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Prevalence of Pulmonary Hypertension in The Setting Of Lv Dysfunction Using Echocardiography

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Abstract

Background: Pulmonary hypertension is a serious condition marked by increased pressure in the pulmonary arteries, which strains the right side of the heart and may lead to right heart failure. A major cause is left ventricular dysfunction, where poor pumping leads to raised left atrial pressure and backward transmission into the lungs. This causes pulmonary congestion and vascular remodeling, eventually increasing pulmonary vascular resistance and resulting in pulmonary hypertension.

Objective: To determine the prevalence of pulmonary hypertension in patients with left ventricular dysfunction using echocardiography.

Methods: A hospital-based cross-sectional observational study will be conducted on patients with left ventricular dysfunction. Transthoracic Doppler echocardiography will be used to estimate pulmonary artery pressures and classify patients based on the presence or absence of pulmonary hypertension.

Results: The study will determine the prevalence of pulmonary hypertension in patients with left ventricular dysfunction using echocardiography. Final statistical results will be reported after completing data collection and analysis

Conclusion: Pulmonary hypertension is a common and clinically important complication of left ventricular dysfunction that significantly worsens patient outcomes by increasing morbidity and mortality. Early detection using echocardiography is

essential for timely diagnosis and management.

Introduction

Pulmonary hypertension is prolonged heart condition where the pressure in the pulmonary arteries increased. As the pressure in these arteries rises above normal levels, changes happen in both the lungs and the heart. These changes can eventually lead to serious problems with the right side of the heart. PH is not just a random finding in the heart; it is a serious condition that affects how a person feels, makes hospital stays longer, and increases the risk of death. The most common and important cause of PH worldwide is when the left ventricle, which is the main pumping chamber of the heart, doesn't work properly. Left ventricular dysfunction means the left ventricle can't pump enough blood through the body to meet the body's needs. This can happen due to various reasons like reduced blood flow to the heart muscle, long-term high blood pressure, damaged heart valves, diseases of the heart muscle, inflammation of the heart, or structural problems in the heart. Portal vein is a blood vessel which carries blood from many organs to the abdomen (belly) to liver. The liver filters and processes blood before it returns to the heart and recirculates through the body. Usually, veins carry blood towards the heart rather than the other organs. The hepatic portal system (portal venous system) is an exception to this rule. The veins in this system send blood to the liver, portal vein is the main blood vessel in this system. To understand the portal vein and its job, it helps to learn a bit about the portal venous system. The portal venous system is a network of veins that drain blood from the following organs of the belly, Small intestine, Large intestine, Stomach, Pancreas, Spleen, and Gallbladder. Numerous smaller veins in portal venous system collect blood from all of these organs and deliver it to your portal vein. You can think of the portal vein as a pit lane at a racetrack. It leads to your liver, which serves as a pit stop for blood to receive necessary maintenance. By the time blood reaches to the portal vein, it's full of nutrients that need processing before the body can use them. This blood also contains toxins, or harmful substances the body doesn't need. The liver "tunes up" your blood by converting nutrients to forms the body can use right away or store. It also removes toxins from your blood. After this tune-up, blood is ready to go back to the heart. So, it leaves your liver and travels towards the inferior vena cava, which delivers blood to the top right chamber of your heart (right atrium)[i].

Portal vein begins just behind the neck of your pancreas and in front of the inferior vena cava. It extends to the porta hepatic (liver hilum). This is an opening in the liver that allows blood vessels and other structures to enter and leave the liver. Portal vein forms at the point where the superior mesenteric vein (SMV) and splenic vein meet. From there, the portal vein travels upward and toward the right, behind the hepatic artery, until it reaches to liver. As it enters the liver hilum, the portal vein splits into two branches. These is a left portal vein and right portal vein. These branches further divide into additional branches that travel to different parts of the liver. The most common anatomical variation is portal vein trifurcation. This is when the portal vein splits into three branches, rather than two, as it enters the hilum. In this case, the branches are the; Left portal vein. Right anterior portal vein. Right posterior portal vein. This variation is not dangerous, but the healthcare provider needs to know about it before performing some surgeries and procedures. Providers run imaging tests prior to surgery to learn the anatomy and adjust techniques as needed. Portal vein is typically 8 centimeters (cm) long and no more than 13 millimeters (mm) wide[ii].

