INTRODUCTION

The broad systemic inflammatory condition known as chronic obstructive pulmonary disease (COPD) is marked by airflow restriction and enduring respiratory symptoms. It is frequently not completely reversible. Genetic predisposition and exposure to environmental factors, such as air pollution and tobacco smoke, interact intricately to cause COPD. One of the leading causes of death and morbidity is COPD, and during the next years, its incidence will rise. Numerous cardiovascular (CV) and non-cardiovascular comorbidities, including hypertension, diabetes, cognitive decline, osteoporosis/osteopenia, chronic kidney disease (CKD), and cancer, are highly connected with the individual burden and health expenditures associated with COPD. Patients with COPD in particular are more likely than the general population to acquire chronic kidney disease (CKD). The most prevalent risk factors for new development of CKD include overweight, diabetes, arterial hypertension, and age. Due to the activation of pro-inflammatory and pro-oxidant pathways that cause pathologic alterations in renal circulation, atherosclerotic damage is a part of the pathophysiology of chronic kidney disease (CKD). The most well-known cytokine in this context is interleukin-6 (IL-6), which is generated by pulmonary cells and travels throughout the body to target other organs, so inducing more inflammation. Not only should IL-6 be taken into account, but also interleukin-1β (IL-1β), interleukin-8 (IL-8), interleukin-8 (IL-8),
interleukin-10 (IL-10) and tumor necrosis factor-α (TNF-α). Important mediators, IL-6, IL-1, TNF-α, and monocyte chemoattracting protein (MCP-1) can cause inflammatory renal tubular injury by activating platelet aggregating factor (PAF-6) and drawing in monocytes and macrophages. Renal ischemia can also increase pulmonary vascular permeability and impact the production of the enzyme angiotensin converting enzyme (ACE), which can hinder the operation of ion channels that are in charge of reabsorption of fluids at the level of pulmonary alveoli. The assessment of renal albumin excretion serves as the fundamental basis for the diagnosis of renal injury. Anomalies in blood tests, imaging changes, and urine sediment changes are further markers of kidney impairment. The estimated glomerular filtration rate (eGFR), whose fluctuation corresponds with renal function decline, is a useful metric used to assess the decline in renal function in large research populations.[5–7]

In fact, the co-occurrence of CKD among COPD patients has a detrimental effect on overall mortality as well as global health status. Specifically, new research indicates that uric acid (UA) levels may be an independent predictor of exacerbations and early death in COPD patients with CKD and hyperuricemia. The pathophysiologcal connection between UA and COPD is still unclear, though. Notably, after 30 days following the start of a COPD exacerbation, UA levels above 6.9 mg/dL are an independent predictor of death; however, this is not the case at one year 8. Furthermore, in the next thirty days, patients with elevated UA levels need more frequent admissions to the critical care unit, longer hospital stays, and noninvasive ventilation.[8]

Additionally, higher plasma UA concentrations are linked to a higher chance of returning to the hospital after a year due to COPD exacerbations.[8]

It’s interesting to note that the Takahata research indicates a strong negative association between forced expiratory volume in the first second (FEV1),[9] and serum UA levels and forced vital capacity (FVC). This finding is in line with the finding that, although a direct causative association has not been shown, hyperuricemia has been linked to reduced lung function and an increased risk of respiratory symptoms in people with COPD. And lastly, further data suggests that the correlation between serum UA and creatinine, as well as the assessment of UA levels, can both be accurate indicators of the likelihood that COPD patients’ condition would worsen and worsen.[10] Therefore, it is conceivable that elevated UA levels might have a detrimental effect on clinical result in individuals with COPD. Taking into account the aforementioned issues, their primary goal was to look into the parameters that predict the development of CKD in COPD patients, specifically focusing on the impact of UA. The other goal was to look for variables linked to the quick drop in eGFR within the same research cohort.

