

Research Article

Relationship Between Pulmonary Artery Pressure and Left Atrial Enlargement: A Retrospective Study

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Abstract

Left atrial enlargement (LAE) and elevated pulmonary artery pressure (PAP) are interrelated hemodynamic markers frequently encountered in cardiovascular disease. This retrospective study evaluates their association in 120 patients undergoing transthoracic echocardiography.

The mean pulmonary artery systolic pressure was 42.6 ± 11.8 mmHg. Left atrial diameter averaged 4.6 ± 0.7 cm, while left atrial volume index (LAVI) was 38.2 ± 9.5 mL/m². Patients demonstrated a significant positive correlation between PAP and LA diameter ($r = 0.61$, $p < 0.001$), and a stronger correlation with LAVI ($r = 0.74$, $p < 0.001$).

Subjects with elevated PAP (≥ 40 mmHg) showed significantly increased LAVI (45.3 ± 8.9 mL/m²) compared to those with normal PAP (31.5 ± 6.8 mL/m², $p < 0.001$). Multivariate regression confirmed LAVI as an independent predictor of elevated PAP ($\beta = 0.68$, $p < 0.001$), even after adjusting for age, hypertension, and left ventricular diastolic dysfunction.

These findings highlight a strong structural-hemodynamic relationship between left atrial remodeling and pulmonary vascular pressure elevation. LA enlargement, particularly volumetric assessment, may serve as an early marker of pulmonary hypertension progression and left heart disease severity.

Integration of LA metrics in routine echocardiographic assessment may improve early detection and risk stratification in cardiopulmonary disorders.

Keywords: Pulmonary artery pressure, left atrial enlargement, echocardiography, diastolic dysfunction

1. Introduction

Pulmonary artery pressure represents a fundamental component of cardiovascular hemodynamics, reflecting the pressure load imposed on the right ventricle and pulmonary circulation. In clinical practice, pulmonary artery systolic pressure (PASP) estimated by Doppler echocardiography has become a widely accepted non-invasive surrogate for pulmonary hemodynamic assessment. Elevation of PAP is commonly associated with a broad spectrum of cardiovascular and pulmonary disorders, including left heart disease, chronic obstructive pulmonary disease, thromboembolic disease, and systemic metabolic disorders [1–3].

Left atrial enlargement is a structural adaptation that develops in response to chronic pressure and volume overload conditions. The left atrium functions as a reservoir, conduit, and contractile chamber that regulates pulmonary venous return into the left ventricle. When left ventricular filling pressures increase, the left atrium is subjected

to persistent hemodynamic stress, leading to progressive dilation and structural remodeling. This process is commonly observed in systemic hypertension, mitral valve disease, and heart failure with preserved ejection fraction [4–6].

The relationship between left atrial enlargement and pulmonary artery pressure is physiologically mediated by backward transmission of elevated left ventricular filling pressures. Increased left atrial pressure is transmitted to the pulmonary veins, resulting in pulmonary venous congestion and subsequent elevation of pulmonary arterial pressure. This mechanism forms the basis of post-capillary pulmonary hypertension, which is the most common form of pulmonary hypertension encountered in clinical practice [7–9].

In recent years, echocardiographic evaluation of left atrial volume index (LAVI) has gained importance as a more accurate marker of chronic diastolic burden compared to linear left atrial diameter. LAVI reflects cumulative exposure to elevated filling pressures over time and has been shown to correlate strongly with adverse cardiovascular outcomes, including atrial fibrillation, heart failure, and mortality [10–12].

Despite increasing recognition of the interplay between left atrial remodeling and

pulmonary vascular pressure, most clinical studies evaluate these parameters independently. There remains a lack of integrated analysis assessing the direct quantitative relationship between PAP and LAE in real-world echocardiographic populations. This gap limits the ability to fully understand the progression from left-sided cardiac dysfunction to pulmonary vascular remodeling [13–15].

Furthermore, comorbid conditions such as systemic hypertension, diabetes mellitus, ischemic heart disease, and chronic kidney disease contribute significantly to both left atrial enlargement and elevated pulmonary pressure. These conditions induce myocardial fibrosis, vascular stiffening, endothelial dysfunction, and neurohormonal activation, which collectively exacerbate cardiac remodeling processes [16–18].

Given the increasing burden of cardiovascular disease worldwide and the widespread use of echocardiography in routine clinical evaluation, understanding the relationship between PAP and LAE has important diagnostic and prognostic implications. Identifying early structural markers of pulmonary hypertension could improve risk stratification and guide early therapeutic intervention [19–20].

Therefore, this retrospective study aims to evaluate the association between pulmonary artery pressure and left atrial enlargement using echocardiographic data from a real-world clinical population, with emphasis on structural and functional cardiac interactions.

2. Methodology

This retrospective observational study included 120 adult patients who underwent transthoracic echocardiography at Gulab Devi Teaching Hospital, Lahore a tertiary care cardiac center. Patients were selected based on availability of complete echocardiographic measurements including pulmonary artery systolic pressure, left atrial diameter, and left atrial volume index.

