Section: General Medicine

Original Research Article

INCIDENCE OF PULMONARY HYPERTENSION IN CHRONIC KIDNEY DISEASE ON MAINTENANCE HAEMODIALYSIS

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ABSTRACT

Background: Chronic kidney disease (CKD) carries a substantial cardiovascular burden, and pulmonary hypertension (PH) is an important yet frequently under-recognised complication in patients on maintenance haemodialysis. Echocardiography provides a reliable non-invasive method for its assessment. The objective is to estimate the incidence of PH among CKD patients on maintenance haemodialysis and to evaluate its association with clinical, laboratory and echocardiographic parameters.

Materials and Methods: This hospital-based observational study was conducted in the Department of Medicine, Katihar Medical College and Hospital, over eighteen months (March 2024–August 2025). One hundred CKD patients with an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m² were enrolled by consecutive sampling. PH was assessed by two-dimensional echocardiography, systolic pulmonary artery pressure being derived from the modified Bernoulli equation, and patients were grouped by the presence or absence of PH. Data were analysed in SPSS version 26.0 using Student's t-test and the chi-square test, with $P<0.05$ considered significant.

Results: PH was present in 50 of 100 patients (50%). Advanced renal disease predominated, with CKD stage 4 in 54% and serum creatinine of 7 mg/dL or more in 35%. Patients with PH showed a higher mean serum creatinine (6.09 versus 5.75 mg/dL) and a lower mean eGFR (33.06 versus 36.55 mL/min/1.73 m²). Left ventricular hypertrophy was present in 67% overall and was more frequent among patients with PH (70%), and mitral regurgitation was seen in 35%. Higher creatinine, lower eGFR, left ventricular hypertrophy and valvular abnormalities were more common in the PH group.

Conclusion: PH is highly prevalent among CKD patients on maintenance haemodialysis and is associated with worsening renal function, anaemia, left ventricular hypertrophy and valvular changes. Routine echocardiographic screening enables early detection and timely management, which may reduce cardiovascular morbidity and mortality.

Keywords: Chronic kidney disease, Pulmonary hypertension, Renal dialysis, Echocardiography, Left ventricular hypertrophy.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive and largely irreversible loss of renal function persisting for more than three months, characterised by structural or functional renal abnormalities with or without a reduced glomerular filtration rate. It is a major global health problem, and cardiovascular disease remains the leading cause of death in this population.[1]

Among the cardiovascular complications of CKD, pulmonary hypertension (PH) is increasingly

recognised as a frequent and clinically important, but often under-diagnosed, condition, particularly in patients receiving maintenance haemodialysis.[2] PH is defined by an abnormal elevation of pulmonary arterial pressure and, in CKD, is classified under Group 5 of the World Health Organization classification, in which multiple and often unclear mechanisms operate.[3]

The pathogenesis of PH in CKD is multifactorial. Chronic volume overload, persistent anaemia, endothelial dysfunction, vascular calcification, arteriovenous fistula–related high cardiac output, left ventricular diastolic dysfunction, chronic inflammation and retention of uraemic toxins all contribute to increased pulmonary vascular resistance and pressure.[2,4] The resulting disease reduces exercise tolerance and causes dyspnoea, right ventricular dysfunction, recurrent hospitalisation and increased mortality.[5] Several of these interrelated vascular and haemodynamic mechanisms are summarised in [Figure 1].

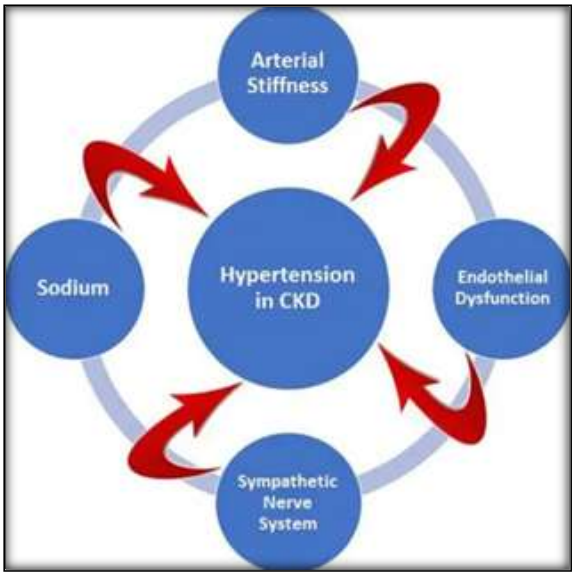


Figure 1: Interrelated mechanisms contributing to vascular dysfunction and raised pressure in chronic kidney disease, including arterial stiffness, sodium retention, endothelial dysfunction and sympathetic overactivity.

