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The lipid profile parameter in Chronic obstructive pulmonary disease patients and correlation with severity of disease

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Abstract

COPD, a chronic respiratory illness with high mortality and morbidity worldwide, may be exacerbated by dyslipidemia. The aim of study is to assess prevalence of dyslipidemia in COPD patients and to evaluate its correlation with COPD severity. Between 2023 and 2024, Baghdad teaching hospital respiratory diseases outpatient clinic recruited 70 COPD patients for cross-sectional research. Anyone over 40 with COPD was sampled. Patients' lung function and lipid levels were documented. The study of 70 COPD patients found significant differences in demographics and clinical metrics across three severity groups (A, B, E). Key findings include higher mean age and lower mean FEV1/FVC% in the most severe group E, and significant correlations between lung function metrics (FEV1%, FEV1/FVC%, PEF%) and lipid profiles. Group E also exhibited the lowest serum HDL and highest triglycerides among the groups. In conclusion; Dyslipidemia is a common condition in COPD-patients that may correlate negatively with disease severity and lung functions.

Keywords: COPD dyslipidemia, lung functions, triglycerides

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Introduction

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation due to airway and alveolar abnormalities, primarily caused by chronic inflammation from prolonged exposure to noxious particles or gases, especially cigarette smoke. This inflammation results in airway narrowing and decreased lung recoil, presenting with symptoms ranging from cough, dyspnea, and sputum production to severe respiratory failure. The etiology of COPD primarily involves cigarette smoking, though non-smokers can also develop COPD due to exposure to biomass, occupational hazards, passive smoking, or due to genetic factors like alpha-1 antitrypsin deficiency, which is particularly suspected in patients who present with liver damage and predominantly affects the lower lobes of the lungs [1-5]. COPD is prevalent in individuals over 40 years old, with global estimates suggesting 174 million cases and 3.2 million deaths in 2015, making it the third most common cause of death worldwide. Its prevalence is likely underestimated due to frequent underdiagnosis. In regions like the Middle East and North Africa, prevalence varies

significantly, with Iraq reporting a prevalence of 15.1% among adults [6,7]. Pathophysiologically, COPD involves oxidative stress and protease-antiprotease imbalances, with structural changes such as emphysema where the destruction of alveolar sacs leads to obstructive physiology and bronchitis affecting the tracheobronchial tree. The disease progression is marked by FEV1 decline and impaired gas exchange, often visualized as lung hyperinflation on imaging studies [1,2]. Acute exacerbations, typically triggered by infections or environmental irritants, are managed with corticosteroids and bronchodilators, highlighting the dynamic nature of COPD's impact on patient health [1,8]. Evaluation of COPD includes a detailed history, physical examination, and confirmatory spirometry tests. Spirometry is crucial, with a FEV1/FVC ratio less than 0.7 confirming diagnosis. The disease severity, assessed by the GOLD classification system, dictates the management strategy which involves pharmacological treatments such as bronchodilators (beta2-agonists, antimuscarinics), inhaled corticosteroids, and newer agents like PDE4 inhibitors and antibiotics for exacerbation management [9-11]. Non-pharmacological interventions include smoking cessation and pulmonary rehabilitation. The goal is to control symptoms, improve quality of life, and reduce exacerbations and mortality 2. Differential diagnoses for COPD include asthma, asthma-COPD overlap syndrome, and other conditions that mimic its symptoms such as interstitial lung diseases and cardiovascular disorders. The prognosis is variable and heavily dependent on treatment adherence and avoidance of exacerbating factors. The BODE index is often used to estimate mortality risk based on BMI, airflow obstruction, dyspnea, and exercise capacity [1,12]. The complications of COPD, including acute and chronic respiratory failure, pulmonary hypertension, and cor pulmonale, further complicate management and highlight the need for comprehensive care strategies. Patient education on inhaler use, symptom monitoring, and lifestyle adjustments form an integral part of long-term management to slow disease progression and improve outcomes [1,13]. Dyslipidemias, often found in COPD patients, exacerbate cardiovascular disease risks. They are characterized by abnormal cholesterol and triglyceride levels, impacting overall health and mortality. Managing dyslipidemia involves lifestyle changes and medications like statins, which are crucial for reducing cardiovascular disease risks in COPD patients [14,15]. Aim of study to assess prevalence of dyslipidemia in COPD patients and to evaluate its correlation with COPD severity.

