Section: General Medicine

Original Research Article

STUDY OF PULMONARY HYPERTENSION IN CHRONIC KIDNEY DISEASE PATIENTS BY ECHOCARDIOGRAPHY

Swapnil Sanjaykumar Kolhe[1], Shivani Bansal[2], Ashok Kumar[3], Dewansh Rana[4], Saahil Sharma[5], Henilkumar Amrutbhai Ahir[6]

[1]Junior Resident, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
[2]Professor, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
[3]Professor & Head, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
[4]Junior Resident, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
[5]Junior Resident, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
[6]Junior Resident, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.

Received *: 05/08/2024*
Received in revised form *: 02/11/2024*
Accepted *: 19/11/2024*

Corresponding Author:
Dr. Swapnil Sanjaykumar Kolhe, Junior Resident, Department of General Medicine, Santosh Medical College, Santosh Deemed to be University, Ghaziabad, Uttar Pradesh, India.
Email: swapnil22396@gmail.com

DOI: 10.70034/ijmedph.2025.2.268

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (2); 1495-1502

ABSTRACT

Background: Chronic Kidney Disease (CKD) is a severe renal disease causing severe organ system issues, including uremia, metabolic bone disease, neuropathy, hypertension, and cardiovascular disorders, often accompanied by pulmonary hypertension. **Objective:** To evaluate pulmonary hypertension in chronic kidney disease patients using echocardiography, assess its severity in various stages, and assess its progression in dialysis patients.

Materials and Methods: This was a hospital-based observational cross-sectional study that total 167 patients diagnosed with chronic kidney disease (CKD) over 18 years old and on maintenance dialysis. The participants were selected based on age and education level, and written informed consent was obtained from all participants. Patients underwent demographic details, routine investigations, including blood count, urea, creatinine, uric acid, calcium, phosphorus, sodium, potassium, albumin, lipid profile, and urinary examination. CKD was diagnosed if patients had a history of decreased GFR for 3 months or more or as per sonographic findings.

Results: Total 167 patients with chronic kidney disease (CKD) and found a prevalence of pulmonary hypertension (HTN) at 43.7%. The incidence was higher in males (47.5%) than females (38.2%). Patients under 60 years old had 42.3% pulmonary HTN, while those over 60 had 47.7%. The incidence was significantly higher in dialysis patients (68.6%) and those on AV fistula (92%). LV hypertrophy was more evident in CKD patients with pulmonary HTN compared to those without pulmonary HTN. Heart failure with reduced ejection fraction (HFrEF) was present in 26 (35.6%) of 73 patients with pulmonary HTN, while heart failure with preserved ejection fraction (HFpEF) was evident in 87.7%. The severity of pulmonary hypertension increased with age, with higher severity associated with later stages of CKD.

Conclusion: Pulmonary hypertension, a prevalent condition in 43.7% of chronic kidney disease (CKD) patients, is linked to renal function progression and pulmonary vascular complications. Targeted therapies can improve pulmonary vascular resistance and severity, but larger-scale studies are needed to better understand this condition.

Keywords: Chronic kidney disease, renal disease, Pulmonary Hypertension, Echocardiography.

INTRODUCTION

Chronic Kidney Disease (CKD) is a terminal stage of renal parenchymal disease, causing significant morbidity and mortality. The Global Burden of Disease study reported a 20% increase in deaths attributed to CKD worldwide in 2019, with an estimated 1.4 million fatalities. This is one of the leading causes of mortality, particularly in low and middle-income countries.[1,2] CKD affects all bodily organ systems, leading to the deterioration of the kidney's ability to excrete waste, regulate metabolism, and produce hormones. This often results in uremia, which manifests as anemia, metabolic bone disease, neuropathy, myopathy, endocrine irregularities, hypertension, dyslipidemia, acidosis susceptibility to infections, and cardiovascular disorders.[3]

Pulmonary hypertension (PH) is a common co-morbidity in patients with chronic kidney disease (CKD) and end-stage renal disease. The incidence of PH varies between 27% and 58% in individuals with end-stage renal disease.[4] Patients with earlier stages of CKD have a lower prevalence of PH, ranging from 8% to 39%.[5] PH is a hemodynamic and pathophysiological condition characterized by an elevation in mean pulmonary arterial pressure (mPAP) equal to or greater than 25 mmHg. It is categorized into five clinical groups: primary forms, left heart disease, lung diseases and hypoxia, chronic thromboembolic pulmonary hypertension, and PH with unclear or multifactorial mechanisms, including chronic renal failure on dialysis.[6,7]

