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Prevalence and Risk Factors of Pulmonary Hypertension in Chronic Kidney Disease Patients A Cross Sectional Study on the Impact of Hemodialysis and Biochemical Markers

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HIGHLIGHTS

1. High pulmonary hypertence in CKD
2. Hemodialysis impacts pulmomary hypertension risk
3. Biochemical maekers Linked to severity
4. Increased risk with CKD progression
5.Cross sectional study supports association

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ABSTRACT

Background: Chronic kidney disease (CKD) presents a significant public health challenge, often complicated by comorbid conditions such as pulmonary hypertension (PH), especially in patients on hemodialysis (HD). Understanding the prevalence, risk factors, and biochemical markers associated with PH in CKD patients can inform better management strategies and improve outcomes. **Objective:** To assess the prevalence of PH among CKD patients, identify key risk factors associated with its development and progression, and evaluate the impact of CKD duration, HD exposure, and biochemical markers on PH severity. **Methods:** A cross-sectional study was conducted involving 250 CKD patients in a tertiary care center. Patients' demographic and clinical data were collected, with PH diagnosed through echocardiography based on elevated right ventricular systolic pressure. Biochemical markers, including serum creatinine and calcium-phosphorus (Ca × P) product, were measured to analyze their correlation with PH presence and severity. Statistical analyses included chi-square tests for categorical variables and logistic regression for adjusted associations. **Results:** The prevalence of PH in CKD patients was 77.2%, with higher rates in patients with diabetes mellitus and hypertension. PH prevalence correlated significantly with CKD duration, HD duration, and higher serum creatinine and Ca × P product levels ($p < 0.05$). Notably, patients on HD for over 12 months had a PH prevalence of 95.6%, indicating that prolonged HD exposure is a significant risk factor. Anemia showed a negative association with PH severity, although this was not statistically significant. **Conclusions:** PH is highly prevalent in CKD patients, particularly among those with prolonged disease duration, extended HD treatment, and elevated metabolic markers. These findings highlight the need for integrated CKD management approaches that include routine PH screening, management of biochemical imbalances, and anemia control. Further research should aim to clarify the causal pathways of PH in CKD and evaluate interventions that may mitigate PH risk in this population.

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INTRODUCTION

Chronic kidney disease (CKD) is a significant and escalating public health challenge globally, affecting over 10% of the adult population worldwide. Defined by the presence of renal impairment or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for three months or more, CKD is often asymptomatic until its advanced stages. Diabetes and hypertension are recognized as primary contributors, especially in regions where access to healthcare is limited or the population faces high socioeconomic challenges. The disease places an enormous strain on both individuals and healthcare systems, particularly as it often leads to end stage renal disease (ESRD) and heightened cardiovascular risk (1-3).In India, the prevalence of CKD is rising, with projections estimating that nearly 115 million people may be affected. The prevalence in India underscores the need for early detection and intervention, given the country's burden of diabetes and hypertension, the leading causes of CKD in the region(4).

Pulmonary hypertension (PH) in CKD patients is an emerging concern that compounds the morbidity associated with kidney disease. PH in CKD, particularly in stages 4 and 5, is attributed to volume overload, increased cardiac output, and vascular calcifications. Studies indicate that PH affects 30-60% of individuals with CKD, with higher prevalence in advanced disease stages. The combination of CKD and PH markedly increases the risk of cardiovascular events and mortality (,). The pathophysiology of PH in CKD is complex and multifactorial, involving not only traditional cardiovascular risk factors but also the metabolic imbalances characteristic of advanced kidney disease. In Indian populations, the incidence of PH in CKD is underreported, yet small cohort studies and hospital-based surveys suggest a high burden, especially among those with uncontrolled hypertension and diabetes(7,8).

In India, CKD remains a frequently overlooked condition despite its increasing prevalence. Factors such as late diagnosis, limited nephrology services, and socioeconomic barriers exacerbate disease progression and complications, including PH. Addressing these challenges requires improving awareness and access to early diagnosis, emphasizing the integration of CKD and PH management into routine healthcare, and supporting research to establish accurate prevalence and incidence rates within Indian demographics(8-11).

MATERIALS AND METHODS

Study Design and Population:

This cross sectional observational study was conducted on a cohort of 250 patients diagnosed with chronic kidney disease (CKD) who were receiving treatment in a tertiary care center. Inclusion criteria encompassed patients with confirmed CKD diagnoses across various etiologies including diabetes mellitus, hypertension and other less common causes as defined by the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines (National Kidney Foundation, 2002). Exclusion criteria were set to remove patients with known primary pulmonary conditions or recent acute cardiac events to ensure specificity in evaluating the relationship between CKD and pulmonary hypertension (PH).

