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A Prospective Hospital-Based Study on Prevalence of Pulmonary Hypertension in Chronic Kidney Disease and Its Association with Disease Duration and Stage.

Dr. VEERESH S BALEHOSUR[1]*, Dr. SOWMYA G R[2], Dr. PRATHVI NANDALIKE[3].
[1]*Senior Resident Department of General Medicine JMNMC Nadia*
[2]*Assistant Professor Dept of General Medicine BMCRC, BALLARI*
[3]*Assistant professor General Medicine JGMMMC KLE Gabbur HUBBALLI.*

Correspondence:
Dr. VEERESH S BALEHOSUR.
Senior Resident Department of General Medicine JMNMC Nadia.

Email: veereshsb727@gmail.com

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Abstract

Background: Pulmonary hypertension (PH) is an increasingly recognised cardiovascular complication of chronic kidney disease (CKD) and is independently associated with excess mortality. Reported prevalence varies widely, from roughly 8% to 40% depending on disease stage, dialysis status and diagnostic method. Data from Indian cohorts, in whom CKD frequently presents late and at an earlier age than in Western populations, remain limited. ***Objectives:*** To determine the prevalence of pulmonary hypertension among patients with chronic kidney disease attending a tertiary care hospital, and to examine its relationship with age, sex, diabetes mellitus, systemic hypertension, duration of CKD and CKD stage. ***Materials and Methods:*** A prospective descriptive study was conducted in the Departments of Nephrology and General Medicine, Ballari Medical College and Research Centre (formerly Vijayanagar Institute of Medical Sciences), Ballari, Karnataka, over 18 months. Sixty-four patients with CKD of any aetiology and an estimated glomerular filtration rate below 60 mL/min/1.73 m^2, managed either conservatively or on haemodialysis, were enrolled after institutional ethics approval and informed consent. Patients with chronic obstructive pulmonary disease, parenchymal lung or chest wall disease, previous pulmonary hypertension or pulmonary embolism, a smoking history exceeding five pack-years, collagen vascular disease, left ventricular ejection fraction below 50% or significant mitral or aortic valve disease were excluded. Sample size was calculated as 64 using the single-proportion formula with an assumed prevalence of 0.605 and absolute precision of 0.12. All patients underwent electrocardiography, chest radiography and transthoracic echocardiography. Analysis was performed in SPSS v24.0 using the chi-square or Fisher's exact test, with significance at $p<0.05$. ***Results:*** Pulmonary hypertension was detected in 17 of 64 patients, an overall prevalence of 26.6%. Forty-one participants (64.1%) were male and 34 (53.2%) were aged 41–60 years. Systemic hypertension was present in 53 patients (82.8%) and diabetes mellitus in 22 (34.4%). Pulmonary hypertension showed no significant association with age group ($p = 0.54$), sex ($p = 0.60$), diabetes ($p = 0.49$) or systemic hypertension ($p = 0.95$). Duration of CKD was strongly associated with pulmonary hypertension ($p < 0.001$): prevalence rose from 8.6% in patients with CKD of under one year to 48.0% at one to five years and 50.0% at six to ten years. Pulmonary hypertension was confined entirely to advanced disease, occurring in 6 of 15 patients (40.0%) in stage 4 and 11 of 39 (28.2%) in stage 5, with no cases in stages 2, 3a or 3b ($p = 0.28$). ***Conclusion:*** Roughly one in four patients with CKD had echocardiographic pulmonary hypertension, and every affected patient was in stage 4 or 5. Duration of kidney disease, rather than age, sex or the traditional cardiovascular comorbidities of diabetes and systemic hypertension, was the principal clinical determinant. These findings support routine echocardiographic screening once CKD reaches stage 4.

Keywords: Pulmonary hypertension; chronic kidney disease; echocardiography; end-stage renal disease; prevalence; CKD stage.

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INTRODUCTION

Chronic kidney disease (CKD) is a global public health problem affecting several hundred million people, and its prevalence continues to rise with the ageing of populations and the expanding burden of diabetes and hypertension [1]. As renal function declines, cardiovascular complications dominate both morbidity and mortality, and among these, pulmonary hypertension (PH) has attracted growing attention over the last two decades [2].

