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Original Article

Pulmonary Hypertension among Individuals with Chronic Kidney Disease: Predictive Factors and Long-Term Clinical Implications

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Abstract:

Background: Pulmonary hypertension (PH) is a significant cardiovascular complication of chronic kidney disease (CKD), associated with increased morbidity and mortality, but its prevalence, predictors, and outcomes remain underexplored in developing countries.

Objective: To assess the prevalence, severity, and predictors of PH in CKD patients and evaluate its impact on clinical outcomes over 12 months.

Methods: This observational cross-sectional study with prospective follow-up was conducted at Sylhet Women's Medical College Hospital from January 2023 to December 2024. A total of 200 CKD patients (stages III–V) were enrolled. Transthoracic echocardiography was used to diagnose PH (PASP ≥35 mmHg) and assess severity. Demographic, clinical, laboratory, cardiovascular, and dialysis-related data were collected. Patients were followed for 12 months to evaluate cardiovascular hospitalizations, all-cause mortality, and dialysis-related complications.

Result: PH was present in 41% of patients, with prevalence increasing with CKD stage (Stage III: 25.7%, Stage IV: 41.7%, Stage V: 55.7%, p<0.001). Dialysis dependency in stage V CKD was strongly associated with PH (76.0% vs. 30.0% in non-dialysis patients, p<0.001). PH was mild in 42.5%, moderate in 46.3%, and severe in 11.2% of affected patients. Independent predictors of PH included LV diastolic dysfunction (OR: 3.8, 95% CI: 2.0–7.2), hemoglobin <10 g/dL (OR: 2.6, 95% CI: 1.4–5.0), age >55 years (OR: 2.2, 95% CI: 1.2–4.1), AV fistula presence (OR: 2.3, 95% CI: 1.2–4.5), and poorly controlled hypertension (OR: 2.0, 95% CI: 1.1–3.6). At 12 months, cardiovascular hospitalizations (36.3% vs. 14.2%, p<0.01) and all-cause mortality (16.3% vs. 5.0%, p=0.03) were higher in patients with PH. Dialysis-related complications were more frequent but not statistically significant.

Conclusion: PH is prevalent in CKD, particularly in advanced stages and dialysis-dependent patients, and is associated with worse cardiovascular outcomes. LV diastolic dysfunction, anemia, older age, AV fistula, and uncontrolled hypertension are significant predictors.

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Introduction:

Chronic kidney disease (CKD) has emerged as a significant public health challenge worldwide.

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It is estimated that approximately 9.1% of the global population is affected, translating to more than 850 million individuals living with various stages of kidney dysfunction.[1] Beyond the progressive loss of renal function, CKD is characterized by a host of systemic complications that contribute substantially to its overall disease burden. Among these, cardiovascular disease remains the most common and devastating, serving as the leading cause of morbidity and mortality in this vulnerable population.[2]

Traditionally, the cardiovascular manifestations of CKD have been understood in terms of accelerated atherosclerosis, left ventricular hypertrophy, and heart failure. However, in recent years, there has been increasing recognition of pulmonary hypertension (PH) as another important, yet underappreciated, cardiovascular complication in both CKD and end-stage renal disease (ESRD).[3] Pulmonary hypertension, defined by a mean pulmonary artery pressure (mPAP) ≥25 mmHg as measured invasively by right heart catheterization, can also be suspected non-invasively through echocardiographic estimation of pulmonary artery systolic pressure (PASP). This makes echocardiography a valuable screening tool in clinical practice.

The reported prevalence of PH among individuals with CKD is highly variable, reflecting differences in study design, patient populations, and diagnostic methods. Published reports suggest that between 13% and 50% of pre-dialysis CKD patients exhibit PH, while in those receiving maintenance hemodialysis, the prevalence may rise to as high as 70%.[4,5] These figures underscore the fact that PH is far from a rare finding in patients with kidney disease; rather, it appears to be a frequent and clinically significant complication.

The underlying mechanisms responsible for the development of PH in CKD are complex and multifactorial. Factors such as chronic volume overload, left ventricular systolic and diastolic dysfunction, impaired endothelial function, vascular calcification, the hemodynamic effects of arteriovenous fistulas, and anemia are all thought to contribute to the increased pulmonary pressures observed in this population.[6,7] Even more concerning is the fact that the presence of PH in CKD has been consistently associated with worse clinical outcomes. Studies have demonstrated that patients with CKD and concomitant PH are at higher risk of hospitalizations, exhibit poorer quality of life, and face significantly increased risk of cardiovascular and all- cause mortality.[8]

Despite the growing evidence from Western populations, there remains a notable paucity of data specific to South Asia, where the burden of both CKD and cardiovascular disease is rising rapidly. Socioeconomic constraints, late presentation, limited access to renal replacement therapy, and regional variation in comorbidity profiles mean that patterns of disease observed in other parts of the world may not be entirely generalizable to this region. Moreover, information regarding the predictive factors associated with the development of PH in CKD patients, as well as the long- term consequences of this condition in resource- limited settings, remains limited.