The splenic vein drains the spleen, part of the pancreas, and part of the stomach. The splenic vein is formed by splenic tributaries emerging at the splenic hilum in the splenorenal ligament at the tip of the tail of pancreas. It runs in the splenorenal ligament in close association with the splenic artery. Behind the neck of the pancreas, it confluences with the superior mesenteric vein to form the portal vein. Portal vein delivers blood from organs to belly than in liver for processing. Therefore, it's vital of

the portal venous system. It's the main passageway for blood to enter the liver. All the other veins in the hepatic portal system ultimately converge (join paths) and lead to the portal vein. So, it needs to be healthy and working at its best for the whole system to work as it should. Portal vein has several tributaries, or veins that drain into it. These include; Superior mesenteric vein, which drains several organs in the middle of belly, including your small intestine. Splenic vein, which drains your spleen. Inferior mesenteric vein, which drains portions of large intestine. Gastric veins, which drain your stomach. Cystic veins, which drain into gallbladder[iii].

The noticeable symptoms of portal hypertension are usually related to new, enlarged, leaky and bleeding veins, such as: Blood in vomit, Blood in poop,Bloated stomach with rapid weight gain (from fluid),Swelling in your legs and feet (edema),Mental confusion or disorientation. The causes of portal hypertension includes; Cirrhosis of the liver, Granulomas of the liver, Blood clots. The most direct way to diagnose portal hypertension is to measure the pressure of the liver veins, which involves inserting a tiny catheter into a vein. But this is a difficult procedure and not usually necessary. Healthcare providers can recognize portal hypertension by its clinical signs and symptoms. Imaging tests and blood tests can help confirm their suspicions. The healthcare provider will suspect portal hypertension if you have; Enlarged varices,Enlarged spleen, Gastrointestinal bleeding, Ascites (fluid in abdominal regions).,Signs of mild cognitive impairment, Low blood cell counts, Easy bleeding and bruising[iv].

Portal vein thrombosis (PVT) is the formation of a blood clot (thrombus) that narrows or blocks the portal vein. This vein carries blood to the liver from organs in the abdomen (belly). Clots can also develop in the portal vein's branches (inside your liver) or the blood vessels that drain into the portal vein (superior mesenteric vein and splenic vein).Portal vein thrombosis is a serious condition, but it often causes no symptoms. So, you may not know you have a blood clot until a healthcare provider finds it through testing or you develop complications, like portal hypertension. Such complications are serious and life-threatening without treatment. Common symptoms include vomiting with blood and rectal bleeding (blood in your poop). If you have these symptoms, seek medical care immediately. People with cirrhosis face a higher risk of portal vein thrombosis and its complications. Many other medical conditions, like certain blood clotting disorders, can also raise your risk. It's important to consult with a healthcare provider if you have risk factors for portal vein thrombosis. Your healthcare provider will make sure you receive appropriate screenings to diagnose and treat issues as early as possible. Types of portal vein thrombosis; Cirrhotic portal vein thrombosis; this is the formation of a portal vein thrombus in people who have cirrhosis (scarring of the liver)[v]. This type is more common. Non-cirrhotic portal vein thrombosis: This is the formation of a portal vein thrombus in people who don't have cirrhosis[vi].

Symptoms of portal vein hypertension can include; Vomit that contains blood, Rectal bleeding (blood in your poop), Bloated stomach, Swelling (edema) in your legs and feet, Cramping in your belly. A blood clot may form in your portal vein due to; Blood that's prone to clotting (hypercoagulable state), Slow blood flow through your liver, Damage to the lining (endothelium) of your portal vein. Healthcare providers diagnose this condition through a comprehensive physical exam and testing. They'll also ask about your medical history to learn if you have risk factors for portal vein thrombosis[vii].