The exact mechanisms of pulmonary hypertension (PH) in chronic kidney disease (CKD) patients are not well understood, and there is less knowledge about the pathophysiologic distinctions between PH in chronic CKD and similar patients. Potential causes include imbalances between blood vessel constricting and dilation substances, disturbances in mineral and bone metabolism, high-flow arteriovenous fistulas, anemia, pulmonary embolisms, excessive fluid accumulation, and impaired left ventricle function.[8-10] Risk factors for CKD include volume overload, arteriovenous fistula, sleep-disordered breathing, exposure to dialysis membranes, endothelial dysfunction, vascular calcification, and severe anemia.[11]

Pulmonary hypertension (PH) is a condition where patients may remain asymptomatic until right ventricular dysfunction manifests, leading to worsening fatigue, dyspnea, and syncope.[12] Right heart catheterization (RHC) is the gold standard method for assessing PH, but transthoracic echocardiography is now recommended for PH screening due to its noninvasive nature and risk.[13,14] Transthoracic echocardiography provides direct and indirect signs of elevated pulmonary artery pressure (PAP) and may provide key information on the etiology and prognosis of PH. Doppler echocardiography can estimate PAP, but it has limitations as it cannot effectively identify left ventricular filling pressure. PH increases the risk of cardiovascular events and mortality. Epidemiologic data on PH prevalence in chronic kidney disease patients is scarce, especially in India. To prevent PH development and improve long-term outcomes, measures to prevent its development can be implemented. This study aimed to determine the incidence of pulmonary hypertension in chronic kidney disease patients using echocardiography.

The study aims to evaluate the severity of pulmonary hypertension in patients with chronic kidney disease using echocardiography, focusing on patients with evolving CKD stages who are on dialysis and assessing progression or decline.

MATERIALS AND METHODS

The was a hospital-based observational cross-sectional study conducted at Santosh Medical College and Hospital in Ghaziabad, India, from 2023 to 2024. Total 167 patients diagnosed with chronic kidney disease (CKD) over 18 years old and on maintenance dialysis. Participants were selected based on their age and education level. Written informed consent was obtained from all participants, regardless of their background or education. Exclusion criteria include those with chronic obstructive pulmonary disease, structural lung disease, structural heart disease, or Hepato-renal and Cardio-renal syndrome.

The study recorded demographic details of all study subjects, including age, sex, weight, and comorbidities. Patients underwent routine investigations, including blood counts, urea, creatinine, uric acid, calcium, phosphorus, sodium, potassium, albumin, lipid profile, and urinary examination. CKD was diagnosed in patients with a history of decreased GFR for 3 months or more, estimated from the Cockcroft-Gault formula.

GFR, mL/min $=\frac{(140-age)*weight(in\ kgs)}{(72\times(in\ mg/dL)}$ to be multiplied by 0.85 in case of females

ECG was recorded by using BPL machine. All patients were subjected to echocardiography (Philips HD 15 machine) to determine the presence of pulmonary artery hypertension. Pulmonary artery systolic pressure (PASP) was calculated by using tricuspid regurgitation jet velocity (T.R. JET) in Doppler echocardiography and applying Bernoulli equation.

- PASP = 4 × (TRV) 2 + right atrial pressure (RAP)

RAP-10 mmHg.

Pulmonary HTN was defined when mean pulmonary artery systolic pressure (PASP) exceeded 30 mmHg and severity was defined as following:

- Mild Pulmonary HTN: PASP >30 to <35 mm hg
- Moderate Pulmonary HTN: PASP is 35 to 50 mm hg

❖ Severe Pulmonary HTN: PASP >50 mm hg

Echocardiography was utilized to evaluate right atrial and ventricular dimensions, while TAPSE and TDI were used to measure flow across tricuspid and pulmonary valves.

The study collected data using a study proforma, which was filtered and analyzed using GraphPad Prism. The data was presented as baseline patient characteristics, with categorical and continuous variables. Quantitative variables were compared using the student t-test for parametric data and the Mann-Whitney U test for non-parametric data. Analysis of variance (ANOVA) was used to examine significant differences between groups, with a P value of <0.05 considered statistically significant. Percentage rounding was done at one decimal place.

