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Clinical and Echocardiographic Profile of Patients with Pulmonary Hypertension at a Tertiary Care Centre: A Cross-Sectional Study

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Abstract

Background: "Pulmonary hypertension (PH) is a progressive disorder defined by a mean pulmonary arterial pressure ≥20 mm hg at rest." It includes a broad range of causes, such as systemic illnesses, thromboembolic disease, chronic lung problems, and left heart disease. In India, diagnosis is often delayed due to overlap with conditions such as interstitial lung disease, rheumatic heart disease, and COPD.

Objective: To evaluate the clinical presentation and echocardiographic profile of patients with pulmonary hypertension in a tertiary care setting.

Methods: A prospective hospital-based observational study was conducted from May 2023 to October 2025 among 100 patients, of whom 52 were diagnosed with PH using transthoracic echocardiography. Pulmonary artery systolic pressure (PASP) was used to categorize PH severity. Functional classification was based on the New York Heart Association (NYHA) criteria, and right heart function was evaluated using echocardiographic parameters, including RV systolic dysfunction and TAPSE.

Results: Among PH patients,65.4% were male. Dyspnoea on exercise was most common symptom. Hypertension (42.3%), diabetes (44.2%), and smoking (65.4%) were common comorbidities. Clinical findings included elevated jugular venous pressure (88.5%), pedal edema (86.5%), loud P2 (98%). Echocardiography showed RV systolic dysfunction in 63.5% and severe PAH in 8.1%. Most patients were NYHA class II–IV.

Conclusion: PH presents late with significant right heart involvement, commonly with etiologies like COPD and left heart disease. Early echocardiographic screening and clinical vigilance are essential for timely diagnosis and improved outcomes. Echocardiography is non-invasive tool for the screening, diagnosis, risk stratification, and monitoring of PH.

Keywords: Pulmonary hypertension, echocardiography, chronic lung disease

Introduction

"Elevated pulmonary arterial pressure (≥20 mm Hg at rest) and increased pulmonary vascular resistance are the hallmarks of pulmonary hypertension (PH)", a progressive and potentially fatal illness that causes right ventricular failure and considerable morbidity and death[1]. Based on underlying pathophysiology, the World Health Organisation (WHO) divides PH into five main categories: pulmonary arterial hypertension (PAH), left heart disease-related PH, lung disease-related PH, chronic thromboembolic PH

(CTEPH), and PH with unknown or mixed causes[2,3]. This categorisation is crucial for directing diagnostic assessment and treatment.

Approximately 1% of adults worldwide suffer from PH, with prevalence greater in those 65 and over (10%). Globally, PAH, a particular form of PH, affects around 15–50 people per million. Five-year survival rates vary from 57% to 75%, depending on the kind and severity of the condition, and mortality is still high[1,4]. There is little information on PH in India, but what is known indicates that the prevalence of related illnesses, such as rheumatic heart disease, interstitial lung disease, COPD, and congenital heart disease, is increasing. According to studies, 20–30% of Indian patients with lung or left heart conditions go on to develop PH, highlighting the importance of early detection and focused screening[5].

The underlying aetiology, illness severity, and related comorbidities all affect how PH manifests clinically. The most prevalent first symptom is exertional dyspnoea, which develops gradually and is sometimes mistaken for other cardiopulmonary disorders such as deconditioning, left heart failure, or COPD[6]. Patients may have exhaustion, chest discomfort, syncope, lower extremities oedema, and symptoms of right heart failure as the illness progresses. Due to the ambiguous presentation, many patients receive a diagnosis only after their right ventricular failure has advanced, which results in considerable delays in diagnosis[7].

For risk stratification and prognostic evaluation in PH, a thorough clinical and echocardiographic assessment is essential. A loud P2, a systolic murmur of tricuspid regurgitation, right ventricular heave, and systemic venous congestion, including jugular venous distension, hepatomegaly, and peripheral oedema, are among the clinical examination findings associated with PH[8,9]. A greater mortality risk is associated with echocardiographic parameters reflecting right ventricular function, namely lower tricuspid annular plane systolic excursion (TAPSE <16 mm) and right ventricular fractional area change (RVFAC <35%)[10].

