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TO STUDY THE PREVALENCE OF PULMONARY HYPERTENSION AMONGST CKD PATIENTS IN BUNDELKHAND REGION

General Medicine

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ABSTRACT

Introduction: Pulmonary hypertension (PH) is a recently recognized complication of chronic kidney disease (CKD), especially in end-stage renal disease. It has prevalence estimates of 30%–50% and is an independent predictor of increased mortality in CKD patients.

Aim: The aim of this study the clinical and diagnostic profile of pulmonary hypertension in patients of CKD treated with different strategies of management i.e. (Conservative, haemodialysis and CAPD).

Material And Methods: Our study is a prospective observational study & will be conducted in M.L.B. Medical College, Jhansi, including a minimum sample size of 100 Chronic Kidney Disease (CKD) patients who are coming to Departments of General Medicine, M.L.B Medical College, Jhansi (U.P.) in our outdoor and indoor during the period from March. 2018 to Oct.2019 as per our selection criteria.

Result: Out of the total 120 patients included in the study, 43 (35.83%) patients had PH. Majority, 21 (48.83%) of CKD patients showed mild PASP followed by 15 (34.88%) patients moderate group and 7 (16.27%) severe PH. There was no effect of age on prevalence of PH. Out of the total 43 patients with PH 31 (72.09%) were males. PH was more common in males than females and statistically significant (p =.022). There was statistically significant association between CKD stages and PH (p <.001). None of the patients in CKD stage II revealed PH, while 1(2.32%), 3(7%), and 39 (90.7%) patients of CKD stage III, IV, and V, respectively, had PH. 20 out of 34 diabetics (58.82%) had PH whereas 26 out of 47 hypertensive's (55.31%) had PH. There was a strong association between diabetes and hypertension with PH (p <.001)

Conclusion: Mortality of CKD Patients with PH was strongly associated with presence of HF, HTN, age>60 years(63.63%), severity of PH (71.42%0, Hb <10gm/dl (50%),BUN>45mg/dl(45.6%), S.creatinine >5mg/dl(44.82%) and Ca x P product >55mg2/dl2 (51.85%) and statistically significant (p<.05)

KEYWORDS

Calcium Phosphate Product, Chronic Kidney Disease, Ejection Fraction, Pulmonary Hypertension

INTRODUCTION

Chronic kidney disease (CKD) describes persistent abnormal kidney function or structure. Chronic kidney disease is common, often unrecognised, expensive to treat and associated with an increase in mortality at all ages when the glomerular filtration rate (GFR) is <60ml/min[1].

Strategies aimed at the early identification and prevention of progression of CKD are required. Hypertension and diabetes are responsible for up to two-thirds of cases. CKD also significantly increases the patient's risk of cardiovascular events, especially when the GFR is <45ml/min. The overall prevalence of CKD in the SEEK India cohort was 17.2% in Indian population. Prevalence of CKD stages 1,2,3,4 & 5 was 7%, 4.3%, 4.3%, 0.8% & 0.8% respectively. The larger population with earlier stages of CKD bears a very high burden of cardiovascular morbidity and mortality despite latest advances in the case of the patients[2].

Pulmonary arterial hypertension (PAH) is a newly recognized cardiovascular complication in patients with renal disease. Pulmonary hypertension (PHT) is a hemodynamic state defined by resting mean pulmonary artery pressure at or above 25 mmHg complicating heart, lung or systemic disease. In a recent review, the prevalence of PHT in ESRD patients was reported to be around 40–50%1[3].

Presence of pulmonary hypertension has been recently suggested to be associated with a high morbidity and mortality in patients of CKD[4]. The risk of pulmonary hypertension appears to be higher among patients with ESKD who are on long term hemodialysis in compared with peritoneal dialysis and non-dialyzed patients. Based on echocardiographic studies, the prevalence of PH estimated to be around 17–56%[5] and is an independent predictor of mortality in such patients[6-7].