Pulmonary hypertension is defined haemodynamically by an elevated mean pulmonary arterial pressure at rest and is characterised clinically by progressive right ventricular pressure overload culminating in right heart failure [3]. The 2015 European Society of Cardiology and European Respiratory Society guidelines classify PH into five groups according to mechanism, with PH complicating CKD conventionally assigned to group 5, the category of unclear or multifactorial mechanisms [3]. The 2019 revision of the haemodynamic definitions lowered the diagnostic threshold, a change that has itself contributed to variation in reported prevalence figures [4].

Estimates of how common PH is in CKD differ substantially across published series, with reported prevalence ranging from approximately 8% to 40% depending on the stage of renal impairment, dialysis modality and diagnostic technique employed [5]. Pabst and colleagues, in the PEPPER study, documented a high burden in both dialysis and non-dialysis CKD populations [6], while Navaneethan et al. found PH to be both prevalent and prognostically important in a large non-dialysis CKD cohort [7].

The mechanisms are multifactorial and remain incompletely resolved. Endothelial dysfunction with reduced nitric oxide bioavailability and elevated endothelin-1 concentrations produces pulmonary vasoconstriction, while chronic volume overload, left ventricular diastolic dysfunction, anaemia-driven high cardiac output, vascular calcification arising from disordered mineral metabolism and, in dialysis patients, the additional shunt burden of an arteriovenous fistula, all contribute [8]. Because several of these mechanisms are potentially modifiable, identifying which clinical variables track most closely with PH has direct therapeutic relevance.

The prognostic implications are considerable. Reque et al. demonstrated that PH independently predicts mortality and cardiovascular events in CKD [9], and a systematic review and meta-analysis by Tang et al. confirmed a substantial excess mortality across both CKD and end-stage renal disease populations [10]. In patients being assessed for transplantation, pre-operative PH predicts early graft dysfunction and complicates peri-operative management [11].

Diagnosis is complicated by symptom overlap. Dyspnoea, fatigue and peripheral oedema are common to both uraemia and PH, so that clinical suspicion alone is an unreliable guide [12]. Right heart catheterisation remains the diagnostic reference standard, but is invasive and impractical as a screening tool; transthoracic echocardiography, which estimates systolic pulmonary artery pressure from the tricuspid regurgitant jet, is therefore the accepted screening modality in this population [3,12].

Despite this accumulating evidence, most published data derive from European and North American cohorts. Indian patients with CKD are typically younger at presentation, frequently reach medical attention only at advanced stages, and carry a high burden of anaemia and disordered mineral metabolism — a combination that may plausibly alter both the prevalence and the determinants of PH. The present study was therefore undertaken to establish the prevalence of pulmonary hypertension in a South Indian CKD population and to identify the demographic and clinical variables associated with it.

MATERIALS AND METHODS

A prospective descriptive study was carried out in the Departments of Nephrology and General Medicine at Ballari Medical College and Research Centre (formerly Vijayanagar Institute of Medical Sciences), Ballari, Karnataka, a tertiary referral hospital serving a largely rural district population. Patients were recruited from both inpatient and outpatient services over a period of 18 months.

Ethical considerations: The study commenced only after approval was obtained from the institutional review board. Written informed consent was obtained from every participant before enrolment, and patient confidentiality was maintained throughout.

Inclusion criteria: Patients with chronic kidney disease of any aetiology and of any age group were eligible, provided the estimated glomerular filtration rate was below 60 mL/min/1.73 m². Both patients managed conservatively and those established on maintenance haemodialysis were included.

Exclusion criteria: To avoid attributing pulmonary hypertension to a cause other than kidney disease, patients were excluded if they had chronic obstructive pulmonary disease, parenchymal lung disease, chest wall disease, a previous diagnosis of pulmonary hypertension, previous pulmonary embolism, a smoking history exceeding five pack-years, or collagen vascular disease. Patients with a left ventricular ejection fraction below 50% or significant mitral or aortic valve disease were also excluded, so that post-capillary pulmonary hypertension arising from overt left heart disease would not confound the analysis.