RESULTS

The study analyzed 167 patients with chronic kidney disease (CKD), with a total of 167 patients having pulmonary hypertension (HTN). The prevalence of pulmonary HTN was 43.7%, with mild, moderate, and severe HTN accounting for 16.4%, 52.1%, and 31.5% respectively. Males had a higher incidence of pulmonary HTN (47.5%) compared to females (38.2%). Patients under 60 years old had 42.3% pulmonary HTN, while those over 60 years old had 47.7%. The incidence of pulmonary HTN was statistically significant for dialysis patients (68.6%), while those on AV fistula had 92%. LV hypertrophy was more evident in CKD patients with pulmonary HTN compared to those without pulmonary HTN. Heart failure with reduced EF (HFrEF) was present in 26 (35.6%) of 73 patients with pulmonary HTN, while heart failure with preserved ejection fraction (HFpEF) was evident in 87.7% of patients with pulmonary HTN. The prevalence of pulmonary HTN increased gradually with deterioration of renal function, and the correlation between pulmonary HTN and CKD stages was found to be significant. [Table 1]

A study of 73 patients with pulmonary hypertension found that 71.2% were under 60 years old, while 28.8% were over 60 years old. The gender distribution was not significant, with 58.3% in mild pulmonary hypertension being male, compared to 68.4% in moderate and 60.9% in severe pulmonary hypertension. The severity of pulmonary hypertension increased with age, with 83.3% in mild pulmonary hypertension being less than 60 years old, 81.6% in moderate, and 47.8% in severe pulmonary hypertension. The severity of pulmonary hypertension was significantly associated with the later stages of CKD, with higher severity associated with later stages. As the CKD stage increased, the severity of pulmonary hypertension also increased. The study highlights the importance of understanding the age distribution and severity of pulmonary hypertension in managing this condition. [Table 2]

Total 73 pulmonary hypertension patients revealed that 41.1% had diabetes, 78.1% had systemic hypertension, 37% were habitual smokers, and 34.2% were alcohol users, with 63% on dialysis. [Table 3]

All patients who had arteriovenous fistula as an access for dialysis had various degrees of PH. 30.4% in the severe pulmonary HTN category had AV fistula as compared to 16.7% and 36.8% in mild pulmonary HTN and moderate pulmonary HTN category respectively. 73.7% in the moderate pulmonary HTN category were under dialysis management as compared to 60.9% in severe pulmonary HTN and 33.3% in mild pulmonary HTN category. [Table 4]

After three months, 52 patients with pulmonary hypertension (PH) underwent follow-up echocardiography. LV hypertrophy was detected in 19.2% of patients, and heart failure with reduced EF was present in 30.8%. Pericardial effusion was observed in 21.2% of patients, and RA-RV dilation was observed in 71.2%. Out of 23 severe PH patients, 14 underwent echocardiography, while 9 were lost. 48.1% had moderate pulmonary HTN. [Table 5]

Table 1: Distribution of demographic profile, Risk factors, clinical profile and CKD stages with respect to pulmonary HTN

	Pulmonary HTN (n=73)	No Pulmonary HTN (n=94)	p-value
Age group			
< 60 years	52 (42.3%)	71(57.7%)	$\chi2 = 0.391$; P = 0.5315
≥ 60 years	21(47.7%)	23(52.3%)	
Gender			
Male	47(47.5%)	52(52.5%)	$\chi2 = 1.398$; P = 0.2369
Female	26(38.2%)	42(61.8%)	
Risk Factors			
DM	30(48.4%)	32(51.6%)	P = 0.3493
HTN	57(61.3%)	36(38.7%)	P < 0.001
Smoking	27(50.9%)	26(49.1%)	P = 0.1989
Alcohol	25(46.3%)	29(53.7%)	P = 0.6418
Parameters			
Dialysis	46(68.6%)	21(31.4%)	P<0.001

AV fistula	23(92.0%)	2(8.0%)	P<0.001
ECHO parameters			
LV hypertrophy	18 (24.7%)	12(12.8%)	P = 0.0470
HFrEF	26(35.6%)	5(5.3%)	P < 0.001
HFpEF	64(87.7%)	68(72.3%)	P = 0.015
RV dilation	61(83.6%)	0 (0.0%)	P < 0.001
RA dilation	61(83.6%)	0(0.0%)	P < 0.001
Pericardial effusion	23(31.5%)	4(4.3%)	P < 0.001
CKD stages			
Stage 1(n=16)	2(12.5%)	14(87.5%)	$\chi2 = 18.4863$ P = 0.0009
Stage 2 (n=32)	9(28.1%)	23(71.9%)	
Stage 3(n=29)	11(37.9%)	18(62.1%)	
Stage 4 (n=40)	19(47.5%)	21(52.5%)	
Stage 5(n=50)	32(64.0%)	18(36.0%)	
Total (n=167)	73(43.7%)	94(56.3%)	

Table 2: Distribution of demographic profile, clinical profile and CKD stages with respect to severity of pulmonary HTN