Method

Between 2023 and 2024, a cross-sectional study was conducted on 70 patients with Chronic Obstructive Pulmonary Disease (COPD) at the respiratory diseases outpatient clinic of Baghdad Teaching Hospital. This study received ethical approval from the Council of Baghdad University College of Medicine – Respiratory Medicine Department. The research focused on patients over 40 years old with a confirmed diagnosis of COPD, characterized by clinical symptoms such as dyspnea, chronic cough, and/or sputum production, along with spirometric evidence of airflow limitation (post-bronchodilator FEV1/FVC less than 0.70) and stable disease. Patients excluded from the study were those in acute or recent exacerbations (within the last three months), those with other respiratory diseases like asthma or tuberculosis, those with uncontrolled conditions such as diabetes mellitus,

congestive heart failure, end-stage renal disease, hepatic failure, and connective tissue diseases, as well as those unwilling to participate or those using lipid-lowering medications such as statins. The methodology involved obtaining verbal consent from participants before enrollment. Each patient underwent a thorough evaluation that included clinical history, physical examination, lung function tests post-bronchodilator inhalation, and lipid profile assessments. Demographic and clinical parameters were meticulously recorded. Lung function parameters such as FEV1%, FEV1/FVC%, and PEF% were measured according to the GOLD guidelines using spirometry post-bronchodilator administration. The degree of dyspnea was assessed using the modified British Medical Research Council (mMRC) scale. Patients were then classified into three groups based on the number of exacerbations and mMRC scale results using the ABE approach. Serum cholesterol, LDL, HDL, VLDL, and triglycerides were assessed using test-specific kits in a fully automated immunoassay system. Statistical analysis was conducted using Microsoft Excel 2010 and SPSS version 26. Data were presented in means with ranges for quantitative measurements and frequencies for qualitative data. Comparative analyses among the three patient groups were performed using one-way ANOVA for quantitative variables and chi-square tests for qualitative variables. Pearson Correlation tests were utilized to evaluate associations between various variables, enhancing the understanding of the relationships within the data. This comprehensive approach allowed for a detailed examination of the impact of COPD on lung function and lipid profiles, contributing valuable insights into the management and treatment of this complex disease.

Results

The study examined 70 COPD patients, distributed as 46 (65.7%) males and 24 (34.3%) females, with 46 (65.7%) currently smoking and 24 (34.3%) former smokers. These participants were categorized into three groups based on disease severity: A (n=32), B (n=28), and E (n=10). Relevant demographic and laboratory data are detailed in table 1, and figures 1, 2, and 3. Notably, the average age in group E was significantly higher than in group A (68.8 years vs. 59.06 years, p-value 0.00098). The proportion of males was significantly greater in group A compared to groups B and E (81.3% vs 50% and 60%, respectively, p-value 0.03). Group E also had a significantly lower average BMI compared to group A (21.16 kg/m^2 vs 24.34 kg/m^2, p-value 0.016).

Regarding pulmonary function tests, group E showed significantly lower mean O2 saturation%, FEV1%, FEV1/FVC%, and PEF% compared to groups A and B. Specific p-values indicated significant differences between groups for each parameter: O2 saturation% (p-value 0.002 for A vs B, p-value <0.0001 for A vs E, p-value 0.009 for B vs E); FEV1% (p-value 0.00001 for A vs B, p-value <0.00001 for A vs E, p-value 0.00001 for B vs E); FEV1/FVC% (p-value <0.00001 for A vs E, p-value 0.00001 for B vs E); PEF% (p-value 0.001 for A vs B, p-value <0.00001 for A vs E, p-value 0.00001 for B vs E).

In terms of lipid profiles, mean serum HDL was notably lower in group E compared to group A (35.02 mg/dl vs 39.90 mg/dl, p-value 0.03), and average serum triglycerides were significantly higher in

groups B and E than in group A (175.01 mg/dl and 173.24 mg/dl vs 152.81 mg/dl, p-value <0.001). Other lipid profile differences among the groups were not statistically significant.