The presence of PH was associated with a 38% increased risk of overall mortality and a 23% increased risk of cardiovascular events in CKD patients[8]. Several mechanism have been proposed, the most logical of which is that PH in this population may relate to increased flow through the pulmonary vascular bed from chronic volume overload (from CKD itself) and increased venous return (from AV access). Additionally, CKD, especially end-stage renal disease, by itself has also been proposed to cause pulmonary vascular remodeling and PH. Possible mechanisms that have been suggested include endothelial dysfunction due to increased oxidative stress from uremic toxins, chronic inflammation.

PH-associated reduced capacity of the right heart to maintain adequate left heart filling pressures in the face of intermittent dialytic fluid removal could contribute to the intradialytic myocardial stunning, ischaemia and myocardial fibrosis is considered a major contributor to sudden cardiac death in the HD population.

The associations between CKD, PH, and adverse cardiovascular events have been explored mostly in patients with end-stage renal disease or postrenal transplant, and limited data are available in earlier stages of CKD[9-10]

Understanding the link between earlier stages of renal disease and PH may allow for timely targeted therapy and prevention of disease progression[11]. In Bundelkhand region we have large number of people suffering from renal disease including chronic renal failure, nephrotic syndrome, nephrolithiasis.

There seems to be a significant burden of morbidity and mortality in patients of renal disease including CKD in this region.

Thus we have planned this study as there is sincere need to evaluate

whether prevalence of cardiovascular disease, especially pulmonary hypertension is associated with a greater risk of adverse outcome in patients suffering from CKD in Bundelkhand region.

AIMS AND OBJECTIVES

The aims and objective of this prospective observational study are as follows:-.

1. To study the prevalence of pulmonary hypertension amongst CKD patients in Bundelkhand region.
2. To study the clinical and diagnostic profile of pulmonary hypertension in patients of CKD treated with different strategies of management i.e. (Conservative, haemodialysis and CAPD).
3. To study the correlation between earlier stages of CKD and pulmonary hypertension.
4. To assess the risk of heart failure in patients of CKD with pulmonary hypertension.

MATERIALS AND METHODS

Our study is a prospective observational study & will be conducted in M.L.B. Medical College, Jhansi, including a minimum sample size of 100 Chronic Kidney Disease (CKD) patients who are coming to Departments of General Medicine, M.L.B Medical College, Jhansi (U.P.) in our outdoor and indoor during the period from March. 2018 to Oct.2019 as per our selection criteria.

The patients would be selected on the basis of definition of Chronic kidney disease as per 'NKF KDOQI guidelines' which is follows :

- Kidney damage for >3 months, as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifest by either.
- Pathological abnormalities or marker of kidney damage including abnormalities in the composition of the blood or urine or abnormalities in imaging tests.
- GFR<60 mL/min/1.73 m2 for >3 months, with or without kidney damage.

INCLUSION CRITERIA:

- All CKD patient >12 yrs of age, either on conservative, haemodialysis or continuous ambulatory peritoneal dialysis, will be included.

EXCLUSION CRITERIA:

- Pregnant females Smokers
- Any cardiovascular and pulmonary diseases lead to pulmonary
- hypertension as well as patients with chronic coronary heart disease,
- Previous pulmonary embolism,
- Collagen vascular disease, scleroderma
- Patients < 12 yrs of age.
- HIV
- Volume overload at the time of echocardiography were excluded.

METHODOLOGY:

Once all the criteria will be meet out, a written informed consent will be taken from patients and patients will be enrolled.