The main non-invasive method for PH screening, diagnosis, and risk assessment is echocardiography. With suggestive findings including TRV >2.8 m/s, right ventricular dilatation, interventricular septal flattening, right atrial enlargement, decreased pulmonary artery acceleration time (PAAT), and inferior vena cava (IVC) dilatation with decreased collapsibility, it offers crucial insights into right ventricular function, pulmonary haemodynamics, and structural abnormalities. Transthoracic echocardiography (TTE) is commonly used in patients with pulmonary hypertension to assess ularight ventricle's structure and function and to monitor pulmonary artery pressure. These metrics aid in distinguishing among subtypes of PH, including CTEPH (Group 4 PH), lung disease-related PH (Group 3 PH), and left heart disease-related PH (Group 2 PH)[10,11].

Due to a lack of knowledge, restricted access to specialist diagnostic facilities, and symptom overlap with other cardiopulmonary disorders, pulmonary hypertension is underdiagnosed and underreported in India.

It is increasingly being recognised by patients with congenital heart disease, valvular heart disease, and interstitial lung disorders, all of which are common in the Indian population[12]. A nationwide registry and epidemiological studies are critically needed to determine the true prevalence and clinical subtypes of PH in India. Additionally, early detection using echocardiographic screening and risk stratification is essential to enable timely interventions and decrease disease progression in settings with limited resources. To improve patient outcomes, optimise the use of healthcare resources, and drive focused public health measures, a thorough understanding of the clinical and echocardiographic characteristics of PH in India is essential.

Material and Method

The Department of General Medicine at Rajarajeshwari Medical College and Hospital in Bangalore, India, conducted an observational cross-sectional study from May 2023 to October 2025. Patients with clinical symptoms of pulmonary hypertension who attended the outpatient and inpatient departments throughout the research period and were at least 18 years old were included. Each participant gave written informed permission prior to enrolment. Critically sick patients and those who refused to provide permission were not allowed to participate in the trial.

Volume 9, Issue 3; May-June 2026; Page No 193-205

The sample size was calculated using the Yamane formula for a known population based on the average number of pulmonary hypertension cases over the preceding three years. Assuming a population size of 126, a 95% confidence level, and a margin of error of 0.005, the estimated sample size was 96. To ensure adequate representation, 100 eligible patients attending the outpatient department or admitted to general medicine wards were enrolled.

Ethical approval was obtained from the institutional ethics committee, and informed consent was obtained from all participants prior to their participation. The study was conducted in accordance with the principles of the Declaration of Helsinki. Detailed history, clinical symptoms related to each system, past history of any chronic diseases, detailed general physical systemic examination to find out the cause of pulmonary hypertension and investigation done as predesigned Protocol and 2D echo parameters, which include TR velocity, RV/LV diameter, PASP, TAPSE and PA diameter seen on PHILIPS Epiq 7c 2D echo machine. Patients were categorized according to the world health organization classified into five groups (**Group 1**: Pulmonary arterial hypertension (PAH), **Group 2**: PH due to left-sided heart disease, **Group 3**: PH due to lung disease, hypoxia, or both, **Group 4**: PH due to pulmonary artery obstruction, **Group 5**:PH with multifactorial or unclear mechanisms) and severity related to PASP (**Mild**: 35-44 mmHg, **Moderate**: 45-59mmHg, **Severe**: >60 mmHg) of pulmonary hypertension assessed by using 2D echo parameter[13].

Data was collected and compiled in MS Excel. Statistical analysis was performed using SPSS for Windows version 26.0. The description of data will be in the form of mean (±) SD for quantitative data and frequency and proportion for qualitative data.

Result:

The study comprised one hundred participants with pulmonary hypertension. With a mean age of 52.79 ± 13.6 years, the majority of patients were between 41 and 60 years old (54%), followed by those between 31 and 40 years old (17.0%). At 6.0% and 11.0%, respectively, younger patients (20–30 years old) and those over 70 accounted for smaller proportions. At 57%, men made up a tiny majority, while women made up 43%. A history of smoking was reported in 32% of participants. Only 4% of the patients had biofuel exposure. 62% of people had a normal body mass index (BMI), compared to 18% who were overweight, 18% who were obese, and 2% who were underweight **(Table 1).**

A significant incidence of right heart involvement or systemic venous congestion was indicated by clinical symptoms, including peripheral oedema in 60% of patients and elevated jugular venous pressure (JVP) in 63%. Significant pressure or volume overload in the corresponding cardiac chambers was indicated by the presence of ventricular gallops (LV S4/RV S3), left parasternal heave, and a loud pulmonary component of the second heart sound (P2) in 50% of patients. 21% and 12% of patients had clubbing and cyanosis, respectively. Notably, 10% of people had mitral stenosis (MS)-specific symptoms, which might indicate misread characteristics or concurrent left-sided valvular illness.