Correlation analysis showed FEV1% was significantly associated with serum cholesterol (R -0.341, p-value 0.04) and triglycerides (R -0.395, p-value 0.01). FEV1/FVC% correlated with serum HDL (R 0.249, p-value 0.03), and PEF% negatively correlated with serum triglycerides (R -0.356, p-value 0.003), as illustrated in table 2.

Table 1.

(Demographic and laboratory parameters of the enrolled patients).

Variable	Group A (n=32)	Group B (n=28)	Group E (n=10)	*P-value*
Age (years) Mean (range)	59.06(46-68)	63.21(46-80)	68.8(65-74)	0.002
Sex n (%) (male/female)	26(81.3%)/6(18.7%)	14(50%)/14(50%)	6(60%)/4(40%)	0.03
BMI (kg/m^2) Mean (range)	24.34(18.7-30.2)	23.56(18.2-30.2)	21.16(19.3-23.4)	0.04
Smoking status n (%) (smoker/ex-smoker)	20(62.5%)/12(37.5%)	22(78.6%)/6(21.4%)	4(40%)/6(60%)	0.07
O_2 saturation (%) Mean (range)	98.68(97-99)	97.35(94-99)	96.2(95-98)	<0 .00001
FEV1(%) Mean (range)	66.62(56-91)	54.35(41-66)	42.2(37-49)	<0 .00001
FEV1/FVC (%) Mean (range)	71.12(50-95)	62.64(44-93)	44.2(40-50)	< 0.00001
PEF (%) Mean (range)	66.87(43-78)	57.28(45-67)	43.8(39-51)	< 0.00001
mMRC score Mean (range)	1.12(0-2)	2.78(2-3)	3.6(3-4)	< 0.00001
Serum Cholesterol (mg/d) Mean (range)	204.325(160.3-250.6)	216.42(180.3-294.3)	224.38(180-270)	0.16
LDL (mg/dl) Mean (range)	100.4(87.3-121.3)	102.91(87.9-113.9	103.84(92.4-120.3)	0.39
HDL (mg/dl) Mean (range)	39.90(30.6-48.9)	36.01(25-44.1)	35.02(23-46.1)	0.01
VLDL (mg/dl) Mean (range)	30.01(19.8-48)	30.19(19.6-40.4)	32.56(25.6-43.2)	0.50
Triglycerides (mg/dl) Mean (range)	152.81(129.4-181.7)	175.01(143.3-231.2)	173.24(159.3-193)	< 0.00001

**BMI; body mass index, FEV1; forced expiratory volume in one second, FVC; forced vital capacity, PEF; peak expiratory flow, mMRC; Modified Medical Research Council, LDL; low density lipoprotein, HDL; high density lipoprotein, VLDL; very low-density lipoprotein, p-value was considered significant at less than 0.05.*

*Post-Hoc analysis

- Age; *p-value* 0 .00098 for A vs E
- BMI; *p-value* 0 .016 for A vs E
- O_2 saturation%; *p-value* 0.002 for A vs B, *p-value* less than 0.0001 for A vs E, *p-value* 0.009 for B vs E
- FEV1%; *p-value* 0.00001 for A vs B, *p-value* less than 0.00001 for A vs E, *p-value* 0.00001 for B vs E
- FEV1/FVC%; *p-value* less than 0.00001 for A vs E, *p-value* 0.00001 for B vs E
- PEF%; *p-value* 0.001 for A vs B, *p-value* less than 0.00001 for A vs E, *p-value* 0.00001 for B vs E
- mMRC score; *p-value* less than 0.00001 for A vs B, *p-value* less than 0.00001 for A vs E, *p-value* 0.00023 for B vs E
- HDL; *p-value* 0 .03 for A vs E
- Triglycerides; *p-value* 0.0005 for A vs B, *p-value* less than 0.001 for A vs E

Table 2: (Pearson Correlation of lung functions and BMI with lipid profile).

