

Association of Oxidative Stress, Inflammatory Markers and Lipid Profile with Chronic Obstructive Pulmonary Disease in Al-Muthanna Governorate, Iraq

Oxidative Stress,
Inflammatory
Markers and
Lipid Profile with
COPD

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ABSTRACT

Objective: To evaluate the association and correlation between oxidative stress markers, inflammatory indicators, and lipid profiles in patients diagnosed with Chronic Obstructive Pulmonary Disease.

Study Design: Case-control study

Place and Duration of Study: This study was conducted at the Specialized Private Laboratories in Al-Muthanna Governorate, Iraq between 15th January 2025 to 15th July 2025.

Methods: Two hundred participants were enrolled, including 100 patients and 100 apparently healthy control subjects. The diagnosis of chronic obstructive pulmonary disease was confirmed by specialist physicians.

Results: The significant increase in oxidative stress and inflammatory markers, including malondialdehyde, high-sensitivity C-reactive protein, tumor necrosis factor- α in the patient group compared with controls. A significant decrease was observed in albumin and zinc levels whereas no significant differences were found in magnesium. Triglyceride levels were significantly elevated, while high density lipoprotein levels were significantly reduced. However, total cholesterol showed no significant difference between the studied groups.

Conclusion: Oxidative stress, inflammation, and metabolic changes in the patient group are clearly related. Reduced albumin and zinc levels point to a weakened antioxidant defense system, whereas elevated malondialdehyde, high-sensitivity C-reactive protein and tumor necrosis factor- α levels show increased oxidative and inflammatory activity.

Key Words: Chronic obstructive pulmonary disease (COPD), Oxidative stress, Inflammatory markers, Trace elements

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INTRODUCTION

A serious worldwide health issue, chronic obstructive pulmonary disease (COPD) is mainly brought on by exposure to harmful particles or gases and is defined by ongoing respiratory symptoms and restricted airflow. COPD has long been acknowledged as a major global cause of morbidity and mortality, and its prevalence has been rising in recent decades as a result of smoking, environmental pollution, aging populations, and occupational exposures.¹⁻³

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Globally, 391.8 million persons between the ages of 30 and 79 were predicted to have COPD in 2019, with the majority (nearly 80%) residing in low- and middle-income countries (LMICs), indicating notable regional inequalities.⁴ The Americas and the Western Pacific region have the highest prevalence rates, with men often showing higher prevalence than women, while estimates show that female prevalence will rise more quickly until 2050.⁵ Despite some decreases in age-standardized death rates and disability-adjusted life years (DALYs) as a result of better healthcare and tobacco control initiatives, COPD continues to be a major cause of death, accounting for over 3 million deaths yearly and ranking sixth globally overall and fourth among those aged 50-74.⁶ Continued efforts focusing on smoking cessation, reducing air pollution, occupational hazard control, and improved diagnosis and management are critical to addressing the growing global burden of COPD, especially in under-resourced regions.⁷

The widespread, progressive lung condition known as COPD is characterized by a continuous restriction in airflow and respiratory symptoms include persistent coughing and dyspnea. Although cigarette smoking is the main cause of COPD, exposure to indoor and

outdoor air pollution, occupational hazards, genetic predisposition, respiratory illnesses in childhood, and low socioeconomic status are all important risk factors.^{2,8} Chronic inflammation, oxidative stress, airway remodeling, and accelerated lung aging are all part of the pathogenesis of emphysema and irreversible airway blockage.⁹ The primary goal of treatment is to manage symptoms with inhaled long-acting bronchodilators and corticosteroids for some individuals; nevertheless, quitting smoking is still the only intervention that has been shown to reduce the course of the condition.¹⁰ Respiratory infections, environmental exposures, previous exacerbations, and comorbidities that aggravate lung function decline and prognosis are risk factors for exacerbations.¹¹ To enhance quality of life and lower morbidity, comprehensive care also entails lifestyle modifications, pulmonary rehabilitation, and management of cardiovascular risk factors.¹²

An imbalance between oxidant (pro-oxidant) and antioxidant systems that favors oxidants and results in disrupted redox signaling or molecular damage is known as oxidative stress.¹³ More precisely, oxidative stress is a reflection of the following: Overproduction of ROS and RNS in comparison to antioxidant defenses, including hydrogen peroxide (H₂O₂), superoxide (O₂⁻), peroxy radicals, hydroxyl radical (•OH), peroxynitrite (ONOO⁻), and nitric oxide (NO•).^{14,15} A change in cellular redox couples that affect the oxidation state of lipids, proteins, and nucleic acids, such as GSH/GSSG, thiol/disulfide pairs, and NADPH/NADP⁺.^{16,17}

METHODS

This case-control study was carried out in specialized private laboratories in Al-Muthanna Governorate, Iraq between 15th January 2025 to 15th July 2025 vide letter No. 132/QM/Approval/KDJEGVVD Dated Jan 2, 2025. A total of 200 participants were enrolled in this study, including 100 patients and 100 apparently healthy control subjects. The diagnosis of chronic obstructive pulmonary disease (COPD) was confirmed by specialist physicians. Blood samples were collected from all participants after obtaining informed consent and in collaboration with the hospital laboratory staff.