The lack of ascites in any of the cases suggests that this cohort does not have advanced right heart failure or related hepatic congestion. All patients had dyspnoea as a symptom, which was followed by tiredness (86%), cough (47%), chest discomfort (12%), syncope (11%), and haemoptysis (8%) **(Table 2).**

Group 3 PH was the most prevalent (32%), closely followed by Group 1 (31%) and Group 2 (25%), according to the WHO categorisation **(Figure 1).** 2% of patients were in Group 4 and 10% in Group 5. The majority of patients (88%) were in grades 1-3, whereas just 12 patients were in grades 4 and 5. Using transthoracic echocardiographic data, the degree of pulmonary hypertension was evaluated. According to the tricuspid regurgitation (TR) jet velocity, 50% of the study group had severe pulmonary hypertension (Vmax >3.4 m/s), 22% had mild pulmonary hypertension (Vmax ≤2.8 m/s), and 28% had moderate pulmonary hypertension (Vmax 2.9–3.4 m/s) **(Table 3).** When categorised by estimated pulmonary artery systolic pressure (PASP), similar results were observed: 50% had severe pulmonary hypertension, 28% had moderate pulmonary hypertension, and 22% had light pulmonary hypertension. This pattern suggests that the research group has a high prevalence of severe clinical presentation **(Table 4).**

Additional subgroup analysis **(Figure 2)** provided important insights: Portal hypertension (45.16%) was the most prevalent subtype in Group 1 (PAH),

Volume 9, Issue 3; May-June 2026; Page No 193-205

followed by idiopathic PAH (12.9%) and congenital heart disease (22.58%). Autoimmune causes were rare. Heart failure (36%) and mitral stenosis (40%) were the main causes in Group 2. Group 3 was the most common cause of COPD (43.75%), with interstitial lung disease (9.38%) and obstructive sleep apnoea (21.88%) being additional significant contributions. Group 5 had a variety of reasons, such as CKD-ESRD (30%) and hypothyroidism (20%), suggesting a complex pathophysiology in this group, while all Group 4 patients were linked to chronic pulmonary thromboembolism.

With a mean tricuspid regurgitation (TR) velocity of 3.286 ± 0.400 m/s, which is higher than the upper normal limit of 2.8 m/s, and a significantly elevated mean pulmonary artery systolic pressure (PASP) of 54.293 ± 14.928 mmHg, the echocardiographic evaluation supported the presence of pulmonary hypertension; the RV/LV diameter ratio of 0.968 ± 0.0920, indicating right ventricular enlargement; the mean tricuspid annular plane systolic excursion (TAPSE) was reduced to 15.4974 ± 2.05493 mm, indicating impailed right ventricular systolic function. **(Table 5)**

Among the 100 participants, severe pulmonary hypertension was the most common (50%), followed by moderate (28%) and mild pulmonary hypertension (22%). In mild cases, Group 1 (PAH), Group 2 (LHD), and Group 3 (LD) each accounted for 7%, while Group 5 (MF) accounted for 1%, with no cases of Group 4 (CTEPH). Among moderate cases, Group 1 (13%) was the most common, followed by Group 3 (7%), Group 2 (5%), and Group 5 (3%), with no Group 4 cases. In severe pulmonary hypertension, Group 3 (LD) was most frequent (18%), followed by Group 2 (13%), Group 1 (11%), Group 5 (6%), and Group 4 (2%) **(Table 6).**

Discussion

"Pulmonary hypertension (PH) is a progressive clinical disorder characterised by elevated pressure in the pulmonary arteries, often leading to considerable morbidity and reduced life expectancy." This increased pressure can result in right ventricular dilation and hypertrophy. It usually presents on chest radiography as an enlarged main pulmonary artery and its central branches, accompanied by noticeable narrowing of the peripheral pulmonary vessels. Echocardiographic findings commonly include right atrial and ventricular enlargement, with normal or reduced left ventricular size, and interventricular septal thickening, while Doppler assessment of tricuspid regurgitation velocity allows non-invasive estimation of right ventricular systolic pressure using the modified Bernoulli equation[14–16]. The present study assessed the demographic characteristics, clinical profile, etiological distribution and echocardiographic features of patients with PH and compared these findings with previously published literature.

