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# A STUDY OF THE BIOCHEMICAL PARAMETERS OF COPD PATIENTS IN ASSOCIATION WITH SMOKING STATUS

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## ABSTRACT

**Introduction:** Chronic obstructive pulmonary disease (COPD) is defined as a preventable disease, caused mainly by chronic inflammation of the airways, characterized by persistent symp- toms such as cough and dyspnea, and modification of lung function parameters shown on spirometry. Smoking is the most incriminated and studied risk factor for COPD, but other factors such as air pollution, are also linked to this disease. The World Health Organization (WHO) states that over 400 million people suffer from COPD and that this disease is the third highest cause of mortality worldwide.

**Aims:** The aim of this study was to investigate the lipid profile and biochemical parameters of COPD patients in relation to smoking status.

**Materials and Methods:** The present study was a observational study. The present study was conducted between 2020–2021 in the Department of Biochemistry, Midnapore Medical College, Paschim Medinipur.

**Result:** In our study, 52 patients suffering from COPD were included; clinical features and biological variables were statistically assessed. Shows the characteristics of the patients included in the study and shows the descriptive statistics of the biochemical parameters of the patients included in the study. The median age of the actively smoking patient was 65 years old. A predominance of the male gender was observed in all groups; in the smoker's group, 88% of the participants were male.

**Conclusion:** We found that COPD is considered a systemic disease, and tobacco consumption is an important contributor to the development of this pathology. Tobacco smoking was also associated with dyslipidemia.

**Keywords:** Tobacco consumption; smoking status; COPD; lipid profile; biochemical parameters and uric acid.

## INTRODUCTION

Chronic tobacco use is defined as tobacco or nicotine dependence. Assessment of nicotine dependence may be performed clinically or paraclinically. The clinical assessment is based on the determination of smoking status, type of tobacco product used, tobacco use and nicotine dependence. By investigating the number of cigarettes smoked, we can determine the status of smoking, respectively: non-smoker, occasional smoker, regular smoker or former smoker. Tobacco use can be defined as the number of cigarettes smoked in a day or the number of packs of cigarettes/years (PY). Nicotine dependence can be diagnosed using the Fagerström nicotine dependence test. Paraclinical assessment of tobacco dependence can be performed by laboratory biochemical tests, which highlight the presence of biomarkers of exposure to tobacco smoke. These tests include the concentration of carbon monoxide in the exhaled air and the level of cotinine, a metabolite of nicotine, which can be identified in urine, serum, saliva, etc. The most commonly used biomarkers in research to confirm or deny self-reported abstinence are carbon monoxide in exhaled air, cotinine (a metabolite of nicotine, measurable in plasma, saliva, urine, hair and intranasally), but also markers such as anatabine, anabazine, thiocyanate, uric acid (UA) and nitric oxide [1]. Another biomarker of exposure to tobacco smoke is uric acid,

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the degradation product of nucleic acids and the final product of purine oxidation. It is transported by plasma from the liver to the kidneys where it is filtered and then excreted in a percentage of 70%, the rest being degraded in the gastrointestinal tract. Uric acid may vary depending on age, diet, sex, genetic factors, exercise, menopause, etc. UA concentration is thought to reflect the antioxidative capacity of the body because it is the most abundant aqueous antioxidant. Smoking causes oxidative stress and uric acid is responsible for up to 60% of serum free radical scavenging [2].

Lipid metabolism seems to be affected by smoking, with studies showing a correlation between this habit and high levels of triglycerides and low levels of high-density lipoprotein cholesterol (HDL-CHOL) [3]. The mechanism by which smoking alters the lipid profile is still not fully understood, but several hypotheses have been proposed in the specialized literature. One of these hypotheses states that nicotine stimulates the production and secretion of growth hormones, catecholamines and cortisol, which causes an increase in the serum concentrations of free fatty acids, which triggers the hepatic secretion of triglycerides and very-low-density lipoprotein (VLDL-C). In one study, Khojah and Ahmed found that smokers compared to non-smokers have higher levels of triglycerides, cholesterol, VLDL-C, low-density lipoprotein cholesterol (LDL-CHOL) and lower levels of high-density lipoprotein cholesterol (HDL-CHOL). The effects of chronic tobacco consumption (smoking and chewing) on the lipid profile of 75 individuals divided in three groups (I: non-smokers and non- chewers; II: smokers and non-chewers and III: chewers and non-smokers) were studied by Rao and Subash and the results showed that the levels of cholesterol, VLDL-C, LDL-CHOL and triglycerides were higher in group II and III compared to group I, and on the other hand, HDL-CHOL had decreased levels in the same groups of patients [4]. These findings seem to reflect the impact that smoking has on lipid metabolism. In smokers, a lower activity of lecithin cholesterol acyltransferase (LCAT) and lipoprotein lipase (LPL) and a higher activity of cholesterol ester transfer protein (CETP) was observed[5].