PAP was estimated using tricuspid regurgitation velocity and the modified Bernoulli equation. Left atrial measurements were obtained according to standardized echocardiographic guidelines recommended by the American Society of Echocardiography. Patients with congenital heart disease, primary pulmonary arterial hypertension, acute pulmonary embolism, or prior cardiac surgery were excluded to eliminate confounding hemodynamic influences.

Sample size estimation was performed using Epi Info StatCalc software, assuming a moderate correlation coefficient ($r = 0.3$ –

0.4), 95% confidence interval, and 80% power, resulting in a minimum required sample size of approximately 100 patients. Data included demographic variables, comorbid conditions, and echocardiographic

parameters. Statistical analysis included Pearson correlation, independent sample t-test, and multivariate linear regression. A p-value < 0.05 was considered statistically significant.

3. Results

Table 1: Baseline Demographic and Clinical Characteristics (n = 120)

Variable	Value
Age (years)	56.8 ± 13.4
Male sex	62 (51.7%)
Female sex	58 (48.3%)
Hypertension	78 (65.0%)
Diabetes mellitus	46 (38.3%)
Ischemic heart disease	41 (34.1%)
Smoking history	39 (32.5%)
Dyslipidemia	52 (43.3%)

The study population represents a typical cardiology outpatient cohort with significant cardiovascular risk burden, predisposing to both left atrial remodeling and pulmonary pressure elevation.

Table 2: Echocardiographic and Hemodynamic Parameters

Parameter	Mean ± SD
Pulmonary artery systolic pressure (mmHg)	42.6 ± 11.8
Left atrial diameter (cm)	4.6 ± 0.7
Left atrial volume index (mL/m ²)	38.2 ± 9.5
Left ventricular ejection fraction (%)	54.3 ± 7.6

Parameter	Mean $\pm$ SD
E/e' ratio	13.8 $\pm$ 4.2
Tricuspid regurgitation velocity (m/s)	3.1 $\pm$ 0.5

Elevated PAP and increased LA structural indices suggest coexistence of diastolic dysfunction and early pulmonary vascular involvement in the majority of patients.

Table 3: Comparison of Echocardiographic Parameters Based on Pulmonary Artery Pressure

Parameter	Normal PAP (<40 mmHg) n=52	Elevated PAP ($\geq$ 40 mmHg) n=68	p-value
LA diameter (cm)	4.1 $\pm$ 0.5	4.9 $\pm$ 0.6	<0.001
LA volume index (mL/m ²)	31.5 $\pm$ 6.8	45.3 $\pm$ 8.9	<0.001
E/e' ratio	10.2 $\pm$ 2.8	16.4 $\pm$ 3.9	<0.001
LVEF (%)	56.1 $\pm$ 6.9	52.8 $\pm$ 8.1	0.03
TR velocity (m/s)	2.6 $\pm$ 0.3	3.5 $\pm$ 0.4	<0.001

- PAP vs LA diameter $\rightarrow r = 0.61, p < 0.001$
- PAP vs LA volume index $\rightarrow r = 0.74, p < 0.001$
- PAP vs E/e' ratio $\rightarrow r = 0.69, p < 0.001$

Regression Analysis

Predictor Variable	β Coefficient	p-value
Left atrial volume index	0.68	<0.001
E/e' ratio	0.52	<0.001
Age	0.21	0.04
Hypertension	0.18	0.06

Left atrial volume index emerged as the strongest independent predictor of elevated pulmonary artery pressure.

4. Discussion

The present retrospective analysis demonstrates a strong and statistically significant relationship between pulmonary artery pressure and left atrial enlargement. The observed correlation supports the well-established hemodynamic continuum linking left ventricular dysfunction, left atrial remodeling, and pulmonary vascular pressure elevation.[13–14].

Left atrial enlargement is a direct consequence of chronic elevation in left ventricular filling pressures. Over time, this leads to structural remodeling characterized by atrial dilation, fibrosis, and reduced compliance. These changes are more accurately captured by left atrial volume index than linear diameter, explaining the stronger correlation observed in this study.[17-18].

The significant association between PAP and E/e' ratio further reinforces the role of diastolic dysfunction as a central mechanism in the development of post-capillary pulmonary hypertension. Elevated filling pressures increase left atrial pressure, which is transmitted backward into the pulmonary circulation.[19].

Comorbid conditions such as hypertension and diabetes mellitus accelerate myocardial remodeling through fibrosis, endothelial dysfunction, and inflammatory pathways. These systemic influences amplify both left atrial enlargement and pulmonary pressure elevation.

The findings also highlight the clinical importance of early echocardiographic detection of left atrial enlargement. LA structural changes may precede overt pulmonary hypertension, making them a valuable early marker in asymptomatic patients.[20].

From a clinical perspective, integrating left atrial metrics into routine echocardiographic assessment can improve early identification of patients at risk of pulmonary hypertension progression and heart failure development.

Overall, this study supports an integrated cardio-pulmonary remodeling model in which left atrial structural changes and pulmonary artery pressure elevation represent interconnected manifestations of progressive cardiovascular disease.

5. Conclusion

Pulmonary artery pressure shows a strong positive relationship with left atrial enlargement in echocardiographic patients.

Left atrial volume index is a superior marker compared to diameter for assessing hemodynamic burden.

Early detection of LA remodeling may enhance risk stratification for pulmonary hypertension and left heart disease progression.

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