1. Demographic Characteristics

In the present study, pulmonary hypertension (PH) was predominantly observed among middle-aged or elderly individuals, with a mean age of 52.79 ± 13.6 years. Most patients belonged to the fifth and sixth decades of life, indicating that PH tends to present later in life, particularly when associated with chronic cardiopulmonary or systemic conditions. The findings are comparable to those reported by Kalyan et al., who also observed a similar age distribution. However, Kulkarni et al. reported a younger study population, which may be attributed to a higher proportion of idiopathic pulmonary arterial hypertension and to referral to specialised centres [17,18].

The current study's gender analysis revealed a modest male predominance, consistent with Kalyan et al.'s findings, which noted a similar age distribution and male preponderance. As a result of regional variations in study population characteristics, referral patterns, and etiological profiles, Kulkarni et al. reported a younger cohort with a female preponderance[17,18]. About one-third of the patients had a history of smoking, which suggests that tobacco exposure contributes to chronic lung illness and future PH. This is lower than the prevalence reported by Papolos et al., but greater than that noted by Kulkarni et al. Only a tiny percentage of individuals reported exposure to biofuels, suggesting its minimal significance in the cohort [17,19].

The majority of patients (62%) in the current study had normal body mass index (BMI), according to the study. While Papolos et al. found a greater prevalence of obesity (21%) in their study, overweight and obese persons accounted for 18% of the population, while only 2% were underweight, indicating that extremes of body weight were rather uncommon[19].

Volume 9, Issue 3; May-June 2026; Page No 193-205

2. Clinical presentation and physical findings:

The most often reported symptom, dyspnoea, was present in all patients, indicating that it is a prominent feature of pulmonary hypertension. Weariness was the second most common symptom (86%), and around half of the volunteers had a cough. Less common symptoms were chest pain (12%), syncope (11%), and haemoptysis (8%). The symptom pattern of the current investigation is consistent with previous research findings. Kulkarni et al. and Kalyan et al. both reported dyspnoea as the most prevalent symptom[17,19]. Notable findings included a higher frequency of underlying parenchymal lung disease, with wheezing in 75.4%, crepitus in 61.4%, and cyanosis in 50%. Additionally, our research supports the findings of Harikrishnan et al., who found that dyspnoea during exercise was the most common symptom, followed by syncope and weariness[20].

Clinical examination in the current study showed peripheral oedema in 60% of patients and elevated jugular venous pressure (JVP) in 63%, reflecting common signs of right-heart involvement. 21% of patients had clubbing, and 12% had cyanosis, both of which are signs of more severe illness. In comparison, a high prevalence of raised jugular venous pressure (91.8%), peripheral oedema (88.59%), 77 loud P2 (96.78%), tricuspid regurgitation murmur (47.20%), and left parasternal heave (44.09%) were reported in the Kulkarni et al. study[17]. Similarly, in the Kalyan et al. study, raised jugular venous pressure (JVP) was observed in 80.7% of patients, and peripheral oedema in 87.7%, indicating significant right heart dysfunction and volume overload [18]. Loud P2, a key auscultatory finding, was present in 44.4% of patients in their study, and ascites was seen in 23.7%, clubbing in 8.8%, and pallor in 5.3%, with syncope present in 3.5% of their cohort.

3. Etiological Distribution of Pulmonary Hypertension Based on WHO Classification and Specific Causes:

According to WHO standards, the most prevalent cause of PH in the current study was lung disease (Group 3), which was most common (32%), followed by pulmonary arterial hypertension (Group 1) at 31% and PH associated with left heart disease (Group 2) at 25%. 10% of patients had multifactorial aetiology (Group 5), whereas only 2% of the study group had chronic thromboembolic PH (Group 4). However, Group 1 PH was the most common (65%) according to Papolos et al., followed by Group 2 (15%), Group 3 (9%), Group 4 (6%), and Group 5 (2%)[19]. Variations in patient profiles, underlying comorbidities, and referral patterns within the research setting might be the cause of this discrepancy.