Transthoracic echocardiography is the most widely used non-invasive tool for screening and for estimating pulmonary artery pressure, left ventricular function and associated cardiac abnormalities, although right heart catheterisation remains the diagnostic gold standard.[6,7] Early recognition is important, because optimisation of fluid status, correction of anaemia, treatment of cardiac dysfunction and appropriate dialysis planning can improve outcomes.

CKD carries one of the highest disease burdens in India, affecting an estimated 10–15% of adults, driven by diabetes mellitus, hypertension and late presentation.[8,9] Despite its clinical importance, PH remains under-recognised in CKD, especially in resource-limited settings, and data from eastern India are scarce. The present study was therefore undertaken to estimate the incidence of PH among CKD patients on maintenance haemodialysis and to examine its association with clinical, laboratory and echocardiographic parameters.

MATERIALS AND METHODS

Study design and setting: This was a hospital-based observational study conducted in the Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar, over a period of eighteen months from March 2024 to August 2025. The study was designed and reported in accordance with the STROBE statement for observational studies.

Study population and sampling: Patients diagnosed with CKD attending the inpatient, outpatient and intensive care units of the Department of Medicine formed the study population. A total of 100 patients were enrolled by consecutive sampling according to the eligibility criteria. CKD was diagnosed on the basis of an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m², elevated serum creatinine, abnormal urinalysis and ultrasonographic evidence of chronic renal changes.

Eligibility criteria:

Inclusion criteria

Patients diagnosed with CKD; those on haemodialysis or conservative management; an eGFR below 60 mL/min/1.73 m²; all age groups; and those who provided written informed consent.

Exclusion criteria: chronic obstructive pulmonary disease; parenchymal lung disease; chest-wall disease; previous pulmonary hypertension; previous pulmonary embolism; smokers with more than five pack-years; collagen vascular disease; a left ventricular ejection fraction below 50%; significant mitral or aortic valve disease; and acute kidney injury of any cause.

Study procedure: After consent, detailed demographic data, clinical history including duration of CKD and dialysis status, comorbidities and physical examination findings were recorded in a pre-designed proforma. Routine investigations including serum creatinine, serum albumin, haemoglobin, serum calcium, blood urea and urinalysis were performed in the central laboratory using standard methods. A chest radiograph (postero-anterior view) and ultrasonography of the kidney, ureter and bladder were obtained. All patients underwent two-dimensional echocardiography performed by experienced cardiologists to assess pulmonary artery pressure, left ventricular ejection fraction, cardiac chamber dimensions and valvular abnormalities. Systolic pulmonary artery pressure was estimated using the modified Bernoulli equation (systolic pulmonary artery pressure = 4 × [tricuspid regurgitant jet velocity]² + estimated right atrial pressure). Patients were classified into two groups, CKD with PH and CKD without PH. All patients were on

maintenance haemodialysis or conservative management; the principles underlying haemodialysis are shown in [Figure 2].

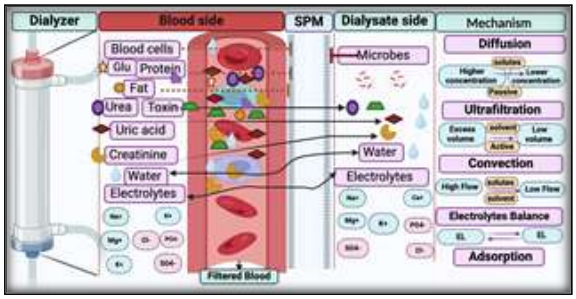


Figure 2: Principles of haemodialysis across the semipermeable dialyzer membrane, showing removal of solutes and fluid by diffusion, ultrafiltration and convection with maintenance of electrolyte balance.

Outcomes: The primary outcome was the prevalence of PH among CKD patients. Secondary outcomes were the associations of PH with demographic variables, laboratory parameters, duration and stage of CKD, and cardiac structural and functional abnormalities.

Statistical analysis: Data were entered in Microsoft Excel and analysed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency and percentage. Student's t-test, the chi-square test, Fisher's exact test, correlation analysis, odds ratios with 95% confidence intervals and multivariate logistic regression were applied as appropriate. A P value below 0.05 was considered statistically significant.

Ethical considerations: The study was conducted after obtaining approval from the Institutional Ethics Committee of Katihar Medical College and Hospital. Written informed consent was obtained from all participants. Participation was voluntary, confidentiality was maintained and data were coded and securely stored. No additional risk was imposed on patients, as all investigations formed part of routine clinical care.

