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Research Article

Evaluation of Oxidative Stress Markers (Tac, Tos, Gsh) In Patients with Copd and Their Correlation with Cardiovascular Risk

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ABSTRACT

Objectives: To evaluate oxidative stress markers—Total Antioxidant Capacity (TAC), Total Oxidant Status (TOS), and reduced glutathione (GSH)—in the patients with the chronic obstructive pulmonary disease (COPD) and to mainly determine their actual correlation with the cardiovascular risk.

Study Design and Setting: This descriptive cross-sectional study was conducted in the Department of Pulmonology and Internal Medicine, Jinnah Hospital, Lahore from 1 November 2024 to 30 April 2025.

Methodology: A total of 140 diagnosed COPD patients were mainly being enrolled using the consecutive sampling. Oxidative stress markers including the TAC, TOS, as well as serum GSH levels were measured using the standardized spectrophotometric form of methods. To aid in the estimation of the cardiovascular risk, the Framingham Risk Score (FRS) and assessment of the lipid profile and the blood pressure were counted. Cardiovascular risk parameters and the oxidative stress markers were analysed using correlation. All the participants were provided with informed consent and the ethical approval was obtained (ERC No: 2023-45).

Results: COPD patients demonstrated significantly elevated TOS levels and reduced TAC and GSH levels compared to reference values. It was discovered that patients with high risk cardiovascular patients were significantly higher in TOS ($p<0.001$) and significantly lower in TAC and GSH ($p<0.001$). TOS was exerting a strong negative connection with Framingham Risk Score ($r= -0.58$), TAC and GSH made mild negative correlations ($r= -0.46$ and $r= -0.49$ respectively).

Conclusions: COPD patients exhibit significant oxidative imbalance that correlates positively with cardiovascular risk.A possible indicator of assisting COPD patients (exposed to cardiovascular complications) could be the indicators of oxidative stress.

Keywords: Cardiovascular Diseases, Chronic Obstructive Pulmonary Disease, Glutathione, Oxidative Stress, Total Antioxidant Capacity, Total Oxidant Status.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is one of the progressive inflammatory airway disorder was mainly being characterized by the persistent airflow limitation and the chronic respiratory symptoms MS is an oxidative stress which is one of the major causes of COPD. Cigarette smoke and environment contaminants result in overproduction of excessive reactive oxygen species (ROS), which overburdens endogenous antioxidants and causes aerial inflammation, tissue remodelling and maladaptation of endothelial systems systemically.

The accumulative measure of the antioxidant defences is the total antioxidant capacity (TAC), and the most significant intracellular antioxidant to counter oxidative cell damage is GSH so it has been seen to be decreased in COPD patients with an inverse relationship with disease severity.

Cardiovascular disease (CVD) is one of the most common form of comorbidities in COPD.[9] Mechanisms linking the COPD and the CVD include: Chronic systemic inflammation, endothelial dysfunction, Oxidative stress, Sympathetic overactivity, Hypoxia-induced vascular injury.

It has been reported that pulmonary dysfunction is mechanistically correlated with cardiovascular risk where the oxidative stress is mechanistically imitated.10 TOS increased TAC reduced.

Increasing evidence indicates that regional data analysis of oxidative stress events among patients with COPD and a cardiovascular relationship, are limited (Miklós *et al*., 2023). The proposed study will therefore be in a position to evaluate the concentrations of TAC, TOS, and GSH in COPD patients and determine their relationship with cardiovascular risks.

Objective

To evaluate the oxidative stress markers (TAC, TOS, GSH) in the patients with the COPD and determine their correlation with the cardiovascular risk.

METHODOLOGY

This descriptive cross-sectional study was mainly conducted at the Department of Pulmonology and Internal Medicine, Jinnah Hospital, Lahore from 1 November 2024 to 30 April 2025. . The enrolled patients were 140 with COPD as per GOLD criteria and a consecutive sampling was done.

The estimation of the preferred sample size was included with 95% confidence interval, and 5 percentage of the known margin of error and given prevalence of oxidative stress which was assumed to be 50 population, 134 was determined as the minimum required sample size; 140 sampled population was considered to account the losses of samples (Jaruga *et al*., 2025).