Data Collection:

Patients' demographic data (age, gender, duration of CKD, and treatment history) were collected through structured interviews and clinical records. Comprehensive clinical assessments, including duration of CKD, history of hypertension, diabetes, and other CKD etiologies, were obtained from medical records. Pulmonary hypertension was assessed through echocardiography following guidelines from the American Society of Echocardiography, to estimate pulmonary arterial pressure. Hemodialysis (HD) duration was also recorded for patients on HD, categorized as less than 6 months, 6-12 months, and over 12 months.

Pulmonary Hypertension Evaluation:

PH was diagnosed based on echocardiographic findings indicating elevated right ventricular systolic pressure (RVSP) above the threshold of 35 mmHg, which is the widely accepted diagnostic criterion for PH in CKD patients. PH severity was classified into mild, moderate, and severe categories based on RVSP levels, aligned with clinical classification standards used in previous studies (Abraham et al., 2019). Echocardiograms were conducted by certified technicians blinded to patients' clinical histories to avoid bias.

Biochemical and Clinical Measurements:

Routine blood tests were performed to assess kidney function and biochemical markers associated with CKD and PH risk, including serum creatinine, hemoglobin, blood urea nitrogen (BUN), and the calcium phosphorus (Ca x P) product. Data on serum creatinine and Ca x P were categorized into clinically significant thresholds to analyze their relationship with PH severity. Blood samples were analyzed using standardized laboratory methods, and laboratory staff were blinded to PH status. Data regarding biochemical parameters were collected and analyzed to establish correlations with PH prevalence and severity.

Statistical Analysis:

Data were analyzed using SPSS version 25. Descriptive statistics were used to summarize demographic, clinical, and biochemical characteristics. PH prevalence across CKD etiologies and duration categories was evaluated using chi square tests, with statistical significance set at $p < 0.05$. Correlation analysis between PH and CKD duration, HD duration, and biochemical variables (e.g., serum creatinine, Ca x P product) was conducted using Pearson's correlation coefficient. Logistic regression was employed to adjust for potential confounders, including age, sex, and comorbid conditions like diabetes and hypertension. Results are presented as odds ratios (OR) with 95% confidence intervals (CI).

Ethical Considerations:

The study was conducted in accordance with the ethical standards of the institutional research committee and the 1964 Helsinki declaration. Informed consent was obtained from all participants prior to their inclusion in the study. Personal health data confidentiality was strictly maintained throughout data collection, analysis, and reporting phases.

RESULTS

In this study of 250 chronic kidney disease (CKD) patients, pulmonary hypertension (PH) was obse-rved across various etiologies. Diabetes Mellitus (DM) had the highest prevalence of PH, with 87.5% (70 out of 80 patients) exhibiting the condition followed by Undetermined etiology at 83.3% (25 of 30 patients) and Hypertension (HTN) at 66.7% (40 of 60 patients). Other causes like Obstructive Uropathy and Chronic Glomerulonephritis showed PH prevalence rates of 66.7% and 60.0%, respectively. The presence of PH was significantly associated with DM and HTN ($p < 0.001$), suggesting a strong correlation between these conditions and elevated pulmonary pressures in CKD patients. The overall analysis indicates a considerable burden of PH among CKD patients, particularly in those with diabetes and hypertension as primary etiologies.

Table 1: Pulmonary Hypertension (PH) and Etiology of CKD

Etiology	Total (n)	PH Present (n, %)	p-value
Diabetes Mellitus (DM)	80	70 (87.5%)	<0.001
Hypertension (HTN)	60	40 (66.7%)	<0.001
Undetermined (UNDETR)	30	25 (83.3%)	0.369
Obstructive uropathy (OU)	15	10 (66.7%)	0.069
Chronic Glomerulonephritis (CGN)	20	12 (60.0%)	0.134
Other	20	15 (75.0%)	0.827
Chronic tubulointerstitial disease	10	7 (70.0%)	
Polycystic kidney disease	6	5 (83.3%)	
Genitourinary TB	5	4 (80.0%)	
Reflux disease	4	3 (75.0%)	
Ischemic nephropathy	3	2 (66.7%)	
Total	250	193 (77.2%)	

In this study, the incidence of pulmonary hypertension (PH) in CKD patients increased with the duration of CKD. Among patients with CKD duration under 6 months, 33.3% (10 out of 30) had PH, whereas 59.1% (65 out of 110) of patients with CKD duration between 6 and 12 months were affected.Notably the prevalence was highest in patients with CKD lasting over 12 months, where 86.4% (95 out of 110) showed PH. This trend suggests a significant correlation between longer CKD duration and the increased likelihood of developing PH, indicating that prolonged kidney disease exacerbates the risk of pulmonary complications.

