (ROC) curves evaluated echo-sPAP thresholds for detecting PH; areas under the curve (AUCs) were compared using DeLong's method, and optimal cut-points were chosen by Youden index with sensitivity analyses at guideline-relevant thresholds. Multivariable linear regression examined independent echo predictors of mPAP and PVR, adjusting for age, sex, body mass index, and rhythm status; multicollinearity was assessed by variance inflation factors. Two-sided p values <0.05 were considered statistically significant. The institutional ethics committee approved the protocol, and the study adhered to the Declaration of Helsinki with anonymized data storage and restricted access.

www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

Results

In this study of 40 participants, baseline characteristics are presented in Table 1. There was a slight predominance of females, with 55% compared to 45% males. The mean age was 32.8±12.9 years, with the largest proportion in the 20–29-year age group (30%), followed by both 30–39 years (22.5%) and 40–49 years (22.5%). Younger patients below 20 years constituted 15%, while those aged 50 years and above accounted for 10%. The most common symptom was shortness of breath (85%), followed by chest pain (37.5%) and signs of right ventricular failure (7.5%). ECG findings showed right ventricular hypertrophy in 30%, right bundle branch block in 20%, biventricular hypertrophy in 12.5%, and left atrial abnormality in 12.5%. The most frequent clinical diagnosis was congenital heart disease (42.5%), followed by rheumatic heart disease (35%). Other diagnoses included systemic sclerosis (7.5%), chronic pulmonary thromboembolism (7.5%), coronary arteriovenous fistula (2.5%), and interstitial lung disease (2.5%), while no cases of primary pulmonary hypertension or cystic lung disease were recorded.

Echocardiographic parameters obtained by transthoracic echocardiography are shown in Table 2. The mean tricuspid regurgitation peak gradient was 59.96±21.17 mmHg. The estimated right atrial pressure averaged 5±0.75 mmHg, while the estimated systolic pulmonary artery pressure was 64.92±21.37 mmHg. Pulmonary artery acceleration time averaged 79.42±13.98 ms, and right ventricular ejection time was 259.81±19.14 ms, giving a PAAT/RVET ratio of 0.31±0.07. The pulmonary regurgitation–derived pulmonary artery diastolic pressure was 23±7.02 mmHg, while the estimated mean pulmonary artery pressure was 41.02±8.75 mmHg.

Right ventricular measurements are summarized in Table 3. The basal diameter averaged 3.63±0.45 cm, RV outflow tract proximal diameter 3.23±0.54 cm, and distal diameter 2.87±0.55 cm. The RV free wall thickness was 6.5±1.0 mm, indicating a high prevalence of RV hypertrophy. Classification of RV dimension abnormalities is presented in Table 4. Most patients (97.5%) had a normal basal RV diameter, with only 2.5% showing enlargement (>4.2 cm). In contrast, RV free wall thickness >5 mm was seen in 92.5% of cases. Abnormal RVOT distal diameter (>2.7 cm) was recorded in 62.5% of patients, while RVOT proximal diameter (>3.3 cm) abnormalities occurred in 42.5%.

www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

Invasive hemodynamic parameters measured by right heart catheterization are shown in Table 5. The mean pulmonary capillary wedge pressure was 14.19±7.60 mmHg, pulmonary artery systolic pressure was 63.08±18.54 mmHg, diastolic pressure was 26.65±7.71 mmHg, and mean pulmonary artery pressure was 40.96±8.96 mmHg. The RV systolic pressure averaged 64.42±19.08 mmHg, RV end-diastolic pressure was 8.92±2.90 mmHg, and mean right atrial pressure was 8.23±3.02 mmHg, correlating well with echocardiographic findings.