By expanding on the aetiologies in the current study. The most frequent cause of PAH in Group 1 was portal hypertension (45.1%), which was followed by idiopathic PAH (12.9%) and congenital heart disease (22.6%). Group 2's top causes were heart failure (36%) and mitral stenosis (40%), underscoring the critical role that cardiac and valvular disease play in the pathogenesis of PH. Group 3 was predominantly COPD patients (43.7%), with notable contributions from sleep apnoea (21.9%), interstitial lung disease, pulmonary fibrosis, and autoimmune disorders, in keeping with known links between COPD and PH. Group 4 only comprised individuals who had persistent pulmonary thromboembolism. The multifaceted character of PH in Group 5 is reflected in the variety of causes, with chronic kidney disease-end-stage renal disease (CKD-ESRD) being the most frequent (30%), followed by hypothyroidism, rheumatoid arthritis, and sarcoidosis (each 20%).

Similarly, Kalyan et al. reported that Group 2 PH was the most common (47.4%), followed by Group 3 PH (43.9%). Interestingly, in their group, COPD was the most prevalent independent etiological factor (35.1%)[18]. In contrast, IPAH accounted for 69.8% of cases in the Kulkarni et al. study, followed by PAH from left heart disease (16.9%) and valvular heart disease (10.7%); however, COPD and ILD accounted for only 3.6% and 1.2%, respectively[17]. In contrast, Harikrishnan et al. reported IPAH in 52.7%, hyperlipidaemia in 27%, CKD in 23%, atrial fibrillation in 19%, and COPD in 14%[20].

4. Echocardiographic Parameters in Pulmonary Hypertension

Echocardiographic evaluation in the current investigation gave vital information about pulmonary pressures and cardiac function. Elevated right-sided pressures were indicated by the mean tricuspid regurgitation (TR) velocity of 3.12 m/s. Correspondingly, the mean pulmonary artery systolic pressure (PASP) was 46.79 mmHg, indicating moderate pulmonary hypertension. The mean RV/LV diameter ratio was 0.968, pointing to right ventricular

Volume 9, Issue 3; May-June 2026; Page No 193-205

enlargement or strain. The pulmonary artery diameter averaged 27.38 mm, consistent with vascular remodelling seen in PH.

Similarly, Papolos et al. reported a mean ePASP of 68.7 mmHg and a mean ePAC of 0.3 cm/mmHg; lower ePAC values were strongly associated with higher mortality, and multivariable analysis in their study confirmed ePAC as an independent predictor of all-cause mortality[19]. Comparatively, Kulkarni et al.'s study included only patients with PASP >50 mmHg and 95.41% confirmed PH on right heart catheterisation (RHC)[17]. In another study by McQuillan et al., the mean peak tricuspid jet velocity was 2.6 ms and the tricuspid insufficiency pressure gradient (TIPG) was 18.0 _4.7 mmHg (95% CI 8.8-27.2 mmHg) among 3,212 patients with otherwise normal Transthoracic echocardiography (TTE) and no clinically diagnosed illnesses that could cause high pulmonary artery pressure (Ppa), whereas age, body mass index (BMI), sex, left ventricular ejection fraction, and clinical referral category all had independent effects on tricuspid velocity, according to multiple linear regressions in their study[21].

At the same time, a study by Sitbon et al. investigated the prevalence of pulmonary arterial hypertension (PAH) among individuals living with HIV. Out of 7,648 HIV-positive participants who completed a screening questionnaire, 739 reported experiencing dyspnea. From this group, 247 individuals met the inclusion criteria and consented to participate in further evaluation. Among them, 18 patients exhibited a peak tricuspid jet velocity greater than 2.5 m/s, along with dyspnea, consistent with our study; however, only 5 were confirmed to have PAH upon right heart catheterisation (RHC). A post hoc analysis raised the threshold for tricuspid jet velocity to 2.8 m/s or higher, reducing the number of positive echocardiographic findings from 18 to 7 and lowering the false-positive rate to 29%[22].

Given the predominance of longitudinal fibres in the right ventricle (RV), the tricuspid annular plane systolic excursion (TAPSE), a simple echocardiographic measure used to evaluate RV function, reflects the longitudinal movement of the tricuspid annulus. TAPSE levels above 20 mm, as determined by M-mode echocardiography, are considered normal, but lower values suggest RV dysfunction. Despite its simplicity, TAPSE has several drawbacks that limit its accuracy as a global RV performance measure: it relies on angle and load measurements, assesses only a small segment of the RV myocardium, and is affected by total cardiac motion.