	Mild (n=12)	Moderate (n=39)	Severe (n=23)	p-value
Gender				
Male	7(58.3%)	26(68.4%)	14(60.9%)	$\chi2 = 0.5855$; P = 0.7461
Female	5(41.7%)	12(31.6%)	9(39.1%)	
Age group				
<60 years	10(83.3%)	31(81.6%)	11(47.8%)	$\chi2 = 8.991$; P = 0.011
≥ 60 years	2(16.7%)	7(18.4%)	12(52.2%)	
Dialysis				
Yes	4(33.3%)	28(73.7%)	14(60.9%)	P=0.2688
No	8(66.7%)	10(26.3%)	9(39.1%)	
AV fistula				
Yes	2(16.7%)	14(36.8%)	7(30.4%)	P=0.4193
No	10(83.3%)	24(63.2%)	16(69.6%)	
CKD stages				
Stage 1	2(16.7%)	0(0.0%)	0(0.0%)	$\chi2 = 28.91$; P < 0.001
Stage 2	5(41.6%)	4(10.5%)	0(0.0%)	
Stage 3	2(16.7%)	6(15.8%)	3(13.0%)	
Stage 4	3(25.0%)	8(21.1%)	8(34.8%)	
Stage 5	0(0.0%)	20(52.6%)	12(52.2%)	

Table 3: Distribution of risk factors, Dialysis and AV fistula in patients with pulmonary HTN

Risk Factor	Presence	N	%
DM	Yes	30	41.1
	No	43	58.9
HTN	Yes	57	78.1
	No	16	21.9
Smoking	Yes	27	37.0
	No	46	63.0
Alcohol	Yes	25	34.2
	No	48	65.8
Dialysis	Yes	46	63.0
	No	27	37.0
AV Fistula	Yes	23	31.5
	No	50	68.5

Table 4: Mean values of laboratory and ECHO parameters with respect to severity of pulmonary HTN

Parameter	Mild (N=12)	Moderate (N=38)	Severe (N=23)
Hb (gm/dl)	11.9 ± 1.8	12.5 ± 1.2	12.9 ± 1.7
TLC (x 10^3/cu.mm)	9.07 ± 2.05	9.31 ± 1.46	10.5 ± 1.5
Platelet (x 10^5/cu.mm)	2.54 ± 0.73	2.57 ± 0.71	2.29 ± 0.96
RBS (mg/dl)	99.4 ± 23.6	118.7 ± 34.2	129.0 ± 21.8
Total cholesterol (mg/dl)	160.9 ± 34.6	137.1 ± 36.1	149.7 ± 30.4
Triglyceride (mg/dl)	187.8 ± 39.8	184.9 ± 28.8	191.0 ± 41.5
LDL cholesterol (mg/dl)	90.2 ± 32.3	80.8 ± 18.2	78.3 ± 22.3
HDL cholesterol (mg/dl)	38.3 ± 8.6	46.7 ± 9.1	40.9 ± 12.8
B. Urea (mg/dl)	60.8 ± 56.6	47.7 ± 32.3	60.0 ± 39.6
S. Albumin (g/dl)	3.1 ± 1.6	3.5 ± 1.9	4.3 ± 1.7
S. Uric acid (mg/dl)	5.9 ± 1.8	5.4 ± 1.8	6.5 ± 1.9
S. Creatinine (mg/dl)	1.2 ± 0.3	1.7 ± 0.8	2.3 ± 1.4
eGFR (mL/min/1.73 m^2)	58.4 ± 14.1	63.7 ± 18.5	40.5 ± 12.4

Table 5: Echocardiographic findings of patients with pulmonary HTN at 3-month follow-up

Parameter	At 3-month follow-up (N=52)	
	N	%
ECHO findings		
LV hypertrophy	10	19.2
HFrEF	16	30.8
HFpEF	36	69.2
RV dilation	37	71.2
RA dilation	37	71.2
Pericardial effusion	11	21.2
PH Severity		
Mild	18	34.6
Moderate	25	48.1
Severe	9	17.3

DISCUSSION

Chronic kidney disease (CKD) affects over 800 million adults worldwide, accounting for over 10% of the global population. The disease is progressive and degenerative, with CKD Stages 1–2 being 5%, 3–9%, 0.16%, 0.07%, dialysis 0.041%, and kidney transplantation 0.011%. Patients often experience heart failure, coronary heart disease, and cardiac arrhythmias. Pulmonary hypertension (PH) may be prevalent among CKD patients, but epidemiological data on PH prevalence is scarce.

The study, involving 167 patients with chronic kidney disease (CKD), found a male predominance with 59.3% of participants. Lifestyle choices like smoking, excessive alcohol consumption, and high sodium and protein diets can increase the risk of developing CKD. CKD prevalence increases with age, and males may have a higher risk compared to females. The study found a total prevalence of pulmonary hypertension (PH) among CKD patients at 43.7%, which aligns with previous studies.[15-19] However, discrepancies in prevalence estimates can be attributed to differences in definitions of PH, varying degrees of volume overload, and the exclusion of patients lacking echocardiography data. Despite these differences, the study suggests a high prevalence of pulmonary HTN among CKD patients.

