Pulmonary hypertension (PAH) has been reported to be high in CKD. It is an overlooked cardiovascular morbidity in chronic kidney disease. Various pathologic process like endothelial dysfunction, inflammation, anemia, volume overload, left ventricular dysfunction and metabolic and hormonal derangements contribute. (1, 2) AV fistula and procedures such as access thrombectomy increase the risk. (3) Underlying diseases like scleroderma and lupus may also cause PAH. It is important to study this condition as it has impact on morbidity and mortality of CKD and is a potentially preventable condition to some extent.

AIMS:
To assess prevalence of pulmonary hypertension in CKD
To correlate PAH with clinical and biochemical parameters

INCLUSION CRITERIA
All patients of CKD > 12 years of age either on conservative treatment or hemodialysis

EXCLUSION CRITERIA
• Chronic smokers
• Pulmonary hypertension due to other known causes
• Pregnant and postpartum females

MATERIALS AND METHODS
This observational study was conducted in a tertiary care centre of central India over a period of 12 months. 120 patients of CKD whether on conservative management or hemodialysis were included. Aims, procedure and investigative course of the study was explained to patient and relatives. Written informed consent was taken before including in study. Detail history including age, sex, duration of illness, etiology of CKD and type of treatment (conservative vs hemodialysis) were noted. Detailed clinical examination was done with special attention to site of AV fistula in patients on dialysis. Investigations done were complete blood picture, renal function test, serum electrolytes, liver function test, urinalysis and ABG along with X-ray chest, ECG and ultrasound abdomen. Each patient underwent Echocardiography by cardiologist of the institute to detect pulmonary hypertension. Systolic Pulmonary artery pressure were evaluated by Doppler echocardiography using tricuspid regurgitation jet velocity and modified Bernoulli’s equation. AV Fistula flow was assessed by Doppler ultrasound.

RESULTS
Out of 120 patients 28.33% had PAH. PAH was more in group on hemodialysis and in cases with AV fistula. Prevalence of PAH had significant correlation with longer duration of dialysis, low haemoglobin, high blood urea & serum creatinine and low serum bicarbonate. Diabetes and hypertension was not associated with PAH. Serum uric acid was not high in PAH.

Table 1. Association between duration of dialysis and PAH

<table>
<thead>
<tr>
<th>DURATION OF DIALYSIS (yrs)</th>
<th>NUMBER OF CASES (N=79)</th>
<th>CASES WITH PAH</th>
<th>PERCENTAGE (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤1</td>
<td>14</td>
<td>03</td>
<td>21.4</td>
</tr>
<tr>
<td>2-3</td>
<td>46</td>
<td>09</td>
<td>19.5</td>
</tr>
<tr>
<td>3-4</td>
<td>04</td>
<td>03</td>
<td>75</td>
</tr>
<tr>
<td>&gt;4</td>
<td>15</td>
<td>14</td>
<td>93.3</td>
</tr>
</tbody>
</table>

Table 2. Correlation between lab parameters and PAH

<table>
<thead>
<tr>
<th>PARAMETERS( mean)</th>
<th>With PAH</th>
<th>Without PAH</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin(gm%)</td>
<td>8.23</td>
<td>9.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Serum Bicarbonate (mmol/L)</td>
<td>8.61</td>
<td>11.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Blood Urea(mg%)</td>
<td>77.49</td>
<td>62.56</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Serum Creatinine(gm%)</td>
<td>9.31</td>
<td>5.26</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Serum Uric Acid(gm%)</td>
<td>6.33</td>
<td>6.44</td>
<td>0.738</td>
</tr>
</tbody>
</table>

DISCUSSION
Pulmonary hypertension (PAH) has been reported to be high in...
Prevalence had significant positive correlation with duration of hemodialysis and in this subgroup more in those with AV fistula. Our study was 28.33%. Prevalence was more in patients on treatment. Out of 23 patients who had AV fistula 10 had PAH (43.3%) while only 24.7% in group without AV fistula had PAH (p value-0.042, significant). The results were comparable with previous study done by Yigla et al. (5) In these patients PAH may be secondary to hemodynamic modifications related to the creation of an anastomosed fistula caused by a reduced ability of pulmonary vessels to accommodate the AV access-mediated elevated cardiac output, possibly because of a derangement of nitric oxide-endothelin metabolism [5] but its pathogenesis has not been completely elucidated.

In this study we divided CKD patients who are on hemodialysis into 4 groups according to duration of dialysis. Results show greater the duration of dialysis more is the prevalence of PAH. This was also seen in similar study done by Patel P et al. (9) On the contrary, Amin et al. (8) reported that there was no significant difference between patients with PAH and those without PAH in end-stage renal disease patients, who were receiving regular hemodialysis with regard to age, duration of dialysis, serum calcium, phosphorus, alkaline phosphatase, parathyroid hormone.

66 patients in our study had hypertension and 24 patients had diabetes. Although PAH was more common in group with hypertension(33.3% vs 22.2%) and diabetes (31.1% vs 20%) the difference was not statistically significant. In a study by Magdy et al (10) diabetes mellitus and hypertension have a higher prevalence in the pulmonary hypertension subgroup than the normal arterial pressure subgroup.

We tried to compare various lab parameters haemoglobin, serum bicarbonate, blood urea and serum creatinine in both groups. Mean haemoglobin was 8.23gm% in cases with PAH while 9.5gm% in cases without PAH and the difference was significant. This indicates the role of anemia in pathogenesis of PAH in CKD. Similar result is also seen in study by C. J. Rhodes et al (11) and Mazdeh et al. (12) It is well known that iron deficiency influences pulmonary vasoconstrictor response to hypoxia.

Similarly there was significant correlation between serum bicarbonate (8.61 vs 11), blood urea(77.49 vs 62.56)and serum creatinine (9.31 vs 5.26) but serum uric acid didn’t correlate with occurrence of PAH. These findings are comparable to study done by Patel et al (9) in which acidosis and low serum bicarbonate as well as level of blood urea and creatinine was associated with PAH. Mean uric acid was 6.33mg% in cases with PAH and 6.44 mg% among cases without PAH and difference was not significant. Other study done by Norotoshi Nagaya et al (13) shows that serum uric acid increases in proportion to the clinical severity of PAH and has independent association with long term mortality of patients with Primary Pulmonary Hypertension.

CONCLUSION
Prevalence of pulmonary hypertension in chronic kidney disease in our study was 28.33%. Prevalence was more in patients on hemodialysis and in this subgroup more in those with AV fistula. Prevalence had significant positive correlation with duration of dialysis, presence of anaemia, low serum bicarbonate and high blood urea and serum creatinine. There was no significant correlation with presence of diabetes and hypertension and also serum uric acid. Hence we conclude that early renal transplant may be important to prevent morbidity and mortality associated with disease and its treatment. Also correction of anaemia and serum bicarbonate may lead to decrease in prevalence of pulmonary hypertension.

Compliance with ethical standards
None of the authors have conflict of interest.
All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study.

REFERENCES