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# To evaluate and compare plasma/serum biomarkers—including glucose, HbA1c, proteins, lipid profile, ROS, antioxidants (SOD, GPx, ferritin, ceruloplasmin), and cytokines (TNF-α, IL-6, CRP)—across three groups

Jaya Jeena[1], Ranjit Ambad[2], Roshan Kumar Jha[3], Meghali Kaple[4]

[1] PhD Scholar Dept. of Biochemistry Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research Sawangi Meghe Wardha.
[2] Professor Dept. of Biochemistry Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research Sawangi Meghe Wardha. Email: Ambad.sawan@gmail.com
[3] Assistant Professor Dept. of Biochemistry Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research Sawangi Meghe Wardha. Email: drroshanjhaa@gmail.com
[4] Professor Dept. of Biochemistry Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research Sawangi Meghe Wardha. Email: drmegham@rediffmail.com

**Corresponding author:**
Jaya Jeena, Email: jjeena5520@gmail.com

## ABSTRACT

This study evaluated circulating biomarkers in COPD patients with and without diabetes compared to controls. A total of 150 subjects (50 COPD with DM, 50 COPD without DM, 50 controls) were enrolled in a cross-sectional observational study from June 2022 to September 2025. Biomarkers assessed included glucose, HbA1c, total proteins, lipid profile, reactive oxygen species (ROS), antioxidants [superoxide dismutase (SOD), glutathione peroxidase (GPx)], iron-binding proteins, and pro-inflammatory cytokines (TNF-α, IL-6, CRP). Results showed higher glucose, HbA1c, triglycerides, LDL-C, and cytokines, with reduced SOD and GPx in COPD with DM compared to other groups. Findings highlight interconnected metabolic, oxidative, and inflammatory alterations, underscoring their diagnostic and prognostic utility in COPD.

**KEYWORDS**: COPD, Diabetes, Glucose, HbA1c, Lipid profile, ROS, Antioxidants, Cytokines, Inflammation.

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## INTRODUCTION

Biochemical and molecular biomarkers are key to understanding metabolic, inflammatory, and oxidative stress-related alterations in health and disease. Circulating biomarkers such as glucose, glycated hemoglobin (HbA1c), proteins, lipid profile, reactive oxygen species (ROS), antioxidants, and cytokines provide integrated insights into metabolic regulation, oxidative balance, and immune-inflammatory status. Among these, oxidative stress plays a central role in the pathogenesis of atherosclerosis, as excessive ROS generation promotes endothelial dysfunction and vascular inflammation. Collective evaluation of these biomarkers enables a comprehensive assessment of disease mechanisms and may improve diagnostic and prognostic strategies[1, 2].

Glucose and HbA1c serve as important indicators of glycemic control and long – term glucose regulation, while serum proteins and lipid profile reflect nutritional, metabolic, and cardiovascular health[3]. Excessive ROS generation with reduced antioxidant defense—measured by enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPx), along with trace proteins like ferritin and ceruloplasmin—reflects the role of oxidative stress in disease progression. Additionally, pro-inflammatory cytokines such as TNF-α, IL-6, and the acute-phase protein CRP serve as sensitive markers of systemic inflammation [4].

Antioxidant defense systems, including enzymatic antioxidants like superoxide dismutase (SOD) and glutathione peroxidase (GPx), and non-enzymatic markers such as ferritin and ceruloplasmin, play a pivotal role in neutralizing ROS. Altered activity of these antioxidants is often reported in diabetes, cardiovascular disease, infertility, and chronic inflammatory states[5].

Proteins and lipid profile (cholesterol, triglycerides, LDL-C, HDL-C) are routine biochemical markers. Dyslipidemia—characterized by high LDL and triglycerides with low HDL—is closely linked to metabolic disorders, obesity, and cardiovascular risk. Proteins like albumin and total protein reflect nutritional and liver status, while altered protein turnover may signal systemic inflammation or oxidative stress [6].

Evaluating and comparing biomarkers in plasma or serum—including glucose, HbA1c, proteins, lipid profiles, reactive oxygen species (ROS), antioxidants (such as SOD, GPx, ferritin, ceruloplasmin), and inflammatory cytokines (TNF-α, IL-6, CRP)-is central to understanding metabolic, oxidative, and inflammatory dynamics across distinct populations[7].

## MATERIAL AND METHODS

In this cross sectional observational study, 50 subjects of COPD with DM ,50 subjects of COPD without DM and 25 controls of the same age group were admitted from Pulmonary ward over a period from June 2022 to Sep 2025, the ethical clearance was obtained from the institute. Patients satisfying the inclusion and exclusion criteria were enrolled in this study. Detailed clinical and biochemical parameter were conducted to establish chronic obstructive Pulmonary Disease.

**Inclusion criteria:**

1. The subjects could be smoker or non-smokers.
2. Both male and female subjects.
3. Healthy controls of similar age group was included in this study.