Chronic kidney disease (CKD) impairs fluid and electrolyte balance, leading to fluid retention and volume overload, increasing pulmonary vascular resistance and pulmonary hypertension. Inflammatory cytokines and oxidative stressors in CKD cause endothelial injury, promoting vasoconstriction and pulmonary vascular remodeling, key mechanisms in PH pathogenesis. The prevalence of pulmonary hypertension increases with renal dysfunction progression. In our study, the prevalence of PH across CKD stages 1-5 was 12.5%, 28.1%, 37.9%, 47.5%, and 64%, respectively. This difference in PH prevalence across different CKD stages was found to be statistically significant (P=0.0009). Li Z et al,[20] documented similar findings, reporting PH prevalence in CKD stages 1-5D as 2.2%, 6.7%, 7.9%, 15.2%, 20.0%, and 37.5%, respectively. Likewise, Zhang Q et al,[21] reported PH prevalence in CKD stages 1–5 as 14.29%, 33.33%, 38.89%, 40.91%, and 64.47%, respectively. Studies show a consistent trend of increasing pulmonary hypertension prevalence with worsening renal function, strongly indicating a strong association between CKD progression and pulmonary hypertension development.

Pulmonary hypertension in renal dysfunction patients is a complex condition with unclear risk factors. The pathogenesis is attributed to age, cardiac dysfunction, dialysis duration, arteriovenous fistula, dialyzer membrane exposure, chronic fluid overload, persistent anemia, untreated bone mineral disorder, uremic toxins, and uremic vasculopathy.

In our study, we did not find a significant association between sex and pulmonary HTN among CKD patients, which aligns with findings by Tarras et al,[18] (P=0.469). Conversely, Mehta SK et al,[17] reported a more significant prevalence of PH in males (P=0.03). Age did not have a notable effect on the prevalence of PH in our study, consistent with the findings of Mazdeh et al,[22] (P = 0.58), Patel et al,[23] (P=0.402), and Tarras et al,[18] (P=0.40), who also found no correlation between age and pulmonary HTN. CKD is frequently accompanied by other comorbid conditions such as hypertension, diabetes, and cardiovascular disease, which are independently associated with an increased risk of pulmonary HTN. The cumulative effect of these comorbidities further exacerbates the development of pulmonary HTN in CKD patients. While some studies suggested that hypertension and diabetes mellitus may trigger left ventricular diastolic dysfunction, leading to increased pulmonary venous and arterial pressure and potentially contributing to pulmonary HTN development, other studies reported no association of these comorbidities with pulmonary HTN. In our study, we found that hypertension was significantly associated with pulmonary HTN. However, Mehta SK et al,[17] reported a statistically significant association between diabetes and hypertension with pulmonary HTN (P < 0.001). Agarwal et al,[24] similarly found a statistical association with diabetes (p = 0.04) but not systemic hypertension (p = 0.20). In contrast, Fabian et al,[25] demonstrated a

statistically strong association of both diabetes (p = 0.021) and hypertension (p = 0.0074) with PH.

Pulmonary hypertension in dialysis patients may be caused by a multifactorial etiology, including the pulmonary circulation's inability to adapt to increased cardiac output, anemia, or arteriovenous fistula, or augmented pulmonary vascular stiffness due to endothelial dysfunction, resulting in reduced nitric oxide production. In our observations, 68.6% of CKD patients undergoing dialysis were found to have PH, highlighting a significant association between dialysis and the incidence of pulmonary hypertension (P<0.001). Studies show that dialysis patients have a higher prevalence of pulmonary hypertension (PH) and more severe cases compared to conservative treatment,[17] Hemodialysis patients have a higher prevalence of PH at 68.6% and 68.8%, respectively.[19,26] Hemodialysis patients have a higher PASP at 58.9% compared to 22.2% of peritoneal dialysis patients, with higher PASP in HD patients,[27] Patel et al,[23] found that 33% of 41 patients with pulmonary hypertension were receiving hemodialysis. Factors like exposure to dialysis membrane and arteriovenous fistula contribute to pulmonary hypertension development. 92% of patients with arteriovenous fistula had pulmonary hypertension, indicating a significant association between arteriovenous fistula and pulmonary hypertension incidence (P<0.0001).