Inclusion Criteria

- Patients aged 40 years or above
- Confirmed diagnosis of chronic obstructive pulmonary disease (COPD)
- Post-bronchodilator spirometry showing FEV1/FVC ratio < 0.70
- Patients in stable clinical condition
- No history of acute exacerbation or recent clinical deterioration
- Patients willing to provide informed consent

Exclusion Criteria

- Known history of ischemic heart disease
- Presence of chronic liver disease
- Presence of chronic renal disease
- Any form of malignancy
- Acute exacerbation of COPD within the last four weeks
- Current use of antioxidant supplementation
- Patients unwilling to participate

Data Collection

Data were mainly being collected systematically from the patients diagnosed with chronic obstructive pulmonary disease (COPD) who had met the predefined form of eligibility criteria (Silvestrini *et al*., 2024). A written informed consent was received and the demographic and clinical data were collected in a structured data collection proforma. Age, sex, body mass index (BMI) and smoking history (current, former or never smoker) were used as the baseline parameters, years of COPD, comorbidities (hypertension, diabetes mellitus, dyslipidemia), and current and past medication (Wolszczak-Biedrzycka *et al*., 2024). A fine clinical analysis involving the utilization of a calibrated sphygmomanometer in order to gauge blood pressure and anthropometrically study measurements as per standard protocols was conducted.

Necessary data about pulmonary functions were received by the use of spirometry records which proved airflow equestrian based on post bronchodilator FEV1/FVC ratio less than 0.70 in accordance with the GOLD criteria (Lazar-Poloczek *et al*., 2024). The degree of severity of the disease was registered where feasible.

It was observed that the venous blood samples (5 mL) were put in an aseptic bottle after 8-10 hours fasting in the evening. Samples were centrifuged at 3000 rpm within a period of 10 minutes and the serum was separated so as to undergo biochemical analysis. Total antioxidant capacity (TAC) and total oxidant status (TOS) were measured using the standardized colorimetric products in the market in the form of spectrophotometric assays kits (Suksatan *et al*., 2022). The methods of enzymatic recycling tests were used to determine the low GSH concentration. It was performed in institutional central laboratory since all the laboratory tests have been performed in standard conditions to achieve the reliability and reproducibility.

In addition, the fasting lipid profile (total cholesterol, HDL, LDL, triglycerides) and fasting blood glucose were measured with the automated biochemical processes. Cardiovascular risk was calculated by taking the

Framingham Risk Score (FRS) age, sex, patient smoking, systolic blood pressure and lipid values (Ergün *et al*., 2025). To manage the overall error that brings about data recording error, the full and proper cross-verification of all the data was executed before statistical entry.

Statistical Analysis

Statistical analysis was mainly being performed using the Statistical Package for the context of Social Sciences (SPSS) version 25.0. Prior to the analysis of the data, data entry and cleaning of data helped in ensuring that they are accurate and complete before data analysis (Grzeszczak *et al*., 2023). The quantitative variables, which comprised of age, BMI, TAC, TOS, and GSH levels, lipid parameters and Framingham Risk Score, were represented by the mean +- standard deviation (SD) whereby whether the data was distributed normally or not was determined using the Shapiro-Wilk test. The categorical variables, including gender, smoking status, groups with hypertension and cardiovascular risks and so on were presented in frequencies, and percentages.

Variables of normal distribution were compared on one-way analysis of variance (ANOVA) of the markers of oxidative stress in the low-risk, moderate-risk, and high-risk groups on the risk of cardiovascular diseases (Golabi *et al*., 2022). Where ANOVA showed any levels of statistical significance, post hoc tukey tests were utilized to locate the difference in the intergroups. The Kruskal-Wallis test was employed in the instance of non-Normally distributed data.

The strength and direction of the relationship between the oxidative stress markers (TAC, TOS and GSH) and cardiovascular risk score were also tested using Pearson correlation coefficient. The concept of correlation coefficient (r) was perceived as weak (0.1-0.3), moderate (0.3-0.5) or strong (>0.5) (Anwar *et al*., 2025). Oxidative stress markers predictive value was determined using linear regression analysis with control of the potential confounding variables (age, BMI, and smoking status) of data.

Statistical significance of p-value 0.05 was considered to be significant. Calculations of the appropriate confidence intervals have been made at a level of 95. All the tests were two-tailed. The results were summarized in the table form and complemented with the appropriate statistical measurements to simplify and facilitate their understanding (Polat *et al*., 2025).

RESULTS

Baseline Characteristics

A total of 140 patients with confirmed COPD were included in the study. The mean age of the participants was 61.4 ± 9.2 years. Among them, 91 were males (65%) and 49 were females (35%), ensuring that the total sample size remained consistent (n = 140). The mean body mass index (BMI) was 25.6 ± 4.1 kg/m². A majority of the participants were smokers (101 patients, 72%). Hypertension was present in 60 patients (43%), while dyslipidaemia was observed in 55 patients (39%).