Table 2: CKD Duration and Incidence of Pulmonary Hypertension

CKD Duration (months)	PH Absent (n)	PH Present (n)	Total (n)	PH Present (%)
<6	20	10	30	33.3
6-12	45	65	110	59.1
>12	15	95	110	86.4

The severity of pulmonary hypertension (PH) among CKD patients showed a progressive increase with longer CKD duration. For patients with CKD duration under 6 months, 80% (8 out of 10) had mild PH, while 20% had moderate PH, with no cases of severe PH. In those with CKD lasting 6 to 12 months, 38.5% (25 of 65) had mild PH, 46.2% had moderate PH, and 15.4% had severe PH. Notably, patients with CKD duration exceeding 12 months exhibited the most severe profile, with only 15.8% having mild PH, while 47.4% had moderate PH, and a significant 36.8% had severe PH. This trend underscores the increasing severity of PH with prolonged CKD duration, suggesting a strong association between CKD progression and worsening pulmonary outcomes.

Table 3: CKD Duration and Severity of Pulmonary Hypertension

CKD Duration (months)	Total PH Present (n)	Mild PH (n, %)	Moderate PH (n, %)	Severe PH (n, %)
<6	10	8 (80.0%)	2 (20.0%)	0 (0.0%)
6-12	65	25 (38.5%)	30 (46.2%)	10 (15.4%)
>12	95	15 (15.8%)	45 (47.4%)	35 (36.8%)

The incidence of pulmonary hypertension (PH) increased with longer durations of hemodialysis (HD). For patients undergoing HD for less than 6 months, 80% (20 out of 25) had PH. This prevalence rose slightly for those with 6 to 12 months of HD, where 81.8% (45 of 55) were affected. The highest prevalence 95.6% (65 of 68), was observed among patients with HD durations exceeding 12 months. The chi-square test confirmed a significant association between HD duration and PH presence ($p = 0.028$), suggesting that extended HD duration may exacerbate PH risk in CKD patients.

Table 4: Duration of Hemodialysis (HD) and Pulmonary Hypertension

HD Duration (months)	Total (n)	PH Present (n, %)	PH Absent (n, %)
<6	25	20 (80.0%)	5 (20.0%)
6-12	55	45 (81.8%)	10 (18.2%)
>12	68	65 (95.6%)	3 (4.4%)
Total	148	130 (87.8%)	18 (12.2%)

The association between biochemical variables and the presence of pulmonary hypertension (PH) in CKD patients highlights specific risk markers. Among patients with hemoglobin levels below 10 gm/dl, 77.9% (88 out of 113) had PH, although this association was not statistically significant ($p = 0.082$). Similarly, 76.6% (59 of 77) of patients with blood urea nitrogen (BUN) levels exceeding 45 mg/dl had PH, with no significant association ($p = 0.240$). In contrast, a statistically significant corre-lation was observed for serum creatinine levels above 5 mg/dl, where 86.7% (78 of 90) of these patients exhibited PH ($p < 0.05$). Additionally, those with a calcium-phosphorus (Ca x P) product greater than 55 mg^2/dl^2 had a high PH prevalence of 80.0% (20 out of 25), with a strong significance ($p < 0.001$). These findings indicate that elevated serum creatinine and Ca x P product levels are significantly associated with an increased risk of PH among CKD patients.