Portal hypertension itself may or may not be reversible, depending on its cause. If there's an infection or a blood clot that your healthcare provider can treat with medication, portal hypertension can improve and sometimes resolve over time. However, significant scar tissue, such as in the case of cirrhosis, usually won't reverse and leads to persistent portal hypertension. Most treatment is aimed at managing the complications of portal hypertension. Your healthcare provider will address

emergencies first, then move on to longer-term solutions.First-line treatments to stop gastrointestinal bleeding include: Endoscopy. Your healthcare provider will treat acute bleeding with endoscopic therapy. Endoscopic methods include sclerotherapy and banding. Sclerotherapy means injecting a solution into the bleeding varices to control the bleeding. Banding means tying off bleeding varices with tiny rubber bands.Medication. Your healthcare provider may prescribe medications in combination with endoscopic therapy to reduce the pressure in your varices and reduce the risk of recurrent bleeding. Beta-blockers can reduce portal pressure, while vasoconstrictors can help reduce dilated blood vessels. Other first-line treatments for complicated portal hypertension may include; Oxygen therapy for hepatopulmonary syndrome, Dialysis for hepatorenal syndrome, Medication to treat hepatic encephalopathy, Paracentesis to remove excess fluid from ascites and test it for peritonitis. When bleeding or other complications persist, healthcare providers may move on to second-line treatments[viii].

As the left ventricle weakens, blood starts to pool in the left atrium and the pulmonary veins. This causes pressure to push back towards the lungs, leading to congestion in pulmonary blood vessels over time, this increased pressure of the pulmonary veins cause's changes in the lung blood vessels, making their walls thicker and increasing resistance in the blood flow. This process leads to pulmonary hypertension. This is a key link between left ventricular dysfunction and pulmonary hypertension. Having pulmonary hypertension in people with left ventricular issues can greatly affect their health outcomes. Symptoms like difficulty in breathing, reduced ability to exercise, tiredness, lightheadedness, and swelling in the legs are common. Pulmonary hypertension can also cause the right ventricle, which isn't designed to handle high pressure for long, to fail. This can lead to enlargement of the right ventricle, leakage of blood through the tricuspid valve, and eventually right heart failure[ix].

Pulmonary hypertension is a major problem that often comes with LV dysfunction, making patients sicker, staying longer in hospitals, and increasing the risk of death from right heart failure. Echocardiography is a crucial for the early detection of pulmonary hypertension, but there's a lack of research in Pakistan on how common it is when left ventricular dysfunction is present. Most studies look at heart failure in general, without focusing on pulmonary hypertension, which creates a big gap in our understanding. This gap makes it hard for the healthcare professionals to predict risks, choose the best treatments, and use resources wisely for these high-risk patients. So, this study aims to find out how common pulmonary hypertension is in the patients with left ventricular dysfunction using echocardiography, to fill this knowledge gap and improve patient outcomes. By doing this, we can better understand the problem and make informed decisions about patient care and resource allocation, which is very critical for effective management and treatment. The study's findings will help the healthcare professionals to identify patients at risk, provide timely interventions, and ultimately reduce the burden of PH in patients and the healthcare systems[x].

Over time, this chronic pressure overload causes structural changes in the lung vasculature, increasing vascular resistance and triggering the Pulmonary Hypertension. This progression eventually leads to right ventricular hypertrophy and failure, significantly worsening the patient's prognosis. Despite this established link, the specific prevalence of PH in this sub-population remains underexplored in local settings. The Diagnostic Gap and Role of Echocardiography while cardiac catheterization is in the way to diagnose pulmonary hypertension it is a procedure that requires putting things into the body and is not available in many places. Echocardiography is an alternative because it is not invasive and does not use radiation to estimate the blood pressure in the pulmonary artery and check how the heart is working. However with this technology available there is a big gap in diagnosis and research. The document says that research from around the world shows that between 30%and 80% of people with ventricular dysfunction have

pulmonary hypertension but these numbers are all over the place depending on how the diagnosis is made and who the patients are.[xi].

Problem Statement:

The Clinical Burden and Pathophysiological Link Pulmonary hypertension (PH) is a progressive and serious condition characterized by elevated blood pressure in the pulmonary arteries, which places excessive strain on the right ventricle (RV). The synopsis identifies Left Ventricular (LV) dysfunction as the most common and significant cause of Pulmonary Hypertension in worldwide. When the left ventricle fails to pump efficiently due to ischemic heart disease, hypertension, or cardiomyopathy blood pools in the left atrium and pulmonary veins. This creates a backward transmission of pressure, leading to pulmonary congestion. Over time, this chronic pressure overload causes structural changes in the lung vasculature, increasing vascular resistance and triggering the Pulmonary Hypertension. This progression eventually leads to right ventricular hypertrophy and failure, significantly worsening the patient's prognosis. Despite this established link, the specific prevalence of PH in this sub-population remains underexplored in local settings. The Diagnostic Gap and Role of Echocardiography while cardiac catheterization is in the way to diagnose pulmonary hypertension it is a procedure that requires putting things into the body and is not available in many places. Echocardiography is an alternative because it is not invasive and does not use radiation to estimate the blood pressure in the pulmonary artery and check how the heart is working. However with this technology available there is a big gap in diagnosis and research. The document says that research from around the world shows that between 30%and 80% of people with ventricular dysfunction have pulmonary hypertension but these numbers are all over the place depending on how the diagnosis is made and who the patients are. There is no way to do these screenings and in many cases pulmonary hypertension is not found until the heart is failing badly[xii].