Table 1. Baseline Demographic and Clinical Characteristics (n = 140)

Variable	Mean ± SD / n (%)
Age (years)	61.4 ± 9.2
Male	91 (65%)
Female	49 (35%)
BMI (kg/m²)	25.6 ± 4.1
Smokers	101 (72%)
Hypertension	60 (43%)
Dyslipidaemia	55 (39%)

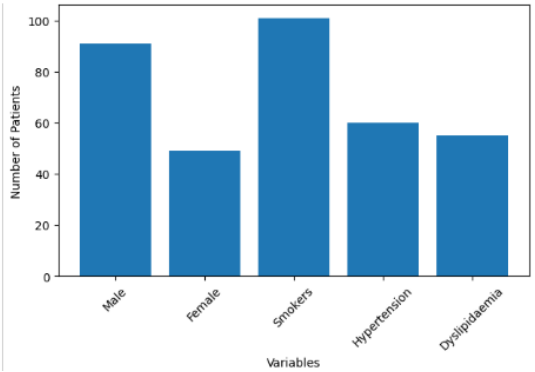

Figure: Baseline Demographic and Clinical Characteristics (n = 140)

Oxidative Stress Profile

Assessment of oxidative stress markers demonstrated a marked oxidative imbalance among COPD patients. The mean total oxidant status (TOS) was 22.6 ± 6.4 μmol H_2O_2 equivalent/L. In contrast, antioxidant parameters were reduced, with a mean total antioxidant capacity (TAC) of 1.12 ± 0.29 mmol Trolox equivalent/L and a mean reduced glutathione (GSH) level of 3.1 ± 0.8 μmol/L.

Table 2. Oxidative Stress Markers in COPD Patients (n = 140)

Parameter	Mean ± SD
TOS (μmol H_2O_2 Eq/L)	22.6 ± 6.4
TAC (mmol Trolox Eq/L)	1.12 ± 0.29
GSH (μmol/L)	3.1 ± 0.8

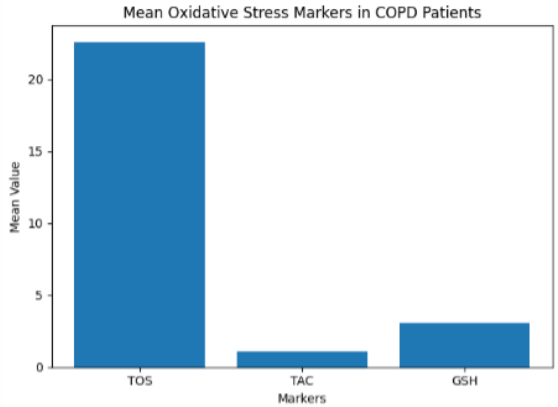

Table 2. Oxidative Stress Markers in COPD Patients (n = 140)

Oxidative Markers across Cardiovascular Risk Categories

When oxidative stress markers were compared across cardiovascular risk categories as determined by the Framingham Risk Score, statistically significant differences were observed. The high-risk group demonstrated significantly elevated TOS levels and significantly reduced TAC and GSH levels compared to the low- and moderate-risk groups ($p < 0.001$ for all comparisons).

Table 3. Oxidative Stress Markers across Cardiovascular Risk Groups

Marker	Low Risk	Moderate Risk	High Risk	P-Value
TOS (μmol H_2O_2 Eq/L)	17.2	21.8	28.5	<0.001
TAC (mmol Trolox Eq/L)	1.35	1.12	0.89	<0.001
GSH (μmol/L)	3.8	3.1	2.4	<0.001

These findings indicate that oxidative burden increases progressively with higher cardiovascular risk, while antioxidant defense capacity declines correspondingly.

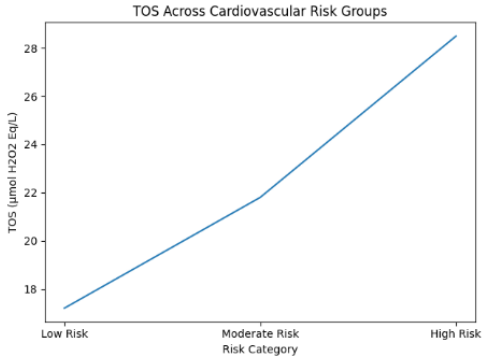

Table 3. Oxidative Stress Markers across Cardiovascular Risk Groups

Correlation Analysis

Pearson correlation analysis demonstrated a strong positive correlation between TOS and the Framingham Risk Score ($r = 0.58$, $p < 0.001$). In contrast, TAC and GSH exhibited moderate negative correlations with cardiovascular risk ($r = -0.46$ and $r = -0.49$, respectively; $p < 0.001$ for both).

Table 4. Correlation of Oxidative Stress Markers with Cardiovascular Risk

Marker	Pearson R	P-Value
TOS	0.58	<0.001
TAC	−0.46	<0.001
GSH	−0.49	<0.001

These results confirm that increased oxidant status is associated with higher cardiovascular risk, whereas stronger antioxidant defense is associated with lower cardiovascular risk in COPD patients.