**MATERIALS AND METHODS**

**Place of Study:** P.G. Department of Medicine, SCBMCH, Cuttack  
**Duration of Study:** December 2020 - November 2022  
**Study Design:** Prospective or observational study  
**Study Population:** The study population will consist of 100 patients of COPD visiting SCB Medical College during the study period.  
**Inclusion Criteria:** Patients who are previously diagnosed to have COPD based on clinical features and spirometry in the department of Pulmonary medicine who got admitted in medicine and Pulmonary medicine wards was included in the study.  
**Exclusion Criteria:** Patients with COPD who have other comorbid illness which are likely to cause renal failure are excluded. These comorbid illness include Diabetes Mellitus, Hypertension, Known renal disease such as renal stones, polycystic kidney disease etc, Cardiac failure, Cirrhosis, Ingestion of nephrotoxic drugs.  
**Method:** Ethical clearance was obtained from the Institutional Ethical Committee and respective authorities before the commencement of the study. The patients fulfilling the inclusion and exclusion criteria, admitted to the Department of Medicine, SCB Medical College, Cuttack, between DECEMBER 2020 to November 2022 was studied and evaluated for RENAL DYSFUNCTION IN COPD PATIENT after obtaining informed consent.

**RESULTS**

In Case, 12 (12.0%) patients were 41-50 years of age, 40 (40.0%) patients were 51-60 years of age and 48 (48.0%) patients were ≥61 years of age.  
In Control, 12 (12.0%) patients were 41-50 years of age, 40 (40.0%) patients were 51-60 years of age and 48 (48.0%) patients were ≥61 years of age.

![Figure 1: Association of Age in Group with Group was Not Statistically Significant (P=1.000).](image)

In Case, 20 (20.0%) patients were Female and 80 (80.0%) patients were Male.  
In Control, 20 (20.0%) patients were Female and 80 (80.0%) patients were Male.  
Association of Sex with Group was not statistically significant (p=1.0000).

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In Case, the mean Age (mean± s.d.) of patients was 59.5000± 6.8306.
In Control, the mean Age (mean± s.d.) of patients was 59.6500± 6.8761.
Distribution of mean Age with Group was not statistically significant (p=0.8772).

In Case, the mean HB (g %)(mean± s.d.) of patients was 11.8940± 2.7940.
In Control, the mean HB (g %)(mean± s.d.) of patients was 11.9200± 2.4402.
Distribution of mean HB (g %) with Group was not statistically significant (p=0.9442).

In Case, the mean TLC (per microl) (mean±s.d.) of patients was 7827.0400± 1921.7490.
In Control, the mean TLC (per microl) (mean±s.d.) of patients was 7872.9000± 1837.3738.
Distribution of mean TLC (per microl) with Group was not statistically significant (p=0.8632).

<table>
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<tr>
<th>Age in group</th>
<th>Case</th>
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<td>12</td>
<td>24</td>
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<td>Row %</td>
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<td>50.0</td>
<td>100.0</td>
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<tr>
<td>Col %</td>
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<td>12.0</td>
<td>12.0</td>
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<tr>
<td>51-60</td>
<td>40</td>
<td>40</td>
<td>80</td>
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<tr>
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<td>48.8</td>
<td>100.0</td>
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<tr>
<td>Col %</td>
<td>40.0</td>
<td>40.0</td>
<td>40.0</td>
</tr>
<tr>
<td>≥61</td>
<td>48</td>
<td>48</td>
<td>96</td>
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<tr>
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<td>48.0</td>
</tr>
<tr>
<td>TOTAL</td>
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<td>50.0</td>
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</tr>
<tr>
<td>Col %</td>
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</table>

Chi-square value: .0000; p-value:1.000

<table>
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<th>GROUP</th>
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<th>Control</th>
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<tr>
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<td>100.0</td>
</tr>
<tr>
<td>Col %</td>
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<td>20.0</td>
<td>20.0</td>
</tr>
</tbody>
</table>
Our analysis revealed that although the age difference between the Control group (59.6500±6.8761) and the Case group (59.5000±6.8306) was greater, it was not statistically significant (p=0.9442). Globally, COPD is a major cause of death (Elmahallawy II et al, 2013). It is linked to a great deal of comorbidities. The 300 control patients in the current study were age and gender matched with illnesses other than COPD; they were aged 64.70±7.12 years; 148 of them (49.3%) were females.

We found that the difference in SR Albumin (g/dl) between the Case [3.9120±0.5945] and Control [3.9100±0.6114] was greater, but the difference was not statistically significant (p=0.9813). There have been few reports of chronic kidney disease (CKD) as a comorbidity of chronic obstructive pulmonary disease (COPD), although Yoshizawa T et al (2015) found cardiovascular illnesses, depression, and osteoporosis as comorbidities of COPD.