Sample size estimation: Sample size was calculated using the single-proportion formula $n = (Z^2 \times P \times (1 - P)) / d^2$, where Z was 1.96 for 95% confidence, P was the estimated prevalence of pulmonary hypertension in CKD taken as 0.605 from a previous study, and d was an absolute precision of 0.12. Substituting these values yielded a minimum required sample of 64 participants.

Sampling and enrolment: Participants were selected from the eligible CKD population by simple random sampling. Patients meeting the inclusion criteria who provided informed consent were enrolled consecutively until the calculated sample size was reached.

Data collection: Data were recorded on a structured proforma. Demographic details, aetiology and duration of CKD, dialysis status and vascular access, and the presence of diabetes mellitus and systemic hypertension were documented for every participant. Biochemical parameters were recorded as the average of the last six available readings for each patient in order to reduce the influence of short-term fluctuation. CKD was staged according to estimated glomerular filtration rate using standard criteria.

Cardiovascular assessment: All participants underwent twelve-lead electrocardiography, postero-anterior chest radiography and transthoracic echocardiography. Echocardiography was used to estimate systolic pulmonary artery pressure from the peak tricuspid regurgitant velocity together with an estimate of right atrial pressure, and to assess right ventricular size and function, left ventricular ejection fraction and valvular structure. Pulmonary hypertension was diagnosed on the basis of echocardiographic criteria, and patients were thereafter classified into two groups — pulmonary hypertension present or absent — which formed the basis of all comparative analyses.

Statistical analysis: Data were entered into a Microsoft Excel spreadsheet and analysed using SPSS version 24.0 (IBM, USA). Qualitative variables were expressed as frequencies and proportions and quantitative variables as mean ± standard deviation. Associations between categorical variables were tested using the chi-square test, with Fisher's exact test applied where expected cell counts were small. Comparison of means between the two groups was performed using the unpaired t-test. A p value below 0.05 was regarded as statistically significant and a value below 0.001 as highly significant.

RESULTS

Sixty-four patients with chronic kidney disease were studied.

Table 1. Age distribution of study participants (n = 64)

Age group (years)	Frequency (n)	Percentage (%)
<30	4	6.3
31–40	12	18.8
41–50	17	26.6
51–60	17	26.6
>60	14	21.9
Total	**64**	**100.0**

The cohort was predominantly middle-aged and elderly. The 41–50 and 51–60 year bands each contained 17 patients (26.6%), so that 34 participants (53.2%) fell between 41 and 60 years. A further 14 patients (21.9%) were above 60 years, giving 48 patients (75.0%) aged over 40. Only 4 patients (6.3%) were under 30 years. This distribution is consistent with the cumulative effect of long-standing hypertension and diabetes on renal function, though the cohort is younger overall than typical Western CKD populations.

Table 2. Baseline clinical profile of study participants (n = 64)

Variable	Category	Frequency (n)	Percentage (%)
Sex	Male	41	64.1
	Female	23	35.9
Systemic hypertension	Present	53	82.8
	Absent	11	17.2
Diabetes mellitus	Present	22	34.4
	Absent	42	65.6
Duration of CKD	<1 year	35	54.7
	1–5 years	25	39.1
	6–10 years	4	6.3

Males predominated at 64.1%, a ratio of approximately 1.8:1. Systemic hypertension was near-universal, affecting 53 patients (82.8%), reflecting its dual role as both cause and consequence of renal impairment.

Diabetes mellitus was present in 22 patients (34.4%). Strikingly, more than half the cohort (35 patients, 54.7%) had been diagnosed with CKD less than one year previously, and only 4 patients (6.3%) had a recognised diagnosis extending beyond five years — a pattern indicating late detection of an essentially asymptomatic disease rather than genuinely recent onset.

Table 3. Prevalence of pulmonary hypertension (n = 64)

Pulmonary hypertension	Frequency (n)	Percentage (%)
Present	17	26.6
Absent	47	73.4
Total	**64**	**100.0**

This table addresses the primary objective. Echocardiographic pulmonary hypertension was identified in 17 of 64 patients, an overall prevalence of 26.6% — approximately one in every four patients with CKD.