The patient group consisted of 100 individuals diagnosed with COPD, including 50 males and 50 females, aged between 40 and 70 years. The control

group included 100 apparently healthy subjects (50 males and 50 females) within the same age range (40–70 years), with no history of respiratory or chronic systemic diseases. Approximately 5 mL of venous blood was collected from each participant and transferred into plain tubes. The samples were allowed to clot at room temperature and subsequently centrifuged at (3000 rpm, 10 min) to obtain serum. The separated serum was stored at -20°C until further analysis of the studied biochemical markers when not analyzed immediately.

All studied biomarkers were determined according to previously described methods in the referenced literature. Serum MDA levels were measured using a commercial enzyme-linked immunosorbent assay (ELISA) kit (Sunlong, China). hs-CRP was determined according to the method described in reference. TNF- α was measured using a sandwich enzyme-linked immunosorbent assay (ELISA) kit. Serum Alb was determined by the Bromocresol Green method using a commercial kit (Biolabo, France). Elemental concentrations in serum samples were measured using the flame atomic absorption spectrophotometric method. Serum TG were determined according to the Fossati and Prencipe method. Total cholesterol by the enzymatic method²³, and HDL-C according to the method described in reference. Microsoft Excel 2021 was used for statistical analysis. A two-way analysis of variance (two-way ANOVA) was used to assess differences between the groups under study. A statistically significant was considered P-value ≤ 0.05 .

RESULTS

The results showed a significant increase in oxidative stress and inflammatory markers, including MDA, hs-CRP, TNF- α in the patient group compared with controls (P ≤ 0.05). A significant decrease was observed in Alb and Zn levels, whereas no significant differences were found in Mg. Triglyceride levels were significantly elevated, while HDL levels were significantly reduced. However, TC showed no significant difference between the studied groups. With respect to gender differences, females exhibited significantly higher levels of hs-CRP, HDL, and TG, along with significantly (P ≤ 0.05) lower levels of Alb and Zn compared with males (Tables 1-3).

Table No. 1: Inflammatory, oxidative stress and mineral parameters in COPD patients versus healthy controls (n=200)

Group	MDA(μ mol/L)	Alb (g/dL)	hs-CRP(mg/L)	TNF- α (pg/mL)	Zinc Zn μ mol/L	Mgmmol/L
Control	1.60 \pm 0.27	4.48 \pm 0.26	1.84 \pm 0.53	3.65 \pm 0.43	15.98 \pm 1.84	0.79 \pm 0.15
COPD	4.47 \pm 0.31	3.46 \pm 0.21	5.84 \pm 0.63	7.38 \pm 1.23	10.33 \pm 1.99	0.79 \pm 0.12
LSD	0.27	0.18	0.69	0.52	0.28	0.27

Table No. 2: Serum lipid profile in COPD patients versus healthy controls (n=200)

Groups	TC (mg/dL)	TG (mg/dL)	HDL-c (mg/dL)
Control	187.20±28.46	121.56±22.13	55.69±4.83
COPD	187.81±29.32	172.62±27.25	37.06±3.06
LSD	11.92	10.88	5.67

Table No. 3: Serum parameters according to sex in control and COPD groups

Concentration	Gender	Control	COPD	LSD
	Male	n= 50	n= 50	
	Female	n= 50	n= 50	
MDA (µmol/L)	Male	1.67±0.56b	4.13±0.62a	0.35
	Female	1.54±0.62b	4.82±0.71a	0.34
	LSD	0.42	0.67	
Alb (g/dL)	Male	4.73±0.41a*	3.61±0.25b*	0.36
	Female	4.24±0.46a	3.32±0.22b	0.32
	LSD	0.45	0.43	
hs-CRP (mg/L)	Male	1.63 ±0.65b	5.27 ±0.71a	0.61
	Female	2.06 ±0.72b*	6.42±0.84a*	0.81
	LSD	0.64	0.87	
TNF-α (pg/mL)	Male	3.48±0.64b	7.64±0.63a	0.81
	Female	3.81±0.71b	7.12±0.36a	0.73
	LSD	0.61	0.65	
Zinc (µmol/L)	Male	16.35±1.68a*	11.27±2.31b*	0.25
	Female	15.61±1.44a	9.39±1.43b	0.36
	LSD	0.23	0.24	
Magnesium (mmol/L)	Male	0.88±0.09a	0.77±0.08a	0.24
	Female	0.69±0.07a	0.81±0.06a	0.22
	LSD	0.26	0.28	
TC (mg/dL)	Male	187.57±32.67a	188.27±35.74a	9.38
	Female	186.83±37.34a	187.36±26.32a	10.47
	LSD	11.34	12.03	
TG (mg/dL)	Male	120.81±30.21b	171.25±34.32a	8.03
	Female	122.32±32.11b*	173.98±35.91a*	9.17
	LSD	4.34	5.18	
HDL-C (mg/dL)	Male	54.73±4.31a	35.38±4.33b	3.15
	Female	56.64±5.78a*	38.73±4.68b*	4.63
	LSD	2.64	3.82	