While Papolos et al. discovered a mean TAPSE of 1.9 cm, the current study's mean tricuspid annular plane systolic excursion (TAPSE) was 15.5 mm, below the usual threshold (>17 mm), indicating decreased right ventricular systolic function[19]. In pulmonary hypertension, TAPSE is very relevant for prognosis. A TAPSE value of less than 1.8 cm was associated with worse outcomes and more severe right ventricular systolic dysfunction, according to a study by Forfia et al. that included 63 patients with PH[23]. The tricuspid annular systolic velocity obtained from tissue Doppler imaging (TDI) also showed predictive value; in the Meluzi et al. study, a systolic velocity of less than 10.8 cm/s was associated with a worse prognosis in a cohort of 139 heart failure patients[24]. Only pericardial effusion and TAPSE have been identified as important prognostic indicators for pulmonary hypertension in current European recommendations, mainly because they are easy to measure in most patients. These two were given priority because of their practicality and wide clinical application, even though additional echocardiographic measures can provide useful prognostic information [25].

Conclusion

The current study demonstrates the diverse clinical and echocardiographic characteristics of pulmonary hypertension patients, with a minor male preponderance and a major effect in those in their fifth and sixth decades of life. Echocardiographic measures continually show right heart strain and high pulmonary pressures, and dyspnoea continues to be the most prevalent presenting symptom. According to our research, the most prevalent etiological categories were lung disease (32%) and PAH (31%), with COPD and portal hypertension being significant underlying causes. A useful non-invasive method for assessing the severity of pulmonary arterial hypertension and cardiac involvement in PH was echocardiography. Serial echocardiographic evaluations are essential for tracking disease progression, assessing therapy efficacy, and identifying early signs of right ventricular failure. PASP/TRV is the most reliable 2D echo. A helpful non-invasive technique for assessing

Volume 9, Issue 3; May-June 2026; Page No 193-205

the extent of cardiac involvement and pulmonary arterial hypertension in PH was echocardiography. Serial echocardiographic evaluations are essential for monitoring the course of the illness, evaluating treatment efficacy, and identifying early indicators of right ventricular failure. According to our research, PASP/TRV are the most accurate 2D echo parameters for assessing pulmonary hypertension severity.

Limitations

There are various limitations to this study. First, because it is a descriptive cross-sectional study conducted at a particular location, the findings may have limited generalizability to other demographics or geographic areas. Even though the sample size is sufficient for observational research, it might not be sufficient to achieve high statistical power for subgroup comparisons or to detect subtle relationships. Furthermore, the absence of long-term follow-up data in the research made it impossible to evaluate clinical outcomes, progression, and mortality associated with different causes of pulmonary hypertension (PH). Due to financial limitations, DLCO, pulmonary function testing, and arterial blood gas analysis were not possible. Additionally, several relevant variables were not thoroughly examined, including genetic predispositions, environmental exposures, and socioeconomic status.

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Volume 9, Issue 3; May-June 2026; Page No 193-205

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Volume 9, Issue 3; May-June 2026; Page No 193-205

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"Table 1: Demographic and Baseline Clinical Profile of Patients with Pulmonary Hypertension (N = 100)"

Variables	Frequency (N)	Percentage (%)
AGE		
20-30 years	6	6.0
31-40 years	17	17.0
41-50 years	22	22.0
51-60 years	32	32.0
61-70 years	12	12.0
>70 years	11	11.0
Total	100	100.0
Mean + SD	52.79 + 13.607	
Gender		
Male	57	57.0
Female	43	43.0
SMOKING HISTORY		
Yes	32	32.0
No	68	68.0
BIOFUEL EXPOSURE		
Yes	4	4.0
No	96	96.0
BMI (kg/m2)		
Underweight (<18.5)	2	2.0
Normal Weight (18.5-24.9)	62	62.0
Overweight (25.0-29.9)	18	18.0
Obese (>30.0)	18	18.0

Volume 9, Issue 3; May-June 2026; Page No 193-205

"Table 2: Clinical Presentation of Patients with Pulmonary Hypertension—Symptoms and Physical Signs (N = 100)"