Pulmonary hypertension (PH) is a common condition characterized by increased afterload on the left ventricle due to elevated pulmonary artery pressures. Chronic pressure overload on the left ventricle leads to hypertrophic remodeling, resulting in increased LV muscle mass. LVH is observed in 24.7% of CKD patients with PH, compared to only 12.8% of CKD patients without pulmonary hypertension (HTN). This suggests a potential interplay between these conditions. Chronic pressure overload on the right heart leads to right ventricular dilation, which is associated with increased right heart strain and disease severity. This dilation is a hallmark feature of PH and reflects the adaptive response of the RV to increased pulmonary vascular resistance. Chronic pressure overload in the pulmonary circulation can result in impaired RV function and right heart failure. Heart failure prevalence in CKD patients ranges from 30% to 40%, with reduced ejection fraction (HFrEF) observed in 35.6% of CKD patients with pulmonary HTN, and preserved ejection fraction in 87.7% of CKD patients with PH. In the similar study by Suresh et al,[16] the prevalence among the patients with and without PH was 38.3% and 6.6%, respectively (P < 0.001). Progressive RV dysfunction, a common consequence of pulmonary HTN, can impair cardiac output and lead to systemic venous congestion. Elevated central venous pressure may contribute to increased transudation of fluid into the pericardial space. The presence of pericardial effusion in patients with pulmonary HTN may have clinical implications, including hemodynamic compromise, cardiac tamponade, or exacerbation of right heart failure. Therefore, regular cardiac imaging, such as echocardiography, is essential for the detection and monitoring of pericardial effusion in pulmonary HTN patients. As evident from our observation, pericardial effusion was found to be significantly more common in CKD patients with pulmonary HTN. Among CKD patients with pulmonary HTN, 31.5% showed signs of pericardial effusion on echocardiography, whereas only 4.3% of CKD patients without pulmonary HTN exhibited this finding. Pericardial effusion may be indicative of increased pressure within the heart or impaired cardiac function, highlighting the impact of PH on cardiac structure and function in CKD patients.

The study analyzed baseline characteristics, demographics, risk factors, and severity of pulmonary hypertension in 73 patients. Results showed that 16.4% had mild pulmonary hypertension, 52.1% had moderate pulmonary hypertension, and 31.5% had severe pulmonary hypertension. The gender distribution was consistent with previous studies. However, males accounted for 58.3% in mild pulmonary hypertension, while females accounted for 41.7%, 31.6%, and 39.1% in mild, moderate, and severe pulmonary hypertension, respectively. No significant gender difference was found in severity categories.[15,28]

Diabetes Mellitus and hypertension can cause microvascular damage in the pulmonary circulation, leading to vascular remodeling, fibrosis, and increased vascular resistance, which increases pulmonary arterial pressure (PH). Patients with diabetes mellitus and hypertension have a higher severity of PH, with a higher percentage of patients having DM and systemic hypertension. The association between smoking, alcohol consumption, and pulmonary hypertension severity is complex and multifactorial. While smoking and alcohol consumption may contribute to PH development and progression, their direct influence on PH severity may not always be straightforward. No significant difference was observed in the presence of smoking or alcohol consumption with respect to PH severity. Factors such as smoking duration and intensity, comorbidities, and genetic predisposition may influence the relationship between smoking, alcohol consumption, and PH severity. Some studies have reported conflicting findings regarding the direct impact of smoking and alcohol on PH severity.

Arteriovenous fistulas are commonly used as vascular access for hemodialysis in CKD patients. However, their presence can lead to hemodynamic changes and alterations in pulmonary circulation, potentially contributing to the development or exacerbation of PH. Additionally, dialysis itself can affect pulmonary hemodynamics, particularly in patients with underlying vascular or cardiac conditions. The presence of AV fistula as an access for dialysis was associated with varying degrees of PH. Among patients with severe pulmonary HTN,

30.4% had AV fistula, compared to 16.7% and 36.8% in the mild and moderate pulmonary HTN categories, respectively. Moreover, a higher proportion of patients in the moderate pulmonary HTN category were managed under dialysis (73.7%) compared to those in the severe pulmonary HTN (60.9%) and mild pulmonary HTN (33.3%) categories. However, there was no significant association between dialysis and severity of PH (P=0.2688). This was Similar to Wang et al,[28] who reported that the moderate PH group comprised a higher proportion of patients undergoing dialysis compared to the mild and severe PH groups (mild vs. moderate vs. severe: 42.4% vs. 74.3% vs. 47.1%, p = 0.004). Additionally, the severe PH group exhibited better kidney function relative to the mild and moderate PH groups. We also find that there was no significant association between severity of CKD and AV fistula (P = 0.4193). This was congruent with Wang et al,[28] (mild vs moderate vs severity; 21.2% vs 28.6% vs 20.6%; P=0.581). But, higher severity of PH suggests a potential association between AV fistula, and dialysis management, highlighting the importance of considering vascular access type and dialysis status in the evaluation and management of PH in patients with CKD. Hence, treating clinician should optimize dialysis parameters, monitoring pulmonary pressures closely in patients with AV fistula, and considering alternative vascular access options when appropriate.