Given these gaps, the present study was designed to provide further insight into this important issue. Specifically, the study aimed to determine the prevalence of pulmonary hypertension among patients with moderate to advanced CKD, to identify the clinical and biochemical predictors associated with its development, and to assess the long-term implications of PH on patient outcomes over a follow-up period of
12 months. Understanding these relationships is crucial for risk stratification, early detection, and the development of strategies to mitigate the adverse effects of PH in CKD patients.

Methods and materials

Study design and settings

The study was an observational, cross-sectional investigation with prospective follow-up, conducted in the Department of Nephrology at Sylhet Women's Medical College & Hospital, Bangladesh, from January 2023 to December 2024.

Sample selection criteria

The study purposively included 200 consecutively enrolled patients with CKD stages III–V, aged 20–75 years, of both sexes, as defined by KDIGO guidelines.9 Eligible participants were adults over 18 years, including pre-dialysis, dialysis-dependent, or recently initiated dialysis patients who were clinically stable and provided written informed consent. Patients with coexisting chronic lung disease, significant valvular or congenital heart disease, chronic thromboembolic PH, severe liver disease, or active infection were excluded.

Diagnostic Methods Echocardiography

All patients underwent transthoracic echocardiography (TTE) performed by an experienced cardiologist. Pulmonary artery systolic pressure (PASP) was estimated using tricuspid regurgitant jet velocity and right atrial pressure based on inferior vena cava

dimensions. Patients were diagnosed with pulmonary hypertension if estimated PASP was ≥35 mmHg, consistent with standard echocardiographic criteria. The severity of PH was categorized as follows: Mild (PASP 36-50 mmHg), Moderate (PASP 51-60 mmHg), and Severe (PASP >60 mmHg), based on the 2022 ESC/ERS Guidelines.10 Left ventricular systolic and diastolic functions were also recorded.

Follow-up and Outcomes measures

For each participant, demographic data (age, sex, body mass index), medical history, and comorbidities such as diabetes, hypertension, anemia, and cardiovascular disease were recorded. Laboratory investigations included hemoglobin, serum creatinine, blood urea, estimated glomerular filtration rate, calcium–phosphate balance, parathyroid hormone, and electrolytes (sodium and potassium). Cardiovascular assessment captured left ventricular ejection fraction, diastolic dysfunction, and valvular abnormalities, while dialysis-related data included duration of dialysis and presence of an arteriovenous fistula. Based on echocardiography, patients were categorized as CKD with pulmonary hypertension (PASP ≥35 mmHg) or CKD without pulmonary hypertension. All patients were prospectively followed for 12 months to document clinical outcomes, including cardiovascular hospitalizations (acute decompensated heart failure, acute coronary syndrome, arrhythmias), all-cause mortality, and dialysis-related complications (vascular access thrombosis, hypotension, recurrent infections). Follow-up data were obtained during outpatient visits and structured telephone interviews for those unable to attend in person.

Statistical analysis

Data were entered and analyzed using SPSS version 26.0 (IBM Corp., Chicago, IL, USA). Continuous variables were presented as mean and standard deviation, and compared between groups using independent t- tests. Categorical variables were expressed as frequencies and percentages and compared using the chi- square test or Fisher's exact test, as appropriate. Multivariate binary logistic regression was performed to identify independent predictors of pulmonary hypertension, adjusting for potential confounders. A p-value <0.05 was considered statistically significant.

Ethical consideration

Written informed consent was obtained from all participants prior to enrollment, ensuring that they understood the study objectives, procedures, and their right to withdraw at any time without affecting their care. The Institutional Review Board (IRB) approval was achieved from the Sylhet Women's Medical College, Sylhet 3100, Bangladesh.(Reference:SWMC/Eth.C/IERB/202142)