Rationale of this Study:

Patients with ventricular dysfunction who develop sudden pulmonary hypertension are in a serious condition and their disease is quite advanced. Finding this out early can helps the doctors to treat them with methods that can stop the left ventricle from getting worse. This approach also saves money on treatment reduces hospital readmissions and slows down the progression of heart failure to its stage. As a result it eases the long-term healthcare burden on society. Improves the results for patients, with left ventricular dysfunction

Material and Methods:

This cross-sectional descriptive study will be conducted at Sheikh Zayed Medical College and Hospital, Rahim Yar Khan, over a period of four months following the approval of the research synopsis. The study sample size will be calculated using an appropriate statistical formula based on the expected prevalence reported in previous studies. The sample size formula used is $n = (Z^2\ P\ (1-P)) / d^2$, and the calculated sample size for this study is 100 participants. A non-probability sampling technique will be employed for the selection of study participants.

The study population will include patients aged 8 to 80 years who present with various cardiac conditions, including grades of systolic and diastolic dysfunction, ischemic heart disease, mitral regurgitation, aortic regurgitation, congenital heart diseases, and pulmonary hypertension. Patients with renal parenchymal diseases, chronic obstructive pulmonary disease (COPD), and eczema will be excluded from the study. Data will be collected using an echocardiography machine equipped with Doppler capability, which will be utilized to assess and record the relevant cardiac parameters required for the study. The collected data will then be analyzed to achieve

the study objectives and determine the prevalence and characteristics of the selected cardiac conditions among the study population.

Statistical Analysis:
The overall model is statistically significant (p = 0.0003), meaning Pulmonary Hypertension is a meaningful predictor of the outcome variable. Conclusion: Pulmonary Hypertension has a statistically significant but little practical effect on the dependent variable, with most variance still unexplained. The overall model is statistically significant (p = 0.0003), meaning Pulmonary Hypertension is a meaningful predictor of the outcome variable. Conclusion: Pulmonary Hypertension has a statistically significant but little practical effect on the dependent variable, with most variance still unexplained. The mean age of the study population was 61.4 ± 12.7 years, with a predominant male representation (58%). The majority of patients (64%) presented with heart failure with reduced ejection fraction (HFrEF), while moderate to severe LV dysfunction was observed in 82% of cases. Mitral regurgitation of at least mild severity was present in 75% of patients.

Results:
A total number of patient is 100 patients with left ventricular dysfunction were enrolled in this cross-sectional study at Sheikh Zayed Medical College and Hospital, Rahim Yar Khan. The demographic and clinical characteristics of the study population are summarized below.

Table 1 Pulmonary Hypertension

Multiple R	0.3573							
R Square	0.1276							
Adjusted R Square	0.1187							
Standard Error	0.8140							
Observations	100.0000							
			ANOVA					
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1.0000	9.5021	9.5021	14.3399	0.0003			
Residual	98.0000	64.9379	0.6626					
Total	99.0000	74.4400						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	0.1448	0.1585	0.9135	0.3632	-0.1698	0.4594	-0.1698	0.4594
Pulmonary Hypertension	0.2862	0.0756	3.7868	0.0003	0.1362	0.4362	0.1362	0.4362

Model significance: The overall model is statistically significant (p = 0.0003), meaning Pulmonary Hypertension is a meaningful predictor of the outcome variable. Conclusion: Pulmonary Hypertension has a statistically significant but little practical effect on the dependent variable, with most variance still unexplained

Table 2 Mitral Regurgitation

Multiple R	0.5652							
R Square	0.3195							
Adjusted R Square	0.3125							
Standard Error	0.8241							
Observations	100.0000							
			ANOVA					
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1.0000	31.2417	31.2417	46.0070	0.0000			
Residual	98.0000	66.5483	0.6791					
Total	99.0000	97.7900						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	0.1759	0.1605	1.0958	0.2759	-0.1426	0.4944	-0.1426	0.4944
Mitral Regurgitation	0.5190	0.0765	6.7828	0.0000	0.3671	0.6708	0.3671	0.6708

Model Fit: The model of result explains 31.95% of the variance in the outcome (R Square = 0.3195), meaning about 68% of the variance remains unexplained. The multiple correlation coefficient is 0.5652, indicating a moderate positive relationship. Conclusion: Mitral Regurgitation is a significant, positive predictor of the outcome.