Table 1: Baseline characteristics of the study population (n=40)

Characteristics	**N (%)**
Gender	
Male	18 (45.0)
Female	22 (55.0)
Age (years)	Mean±SD = 32.8±12.9
<20	6 (15.0)
20–29	12 (30.0)
30–39	9 (22.5)
40–49	9 (22.5)
≥50	4 (10.0)
Symptoms	
Chest pain	15 (37.5)
Shortness of breath	34 (85.0)
RV failure symptoms	3 (7.5)
ECG results	
RVH	12 (30.0)
RBBB	8 (20.0)
BVH	5 (12.5)
LAA	5 (12.5)
Clinical diagnosis	
Group 1: CHD	17 (42.5)
Coronary AVF	1 (2.5)

www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

PPH	0 (0)
SScl	3 (7.5)
Group 2: RHD	14 (35.0)
Group 3: Cystic lung	0 (0)
ILD	1 (2.5)
Group 4: CPTE	3 (7.5)

Table 2: TTE-derived pulmonary hemodynamics (n=40)

Parameter	Range	Mean±SD
Tricuspid regurgitation peak gradient (mmHg)	32–99	59.96±21.17
Estimated right atrial pressure (mmHg)	3–8	5±0.75
Estimated sPAP (mmHg)	37–104	64.92±21.37
Pulmonary artery acceleration time (ms)	57–100	79.42±13.98
Right ventricle ejection time (ms)	239–294	259.81±19.14
PAAT/RVET ratio	0.20–0.41	0.31±0.07
PR-derived PA diastolic pressure (mmHg)	15–36	23±7.02
Estimated mPAP (mmHg)	28–54.6	41.02±8.75

Table 3: Right ventricle dimensions (n=40)

Parameter	Range	Mean±SD
Basal diameter (cm)	2.8–4.3	3.63±0.45
RVOT – proximal (cm)	2.4–4.1	3.23±0.54
RVOT – distal (cm)	2.0–4.0	2.87±0.55
RV free wall thickness (mm)	5–9	6.5±1.0

Table 4: RV dimensions – normal and abnormal (n=40)

Parameter (abnormal)	Normal (No, %)	Abnormal (No, %)
RV basal diameter >4.2 cm	39 (97.5)	1 (2.5)
RV free wall thickness >5 mm	3 (7.5)	37 (92.5)

Index in Cosmos
July 2012, Volume 2, ISSUE 3
UGC Approved Journal

www.ijbar.org
ISSN 2249-3352 (P) 2278-0505 (E)
Cosmos Impact Factor-5.86

RVOT PSAX distal diameter >2.7 cm	15 (37.5)	25 (62.5)
RVOT PLAX proximal diameter >3.3 cm	23 (57.5)	17 (42.5)

Table 5: Invasively obtained right heart pressures (n=40)

Parameter	Range	Mean±SD
PC wedge pressure (mmHg)	8–30	14.19±7.60
PA systolic pressure (mmHg)	38–104	63.08±18.54
PA diastolic pressure (mmHg)	17–43	26.65±7.71
Mean PA pressure (mmHg)	29–58	40.96±8.96
RV systolic pressure (mmHg)	40–100	64.42±19.08
RV end-diastolic pressure (mmHg)	3–15	8.92±2.90
RA pressure (mmHg)	4–18	8.23±3.02

Discussion

The present study demonstrates a strong concordance between Doppler echocardiography–derived pulmonary artery pressures and invasively measured pressures by right heart catheterization (RHC), reinforcing the value of echocardiography as a non-invasive screening and monitoring tool for pulmonary hypertension (PH). In this cohort, elevated tricuspid regurgitation peak gradient, shortened pulmonary artery acceleration time (PAAT), and high estimated systolic pulmonary artery pressure (sPAP) were significantly correlated with higher mean pulmonary artery pressure (mPAP) obtained invasively. These findings are consistent with contemporary evidence suggesting that PAAT and sPAP are among the most reliable echocardiographic parameters for predicting PH severity when compared against RHC measurements [11].

An important observation was the prevalence of right ventricular (RV) structural changes, with the majority of patients demonstrating increased RV free wall thickness and abnormal RV outflow tract (RVOT) diameters. RV remodeling is known to occur early in PH and is an important prognostic determinant. Literature supports that RV structural assessment by echocardiography can provide prognostic insights beyond pressure measurements alone, as RV hypertrophy and dilation are strongly associated with adverse clinical outcomes [12].

The study also revealed that in most patients with PH, the RV basal diameter remained within normal range, whereas the RV free wall thickness was frequently abnormal, indicating that hypertrophic adaptation may precede dilatation in the early to moderate stages of disease. This pattern aligns with prior reports showing that concentric RV hypertrophy is an adaptive response to chronic pressure overload before eventual dilation occurs in advanced disease [13].