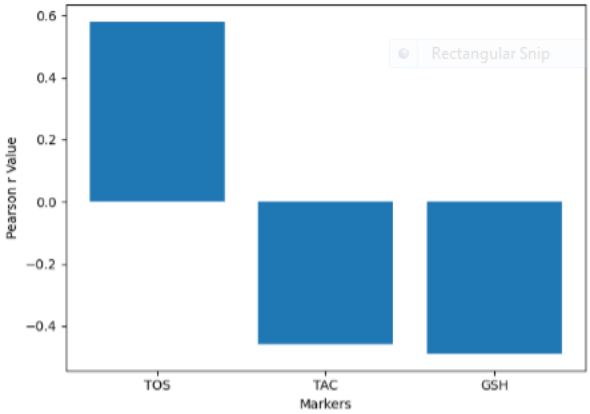

Figure: Correlation of Oxidative Stress Markers with Cardiovascular Risk

DISCUSSION

This study mainly demonstrates a very much significant oxidative imbalance in the COPD patients, characterized by the elevated TOS and the reduced TAC and the GSH levels. These are the results that validate the role of oxidative stress in the pathogenesis of COPD.12 (Fallahzadeh *et al.*, 2026). Strong associations between TOS and cardiovascular risk are high implying that the oxidative load may lead to the dysfunction of endothelium and atherosclerosis.13 TAC and GSH is declared as poor having shown poor antioxidant defines that may instigate the rapid injury to vascularity.14 (Fallahzadeh *et al.*, 2026). Our findings are in line with the past research studies that have discovered the existence of high amounts of oxidative stress in COPD along with its adverse effects on the body that are apparent as heart-related issues.15

CONCLUSION

Patients with COPD experience oxidative stress with TOS high and low of TAC and GSH. The presence of both COPD and cardiovascular disease may have an underlying mechanistic relationship that is oxidative stress. Regular assessment of the indicators of oxidative stress can improve the classification of cardiovascular risks among the COPD patients.

Possible Mechanisms

The observed association between the oxidative stress and the increased cardiovascular risk in patients with COPD may be explained by several interrelated biological mechanisms. Overproduction of the reactive oxygen species (ROS) may cause endothelial damage directly, thereby causing vascular functioning impairment and facilitating the atherosclerotic changes. Senescence of lipids through lipid peroxidation is also promoted by oxidative stress leading to the formation of oxidized low-density lipoproteins, which lead to the development of plaque and vascular stiffness. Moreover, oxidative imbalance caused by the loss of nitric oxide decreases vasodilation and protective properties of nitric oxide, which contributes to endothelial cell dysfunction and vascular resistance. Continuous systemic inflammation, which COPD is typified with, further enhances oxidative damage and leads to cardiovascular remodelling and disease development.

Clinical Implications

The findings of this particular study suggest that the assessment of the oxidative stress markers such as TOS, TAC, and GSH may have some level of potential clinical value in COPD patients. Early detection of oxidative markers would be useful to detect people at high cardiovascular risks before cardiovascular complications appear. Use of oxidative stress assessment in cardiovascular risk stratification models could have a beneficial effect in increasing the predictive validity in this high-risk population. Besides, the possible therapeutic relevance of antioxidant interventions should be taken into consideration because the process of oxidative imbalance regulation, used in the prevention of cardiovascular morbidity in the past, should be considered in the studies (Anwar et al., 2024).

Limitations

Several limitations must be well acknowledged at the time of the findings of this study. The cross-sectional design restricts the actual ability to mainly establish causal relationships between oxidative stress and also the cardiovascular risk. The research was carried out in one centre and therefore, might be restricted on how the study can be generalized to larger populations. The lack of a healthy control group prevents comparative assessment of the level of oxidative stress in patients with COPD and people without the disease. Also, it has a short follow-up time, incapable of determining the outcomes of oxidative stress on the cardiovascular system in the long term and using oxidative markers as predictors of cardiovascular outcomes over time (Alameri et al., 2024).

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this study. The research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Authors' Contribution

All authors contributed significantly to the conception and design of the study. The study protocol was developed collaboratively, and ethical approval was obtained with the involvement of all authors. Data collection was carried out by the primary investigators, including patient recruitment, clinical assessment, and sample acquisition. Laboratory analysis of oxidative stress markers (TAC, TOS, and GSH) was performed with the support of the research team under standardized conditions.

Statistical analysis and interpretation of data were conducted jointly, with particular emphasis on correlation and regression analysis. The initial draft of the manuscript was prepared by the lead author, and all authors critically reviewed, revised, and approved the final version of the manuscript. All authors agree to be accountable for all aspects of the work and ensure the integrity and accuracy of the study.

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