Table Summary of key Findings

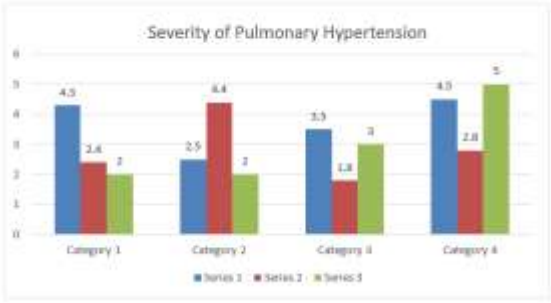
Findings	Results
Overall PH Prevalence	67% (67/100 patients)
PH Prevalence by LV Dysfunction Severity	Mild: 33.3%, Moderate: 66.7%, Severe: 82.5%
PH Prevalence by HF Type	HFrEF: 78.1%, HFmrEF: 54.5%, HFpEF: 35.7%
PH Prevalence by MR Severity	None: 48%, Mild: 57.1%, Moderate: 82.1%, Severe: 100%
Strongest Independent Predictor of sPAP	LVEF (standardized $\beta = -0.48$, $p < 0.001$)
Model Performance	Adjusted $R^2 = 0.398$, $F(5,94) = 13.55$, $p < 0.001$

Table 1: The Table of Distribution of Pulmonary Hypertension Severity

PH Severity	sPAP Range (mmHg)	Frequency (n)	Percentage (%)
No PH	≤35	33	33.0
Mild PH	36-45	28	28.0
Moderate PH	46-60	24	24.0
Severe PH	>60	15	15.0

The findings indicate that 67% of the patients had some degree of pulmonary hypertension, while 33% showed no evidence of PH. Among patients with PH, mild PH was the most common severity level (28%), followed by moderate PH (24%) and severe PH (15%). The decreasing frequency from mild to severe categories suggests that higher levels of pulmonary artery pressure were less common in the study population.

Figure 1: Severity Distribution of Pulmonary Hypertension



Overall, the table demonstrates a substantial burden of pulmonary hypertension among the participants, with the majority exhibiting mild to moderate disease severity. These findings highlight the importance of early detection and management to prevent progression to severe pulmonary hypertension.

Discussion:

The present study demonstrates that pulmonary hypertension (PH) is highly prevalent among 100 patients with left ventricular (LV) dysfunction, affecting 67% of the study population. This finding is consistent with previous international studies and reinforces the concept that PH is a frequent and important complication of left-sided heart disease. The strong association between worsening LV dysfunction and increasing prevalence and severity of PH observed in our cohort reflects the progressive hemodynamic burden imposed on the pulmonary circulation. Patients with severe LV dysfunction exhibited the highest prevalence of PH (82.5%) and the greatest mean systolic pulmonary artery pressure (54.8 ± 14.6 mmHg), highlighting the close relationship between impaired left ventricular performance and pulmonary vascular abnormalities.

The development of PH in LV dysfunction begins with elevated left ventricular filling pressures caused by impaired systolic function. As the failing left ventricle becomes unable to effectively eject blood, left ventricular end-diastolic pressure rises and is transmitted backward to the left atrium and pulmonary veins. This chronic elevation of pulmonary venous pressure increases hydrostatic pressure within the pulmonary capillaries, leading to pulmonary congestion and elevated pulmonary artery pressures. Initially, these changes are passive and reversible, representing isolated post-capillary pulmonary hypertension. At this stage, pulmonary vascular resistance remains normal, and treatment directed at improving left ventricular function and reducing filling pressures may effectively lower pulmonary pressures.