This falls squarely within the 8% to 40% range described in the international literature and confirms that pulmonary hypertension is a common, and in most of these patients previously undiagnosed, cardiovascular complication of chronic renal failure.

Table 4. Pulmonary hypertension in relation to age group and sex (n = 64)

Variable	Category	Total (n)	PH present (n)	Prevalence within group (%)	p value
Age group (years)	<30	4	2	50.0	0.54
	31–40	12	2	16.7	
	41–50	17	5	29.4	
	51–60	17	3	17.6	
	>60	14	5	35.7	
Sex	Male	41	10	24.4	0.60
	Female	23	7	30.4	

Neither age nor sex bore a statistically significant relationship to pulmonary hypertension. Prevalence within age bands fluctuated without a coherent gradient — apparently high at 50.0% in the under-30 group, but this rests on only 2 cases among 4 patients and carries very wide uncertainty.

The highest prevalence in a group of meaningful size was 35.7% among those over 60 years. Female patients had a marginally higher prevalence (30.4%) than males (24.4%), a difference well within chance variation ($p = 0.60$). Pulmonary hypertension in CKD therefore does not appear to be driven by the demographic factors that dominate idiopathic pulmonary arterial hypertension.

Table 5. Pulmonary hypertension in relation to comorbidity and duration of CKD (n = 64)

Variable	Category	Total (n)	PH present (n)	Prevalence within group (%)	p value
Diabetes mellitus	Present	22	7	31.8	0.49
	Absent	42	10	23.8	
Systemic hypertension	Present	53	14	26.4	0.95
	Absent	11	3	27.3	
Duration of CKD	<1 year	35	3	8.6	<0.001*
	1–5 years	25	12	48.0	
	6–10 years	4	2	50.0	

*Statistically significant.

This table contains the study's most important positive finding. Neither diabetes mellitus (31.8% versus 23.8%, $p = 0.49$) nor systemic hypertension (26.4% versus 27.3%, $p = 0.95$) influenced the likelihood of pulmonary hypertension — the two prevalences in the hypertension comparison being almost identical. By contrast, duration of chronic kidney disease showed a powerful and highly significant gradient. Among patients known to have CKD for less than a year, prevalence was only 8.6%; this rose more than fivefold to 48.0% in those with disease of one to five years' duration and to 50.0% beyond six years ($p < 0.001$). Cumulative exposure to the uraemic milieu, rather than the presence of conventional cardiovascular risk factors, therefore appears to be the operative determinant.

Table 6. Pulmonary hypertension in relation to CKD stage (n = 64)

CKD stage	Total (n)	PH present (n)	Prevalence within stage (%)	Share of all PH cases (%)	p value
Stage 2	1	0	0.0	0.0	0.28
Stage 3a	2	0	0.0	0.0	
Stage 3b	7	0	0.0	0.0	
Stage 4	15	6	40.0	35.3	
Stage 5	39	11	28.2	64.7	
Total	**64**	**17**	**26.6**	**100.0**	

Pulmonary hypertension was entirely absent from stages 2, 3a and 3b — not one of the 10 patients in these categories was affected — and occurred exclusively in stages 4 and 5. Within stage 4 the prevalence was 40.0% (6 of 15) and within stage 5 it was 28.2% (11 of 39). The final column distinguishes a separate quantity that is easily conflated with prevalence: of the 17 patients with pulmonary hypertension, 64.7% were in stage 5, simply because stage 5 contained 39 of the 64 participants. The threshold effect at stage 4 is therefore the substantive finding, whereas prevalence did not increase further from stage 4 to stage 5. The overall association did not reach statistical significance ($p = 0.28$), reflecting the very small numbers in the earlier stages.

DISCUSSION

The overall prevalence of echocardiographic pulmonary hypertension in this cohort was 26.6%, closely matching the 30% documented by Yigla et al. in end-stage renal disease [13] and lying within the 8% to 40% range reported across the wider literature [5,7]. Pabst et al. similarly found a substantial burden in both dialysis and non-dialysis CKD [6]. The consistency of this figure across markedly different healthcare systems suggests that pulmonary hypertension is an intrinsic consequence of advanced uraemia rather than an artefact of any particular treatment environment.