**Aims:** The aim of this study was to investigate the lipid profile and biochemical parameters of COPD patients in relation to smoking status.

## MATERIALS AND METHODS

- **Study site:** Department of Biochemistry, Midnapore Medical College, Paschim Medinipur
- **Study design:** A present study was an observational study.
- **Period of study**: 2020–2021Years
- **Statistical Analysis** -This study evaluated the clinical features and biological variables of 52 patients diagnosed with different stages of COPD. Mann–Whitney U test was used to compare the biochemical parameters for two independent groups (smokers vs. non-smokers, smokers vs. ex-smokers, non-smokers vs. ex-smoker). Descriptive statistics in case of smokers/non-smokers/ex-smokers were present in terms of average, standard deviation, median and range. Spearman's rank correlation coefficient was used to measure the strength of the correlations between biochemical parameters corresponding to smokers and non-smokers.

## RESULT AND DISCUSSION

The present study was an observational study. The present study was conducted between 2020–2021in the Department of Biochemistry, Midnapore Medical College, Paschim Medinipur. In this observational study, 52 patients suffering from COPD were included; clinical features and biological variables were statistically assessed. Shows the characteristics of the patients included in the study and shows the descriptive statistics of the biochemical parameters of the patients included in the study. The median age of the actively smoking patient was 65 years old. A predominance of the male gender was observed in all groups; in the smoker's group, 88% of the participants were male

In this study, the associations between smoking status and biochemical indicators in case of patients diagnosed with COPD were investigated. The present study correlates tobacco use and COPD with lipidic profile markers useful in clinical practice. Antioxidant status was evaluated according to the level of uric acid, smoking was

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quantified using packs- years (PY) and other useful parameters were also assessed: glucose, urea and creatinine. The results obtained in the present study point to a low level of uric acid in the case of smoking patients and positive correlation with COPD stages. Studies in this field have shown that UA levels are higher in COPD patients compared to healthy people, suggesting that tissue hypoxia causes an increase in purine catabolism, especially in the advanced stages of the disease [6]. Mouhamed et al. conducted a study about the effects of smoking on uric acid concentrations showing that plasma uric acid concentrations were lower in smokers versus non-smokers. The author attributed this finding to the fact that smoking reduces the endogenous production because of the oxidative stress generated. In fact, serum uric acid acts as a true antioxidant, including against oxidative stress induced by tobacco use. Thus, low serum uric acid concentrations in smokers have been attributed to decreased endogenous production as a result of chronic exposure to tobacco smoke, which is a significant source of oxidative stress. Increased oxidative stress can lead to depletion of antioxidants, including uric acid. In fact, cigarette smoke, due to the large number of chemical substances resulting from combustion (4700 substances, including polycyclic aromatic hydrocarbons, heavy metals, NO) leads to the formation of a strong chemical oxidative stress involving practically all the compounds present in tobacco, leading to the formation of free radicals. In this case, free radicals are able to react with all the constituents of the cell and the matrix, resulting in the so-called state of oxidative stress. Another study showing the same results was conducted by Hanna et al, where correlations between smoking status and uric acid concentrations were also observed [7]. Similar results of serum uric acid concentrations for smokers/non-smokers were presented in recent studies, and low values were associated with oxidative stress that reduces antioxidant levels.

Recent studies regarding imbalances in lipid metabolism point to the fact that smoking as well as oxidative stress are possible mechanisms responsible for the development of dyslipidemia in COPD patients. However, there are limitations to these statements that are related to the severity of the disease, gender, body mass index (BMI) and especially to the intensity of smoking [8]. Furthermore, literature point that smoking is associated with an atherogenic lipid profile, which can also contribute to the production of oxidative stress. Smoking, in its various forms, leads to an increased risk for high total cholesterol serum levels, as well as for high triglycerides levels. In this study, the parameters of the lipidic profile were observed to not be normally distributed (Figure 1a–d) and the statistical results presented in Figure 2 show important correlations especially for total cholesterol, triglycerides, total lipids and LDL-CHOL.