However, prolonged exposure to elevated pulmonary venous pressure initiates a cascade of pathological changes within the pulmonary vasculature. Endothelial dysfunction develops due to chronic mechanical stress and inflammation, resulting in an imbalance between vasodilatory mediators such as nitric oxide and prostacyclin and vasoconstrictive mediators including endothelin-1. This imbalance promotes pulmonary vasoconstriction, vascular smooth muscle proliferation, and structural remodeling of the pulmonary arteries. Histopathological studies have demonstrated medial hypertrophy, intimal fibrosis, collagen deposition, and reduced vascular compliance in these patients. Consequently, pulmonary vascular resistance increases, giving rise to combined pre-capillary and post-capillary pulmonary hypertension (CpcPH), a more advanced and clinically significant form of disease.

The markedly elevated sPAP observed among patients with severe LV dysfunction in the present study suggests that a substantial proportion of these individuals may have progressed beyond simple passive pulmonary hypertension to a stage characterized by pulmonary vascular remodeling. This is clinically important because CpcPH is associated with greater right ventricular afterload, progressive right ventricular dysfunction, worsening exercise intolerance, increased hospitalization rates, and higher mortality. Several studies have shown that the presence of PH in heart failure patients independently predicts adverse outcomes regardless of left ventricular ejection fraction. Therefore, identification of PH in patients with LV dysfunction is not merely a hemodynamic observation but an indicator of more advanced disease and poorer prognosis.

Another notable finding is that not all patients with severe LV dysfunction developed pulmonary hypertension, while some individuals with only moderate impairment demonstrated significantly elevated pulmonary pressures. This observation suggests that factors beyond left ventricular dysfunction contribute to the development and progression of PH. Individual variations in pulmonary vascular responsiveness, genetic susceptibility, endothelial function, inflammatory pathways, duration of elevated filling pressures, age-related vascular changes, and the presence of comorbid conditions such as hypertension, diabetes mellitus, obesity, chronic kidney disease, and sleep-disordered breathing may influence pulmonary vascular remodeling. These factors may explain the heterogeneity observed among patients with apparently similar degrees of LV dysfunction. The findings of this study have important clinical implications, particularly in resource-limited settings such as Pakistan. Since the prevalence and severity of PH increase significantly with worsening LV dysfunction, routine echocardiographic assessment of pulmonary artery pressures should be considered in patients with heart failure. Early detection of PH may facilitate timely optimization of heart failure therapy, closer clinical monitoring, and identification of patients at increased risk for adverse cardiovascular outcomes. Furthermore, recognition of PH may help guide decisions regarding advanced heart failure therapies and improve risk stratification.

Overall, the present study supports existing evidence that pulmonary hypertension is a common consequence of left ventricular dysfunction and that its severity increases progressively with worsening cardiac impairment. The results highlight the importance of understanding the complex interaction between left heart disease and pulmonary vascular remodeling, emphasizing the need for early diagnosis and aggressive management to prevent progression to advanced pulmonary vascular disease and right heart failure.

Comparison with other studies:

For your **research article Discussion section**, you can write the comparison with previous studies as follows:

Comparison with Previous Studies

The present study demonstrated that pulmonary hypertension (PH) was present in 67% of patients with left ventricular (LV) dysfunction. This finding is consistent with previous international studies reporting a high prevalence of PH among patients with left-sided heart disease and heart failure. In the current study, PH prevalence increased progressively with worsening LV dysfunction, reaching 82.5% among patients with severe LV dysfunction.Our findings are comparable to the study conducted by Alrahimi et al. (2025), who reported that approximately 54% of patients with heart failure and reduced ejection fraction (HFrEF) had secondary pulmonary arterial hypertension. Similar to our results, their study demonstrated a strong association between pulmonary hypertension, reduced ejection fraction, elevated filling pressures, and valvular regurgitation.The prevalence observed in our study also falls within the range reported by international literature, where pulmonary hypertension has been documented in approximately 30%–80% of patients with left ventricular dysfunction depending on patient characteristics and diagnostic criteria.Furthermore, our results support the observations of Rosenkranz et al. (2018), who reported that patients with reduced left ventricular ejection fraction are particularly susceptible to developing pulmonary hypertension due to chronically elevated left-sided filling pressures and pulmonary vascular remodeling.