RESULTS

One hundred CKD patients on maintenance haemodialysis were studied. Pulmonary hypertension was present in 50 patients (50%), as reflected by a systolic pulmonary artery pressure of 25 mmHg or more.

The age and sex distribution of the study population is shown in [Table 1]. Elderly patients predominated, with 34% aged more than 75 years and 19% in the 56–65 year group. There was a mild female preponderance, with 58 women (58%) and 42 men (42%).

The clinical, laboratory and renal profile is presented in [Table 2]. A long duration of CKD was common, the largest groups being 5–5.9 years (23%) and 1–1.9 years (22%). Advanced renal impairment predominated: serum creatinine was 7 mg/dL or more in 35% and between 5 and 6.9 mg/dL in a further 34%, while 40% had an eGFR below 30 mL/min/1.73 m². CKD stage 4 was the commonest stage (54%), followed by stage 5 (46%).

Echocardiographic findings are summarised in [Table 3]. Half of the patients (50%) had a pulmonary artery pressure below 25 mmHg, while 28% were in the 40–60 mmHg range and 22% in the 25–39.9 mmHg range. Left ventricular systolic function was largely preserved, 75% having an ejection fraction of 55% or more. Left ventricular hypertrophy was present in 67% of patients and mitral regurgitation in 35%.

The comparison of parameters between patients with and without PH is shown in [Table 4]. The mean age was 48.34 ± 13.57 years in the PH group and 49.18 ± 12.68 years in the non-PH group. Patients with PH had a higher mean serum creatinine (6.09 versus 5.75 mg/dL) and a lower mean eGFR (33.06 versus 36.55 mL/min/1.73 m²), indicating more severe renal dysfunction. Left ventricular hypertrophy was more frequent in the PH group (70%). Overall, higher creatinine, lower eGFR, the presence of left ventricular hypertrophy and valvular abnormalities were more common in patients with PH, indicating an association between PH and both disease severity and cardiac involvement.

Table 1: Age and sex distribution of the study population (n = 100)

Variable	No. of patients (n)	Percentage (%)
Age group (years)		
26–35	12	12.0
36–45	12	12.0
46–55	10	10.0
56–65	19	19.0
66–75	13	13.0
>75	34	34.0
Sex		
Female	58	58.0
Male	42	42.0
Total	100	100.0

Table 2: Clinical, laboratory and renal profile of the study population (n = 100)

Variable	No. of patients (n)	Percentage (%)
Duration of CKD (years)		
0–0.9	8	8.0

1–1.9	22	22.0
2–2.9	16	16.0
3–3.9	18	18.0
4–4.9	13	13.0
5–5.9	23	23.0
Serum creatinine (mg/dL)		
0–2.9	7	7.0
3–4.9	24	24.0
5–6.9	34	34.0
≥7	35	35.0
eGFR (mL/min/1.73 m²)		
0–14.9	12	12.0
15–29.9	28	28.0
30–44.9	27	27.0
45–59.9	33	33.0
CKD stage		
Stage 4	54	54.0
Stage 5	46	46.0

CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Table 3: Echocardiographic parameters of the study population (n = 100)

Variable	No. of patients (n)	Percentage (%)
Pulmonary artery pressure (mmHg)		
0–24.9	50	50.0
25–39.9	22	22.0
40–60	28	28.0
LVEF (%)		
50–54.9	25	25.0
55–59.9	37	37.0
60–64.9	38	38.0
Left ventricular hypertrophy		
Present	67	67.0
Absent	33	33.0
Mitral regurgitation		
Present	35	35.0
Absent	65	65.0

LVEF, left ventricular ejection fraction.

Table 4: Comparison of clinical, laboratory and echocardiographic parameters between CKD patients with and without pulmonary hypertension

Variable	Mean difference	t value	df	P value
Age (years)	0.84	0.320	98	<0.001
eGFR (mL/min/1.73 m²)	3.49	1.257	98	<0.001
Serum creatinine (mg/dL)	−0.34	−0.902	98	<0.001
Haemoglobin (g/dL)	0.12	0.409	98	<0.001
Calcium (mEq/L)	0.06	0.495	98	<0.001
LVEF (%)	0.87	1.079	98	<0.001

df, degrees of freedom; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction. Values as reported by the investigators.

DISCUSSION

In this study of 100 CKD patients on maintenance haemodialysis, pulmonary hypertension was present in half of the cohort. This high burden is consistent with the recognition of PH as a common and clinically significant cardiovascular complication of advanced CKD, and it underscores the value of routine echocardiographic screening in this group.