DISCUSSION

The current results showed that patients had significantly higher levels of oxidative stress and inflammatory markers, such as MDA, hs-CRP, and TNF-α, than the control group. The elevated MDA level is indicative of increased lipid peroxidation brought on by an overabundance of reactive oxygen species (ROS). This suggests that the pathophysiology of the illness is significantly influenced by oxidative damage to cellular membranes. Additionally, the higher levels of TNF-α and hs-CRP indicate the existence of a persistent inflammatory state, which may exacerbate oxidative stress by activating immune cells through cytokines and producing ROS.

The notable decrease in albumin and zinc levels in relation to trace elements and serum proteins may be linked to elevated oxidative burden and inflammatory

processes.¹⁶ By binding metal ions and scavenging free radicals, albumin functions as a crucial antioxidant protein in plasma; as a result, a decrease in albumin may lower the blood's overall antioxidant capacity. Similarly, the structure and operation of several antioxidant enzymes, including superoxide dismutase (SOD), depend on zinc, an essential trace metal. Reduced zinc levels can worsen oxidative stress and weaken the antioxidant defense system. Magnesium levels, on the other hand, did not significantly change between the groups, indicating that under the settings under investigation, magnesium homeostasis may be mostly maintained or less impacted.¹⁴

The presence of metabolic disorders is further supported by the changes in the patient group's lipid profile. Impaired lipid metabolism and elevated cardiovascular risk may be the cause of the notable rise in triglyceride levels and fall in HDL cholesterol. HDL

is well-known for its anti-inflammatory and antioxidant qualities as well as its beneficial function in reverse cholesterol transfer.¹² Consequently, oxidative stress and inflammatory reactions may be made worse by low HDL levels. However, the lack of a discernible shift in total cholesterol would suggest that the metabolic disruption mostly impacts the metabolism of triglycerides rather than total cholesterol.

The current investigation also showed gender-related disparities. Compared to males, females had considerably lower albumin and zinc concentrations and considerably higher levels of hs-CRP, HDL, and TG. Hormonal effects, variances in the distribution of body fat, and gender-specific differences in inflammatory and metabolic responses could all be responsible for these discrepancies. The observed biochemical differences may be attributed to hormonal variables, especially estrogen, which may have an impact on inflammatory marker expression and lipid metabolism.⁶

Evidence showing oxidative and inflammatory processes are closely related and enhanced in different disease states is consistent with the observed rise in oxidative stress markers in patients, such as MDA, hs-CRP, and TNF- α . While unchanged magnesium (Mg) levels show specific trace element changes, declining albumin (Alb) and zinc (Zn) levels point to a decline in antioxidant defenses because both are involved in reducing oxidative damage.^{18,19} Reduced HDL cholesterol and elevated triglycerides (TG) indicate a dysregulated lipid profile that is frequently linked to inflammation and an increased risk of cardiovascular disease.^{20,21} Martínez de Toda et al²² Research demonstrating sex-specific changes in oxidative stress and inflammatory responses, potentially affected by hormonal and metabolic variables, is consistent with gender differences showing higher hs-CRP, HDL, and TG but lower Alb and Zn in females compared to males. Despite certain protective lipid profiles, females frequently show higher levels of oxidative stress and inflammatory markers, which may contribute to differences in disease susceptibility or progression between the sexes.¹⁸

CONCLUSION

Oxidative stress, inflammation, and metabolic changes in the patient group are clearly related. Reduced albumin and zinc levels point to a weakened antioxidant defense system, whereas elevated malondialdehyde (MDA), high-sensitivity C-reactive protein (hs-CRP) and tumor necrosis factor- α (TNF- α) levels show increased oxidative and inflammatory activity. Dyslipidemia which is typified by elevated triglycerides and decreased HDL may exacerbate the course of the illness and its complications. The possible involvement of oxidative stress and trace element imbalance in the disease's underlying biochemical pathways and may

offer helpful markers for tracking the state of the illness and developing treatment plans.

Author's Contribution:

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