- A detailed history will be noted and clinical examination will be done on patients including age; sex; smoking habits; associated comorbidity particularly diabetes mellitus and hypertension; age at time of CKD, etiology of renal failure, duration of dialysis treatment, and access location of AVF.
- Each patient will undergo routine investigations like complete haemogram, serum electrolytes (Na, K, Ca, PO4, UA), renal function tests (BUN, serum creatinine), liver function tests (Total /direct bilirubin, AST, ALT, ALP), urine routine & microscopy, and some specific investigations like arterial blood gas (ABG), Chest X-Ray, ECG, USG abdomen, serum parathyroid level, iron profile, autoimmune work up like ANA, Pro BNP (as and when indicated).
- PFT will be done by Pulmonary Function Equipment (COSMED, REF C09072-02-99, S/N 2015051137) in Department of T.B. and Chest Diseases, MLB Medical College, Jhansi.
- Transthoracic Doppler echocardiography: Every patient will be subjected to complete two-dimensional and Doppler echocardiography study by 2D echo machine (Hitachi – Aloka-SN:M-12253LI, Model:Alpha-6) in Medicine Department of MLB Medical College, Jhansi.
- Echocardiography will be planned to assess the pericardium, valvular anatomy and function, left and right side chamber size and cardiac function. Tricuspid regurgitant flow will be identified by color flow Doppler technique and the maximumjet velocity will be measured by continuous wave Doppler without the use of intravenous contrast. Right ventricular systolic pressure will be estimated.
- Pulmonary hypertension (PH) is defined in this study as sPAP ≥ 30 mmHg. PH is classified into three categories
- Mild - 30–50 mmHg
- Moderate- 50–70 mmHg
- Severe - >70 mmHg (using Chemla formula, mean pulmonary arterial pressure (MPAP) =0.61 PASP + 2 mmHg).

- Right ventricle dimension will be measured by M-Mode echo and right ventricular dilation or cor pulmonale would be said to be present when it exceeded the normal range of 0.9–2.6 cm.
- Left ventricular function will be also assessed by using the following parameters:
  - EF (ejection fraction) = measure of how much end-diastolic value is ejected from LV with each contraction (56%–78%)
  - FS (fractional shortening) = it is a percentage change in LV dimension with each LV contraction (28%–44%).
  - LV mass = left ventricular mass (88–224 g).
  - E/A will be assessed
- Non-contrast computed tomography (NCCT) have a significant role in the clinical evaluation and management of patients with PH. The pulmonary parenchymal findings on CT in patients with PH are variable and depend upon the etiology. If needed patients would be subjected to NCCT chest for evaluation of PH.
- Systolic PAP: A tricuspid regurgitation systolic jet will be recorded from the parasternal or apical window with the continuous -wave Doppler echocardiography probe. This assessment included history, physical examination, chest radiograph, chest CT, complete pulmonary function tests, and measurement of arterial blood gases.
- Doppler sonography: AVF flow measurement in hemodialysis patients by Doppler ultrasound will be done.

STATISTICAL ANALYSIS:

The prevalence of PHT will be calculated using the SPSS software. Clinical, hemodynamic, and metabolic variables will be compared between patients with and without PHT with "t" test. Values will be expressed as mean ± Standard deviation (SD) and as percentage for categorial parameters. Differences between groups will be compared with Student's t-test for parametric continuous variables. Chi-square test will be applied for estimating the occurrence of categorical variables. Pearson's correlation coefficient will be used to test the relationship between PAP and other parameters. A P value <0.05 will be used as the threshold of statistical significance.

RESULT

Out of the total 120 patients included in the study, 43 (35.83%) patients had PH. Majority, 21 (48.83%) of CKD patients showed mild PASP followed by 15 (34.88%) patients moderate group and 7 (16.27%) severe PH. There was no effect of age on prevalence of PH. Out of the total 43 patients with PH 31 (72.09%) were males. PH was more common in males than females and statistically significant (p =.022). There was statistically significant association between CKD stages and PH (p <.001). None of the patients in CKD stage II revealed PH, while 1(2.32%), 3(7%), and 39 (90.7%) patients of CKD stage III, IV, and V, respectively, had PH. 20 out of 34 diabetics (58.82%) had PH whereas 26 out of 47 hypertensives (55.31%) had PH. There was a strong association between diabetes and hypertension with PH (p <.001)

Table 1: Correlation of Age with pulmonary hypertension.