Chronic kidney disease (CKD) and pulmonary hypertension (HTN) are common risk factors, with CKD leading to fluid overload and volume expansion, which can increase cardiac output and preload, leading to elevated pulmonary artery pressures and exacerbating HTN. Common risk factors include hypertension, diabetes, and obesity, which can contribute to the development and progression of both conditions. A significant association was found between CKD stage and pulmonary HTN severity, suggesting a progressive relationship between CKD stage and pulmonary HTN severity.

Pulmonary hypertension can be effectively managed by improving pulmonary vascular resistance and right ventricular function, which can reduce backward pressure on the left ventricle and alleviate hypertrophic changes. Adherence to prescribed medications, lifestyle modifications, and follow-up appointments can help in the regression of left ventricular hypertrophy. A study on 52 patients with pulmonary hypertension found that 19.2% showed left ventricular hypertrophy, while 30.8% had HFrEF. Pericardial effusion was detected in 21.2% of patients, and right atrial-right ventricular dilation was observed in 71.2%. Adherence to treatment regimens and lifestyle modifications can significantly impact the progression of pulmonary hypertension. Some patients may experience periods of stability or improvement in pulmonary hypertension severity over time, especially with appropriate management and treatment.

The study has limitations, including a single-center experience, reliance on indirect echocardiographic estimates for pulmonary hypertension diagnosis, and uncertainty about the exact cause of pulmonary hypertension. The non-interventional nature of the trial made it challenging to establish a cause-and-effect relationship between pulmonary hypertension and mortality. These limitations suggest the need for cautious interpretation and future research, including larger multi-center studies with more rigorous diagnostic criteria and comprehensive assessment of pulmonary hypertension etiology.

CONCLUSION

Pulmonary hypertension is a prevalent condition among chronic kidney disease (CKD) patients, with 43.7% experiencing it. This condition is linked to the stage of CKD, with higher rates in patients undergoing dialysis and those with arteriovenous fistulas. Left ventricular hypertrophy and pericardial effusion are more common in CKD patients with pulmonary hypertension, highlighting its impact on cardiac structure and function. The prevalence increases with deteriorating renal function, highlighting the relationship between CKD progression and pulmonary vascular complications. Targeted therapies can improve pulmonary vascular resistance, artery pressures, and overall pulmonary hypertension severity. Regular monitoring and individualized management strategies are crucial for optimizing outcomes. Larger-scale studies are needed to better understand pulmonary hypertension in CKD.

REFERENCES

1. Nugent RA, Fathima SF, Feigl AB, Chyung D. The burden of chronic kidney disease on developing nations: a 21st century challenge in global health. Nephron Clin Pract. 2011; 118(3):c269-77.
2. Fraser SDS, Roderick PJ. Kidney disease in the Global Burden of Disease Study 2017. Nat Rev Nephrol. 2019; 15(4):193-194.
3. Bello AK, Alrukhaimi M, Ashuntantang GE, Basnet S, Rotter RC, Douthat WG, et al. Complications of chronic kidney disease: current state, knowledge gaps, and strategy for action. Kidney Int Suppl. 2017; 7(2):122-129.
4. Etemadi J, Zolfaghari H, Firoozi R, Ardalan MR, Toufan M, Shoja MM, et al. Unexplained pulmonary hypertension in peritoneal dialysis and hemodialysis patients. Rev Port Pneumol. 2012; 18(1):10-4.
5. Sise ME, Courtwright AM, Channick RN. Pulmonary hypertension in patients with chronic and end-stage kidney disease. Kidney Int. 2013; 84(4):682-92.
6. Galiè N, Hoeper MM, Humbert M, Torbicki A, Vachiery JL, Barbera JA, et al. Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Respir J. 2009; 34(6):1219-63.
7. Simonneau G, Robbins IM, Beghetti M, Channick RN, Delcroix M, Denton CP, et al. Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol. 20090; 54(1):S43-S54.
8. Harp RJ, Stavropoulos SW, Wasserstein AG, Clark TW. Pulmonary hypertension among end-stage renal failure