SYMPTOMS	Frequency	Percentage
Dyspnea	100	100.0
Fatigue	86	86.0
Cough	47	47.0
Chest Pain	12	12.0
Syncope	11	11.0
Hemoptysis	8	8.0
SIGNS		
Raised JVP	63	63.0
Peripheral edema	60	60.0
P2	50	50.0
Left parasternal heave	50	50.0
LVS3/RVS3	50	50.0
Clubbing	21	21.0
Cyanosis	12	12.0
Signs of MS	10	10.0
Ascites	0	0.0

"TABLE 3. Severity of disease in relation with TR velocity (N = 100)"

Severity	Parameter	Frequency	Percentage
Mild Pulmonary Hypertension	TR jet velocity (Vmax) (≤ 2.8 m/s)	22	22.0
Moderate Pulmonary Hypertension	Suggestive of PHT (borderline) (2.9 - 3.4 m/s)	28	28.0
Severe Pulmonary Hypertension	Indicative of Pulmonary Hypertension (> 3.4 m/s)	50	50.0

"Table 4. Grading of severity according to pulmonary artery systolic pressure (PASP) (N = 100)"

Severity	Estimated PASP	Frequency	Percentage

Volume 9, Issue 3; May-June 2026; Page No 193-205

Mild Pulmonary HTN	**35-44 mm Hg**	22	22.0
Moderate Pulmonary HTN	**45-59 mm Hg**	28	28.0
Severe Pulmonary HTN	**≥ 60 mm Hg**	50	50.0

"Table 5 ECHO PARAMETERS"

ECHO PARAMETERS (Normal Value)	**MEAN**	**SD**
TR Velocity (≤ _2.8)	3.286	0.400
PASP (≤35)	54.293	14.928
RV/LV Diameter (<0.9)	0.968	0.0920
TAPSE (≥17 mm)	15.4974	2.05493
PA Diameter (≤25 mm)	27.3837	3.34921

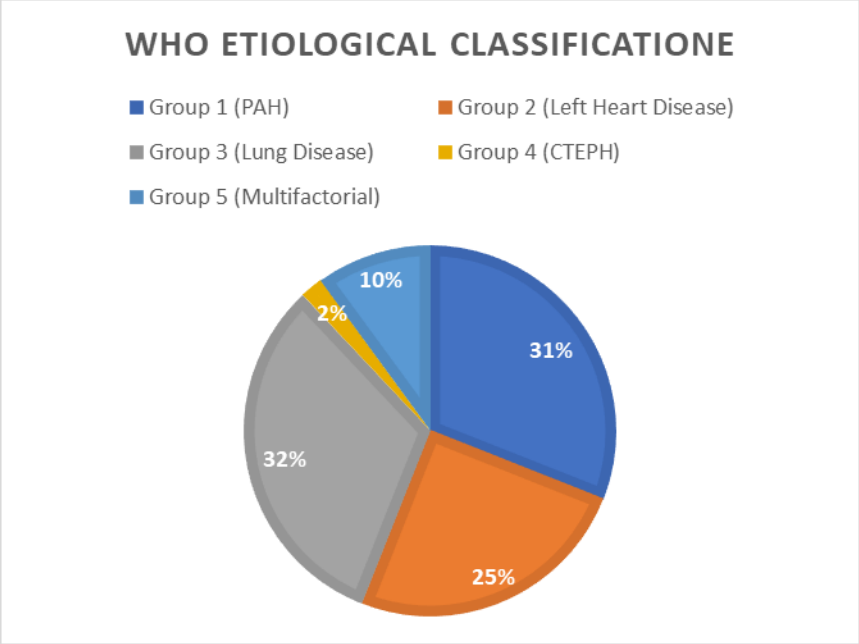
"TABLE 6. Number of patients in each respective groups (N = 100)"

Severity	**Cases (N = 100)**	**Percentage**
Mild Pulmonary Hypertension	22	22
Group 1 (PAH)	7	7
Group 2 (LHD)	7	7
Group 3 (LD)	7	7
Group 4 (CTEPH)	0	0
Group 5 (MF)	1	1
Moderate Pulmonary Hypertension	28	28
Group 1 (PAH)	13	13
Group 2 (LHD)	5	5
Group 3 (LD)	7	7
Group 4 (CTEPH)	0	0
Group 5 (MF)	3	3
Severe Pulmonary Hypertension	50	50
Group 1 (PAH)	11	11
Group 2 (LHD)	13	13
Group 3 (LD)	18	18