Age(Years)	Group	
	PHAbsent	PHPresent
Š20	1	0
21-40	24	9(27.27%)
41-60	33	23(41.07%)
>60	19	11(36.66%)
Total	77	43

Table 2: Distribution of patients according to their Sex.

Sex	PHAbsent	PHPresent	Total
Male	39	31(72.09%)	70
Female	38	12(27.90%)	50
Total	77	43	120

Table 3: Pulmonary hypertension (PH) and etiology of CKD

Etiology	PHPresent(%)	PHAbsent	Total
Diabetes	20(58.82%)	14	34
Hypertension	26(55.31%)	21	47
Undetermined	5(11.90%)	37	42
Obstructiveuropathy	4(33.33%)	8	12
ADPKD	1(25%)	3	4
Total	43		120

Table 4: CKD duration and incidence of pulmonary hypertension CKD duration and incidence of pulmonary hypertension

CKD Duration (inmonths)	PH absent	PH present	TOTAL
<6	47	6(11.32%)	53
6-12	22	20(47.61%)	42
>12	8	17(68%)	25
Total		43	120

Table 5: CKD duration and severity of pulmonary hy pertension

PH(PAPsin mmHg)	CKDdurationinmonths			Total
	<6	6-12	>12	
35-50	4	10	7(33.33%)	21(48.88%)
50-70	2	4	9(60.0%)	15(34.88%)
>70	0	2	5(71.42%)	7(16.27%)
Total	6	16	21	43

Table 6: Clinical and diagnostics profile of pulmonary hypertension in CKD patient treated with different strategies of management.

	PHabsent	PHpresent	Total
Conservative	32	4(11.11%)	36
Hemodialysis	33	34(50.75%)	67
CAPD	12	5(29.41%)	17
Total	77	43	120

Table 7: Severity of pulmonary hypertension in CKD patient treated with different strategies of management.

PH	Conservative	Hemodialysis	CAPD	Total
Mild	3(14.28%)	16(76.19%)	2(9.5%)	21
Moderate	1(6.66%)	13(86.67%)	1(6.66%)	15
Severe	0	5(71.42%)	2(25.56%)	7
Total	4	34	5	43

Table 8: Duration of Hemodialysis (HD) and Pulmonary hypertension

HD duration (inmonths)	PHabsent	PHpresent	Total
<6	20	8(28.57%)	28
6-12	9	10(52.63%)	19
>12	7	16(69.56%)	23
Total	36	34	70

Table 9: Severity of pulmonary hypertension in CKD patients on HD along with duration.

PH	<6	6-12	12	Total
Mild	7(25%)	4(25%)	5(21.73%)	16
Moderate	1(3.57%)	4(25%)	8(34.78%)	13
Severe	0	2(12.52%)	3(13.04%)	5
Total	28	16	23	34

Table 9: Correlation of pulmonary hypertension with AVF in CKD patients.

	PH+	PH–	Total
AVF+	24(61.54%)	15	39
AVF-	10(35.71%)	18	28
Total	34	33	67

Table 10: Biochemical variables and pulmonary hypertension Biochemical variable and pulmonary hypertension

CKDduration (inmonths)	PHabsent(%)	PHpresent(%)	Total	p-value
Hb<10gm/dl	44	40(47.6%)	84	<.001
BUN>45mg/dl	22	40(64.51%)	62	<.05
Sr.Creat.>5mg/dl	27	38(58.46%)	65	<.05
CaxPproduct> 55mg2/dl2	29	37(56.06%)	66	<.05

Table 11: Stages of CKD and pulmonary hypertension.

CKDstages	PHabsent	PHpresent	Total
I-II	0	0	0
III	9	1(2.32%)	10
IV	25	3(7%)	28
V	43	39(90.7%)	82

Table 12: Assess the risk of heart failure in patients of CKD with pulmonary hypertension with CKD duration.