patients following hemodialysis access thrombectomy. Cardiovasc Intervent Radiol. 2005; 28(1):17-22.
9. Santosh S, Chu C, Mwangi J, Narayan M, Mosman A, Nayak R, et al. Changes in pulmonary artery systolic pressure and right ventricular function in patients with end-stage renal disease on maintenance dialysis. Nephrology (Carlton). 2019; 24(1):74-80.
10. Walther CP, Nambi V, Hanania NA, Navaneethan SD. Diagnosis and Management of Pulmonary Hypertension in Patients with CKD. Am J Kidney Dis. 2020; 75(6):935-945.
11. Bolignano D, Rastelli S, Agarwal R, Fliser D, Massy Z, Ortiz A, et al. Pulmonary hypertension in CKD. Am J Kidney Dis. 2013; 61(4):612-22.
12. Hoeper MM, Bogaard HJ, Condliffe R, Frantz R, Khanna D, Kurzyna M, Langleben D, et al. Definitions and diagnosis of pulmonary hypertension. J Am Coll Cardiol. 2013; 62(25):D42-50.
13. Gonzalez-Hermosillo LM, Cueto-Robledo G, Roldan-Valadez E, Graniel-Palafox LE, Garcia-Cesar M, Torres-Rojas MB, et al. Right Heart Catheterization (RHC): A Comprehensive Review of Provocation Tests and Hepatic Hemodynamics in Patients with Pulmonary Hypertension (PH). Curr Probl Cardiol. 2022; 47(12):101351.
14. Sachpekidis V, Karvounis H, Giannakoulas G. The role of echocardiography in the diagnostic work-up ofpulmonary hypertension. Continuing Cardiology Education, 2021; 10(15); 3229.
15. Manjiri N. A Cross Sectional Study of Pulmonary Hypertension in Chronic Kidney Disease Patients by Echocardiography at MGM Medical College, Aurangabad, Maharashtra. Ann Med Health Sci Res. 2022; 12: S1:121-124.
16. Suresh H, Arun BS, Moger V, Vijayalaxmi PB, Murali Mohan KTK. A Prospective Study of Pulmonary Hypertension in Patients with Chronic Kidney Disease: A New and Pernicious Complication. Indian J Nephrol. 2018; 28(2):127-134.
17. Mehta KS, Shirkande AK, Bhurke SP, Pajai AE, Swami RS, Jadhav SN. Pulmonary Hypertension in Various Stages of Chronic Kidney Disease in Indian Patients. Indian J Nephrol. 2019; 29(2):95-101.
18. Tarrass F, Benjelloun M, Hachim K, Benghanem MG, Ramdani B. Pulmonary hypertension in patients with end-stage renal disease. Indian J Nephrol 2005; 15: 223- 226.
19. Moniruzzaman M, Islam MN, Alam MB, Alam ABM. M, Khan MMH, ALI Z, Chowdhury AW, Chowdhury MN. Pulmonary Hypertension in Hemodialysis Patients. Cardiovasc. j. 2012; 4(2): 148-152.
20. Li Z, Liang X, Liu S, Ye Z, Chen Y, Wang W, Li R, Xu L, Feng Z, Shi W. Pulmonary hypertension: epidemiology in different CKD stages and its association with cardiovascular morbidity. PLoS One. 2014; 9(12):e114392. 0119787.
21. Zhang Q, Wang L, Zeng H, Lv Y, Huang Y. Epidemiology and risk factors in CKD patients with pulmonary hypertension: a retrospective study. BMC Nephrol. 2018; 19(1):70.
22. Mahdavi-Mazdeh M, Alijavad-Mousavi S, Yahyazadeh H, Azadi M, Yoosefnejad H, Ataiipoor Y. Pulmonary hypertension in hemodialysis patients. Saudi J Kidney Dis Transpl. 2008; 19(2):189-93.
23. Patel P, Abraham G, Pratap B, Ramalakshmi R, Mathew M, Jeevan JM, et al. Clinical and biochemical parameters in chronic kidney disease with pulmonary hypertension. Indian Journal of Nephrology.2007; 17(1):5.
24. Agarwal R. Prevalence, determinants and prognosis of pulmonary hypertension among hemodialysis patients. Nephrol Dial Transplant. 2012; 27(10):3908-14.
25. Fabbian F, Cantelli S, Molino C, Pala M, Longhini C, Portaluppi F. Pulmonary hypertension in dialysis patients: a cross-sectional italian study. Int J Nephrol. 2010 Sep 30; 2011:283475.
26. Kiykim AA, Horoz M, Ozcan T, Yildiz I, Sari S, Genctoy G. Pulmonary hypertension in hemodialysis patients without arteriovenous fistula: the effect of dialyzer composition. Ren Fail. 2010; 32(10):1148-52.
27. Domenici A, Luciani R, Principe F. Pulmonary hypertension in dialysis patients. Perit Dial Int. 2010; 30(2):251-2.
28. Wang L, Zhang W, Zhang C, Yan Z, Li S, Zhang C, et al. Prognostic effect of pulmonary hypertension in patients with chronic kidney disease: Univariate and multivariate analyses of factors associated with survival. Front Med (Lausanne). 2022; 9:972937.