Table 5: Biochemical Variables and Pulmonary Hypertension

Biochemical Variable	PH Present (n, %)	PH Absent (n, %)	Total (n)	p-value
Hemoglobin <10 gm/dl	88 (77.9%)	25 (22.1%)	113	0.082
BUN >45 mg/dl	59 (76.6%)	18 (23.4%)	77	0.24
Serum creatinine >5 mg/dl	78 (86.7%)	12 (13.3%)	90	<0.05
Calcium-Phosphorus (Ca x P) Product >55 mg^2/dl^2	20 (80.0%)	5 (20.0%)	25	<0.001

The correlation analysis between pulmonary hypertension (PH) and key clinical variables in CKD patients reveals significant associations. A positive correlation was observed between PH and both CKD duration ($r = 0.23$, $p = 0.001$) and hemodialysis duration ($r = 0.40$, $p < 0.001$), suggesting that prolonged disease and treatment duration may increase PH risk. Serum creatinine and calcium phosphorus (Ca x P) product levels also showed strong positive correlations with PH ($r = 0.41$ and 0.40,- respectively; both $p < 0.001$) highlighting these markers as significant contributors to PH development. In contrast, hemoglobin exhibited a negative correlation with PH ($r = -0.32$, $p < 0.001$), indicating that lower hemoglobin levels may exacerbate PH risk. These findings underscore the role of disease duration biochemical markers, and anemia in influencing PH severity in CKD patients.

Table 6: Correlation between Pulmonary Hypertension and other Dependent Variables

Variable	Correlation Coefficient	p-value
CKD Duration	0.23	0.001
Hemodialysis Duration	0.4	<0.001
Hemoglobin	-0.32	<0.001
BUN	0.33	<0.001
Serum Creatinine	0.41	<0.001
Ca x P Product	0.4	<0.001

DISCUSSION

Our study highlights the high prevalence of pulmonary hypertension (PH) among chronic kidney disease (CKD) patients, a finding consistent with other studies that underscore the interplay between progressive renal impairment and elevated pulmonary pressures. In our sample, PH prevalence increased markedly in patients with longer CKD duration and among those undergoing prolonged hemodialysis (HD). Mehta et al. (2019) found similar patterns in an Indian CKD cohort reporting increased PH prevalence with advancing CKD stages, particularly in patients with ESRD. The association between CKD and PH is partly explained by shared risk factors such as volume overload, vascular calcifications, and endothelial dysfunction, all of which exacerbate cardiovascular morbidity in CKD populations(). Such findings stress the importance of early identification of PH in CKD patients, especially those in advanced disease stages.

Our data also reveal a notable impact of HD on PH development, with the highest PH prevalence observed among patients on HD for over 12 months. Previous studies, such as those by Moniruzzaman et al. (2012) and Kiykim et al. (2010), support this finding, showing that prolonged HD exposure is strongly associated with increased PH risk. The mechanisms likely involve repetitive exposure to dialysis membranes and arteriovenous fistula (AVF) creation both of which contribute to endothelial damage and impaired nitric oxide production leading to increased pulmonary vascular resistance(12,13). These results underscore the need to explore alternative dialysis strategies or AVF management to reduce PH progression in HD dependent patients.

Biochemical markers also emerged as significant predictors of PH in our study, particularly serum creatinine and the calcium phosphorus (Ca × P) product. Elevated serum creatinine and Ca × P levels both indicators of impaired kidney function and altered mineral metabolism, were significantly associated with higher PH prevalence. Emara et al. (2013) reported a similar correlation, suggesting that elevated Ca × P products contribute to vascular calcification, which increases pulmonary arterial stiffness and exacerbates PH (10). These findings emphasize the importance of closely monitoring these markers in CKD patients and implementing interventions to manage mineral and bone disorders as part of an integrated approach to PH risk reduction.

The role of anemia, frequently observed in CKD, was also notable in our study as it showed a negative correlation with PH severity. Anemia exacerbates hypoxia, which in turn increases pulmonary vascular resistance and contributes to PH development. Although our findings did not show a statistically significant association between hemoglobin levels and PH prevalence, the observed trends are consistent with studies that document anemia as a contributing factor in CKD related PH, highlighting the potential benefit of anemia management in reducing PH risk (14). Targeted anemia correction strategies could therefore play a critical role in the comprehensive management of PH in CKD patients.

Our findings align with studies such as those by Yigla et al. (2003), which demonstrated a higher mortality rate among CKD patients with PH compared to those without, marking PH as an independent predictor of poor outcomes in CKD. Specifically Yigla et al. found that CKD patients with PH had a mortality rate of 30.4%, compared to only 8.5% among those without PH. This increased mortality risk associated with PH, combined with the high prevalence observed in CKD, underscores the need for clinicians to incorporate routine PH screening and management as part of CKD care particularly for those in advanced stages and on long term HD(14).