CKDwithPulmonaryhypertension (duration)	HF absent	HF present	Total
<6Months	3	3(12.50%)	6
6-12Months	6	8(33.33%)	16
>12Months	3	13(54.16%)	21
Total	12	24(55.81%)	43

Table 13: Correlation of mortality in presence of Heart Failure in patients of CKD with PH

Ph	Discharged	Expired	Total
HFpresent	11	13(30.23%)	24
HFabsent	17	2(4.65%)	19
Total		15	43

DISCUSSION

PH, a disorder characterized by elevated pulmonary artery pressure, is a progressive disorder complicating heart, lung, or systemic diseases, with increased morbidity and mortality regardless of its etiology[12]. Recently it has been found that PH is a strong independent predictor of morbidity and mortality in HD patients. In present study of 67 HD patients, higher mortality OF 26.47% in patients with PH compared with 6.06% among patients without PH[13]. In an observational study of 58 HD patients, with a mean follow-up of 30 months, patients with PH had mortality of 30.4% compared with 8.5% among patients without PH ($p < 0.03$)[14]. Yigla et al[15], in their cohort of CKD patients, reported significantly lower survival than those without PH with 1 year, 3 years, and 5 years survival rates of 78.6% versus 96.5%, 42.9% versus 78.8%, and 25.2% versus 66.4% respectively ($p = 0.0001$). There are very few Indian studies addressing the prevalence of PH in CKD patients. In patients with ESRD, PH has been recognized to be a frequent condition and appears to be independent from cardiovascular disease prevalence[13]. innovative supplementary Construction Material is formed through this study.

The prevalence of PH in CKD patients in the present study was 35.83% with mean PASP of 38.52 ± 7.32 mmHg. Tarras et al[15] found PH prevalence to be as low as 26.74% and Moniruzzaman et al[16] found it to be as high as 68.6%. In another Indian study Patel et al[17] studied 100 patients (69 males, 31 females) who were on conservative management, HD or continuous ambulatory peritoneal dialysis (CAPD). The prevalence of PH in this cohort was 41% and the highest prevalence was in the HD group (33%). The variability in the prevalence of PH among CKD patients in different studies[16]] can be explained by the difference in the ethnicity of the population studied as well as in the study group, regarding stage of CKD, mode of dialysis (HD vs PD), comorbid conditions such as COPD/CHF and inclusion criteria. Though these studies considered different parameters and are not truly comparable, most concluded that there was high prevalence of PH among CKD patients.

There was no effect of age on prevalence of PH in our study. This result was similar to the study by Mazdeh et al[18] ($p = 0.58$) and Tarras et al[15] ($p = 0.37$). Patel et al. also did not find correlation between age and PHT ($p = 0.402$). In the present study PH was more common in males ($p = .022$). Tarras et al[15] ($p = 0.69$) could not find a significant association between sex and PH among CKD patients. Moniruzzaman et al[16] found a male predominance (male to female ratio of 2:1) in their study.

In the present study, statistically significant association between CKD stages and PH ($p < 0.001$) was noted, inferring that advanced the CKD stage is, the higher is the incidence of PH. Being a tertiary referral centre, our patients are usually late referrals and all patients in our study group were in stage III, IV, or V. Yang et al[19] found PH prevalence of 23.76% (24/101) in stage II and 48.15% (13/27) in GFR <60 mL/min/1.73 m 2 group ($p < 0.05$) raising the alarm that PH exists and may be prevalent prior to drop in GFR to <60 ml/min/1.73 m 2. Severe PH was detected in CKD patients in stage-V and stage-VD along with increased prevalence of PH and cardiovascular morbidity as renal disease progressed in study by Li et al[20].