Our prevalence of 50% is comparable with that reported in several haemodialysis cohorts. Nagaraju et al. reported a prevalence of 54% among maintenance haemodialysis patients,[10] Ahmed et al. 58.4%,[11] and Nithiya et al. 59.6% among those on dialysis.[12] Higher and lower estimates have also been described—70% by Khedhiri et al. and 30% by Behzadnia et al.—reflecting differences in sample size, dialysis vintage and the echocardiographic thresholds used to define PH.[13,14]

Renal dysfunction was closely linked with PH in our cohort. Patients with PH had higher serum creatinine and lower eGFR, and advanced CKD predominated overall. A similar relationship between worsening renal function, advanced CKD stage and PH was reported by Ravi et al., who found increasing age, longer disease duration, advanced stage, higher creatinine and anaemia to be significantly associated with pulmonary artery hypertension.[15] These findings support the role of chronic volume overload and progressive vascular dysfunction in the pathogenesis of PH as renal function declines.

Cardiac structural and functional abnormalities were prominent in our patients, with left ventricular hypertrophy in 67% and mitral regurgitation in 35%, both more frequent in the PH group. Comparable

associations between PH and left ventricular hypertrophy, reduced ejection fraction and valvular abnormalities have been described by Khemchandani et al.,[16] Rroji et al.,[17] and Nagaraju et al., the last reporting mitral regurgitation as the echocardiographic finding most strongly associated with PH.[10] Left atrial enlargement and left ventricular diastolic dysfunction raise pulmonary venous pressure and contribute to a post-capillary component of PH, which is common in this population.

Beyond its prevalence, PH is an important prognostic marker in CKD. A meta-analysis by Tang et al. found PH to be significantly associated with increased all-cause and cardiovascular mortality across the spectrum of CKD and end-stage renal disease, with the risk being highest among dialysis-dependent patients.[18] This reinforces the importance of the early detection that echocardiographic screening allows, and of addressing modifiable contributors such as anaemia, volume overload and mineral bone disease.

The mechanisms underlying PH in CKD are interrelated. Persistent volume overload increases pulmonary blood flow and vascular stress; anaemia produces a high-output state that exposes the pulmonary vasculature to prolonged shear stress; endothelial dysfunction from uraemic toxins and chronic inflammation shifts the balance towards vasoconstriction and remodelling; and arteriovenous access further augments cardiac output. Anaemia is a particularly important contributor, and its multifactorial pathogenesis in CKD is outlined in [Figure 3]. Together these processes lead to pulmonary vascular remodelling, sustained pressure elevation and, ultimately, right ventricular strain.

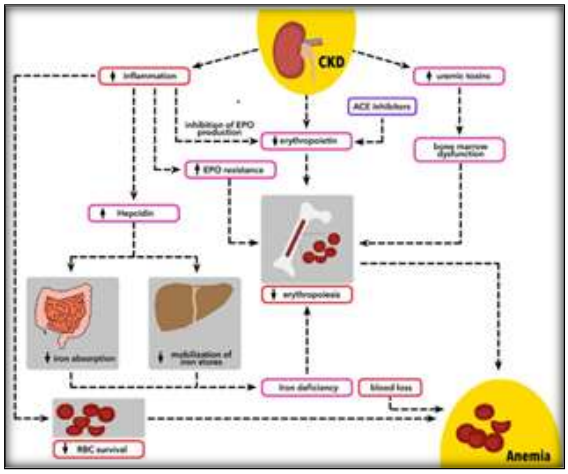


Figure 3: Pathogenesis of anaemia in chronic kidney disease, involving reduced erythropoietin production, uraemic toxins, inflammation, raised hepcidin, impaired iron absorption and reduced red-cell survival.

Limitations: This study has several limitations. It was a single-centre observational study with a modest sample size, which limits generalisability. PH was diagnosed by echocardiography rather than by right heart catheterisation, the gold standard, so pressure estimates may be affected by volume status and dialysis-related haemodynamic fluctuations. The cross-sectional design precludes inference about causality or long-term outcomes. Larger, multicentre and longitudinal studies incorporating right heart catheterisation are needed to confirm these associations and to evaluate whether correcting modifiable risk factors reduces the burden of PH.

CONCLUSION

Pulmonary hypertension is highly prevalent among CKD patients on maintenance haemodialysis and is closely associated with worsening renal function, anaemia, left ventricular hypertrophy and valvular abnormalities. Echocardiographic evaluation plays a key role in early detection. Routine screening of high-risk patients, optimisation of dialysis, correction of anaemia and appropriate management of cardiac abnormalities may help reduce cardiovascular morbidity and mortality and improve long-term outcomes in this vulnerable population.

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