Result

Table 1 presents the baseline characteristics of 200 patients with CKD. Patients with CKD and PH were older (58.4±11.8 vs. 50.1±10.3 years, p<0.001) with higher rates of hypertension (81.3% vs. 54.2%, p<0.001), coronary artery disease (21.3% vs. 10.0%, p=0.026), and CCF (18.8% vs. 8.3%, p=0.031) compared to those without PH. Hypertensive nephrosclerosis was more common in PH (33.8% vs. 18.3%, p=0.014), while chronic glomerulonephritis predominated in non-PH (37.5% vs. 17.5%, p=0.002). Laboratory findings showed lower hemoglobin (9.1±1.5 vs. 10.7±1.4 g/dL, p<0.001) and eGFR (18.9±9.8 vs. 29.5±11.2 mL/min/1.73 m², p<0.001) and higher creatinine, urea, uric acid, phosphate, and parathyroid hormone in the PH group. Echocardiography revealed higher PASP (50.2±8.9 vs. 31.1±5.4 mmHg, p<0.001), lower LVEF (59.3±9.5 vs. 64.5±7.2%, p<0.001), larger left atrial diameter, and more frequent LV diastolic dysfunction (65.0% vs. 27.5%, p<0.001) and LVH (72.5% vs. 41.7%, p<0.001). Dialysis factors were also higher in PH: AV fistula presence (41.3% vs. 10.8%, p<0.001) and longer dialysis duration (12.1±11.5 vs .6.2±8.4 months, p<0.001).

Table 1: Baseline demographic and clinical characteristics of the study population (n=200)

Variables	Total (n=200)	CKD with PH (n=80)	CKD without PH (n=120)	*p*-value
	n (%)	n (%)	n (%)	
Demographic data				
Age (years)	53.2 ± 12.5	58.4 ± 11.8	50.1 ± 10.3	<0.001
Gender (Male)	119 (59.5)	49 (61.3)	70 (58.3)	0.682
Comorbidities				
Diabetes mellitus	71 (35.5)	33 (41.3)	38 (31.7)	0.162
Hypertension	130 (65.0)	65 (81.3)	65 (54.2)	<0.001
Coronary artery disease (IHD)	29 (14.5)	17 (21.3)	12 (10.0)	0.026

Congestive cardiac failure (CCF)	25 (12.5)	15 (18.8)	10 (8.3)	0.031
Stroke	12 (6.0)	7 (8.8)	5 (4.2)	0.182
Causes of CKD				
Diabetic nephropathy	71 (35.5)	33 (41.3)	38 (31.7)	0.162
Hypertensive nephrosclerosis	49 (24.5)	27 (33.8)	22 (18.3)	0.014
Chronic glomerulonephritis	59 (29.5)	14 (17.5)	45 (37.5)	0.002
ADPKD	11 (5.5)	3 (3.8)	8 (6.7)	0.374
Others	10 (5.0)	3 (3.8)	7 (5.8)	0.502
CKD stage				
Stage 3	70 (35.0)	24 (30.0)	46 (38.3)	0.226
Stage 4	60 (30.0)	26 (32.5)	34 (28.3)	0.526
Stage 5	70 (35.0)	30 (37.5)	40 (33.3)	0.550
Laboratory parameters				
Hemoglobin (g/dL)	10.1 ± 1.6	9.1 ± 1.5	10.7 ± 1.4	<0.001
Serum creatinine (mg/dL)	3.2 ± 1.8	3.8 ± 2.1	2.8 ± 1.5	<0.001
eGFR (mL/min/1.73 m²)	25.4 ± 12.3	18.9 ± 9.8	29.5 ± 11.2	<0.001
Blood urea (mg/dL)	128 ± 56	145 ± 62	117 ± 48	0.001
Serum uric acid (mg/dL)	7.8 ± 2.1	8.2 ± 2.3	7.5 ± 1.9	0.023
Serum calcium (mg/dL)	8.9 ± 1.2	8.7 ± 1.3	9.0 ± 1.1	0.085
Serum phosphate (mg/dL)	5.2 ± 1.8	5.8 ± 1.9	4.8 ± 1.6	<0.001
Parathyroid hormone (pg/mL)	285 ± 180	356 ± 192	238 ± 158	<0.001
Echocardiographic parameters				
LVEF (%)	62.4 ± 8.7	59.3 ± 9.5	64.5 ± 7.2	<0.001
PASP (mmHg)	38.5 ± 12.3	50.2 ± 8.9	31.1 ± 5.4	<0.001
Left atrial diameter (mm)	39.2 ± 6.8	42.5 ± 6.9	37.1 ± 5.8	<0.001
LV diastolic dysfunction	85 (42.5)	52 (65.0)	33 (27.5)	<0.001
LVH	108 (54.0)	58 (72.5)	50 (41.7)	<0.001
Medications				
ACEI/ARB	125 (62.5)	48 (60.0)	77 (64.2)	0.546
β-blocker	98 (49.0)	45 (56.3)	53 (44.2)	0.092
Calcium channel blocker	85 (42.5)	38 (47.5)	47 (39.2)	0.249
Diuretic	112 (56.0)	52 (65.0)	60 (50.0)	0.038
Dialysis profile				
Arteriovenous fistula	46 (23.0)	33 (41.3)	13 (10.8)	<0.001
Dialysis duration (months)	8.5 ± 10.2	12.1 ± 11.5	6.2 ± 8.4	<0.001