The exact mechanisms of PH in higher stages of CKD remain poorly understood. PH might be induced and/or aggravated by left ventricular disorders and risk factors typical of CKD, including volume overload, AVF, sleep disordered breathing, exposure to dialysis membranes, endothelial dysfunction, vascular calcification and stiffening, and severe anemia[21]ESRD-related PH, for the first time, was grouped into the 5th subtype (PH with unclear multifactorial mechanisms) of PH by the World Symposium of PH (WSPH)[22]. This group includes PH in CKD without significant cardiac and pulmonary diseases. Ruling out these comorbid conditions—which were found in 40%–70% of patients in most cohorts—typically involves chest radiography, pulmonary function tests, CT scans, and ventilation/perfusion scans[21].

Significant association was seen between CKD duration and PH prevalence ($p < .001$) and and its severity ($p < .05$) in our study. Havlucu et al[23] ($p < 0.05$) and Patel et al[17]. ($p = 0.002$) found similar association between duration of CKD and PH in their study. The greater the duration of the CKD, the longer will be exposure of the patients to the altered cardiovascular physiology including synergistic effects of increased PVR, increased cardiac output, and elevated PCWP, and hence, the chances of having more severe pulmonary hypertension increases[21] There was a statistically significant association between diabetes and hypertension with PH ($p < 0.001$) in this study. In a study conducted by Agarwal et al[24] there was similar statistical association of diabetes ($p = 0.04$) with PH but not of systemic hypertension ($p = 0.2$). However, the study by Fabian et al[25] showed statistically strong association of both diabetes ($p = 0.021$) and hypertension ($p = 0.0074$) with PH. Hypertension and diabetes mellitus, two dominant causes of kidney disease, trigger LV diastolic dysfunction, an alteration bound to increase pulmonary venous and arterial pressure[26]. Chronic volume overload, a factor implicated in LV disorders and in high venous return in patients with CKD, may induce pulmonary venous hypertension by increasing pulmonary blood flow and adversely affecting LV function. Two echocardiographic studies have shown that CKD patients with PH have significantly higher estimates of left-sided filling pressures, with higher estimates of PCWP and left atrial size, suggesting chronic volume overload[24].

Our study showed statistically significant presence of PH in patients treated on HD than treated on CAPD or conservatively ($p < 0.001$). The HD group not only showed higher prevalence of PH but also had more severe PH ($p < .001$) than those on CAPD or conservative treatment. The prevalence of PH among patients on HD was studied by Moniruzzaman et al[16] and Kiykim et al[27] and was found to be 68.6% and 68.8%, respectively. Though, effect of duration of CKD in patients on HD is important in pulmonary hypertension pathogenesis, factors specific to HD like exposure to dialysis membrane, AV fistula contributes to pulmonary hypertension. Uremic patients on chronic HD therapy, via AV access exhibit decreased Nitric oxide production. This endothelial dysfunction, coupled with prolonged elevation of endothelin, reduces capacity of pulmonary circulation to maintain AV access mediated elevation of cardiac output and contributes to pulmonary hypertension[21].

Domenici et al[28] who reported that PH was found in 23/39 (58.9%) of the HD patients and 2/9 (22.2%) of the PD patients; PASP was significantly higher in HD patients than in PD patients ($p < 0.01$). Further, Patel et al[17] demonstrated that 41 patients had PH, of whom 33% were on HD. The prevalence and average PASP is lower in PD patients than in HD patients. In our study group, there were 43 patients had PH, of whom 79.06% on HD and 11.62 % on CAPD. Neutrophil activation secondary to blood–dialysis membrane contact accompanied by reversible neutrophil sequestration in the lung also contributes to causing or worsening microvascular lung disease in HD patients[5].

In the present study, patients with longer duration of HD had higher prevalence of PH. Emara et al[3] ($p < 0.001$) and Patel et al[17] ($P = 0.001$) also found a similar association. The presence of AVF itself, which is accompanied by commonly occurring anemia and fluid overload, further increases the PH[29] There was association between duration on HD and severity of PH in the present study but statistically not significant ($p = .96$). There are many studies similar that of Issa et al[30] and Bozbas et al[31] showing association of duration of HD and PH, but none have studied the association between the duration of HD and severity of PH.