	Cholesterol (R, *p-value*)	LDL (R, *p-value*)	HDL (R, *p-value*)	VLDL (R, *p-value*)	Tridycerides (R, *p-value*)
FEV1%	-0.341, 0.04	-0.132, 0.2	0.165, 0.17	-0.124, 0.30	-0.395, 0.01
FEV1/FVC%	-0.153, 0.2	-0.016, 0.89	0.249, 0.03	-0.046, 0.70	-0.147, 0.22
11PEF%	-0.140, 0.24	-0.020, 0.86	0.327, 0.006	-0.028, 0.82	-0.356, 0.003
BMI	-0.021, 0.86	0.142, 0.24	0.157, 0.19	0.128, 0.29	-0.002, 0.98

**BMI; body mass index, FEV1; forced expiratory volume in one second, FVC; forced vital capacity, PEF; peak expiratory flow, mMRC; Modified Medical Research Council, LDL; low density lipoprotein, HDL; high density lipoprotein, VLDL; very low-density lipoprotein, p-value was considered significant at less than 0.05.*

Figure 1.

(Comparison of mean age in years between the three groups)

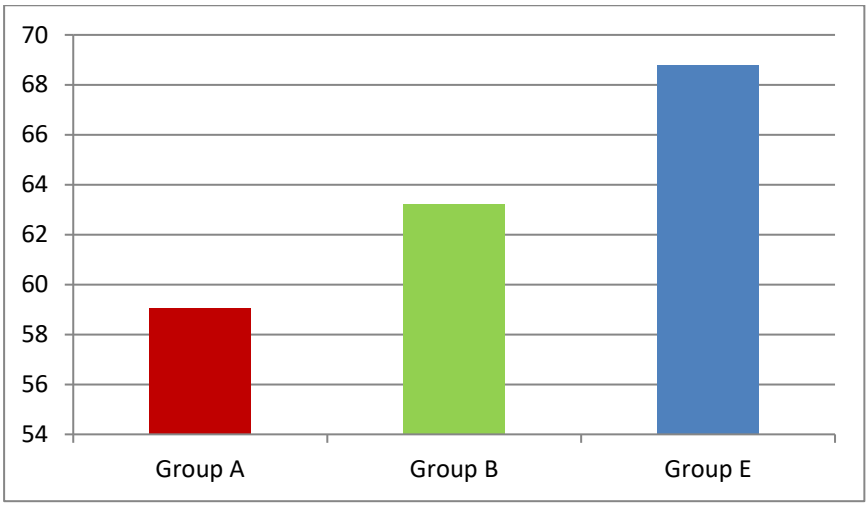


Figure 2.

(Male to female ratio in the three groups)

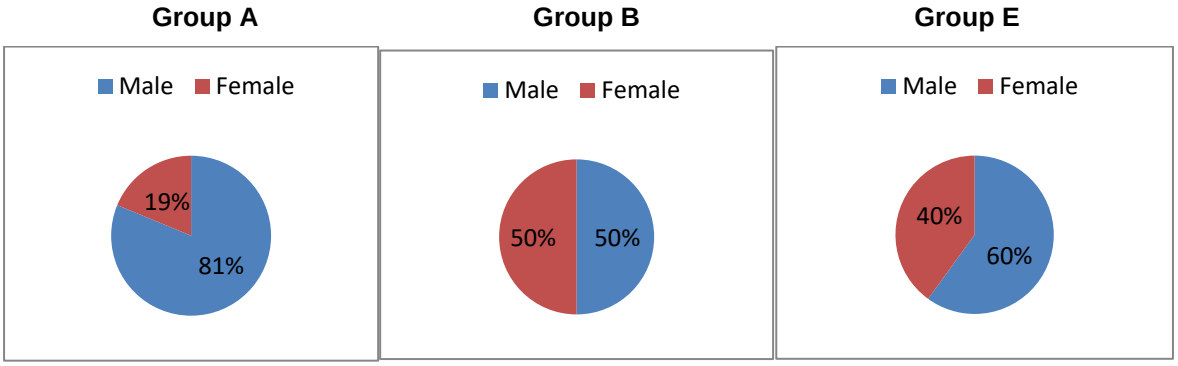
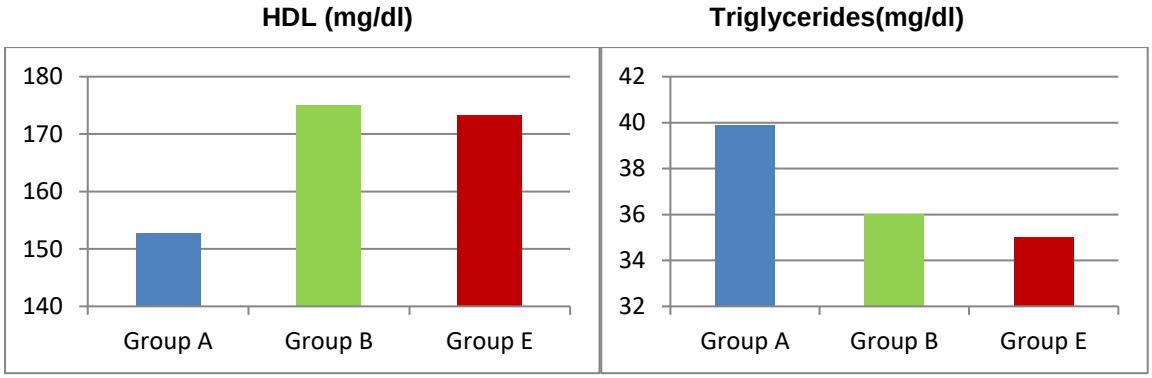