Chi-square, Fisher's exact test and Independent t-test were done; P<0.05 is considered as a significant value

Table 2 shows that the prevalence of PH increases progressively with advancing CKD stage. Among patients with Stage III CKD, 25.7% had PH, which increased to 41.7% in Stage IV and 55.7% in Stage V. The chi-square test shows this trend is statistically significant (p<0.001).

Table 2: Prevalence of PH by CKD stage (n=200)

CKD stage	N	Patients with PH	Patients without PH	p-value
		(n, %)	(n, %)	
Stage III	70	18 (25.7)	52 (74.3)	<0.001
Stage IV	60	25 (41.7)	35 (58.3)	
Stage V	70	39 (55.7)	31 (44.3)	

Chi-square test was done; P<0.05 is considered as a significant value

Table 3 demonstrates a strong association between dialysis dependency and PH in Stage 5 CKD patients. Among non-dialysis patients, 30.0% had PH, whereas 76.0% of dialysis-dependent patients developed PH. The difference is statistically significant (p<0.001).

Table 3: Association between dialysis dependency and PH in Stage 5 CKD patients (n=70)

Stage 5 CKD group	N	Patients with PH	Patients without PH	p-value
		(n, %)	(n, %)	
Non-dialysis	20	6 (30.0)	14 (70.0)	<0.001
Dialysis-dependent	50	38 (76.0)	12 (24.0)	

Chi-square test was done; P<0.05 is considered as a significant value

Table 4 shows the distribution of PH severity among CKD patients with PH. Mild PH (PASP 36–50 mmHg) was present in 42.5% of patients, moderate PH (PASP 51–60 mmHg) in 46.3%, and severe PH (PASP >60 mmHg) in 11.2%.

Table 4: PH severity in CKD patients with PH (n=80)

PH severity	PASP Range (mmHg)	Number of patients (n, %)
Mild	36–50	34 (42.5)
Moderate	51–60	37 (46.3)
Severe	>60	9 (11.2)

Table 5 shows that several factors independently predict PH in CKD patients. LV diastolic dysfunction was the strongest predictor (OR 3.8, 95% CI 2.0–7.2, p < 0.001), followed by anemia with hemoglobin <10 g/dL (OR: 2.6, 95% CI: 1.4–5.0, p<0.01), age >55 years (OR: 2.2, 95% CI: 1.2–4.1, p= 0.02), presence of an AV fistula (OR: 2.3, 95% CI: 1.2–4.5, p=0.03), and poorly controlled hypertension (OR: 2.0, 95% CI: 1.1–3.6, p=0.04).

Table 5: Logistic regression analysis of predictive factors for PH in CKD patients (n=200)

Predictive factors	Odds ratio (OR)	95% CI for OR		p-value
		Lower	Upper	
Age >55 years	2.2	1.2	4.1	0.02
Hemoglobin <10 g/dL	2.6	1.4	5.0	<0.01
LV diastolic dysfunction	3.8	2.0	7.2	<0.001
AV fistula presence	2.3	1.2	4.5	0.03
Poorly controlled HTN	2.0	1.1	3.6	0.04

Multivariate binary logistic regression, P<0.05 is considered as a significant value

Table 6 shows that CKD patients with PH experienced significantly worse clinical outcomes over 12 months compared to those without PH. Cardiovascular hospitalizations were higher in the PH group (36.3% vs. 14.2%, p<0.01), and all-cause mortality was also increased (16.3% vs. 5.0%, p=0.03). Dialysis-related complications were more frequent in the PH group (27.5% vs. 20.8%) but did not reach statistical significance (p=0.25).