In the present study, out of 39patients with AVF, 24 had PH, where as, out of 28 patients without AVF, 10 had PH. This shows there was a strong association between AVF and PH ($p < .05$). Havlucu et al[23] showed similar association ($p < 0.05$); however, Agarwal et al[24] ($p = 0.1$) did not find a similar association. This difference could have been due to the difference in the AVF duration as well as the population studied. In a case report, PH had reversed after ligation of the patient's large fistula[32] The AVF created causes decrease in the systemic vascular resistances with increase in venous return and cardiac output, which helps maintain proper blood flow to all organs and tissues. These adaptations increase pulmonary blood flow and set the stage for PH. Well-performed studies show that pulmonary pressure increases in strict temporal relationship with AVF creation[29] and that PH tends to worsen over time in this population[23].

Various variables were taken into consideration such as hemoglobin levels, BUN, serum creatine, and serum calcium × phosphorus product (Ca × P) , all of which were found to be statistically associated with PH. Significant association of hemoglobin, BUN, serum creatine, and serum Ca × P product was observed in various studies. It is postulated that low hemoglobin levels can contribute to PH by aggravating hypoxia[33]. PH in patients having raised Ca × P product may be attributed to increased stiffness of pulmonary vasculature caused by vascular calcification. Increased uremic load reflected by BUN and serum creatinine level causing release o f inflammatory markers (acute phase reactive protein and cytokines, including IL-1β, TNF-α and IL-6), especially in HD patients can be a factor for development of PH in these patients.

CONCLUSIONS

The prevalence of PH in CKD patients is 35.83%. Prevalence of PH had positive correlation with stage of CKD, duration of CKD, those on hemodialysis, and those with AVF. The severity of PH was also directly proportional to the duration of CKD and duration of hemodialysis.serum creatinine, BUN, Calcium phosphorus product were significantly higher and Hb is lower in CKD patients with PH than without it.there is significant mortality in pt of CKD with PH than without it, risk of mortality increases in presence of heart failure along with PH, but statistically not significant. The present study entitled 'A study of Pulmonary Hypertension in patients of Chronic Kidney Disease in Bundelkhand Region –A Hospital Based Study, a prospective observational study carried in the department of M.L.B. Medical College ,Jhansi during the period from March 2018 to oct 2019.

Out of 120 patients included in this study, 43(35.83%) patients had PH. There was no effect of age in the prevalence of PH. Out of total 43 patients with PH 31(72.09%) are male. PH was more common in male than female (p value<.05). There was strong association of PH with Diabetes and Hypertension (p value <.001). PH was more common in advance stages of CKD-stage V(90.7%), longer duration (>12 month) of CKD(68%),and statistically significant (p value is <.001). Out of 7 patients severe PASP , 5(71.42%) had CKD for >12 month(p value<.05).

PH was more common in CKD patients (P<.001) on HD(50.75%) and longer duration (>12 month) of HD (p<.05). HD was also associated with severity of PH (p<.001).

PH was also more common in CKD Patients with AVF (61.6%), Hb<10gm/dl(47.6%),

BUN>45mg/dl(64.5%),S.Creatinine>5mg/dl(58.46%) and Ca x P product >55mg2/dl2(56.06%) and statistically significant (p<.05).

In CKD Patients with PH had increase mortality (12.5%) compared with 4.17% among patients without PH (p<.001). Presence of HF had increase mortality of 23.26% compared with 4.65% among the patients with PH in absence of HF(p<.001).

Mortality of CKD Patients with PH was strongly associated with presence of HF, HTN, age>60 years(63.63%), severity of PH (71.42%0, Hb <10gm/dl (50%),BUN>45mg/dl(45.6%), S.creatinine >5mg/dl(44.82%) and Ca x P product >55mg2/dl2 (51.85%) and statistically significant (p<.05)

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