Figure 3.

(Comparison of HDL and triglycerides between the three groups)



Discussion

In this cross-sectional study conducted between 2023 and 2024, we analyzed 70 COPD patients from a respiratory diseases outpatient clinic in Baghdad, focusing on the correlation between COPD severity and various demographic and clinical parameters, including lipid profiles. Age proved to be a significant factor, with older patients predominantly in the more severe group E compared to group A. This finding is consistent with existing literature that identifies age not only as a risk factor for the development of COPD but also as a determinant for its progression, due to physiological and immunological changes in the respiratory system with aging [16-18]. The male-to-female ratio of 1.91:1 in our study reflects previous reports of male predominance in COPD populations, which could be attributed to higher smoking rates among males in Iraq [19-21]. Interestingly, despite this male predominance, a significant portion of females (75%) in our study exhibited moderate to severe disease (groups B and E), aligning with literature suggesting increased susceptibility among females to severe COPD due to factors such as lung volume differences, hormonal influences, and possibly genetic predispositions [22,23]. Nutritional status, as reflected by BMI, also correlated with disease severity. Patients in the more severe group E had a lower mean BMI compared to those in group A, a finding supported by previous studies that associate lower BMI with advanced COPD due to factors like increased energy expenditure, postprandial dyspnea, and medication effects [24]. Additionally, lower oxygen saturation percentages in group E suggest a worsening ventilation/perfusion mismatch, a characteristic complication in advanced COPD stages [25]. Lipid profiles revealed elevated levels of cholesterol, LDL, VLDL, and triglycerides, while HDL levels were suboptimal. This dyslipidemia in COPD patients may stem from chronic inflammation, physical inactivity, corticosteroid use, and the oxidative stress from smoking, all of which are known to influence lipid metabolism [26]. Notably, our findings indicated that serum HDL was significantly lower in the more severe group E compared to group A, supporting evidence from other studies that associate lower HDL levels with higher COPD severity [27]. Furthermore, our analysis showed significant correlations between serum lipid levels (cholesterol, HDL, triglycerides) and lung function parameters (FEV1, FVC), highlighting the interplay between COPD and systemic lipid abnormalities. These correlations are consistent with other studies which also reported associations between dyslipidemia and reduced pulmonary function [28]. However, the study's cross-sectional nature limits our ability to ascertain causal relationships between COPD severity and lipid profiles. The small sample size and lack of a control group also constrain the generalizability of our findings, potentially leading to over or underestimation of the observed effects. Additionally, the homogeneous geographic recruitment of participants may limit the applicability of our results to broader populations.

Conclusion

Dyslipidemia is a common condition in COPD-patients. Dyslipidemia may correlate negatively with disease severity and lung functions.

Conflict of Interest

No conflicts of interest were declared by the authors.

Financial Disclosure

The authors declared that this study has received no financial support.

Ethics Statement

Approved by local committee.

Authors' contributions

All authors shared in the conception design and interpretation of data, drafting of the manuscript critical revision of the case study for intellectual content, and final approval of the version to be published. All authors read and approved the final manuscript.

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