Volume 9, Issue 3; May-June 2026; Page No 193-205

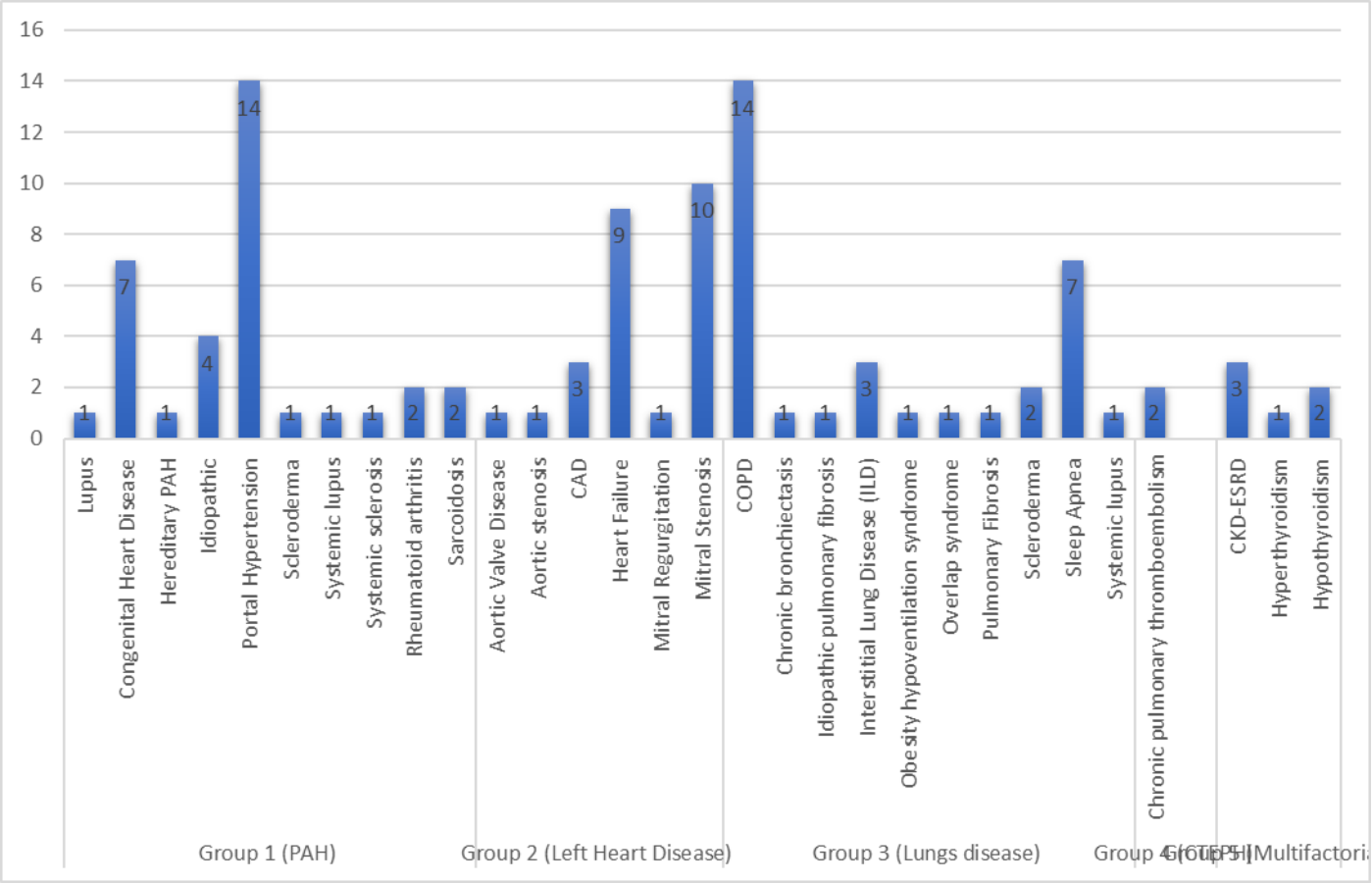
Group 4 (CTEPH)	2	2
Group 5 (MF)	6	6

"Figure 1. Who Etiological Classification"



Volume 9, Issue 3; May-June 2026; Page No 193-205

"Figure 2. Who Etiological Classification With Etiological Subtypes"



Volume 9, Issue 3; May-June 2026; Page No 193-205