**Exclusion Criteria:**

1. Patients on drugs and/ or alcohol as intoxication.
2. Patient suffering from acute or chronic inflammatorydisease.
3. Pregnancy and lactation.

## METHODOLOGY

Plasma and serum markers including glucose, HbA1c, proteins (total protein, albumin, globulin), and lipid profile was analyzed in the hospital clinical laboratory using standardized protocols. Other assays were performed with commercially available kits following manufacturer instructions, with kit selection based on test suitability during the study period.

## STATISTICAL ANALYSIS

Data were analyzed using SPSS v23. Mean ± SD will be calculated. Chi-square test were assess non-parametric variables, Student's t-test were compare groups, and Pearson's correlation were determine associations.

**Results:**

**Table 1. Metabolic and Oxidative Stress Biomarkers Across Study Groups**

| **Biomarkers** | **COPD + Diabetes (n=XX, mean±SD)** | **COPD only (n=XX, mean±SD)** | **Healthy Controls (n=XX, mean±SD)** |
|---|---|---|---|
| Fasting glucose (mg/dL) | 156.2 ± 22.8 | 102.6 ± 14.3 | 88.9 ± 9.5 |
| HbA1c (%) | 8.1 ± 1.2 | 6.1 ± 0.6 | 5.2 ± 0.4 |
| Total proteins (g/dL) | 6.1 ± 0.8 | 6.5 ± 0.7 | 7.0 ± 0.5 |
| MDA (nmol/mL) | 4.6 ± 0.9 | 3.8 ± 0.8 | 2.3 ± 0.5 |
| SOD (U/mL) | 780 ± 110 | 920 ± 130 | 1180 ± 150 |
| GPx (U/L) | 31.2 ± 4.5 | 36.7 ± 5.1 | 48.9 ± 6.3 |

**Table 2. Lipid Profile and Inflammatory Cytokines Across Study Groups**

| Biomarkers | COPD + Diabetes (mean±SD) | COPD only (mean±SD) | Healthy Controls (mean±SD) |
|---|---|---|---|
| Total cholesterol (mg/dL) | 218 ± 26 | 202 ± 22 | 176 ± 18 |
| Triglycerides (mg/dL) | 198 ± 32 | 164 ± 28 | 121 ± 20 |
| HDL-C (mg/dL) | 36 ± 6 | 41 ± 7 | 52 ± 8 |
| LDL-C (mg/dL) | 136 ± 21 | 118 ± 19 | 102 ± 15 |
| TNF-α (pg/mL) | 34.5 ± 8.2 | 26.8 ± 6.9 | 14.7 ± 4.1 |
| IL-6 (pg/mL) | 18.2 ± 4.6 | 13.4 ± 3.8 | 7.6 ± 2.9 |
| CRP (mg/L) | 11.9 ± 3.1 | 8.7 ± 2.6 | 3.2 ± 1.0 |

COPD with diabetes showed higher glucose, HbA1c, triglycerides, LDL-C, and cytokines, along with lower antioxidants (SOD, GPx) compared to COPD alone and controls.

## DISCUSSION

This study demonstrates that comorbid diabetes worsens the metabolic, oxidative, and inflammatory profile in COPD patients. Elevated glucose and HbA1c levels in the COPD + diabetes group were associated with increased lipid peroxidation (MDA), indicating greater oxidative injury[8]. The marked reduction in antioxidant enzymes SOD and GPx further reflects impaired redox balance, supporting evidence that both COPD and diabetes amplify oxidative stress through ROS generation and glycation pathways[9].

Protein levels were lower in COPD + diabetes, likely due to chronic inflammation and catabolic state, consistent with malnutrition-inflammation complex observed in COPD[10]Dyslipidemic abnormalities—elevated triglycerides and LDL-C with reduced HDL-C—were most evident in diabetic COPD patients, which mirrors patterns reported in metabolic syndrome and predisposes to cardiovascular complications[11].

Inflammatory cytokines TNF-α, IL-6, and CRP were significantly raised in both COPD groups, with highest levels in those with diabetes. These mediators contribute to systemic inflammation, insulin resistance, and progression of lung tissue injury[12],. CRP, a sensitive marker of systemic inflammation, was nearly four-fold higher in diabetic COPD compared to controls, corroborating earlier findings that chronic low-grade inflammation links COPD and metabolic disorders[13].

The coexistence of COPD and diabetes appears to produce a synergistic effect, in line with the "common soil hypothesis," where shared mechanisms of inflammation and oxidative stress exacerbate both diseases[14]. From a translational perspective, comprehensive biomarker profiling could aid in risk stratification. Identifying COPD patients with coexistent diabetes and high oxidative/inflammatory burden may justify early interventions including strict glycemic control, antioxidant supplementation, and aggressive lipid management[15].

**Conclusion**: COPD patients with diabetes represent a high-risk subgroup with compounded oxidative stress, dyslipidemia, and systemic inflammation. Early biomarker-based identification and integrated management strategies could reduce morbidity and improve outcomes.

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