Table 6: Outcomes at 12-month follow-up (n=200)

Outcomes	CKD with PH	CKD without PH	p-value
	(n, %)	(n, %)	
Cardiovascular hospitalizations	29 (36.3)	17 (14.2)	<0.01
All-cause mortality	13 (16.3)	6 (5.0)	0.03
Dialysis-related complications	22 (27.5)	25 (20.8)	0.25

Fisher's exact test was done; P<0.05 is considered as a significant value

Discussion:

In this study among the CKD patients, 40% had echocardiographic evidence of PH, consistent with previously reported prevalence rates of 30–50% in pre-dialysis and ESRD populations, especially in Asia and the Middle East. [4,11] PH prevalence increased progressively with CKD stage, rising from 25% in stage III to 56% in stage V, highlighting the close relationship between declining renal function and pulmonary vascular changes. Among stage V patients, dialysis-dependent individuals had a markedly higher prevalence of PH (76.0%) compared to non-dialysis patients (30.0%), indicating the added hemodynamic burden of renal replacement therapy. Regarding severity, nearly half of PH patients (46.3%) had moderate PH (PASP 51–60 mmHg), with mild (42.5%) and severe (11.2%) forms being less common, reflecting clinically significant elevations in pulmonary pressure in most affected CKD patients, in line with current international guidelines.[10]

The independent predictors of PH identified in this study, older age, anemia, LV diastolic dysfunction, presence of an AVF, and poorly controlled hypertension are both biologically plausible and clinically significant. Advancing age is associated with vascular stiffening, reduced cardiopulmonary reserve, and accumulation of comorbidities, all of which increase susceptibility to PH in CKD patients.[6] Anemia, a common CKD complication, elevates cardiac output to maintain oxygen delivery; this chronic high-output state, combined with volume overload, increases pulmonary venous return and pulmonary pressures.[6,11] LV diastolic dysfunction emerged as the strongest predictor. Impaired ventricular relaxation elevates left atrial and pulmonary venous pressures, transmitted backward into the pulmonary circulation, resulting in post-capillary PH. This aligns with prior studies identifying diastolic dysfunction as a crucial determinant of PH in CKD.[12] Dialysis-related factors were also significant. AVF presence more than doubled the likelihood of PH by increasing venous return and cardiac output, elevating pulmonary vascular flow. Longer dialysis duration further contributes via cumulative volume and hemodynamic shifts. Finally, poorly controlled hypertension promotes LV hypertrophy and myocardial stiffening, exacerbating diastolic dysfunction and ultimately facilitating the development of PH.[13] These findings underscore the multifactorial pathophysiology of PH in CKD and highlight potential targets for risk stratification and management.

PH carries substantial prognostic significance in CKD. At one-year follow-up, patients with PH experienced more than twice the risk of cardiovascular hospitalization compared to those without PH (36% vs. 14%), primarily due to acute decompensated heart failure, arrhythmias, and acute coronary syndromes, highlighting the considerable cardiac burden associated with pulmonary vascular dysfunction in CKD. All-cause mortality was also markedly higher in the PH group (16% vs. 5%), confirming PH as a strong independent predictor of poor survival. These findings align with previous studies, including study reported a 39% PH prevalence in CKD and its association with increased mortality[11], and another study demonstrated worsened outcomes in both pre-dialysis and dialysis-dependent patients.[7] Collectively, these results emphasize that PH is not merely a secondary phenomenon but a clinically significant complication that directly contributes to adverse outcomes in CKD. Although dialysis-related complications such as vascular access thrombosis, intradialytic hypotension, and

infections were slightly higher in the PH group (28% vs. 21%) this difference did not reach statistical significance. Larger studies are warranted to determine whether PH indirectly predisposes patients to dialysis-related morbidity or whether these complications occur independently of pulmonary pressures.[14,15]

This study's strengths include providing valuable data from South Asia, where CKD and cardiovascular patterns may differ from high-income countries, and the comprehensive assessment of clinical, laboratory, and echocardiographic parameters, which allowed identification of multiple independent predictors, including dialysis-related factors. Limitations include the non-invasive echocardiographic diagnosis of PH, a moderate single-center sample, and a 12-month follow-up, which may limit generalizability and long-term outcome assessment. Clinically, routine PH screening and early management of modifiable risk factors such as anemia, hypertension, and AV fistula hemodynamics may improve outcomes. Future multicenter studies with right heart catheterization are needed to confirm findings and evaluate targeted interventions

Conclusion:
Pulmonary hypertension is a frequent and clinically important complication in CKD, with prevalence increasing alongside CKD stage and dialysis dependence. Key independent predictors include older age, anemia, left ventricular diastolic dysfunction, presence of an arteriovenous fistula, and uncontrolled hypertension. PH is associated with markedly higher cardiovascular hospitalization and all-cause mortality, highlighting its prognostic significance. These findings emphasize the need for routine echocardiographic screening in high-risk CKD patients and early intervention targeting modifiable factors such as anemia, blood pressure control, and dialysis access management. Further large-scale, multicenter studies with invasive hemodynamic assessment are warranted to improve risk stratification and guide therapeutic strategies.

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