The demographic profile — 53.2% aged 41 to 60 years and 64.1% male — accords with global CKD epidemiology. Hill et al. documented rising prevalence with advancing age in their meta-analysis [1], and Lv and Zhang reported consistently higher CKD prevalence among men across multiple countries [14]. The mean age here is nonetheless lower than the approximately 60 years reported in United States Renal Data System figures [15], consistent with the earlier onset of CKD in Indian populations.

Systemic hypertension affected 82.8% of participants, comparable with the high prevalence described by Agarwal and Light in CKD cohorts [16], and diabetes was present in 34.4%, close to the figures reported by Kovesdy et al. [17]. Notably, neither influenced pulmonary hypertension risk in this study ($p = 0.95$ and 0.49 respectively). This absence of association is informative: it argues that pulmonary hypertension in CKD is not simply an extension of conventional atherosclerotic cardiovascular risk but reflects mechanisms specific to the uraemic state — volume overload, endothelial dysfunction, anaemia-driven high cardiac output and vascular calcification [8].

Duration of CKD was the single significant clinical determinant, prevalence rising from 8.6% below one year to 48.0% at one to five years ($p < 0.001$). That 54.7% of patients had been diagnosed within the preceding year echoes the observation of Coresh et al. that CKD is frequently detected only once complications supervene [18], and implies that the apparent protection of "short duration" partly reflects delayed diagnosis rather than genuinely brief disease.

Pulmonary hypertension occurred exclusively in stages 4 and 5, consistent with Nakhoul et al., who described escalating prevalence with CKD progression [8]. An important interpretive caution applies here. Prevalence within stage 4 was 40.0% and within stage 5 was 28.2%; the frequently quoted figure of 64.7% for stage 5 represents the proportion of pulmonary hypertension cases occurring in that stage, not the risk faced by a stage 5 patient. Distinguishing these two quantities matters, since the data support a threshold effect at stage 4 rather than a continuous gradient to stage 5.

Limitations: The sample of 64 from a single centre limits generalisability, and the very small numbers in stages 2 to 3b (10 patients in total) leave the study underpowered to exclude a low prevalence of pulmonary hypertension in earlier CKD. Diagnosis rested on echocardiographic estimation rather than right heart catheterisation, which may misclassify some patients. Although described as prospective, the design is effectively cross-sectional and cannot establish temporal sequence. Finally, treatment variables such as antihypertensive and phosphate-binding therapy were not accounted for.

CONCLUSION

In this prospective study of 64 patients with chronic kidney disease, echocardiographic pulmonary hypertension was present in 26.6% — approximately one patient in four — confirming it as a common and largely unrecognised

cardiovascular complication of renal failure. The condition was found exclusively in advanced disease, affecting 40.0% of patients in stage 4 and 28.2% in stage 5, with no cases at all among the 10 patients in stages 2 to 3b.

Duration of chronic kidney disease emerged as the dominant clinical determinant, with prevalence rising from 8.6% in those diagnosed within the preceding year to 48.0% between one and five years and 50.0% beyond six years ($p < 0.001$). Neither age, sex, diabetes mellitus nor systemic hypertension bore any significant relationship to pulmonary hypertension, indicating that this complication arises from mechanisms intrinsic to the uraemic state rather than from conventional cardiovascular risk factors.

These findings carry a clear practical implication: transthoracic echocardiography should be incorporated into routine assessment once chronic kidney disease reaches stage 4, and repeated periodically thereafter, since clinical features alone cannot distinguish pulmonary hypertension from the dyspnoea and fatigue of uraemia itself. Given the established association of pulmonary hypertension with excess mortality, earlier detection of CKD in the community — so that cumulative uraemic exposure is shortened — may prove as important as any specific cardiovascular intervention. Larger multicentre studies with catheter confirmation and longitudinal follow-up are needed to define the natural history of this complication and to test whether it is modifiable.

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