The relationship between smoking and serum creatinine levels has been discussed in few studies. What was observed is that the level of serum creatinine increases in active smokers [9], as well as in the case of subjects with various renal diseases and hypertension. Dülger et al. studied the renal function in active and passive smokers and showed that creatinine levels were significantly increased in active smokers ($p < 0.01$) and concluded that kidneys, and in particular the glomerular function, can be affected even in case of passive smoking [9]. Few studies in the literature showed that smoking increases the risk of proteinuria and also the risk of mild hyperfiltration, as well as the risk of mild renal impairment, especially in men and in elderly people. Smoking in general has a negative influence on renal function even in subjects apparently not suffering from a kidney disease, but the adverse renal effects due to smoking are present especially in patients with different kidney disorders as well as in hypertensive patients.

## CONCLUSION

COPD is considered a systemic disease, and tobacco consumption is an important contributor to the development of this pathology. Tobacco smoking was also associated with dyslipidemia. In the case of the patients included in the present study, smoking was associated with changes in the lipid profile of smokers and ex-smokers and these results were highlighted by increased serum triglyceride concentrations, decreased plasma high density lipoproteins and HDL-CHOL and increased LDL-CHOL fraction. Low serum uric acid values in smokers could identify a chemical oxidative stress produced by the effect of toxic compounds in

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tobacco smoke, which in turn could trigger an imbalance of lipid metabolism in smokers. More studies need to be conducted in this field to obtain a better understanding of the mechanisms involved in the alteration of the lipid profile of smoker and ex-smoker patients.

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**Mann–Whitney U test applied for comorbidities and demographic area of COPD patients.**

| Mann-Whitney U Test by Variable Obesity; Marked Tests are Significant at $p < 0.05000$ | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Rank Sum | Rank Sum | U | Z | $p$-Value | Z | $p$-Value | Valid N | Valid N | 2*1sided |
| Total cholesterol | 1224.50 | 153.50 | 125.50 | 0.84 | 0.40 | 0.84 | 0.40 | 45.00 | 7.00 | 0.40 |
| LipT | 1287.00 | 91.00 | 63.00 | 2.52 | 0.01 | 2.52 | 0.01 | 45.00 | 7.00 | 0.01 |
| HDL-CHOL | 1245.00 | 133.00 | 105.00 | 1.39 | 0.16 | 1.39 | 0.16 | 45.00 | 7.00 | 0.17 |
| LDL -CHOL | 1211.00 | 167.00 | 139.00 | 0.48 | 0.63 | 0.48 | 0.63 | 45.00 | 7.00 | 0.64 |
| Triglicerides | 1217.00 | 161.00 | 133.00 | 0.64 | 0.52 | 0.64 | 0.52 | 45.00 | 7.00 | 0.53 |

**Application of the Spearman correlation to biochemical parameters and long-acting beta2-agonists in case of patients with COPD.**

| | UA | Urea | Creatinine | Glucose | Triglicerides | CHOL | HDL-CHOL | LDL-CHOL | COPD Stage | Status of Smoking | PY LABA | VEMS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | LipT | | | | | | | | | | |
| UA | 1 | | | | | | | | | | | |
| Urea | 0.281 | 1 | | | | | | | | | | |
| Creatinine | 0.546 | 0.500 | 1 | | | | | | | | | |
| Glucose | 0.316 | 0.007 | 0.456 | 1 | | | | | | | | |
| Triglicerides | 0.125 | 0.357 | 0.037 | 0.004 | 1 | | | | | | | |
| CHOL | 0.048 | 0.105 | 0.014 | 0.044 | 0.089 | 1 | | | | | | |
| LipT | 0.031 | 0.308 | 0.046 | 0.060 | 0.884 | 0.945 | 1 | | | | | |
| HDL-CHOL | 0.007 | 0.144 | 0.113 | 0.006 | 0.377 | 0.220 | 0.234 | 1 | | | | |
| LDL -CHOL | 0.078 | 0.154 | 0.018 | 0.089 | 0.209 | 0.809 | 0.945 | 0.043 | 1 | | | |
| COPD stage | 0.398 | 0.028 | 0.231 | 0.273 | 0.057 | 0.056 | 0.061 | 0.046 | 0.059 | 1 | | |
| Status of smoking | 0.025 | 0.182 | 0.144 | 0.067 | 0.140 | 0.453 | 0.120 | 0.09 | 0.297 | 0.204 | 1 | |

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| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PY | 0.238 | 0.451 | 0.139 | 0.170 | 0.046 | 0.134 | 0.030 | 0.069 | 0.141 | 0.211 | 0.215 | 1 |
| VEMS | 0.180 | 0.044 | 0.072 | 0.039 | 0.227 | 0.117 | 0.100 | 0.188 | 0.068 | 0.142 | 0.252 | |
| | 0.068 | | 1 | | | | | | | | | |
| LABA | 0.188 | 0.073 | 0.086 | 0.191 | 0.138 | 0.124 | 0.105 | 0.093 | 0.134 | 0.070 | | |