Similarly, Ghio et al. (2017) found that pulmonary hypertension frequently occurs in patients with chronic heart failure and is associated with worsening clinical outcomes, increased hospitalization, and higher mortality rates. The high prevalence of PH in our cohort further reinforces the clinical importance of routine screening in patients with LV dysfunction.Our findings also agree with Lam et al. (2018), who demonstrated that a considerable proportion of patients with both preserved and reduced ejection fraction develop pulmonary hypertension and emphasized the importance of Doppler echocardiography for early detection.An important finding of the present study was the strong relationship between mitral regurgitation (MR) severity and pulmonary hypertension. PH prevalence increased from 48% in patients without MR to 100% in patients with severe MR. These findings are consistent with Ratwatte et al. (2023), who reported that pulmonary hypertension commonly accompanies significant mitral regurgitation and is associated with adverse clinical outcomes, including right ventricular dysfunction and increased mortality.

The current study further supports the conclusions of Vachiéry et al. (2019) and Gerges et al. (2020), who identified pulmonary hypertension due to left heart disease (PH-LHD) as the most common form of pulmonary hypertension worldwide. Both studies emphasized that chronic elevation of left atrial and pulmonary venous pressures eventually leads to pulmonary vascular remodeling and increased pulmonary artery pressure, findings that are reflected in the increasing severity of PH observed among patients with severe LV dysfunction in our study.Overall, the results of the present study are in agreement with the existing literature and provide local evidence from Pakistan that pulmonary hypertension is a frequent and clinically significant complication of left ventricular dysfunction. The observed prevalence of 67% highlights the need for routine echocardiographic assessment and early intervention to reduce disease progression and improve patient outcomes.

Limitations:

This study has several limitations. First, it is a single-center study conducted at Sheikh Zayed Medical College and Hospital, Rahim Yar Khan, which may limit the generalizability of the results. Second, the sample size of 100 patients may not fully represent the broader population. Third, the cross-sectional design allows assessment of prevalence only and cannot establish causal relationships. Fourth, non-probability sampling may introduce selection bias. Fifth, pulmonary hypertension is diagnosed using echocardiography rather than right heart catheterization, the gold standard diagnostic method. Furthermore, the short study duration, potential observer

variability during echocardiographic measurements, and the presence of confounding cardiac conditions may influence the results. Finally, the absence of long-term follow-up prevents assessment of disease progression and clinical outcomes.

Conclusion:

This study highlights the close relationship between left ventricular (LV) dysfunction and pulmonary hypertension (PH). Among the 100 patients studied, PH was present in 67%, indicating that elevated pulmonary artery pressure is a frequent complication of impaired left ventricular function. The findings showed a clear trend in which the prevalence and severity of PH increased as LV function worsened. Patients with severe LV dysfunction had a PH prevalence of 82.5% and a mean systolic pulmonary artery pressure (sPAP) of 54.8 mmHg, whereas patients with mild LV dysfunction had a much lower prevalence (33.3%) and mean sPAP (32.4 mmHg). This suggests that progressive deterioration of left ventricular performance leads to increased pressure within the pulmonary circulation. Physiologically, when the left ventricle is unable to pump blood effectively, blood accumulates in the left atrium and pulmonary veins, causing a rise in pulmonary venous pressure that is transmitted backward into the pulmonary arteries, ultimately resulting in PH.

The study also demonstrated that PH was particularly common among patients with heart failure with reduced ejection fraction (HFrEF), with a prevalence of 78.1%. This finding is consistent with the understanding that reduced ejection fraction reflects impaired systolic function, which contributes to elevated left-sided filling pressures and subsequent pulmonary vascular congestion. In addition, the severity of mitral regurgitation (MR) was strongly associated with the presence of PH. Notably, all patients with severe MR developed PH, and they exhibited the highest mean sPAP (62.7 mmHg). Severe MR causes backward leakage of blood from the left ventricle into the left atrium during systole, increasing left atrial pressure and further elevating pulmonary venous and arterial pressures. These findings emphasize the importance of early detection and management of significant valvular disease to prevent irreversible pulmonary vascular changes and worsening right ventricular function.

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Declaration:

We declare that the thesis titled “Prevalence of Pulmonary Hypertension in the Setting of LV Dysfunction Using Echocardiography” is our original work carried out under the supervision **of** Ms. Rabia Ismaeel and Mr. Muneeb Bin Naveed at the Department of Medical Imaging Technology, Faculty of Allied Health Sciences, and Superior University Lahore**.** This work has not been submitted to any other university for any degree.

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