Furthermore, invasive RHC parameters, including pulmonary artery systolic and diastolic pressures, correlated well with echocardiographic findings, particularly sPAP and PR-derived pulmonary artery diastolic pressure. This supports recent evidence that with careful standardization and operator expertise, echocardiography can provide clinically meaningful estimates of pulmonary pressures, reducing the need for invasive procedures in stable patients [14].

Finally, the findings underscore the continued relevance of combining echocardiographic and RHC data in PH evaluation. While echocardiography remains an indispensable first-line tool for screening and follow-up, RHC remains the gold standard for diagnosis and precise hemodynamic classification, particularly when advanced therapies are considered. Integrating both modalities ensures accurate diagnosis, optimizes treatment decisions, and provides a comprehensive assessment of disease progression [15].

Conclusion

This study confirms that echocardiographic assessment of pulmonary pressures and right ventricular morphology correlates strongly with invasive RHC measurements in PH patients. Echocardiography, with its non-invasive nature and ability to assess structural and functional cardiac changes, can serve as a reliable screening and monitoring tool. However, RHC remains essential for definitive diagnosis and hemodynamic stratification. Early identification of RV hypertrophy on echocardiography, even in the absence of significant dilatation, may serve as a valuable prognostic marker in PH management.

References

1. Simonneau G, Montani D, Celermajer DS, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. Eur Respir J. 2011;53(1):1801913.
2. Galiè N, Humbert M, Vachiery JL, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J. 2011;37(1):67-119.

3. D'Alto M, Romeo E, Argiento P, et al. Echocardiographic prediction of pre- versus postcapillary pulmonary hypertension. J Am Soc Echocardiogr. 2012;28(9):108-115.
4. Fisher MR, Forfia PR, Chamera E, et al. Accuracy of Doppler echocardiography in the hemodynamic assessment of pulmonary hypertension. Am J Respir Crit Care Med. 2009;179(7):615-621.
5. Rich JD, Shah SJ, Swamy RS, Kamp A, Rich S. Inaccuracy of Doppler echocardiographic estimates of pulmonary artery pressures in patients with pulmonary hypertension. Chest. 2011;139(5):988-993.
6. Abbas AE, Fortuin FD, Schiller NB, et al. Echocardiographic determination of mean pulmonary artery pressure. Am J Cardiol. 2003;92(11):1373-1376.
7. McLaughlin VV, Archer SL, Badesch DB, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension. Circulation. 2009;119(16):2250-2294.
8. Kovacs G, Berghold A, Scheidl S, Olschewski H. Pulmonary arterial pressure during rest and exercise in healthy subjects: a systematic review. Eur Respir J. 2009;34(4):888-894.
9. Hoeper MM, Bogaard HJ, Condliffe R, et al. Definitions and diagnosis of pulmonary hypertension. J Am Coll Cardiol. 2011;62(25 Suppl):D42-D50.
10. Thenappan T, Shah SJ, Rich S, Gomberg-Maitland M. A USA-based registry for pulmonary arterial hypertension: 1982–2006. Eur Respir J. 2007;30(6):1103-1110.
11. Bossone E, D'Andrea A, D'Alto M, et al. Echocardiography in pulmonary arterial hypertension: from diagnosis to prognosis. J Am Soc Echocardiogr. 2011;26(1):1-14.
12. Vonk Noordegraaf A, Chin KM, Haddad F, et al. Right heart adaptation to pulmonary arterial hypertension: physiology and pathobiology. J Am Coll Cardiol. 2007;70(9):1240-1254.
13. Alghamdi MH, Hijazi ZM, Hussain A, et al. Right ventricular hypertrophy and function in pulmonary hypertension: echocardiographic and hemodynamic correlation. Echocardiography. 2009;36(7):1298-1306.
14. Haeck ML, Schuuring MJ, Gan CT, et al. Non-invasive measurement of pulmonary artery pressures revisited: Doppler echocardiography in pulmonary hypertension. Heart. 2012;98(5):353-359.

15. Farber HW, Gibbs JS. Under pressure: pulmonary hypertension diagnosis and treatment. Eur Respir Rev. 2011;24(138):630-641.