DISCUSSION

This investigation was either prospective or observational. This study was carried out in the P.G. Department of Medicine, SCBMCH, Cuttack, from December 2020 to November 2022. In all, 200 patients were involved in this investigation. Out of 200 patients in our research, 96 (48.0%) were older than 61 years, a number that was not statistically significant (p=1.000). The population of men was determined to be greater [80 (80.0%)] than that of women [20 (20.0%)], although this difference was not statistically significant (p=1.000).

The population of men was 65.28±6.32 years; 148 of them (49.3%) were females. The 300 COPD patients were aged 64.70±7.12 years; 138 of them (46%) were men, and the remaining patients were females. The 300 COPD patients were 65.28±6.32 years; 148 of them (49.3%) were females.

Our analysis revealed that although the age difference between the Control group (59.6500±6.8761) and the Case group (59.5000±6.8306) was greater, it was not statistically significant (p=0.8772). Globally, COPD is a major cause of death (Elmahallawy II et al, 2013). It is linked to a great deal of comorbidities. Thirty-odd COPD patients were included in the current study.

The mean HB (g%) was found to be higher in the Control group [11.9200±2.4402] than in the Case group [11.8940±2.7940], however the difference was not statistically significant (p=0.9442).

TLC (per microl) was greater in the Control group [7872.9000±1837.3738] than in the Case group [7827.0400±1921.7490] in our investigation, although the difference was not statistically significant (p=0.8632).

We found that the difference in SR Albumin (g/dl) between the Case [3.9120±0.5945] and Control [3.9100±0.6114] was greater, but the difference was not statistically significant (p=0.9813). There have been few reports of chronic kidney disease (CKD) as a comorbidity of chronic obstructive pulmonary disease (COPD), although Yoshizawa T et al (2015) found cardiovascular illnesses, depression, and osteoporosis as comorbidities of COPD.

This study used estimated glomerular filtration rate (eGFR) based on creatinine (Cr) and cystatin C (Cys) levels to examine the prevalence of CKD in COPD patients. A comparison was made between 108 stable COPD outpatients (the COPD group) and 73 patients (the non-COPD control group) who were at least 60 years old and had no prior history of renal disease or COPD regarding the incidence of CKD and the values of several CKD-related markers. The criteria for CKD was an eGFR of less than 60 mL/min/1.73 m2. The COPD group had a considerably higher Cr level, however there was no significant difference in eGFR based on serum Cr (eGFRCr) between the two groups (73.3±25.3 vs 79.7±15.5 mL/min/1.73 m2).
According to Madouros N. et al. (2022), the prevalence of chronic obstructive pulmonary disease (COPD), one of the most prevalent illnesses in the world, rises with age. It frequently coexists with other illnesses, making patient management challenging and costly. Patients with COPD frequently have chronic kidney disease (CKD), which may go undiagnosed, particularly if it is mild. Most investigations revealed a higher mortality risk when both conditions combine.

To support the association, additional investigation is needed, as well as larger prospective studies with matched control groups.

**CONCLUSION**

By 2025, COPD will be the fourth most prevalent chronic illness globally, up from its current seventh position. In the globe, it ranks as the fourth most frequent cause of death. According to estimates, COPD claims the lives of half a million Americans each year. It is anticipated that COPD-related mortality would rise in the next years. Because of the same risk factors, COPD is linked to a number of other comorbid conditions, including musculoskeletal disorders, cancer, and coronary artery disease. It was only recently discovered that COPD and renal failure are related. A few studies in the western population have examined the degree of correlation between COPD and renal failure. The fact that individuals with COPD have less muscle mass, which results in a normal blood creatinine level despite a marked reduction in renal function, may be the cause of the inability to recognize the link between renal failure and COPD. We call this hidden renal failure. This suggests that in COPD patients, the estimated GFR should ideally be determined by estimating the creatinine clearance using established formulae, such as the MDRD formula, which have been verified in extensive research. In these patients, serum creatinine on its own would not be the best indicator of renal function.

**REFERENCES**