The mechanisms underlying PH in CKD patients, especially those on HD, remain complex and multifaceted. Studies have suggested that CKD induced uremic endothelial dysfunction disrupts the balance of vasodilators and vasoconstrictors, which coupled with chronic volume overload, may elevate pulmonary artery pressures (). Our study supports this model by highlighting the strong correlation between HD duration vascular calcification and PH severity. Targeted therapies that aim to restore endothelial function, such as the use of endothelin receptor antagonists, could potentially mitigate PH severity in this population, although further research is needed to explore these therapeutic avenues.

An interesting aspect of our findings is the variability in PH prevalence across CKD etiologies, with the highest rates seen in patients with diabetes and hypertension, which are known risk factors for both CKD and PH. Studies such as those by Agarwal (2012) have similarly documented significant associations between diabetes, hypertension, and PH in CKD populations, attributing this link to the cardiovascular changes driven by these conditions, including left ventricular dysfunction and chronic inflammation. Such findings suggest that in CKD patients with comorbid diabetes and hypertension, early interventions for PH prevention could be particularly beneficial, potentially improving both cardiovascular and renal outcomes().

In conclusion, our study highlights the extensive burden of PH in CKD patients, particularly in those on long term HD or with metabolic imbalances, such as high Ca × P products. These insights underscore the importance of an integrated, multifactorial approach to CKD management that incorporates PH risk assessments, mineral metabolism control, and strategic anemia management. Given the progressive nature of PH and its strong association with adverse outcomes in CKD future research should focus on developing early intervention protocols and exploring pharmacological options that address the unique pathophysiology of PH in this population.

LIMITATIONS

This study, while providing significant insights into the prevalence and risk factors of pulmonary hypertension (PH) in chronic kidney disease (CKD) patients, has several limitations. First, our sample size is limited, which may restrict the generalizability of the findings to larger, more diverse CKD populations. A larger cohort would be more representative and could help clarify the relationships between PH and various CKD related variables. Second, we relied on echocardiography for PH diagnosis rather than right heart catheterization, which is the gold standard for accurately measuring pulmonary arterial pressures. Although noninvasive and widely accessible, echocardiography may be less precise, potentially leading to underestimation or overestimation of PH severity.

Additionally, the study's cross sectional design limits our ability to establish causal relationships between CKD progression, HD duration, and PH development. Longitudinal studies would be needed to determine if

the observed associations are indeed causal and to explore the temporal progression of PH in CKD patients. Our study also excluded patients with CKD stages I and II, focusing only on those with moderate to advanced disease stages which might result in selection bias. Including early stage CKD patients could provide a broader understanding of PH onset across different disease severities.

Furthermore we did not account for confounding factors such as systemic inflammation which is prevalent in CKD and known to contribute to vascular complications. Variables such as C-reactive protein and other inflammatory markers could provide additional insights but were not measured in our cohort. Lastly while we examined the role of biochemical markers like serum creatinine and calcium-phosphorus (Ca × P) product the study did not investigate other potentially relevant factors such as parathyroid hormone levels and bone mineral density which could further elucidate the mechanisms linking mineral metabolism disorders to PH in CKD patients.

Overall, addressing these limitations in future studies would enhance the robustness and applicability of findings related to PH prevalence and risk factors in CKD populations.

CONCLUSION

This study underscores the significant burden of pulmonary hypertension (PH) among chronic kidney disease (CKD) patients, especially as CKD progresses and with extended hemodialysis (HD) exposure. We identified key risk factors, including prolonged CKD and HD duration high serum creatinine and elevated calcium phosphorus (Ca × P) product, all of which correlate strongly with PH prevalence and severity. Our findings suggest that these factors contribute to vascular changes that elevate pulmonary pressures, highlighting the importance of routine PH screening in CKD management, particularly in advanced stages and long term HD patients.

The study emphasizes the need for an integrated approach to managing CKD that addresses not only renal function but also cardiovascular and metabolic complications including mineral and bone disorders. Early intervention strategies focused on managing biochemical imbalances and anemia, alongside alternative dialysis approaches, could potentially mitigate PH progression and improve patient outcomes. Future research with larger and more diverse CKD populations as well as longitudinal studies, is essential to confirm these associations and to explore additional mechanisms driving PH in CKD patients.

In conclusion as PH significantly impacts morbidity and mortality in CKD patients, especially those undergoing HD, targeted interventions and vigilant monitoring for PH can play a crucial role in enhancing the quality of life and reducing cardiovascular risk in this vulnerable population.

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