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Original Research Article

Cross- Sectional Study of-Lipid Profile in Patients with COPD

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Abstract:

Background and Objectives: Chronic Obstructive Pulmonary Disease (COPD) includes persistent bronchitis and emphysema, which is generally caused due to smoking of tobacco. (COPD) is an obstructive lung disorder characterized by airflow limitation that is progressive, associated with severe inflammatory response in the air passages and the lungs due to toxic particles. It interferes with normal breathing and is not fully reversible. Objective of the present study was to estimate lipid profile (Total cholesterol (TCH), Triglycerides (TG), High density lipoprotein (HDL), Low density lipoprotein (LDL), Very low-density lipoprotein (VLDL)) in patients with Chronic Obstructive Pulmonary and to find correlation of these parameters with severity of disease.

Material and Method: The study was conducted in Department of Biochemistry in collaboration with Department of Respiratory Medicine at GMCH, Purnea. Thirty-two patients diagnosed with COPD were selected from OPD and ward of Respiratory Medicine and thirty-two age and sex matched controls were included.

Conclusion: We conclude that smoking significantly affect lipid profile in COPD patients, as it increases LDL, TCH levels and decreases HDL levels. Hence all patients with COPD patients who are smoker's needs to check their lipid profile. COPD is considered as systemic disease with various co morbidities, which may affect the lipid profile. This finding may aid new interest in management of COPD.

Keywords: Lipid profile, COPD, AAT, CVD, TNF-α, CRP and GOLD.

Introduction

Chronic obstructive pulmonary disease (COPD) is an obstructive lung condition categorized by limitation of air flow in lungs which is usually advanced and is related with reactions due to severe inflammation in the lungs more likely because of toxic elements. It interferes with normal breathing and is not fully reversible. Chronic bronchitis and emphysema is related with the diagnosis of COPD. The COPD is predicted to be the third furthermost important reason of mortality by World Health Organization, by 2030. Smoking is a major risk factor and smoking is known to have hyperlipidemic effects. Oxidative stress is implicated in COPD which initiate inflammatory responses in the lungs. Effects other than pulmonary effects include weight loss and skeletal muscle dysfunction. [1] Initially few studies were carried out; Jindal found out prevalence of the disease was 6.2% and 3.9% in males and females respectively in urban areas. Another study by Ray et al reported the prevalence of the disease to be 4.08% and 2.55% respectively in males and females. Recent study by INSEARECH, (Indian Study on Epidemiology of Asthma, Respiratory Symptoms and Chronic Bronchitis in Adults) involved 85105 men and 84470 women from 11 rural and 12 urban cities, reported frequency of longstanding bronchitis in adults more than 35 years of old was 3.49 percent. Prevalence of COPD varies in India from north to south sub-continent. On the basis of the study conducted by INSEARECH, 14.84 million people were suffering from chronic bronchitis. [2] COPD is now considered as a multisystem disease, distinguished into pulmonary as well as systemic inflammation. Harmful gases and particles in the lungs cause pulmonary inflammation. In obese subjects, or those with significant adipose tissue, indicators of inflammation are Tumour Necrosis Factor alpha, Interleukin-6, Interleukin-8, Fas, Fas-L, lipopolysaccharides binding protein are increasingly synthesized by adipocytes. These inflammatory markers have great roles in the pathogenesis of COPD which features increased metabolism, weight loss, and asthenia. Systemic inflammation has become the primary focus to link COPD and cachexia and to explain the presence of COPD in susceptible subjects. Cardiovascular disease, cerebrovascular disease, osteoporosis, diabetes mellitus, depression and the metabolic syndrome are common co-morbid conditions in COPD patient. These co- morbidities increase mortality rate in COPD patient. The occurrence of COPD in India is 4.1%. [3,4,5] In patients with COPD, smoking is considered as main risk factor. Various studies have

concluded disarrangement of lipoprotein parameters as effects of tobacco smoking. HDL levels were found to be lowered in smokers than in nonsmokers. While cholesterol, triglycerides and plasma beta lipoprotein levels were found to be high in smokers than in nonsmokers. [6] The medical consequence of lipid is related generally with various lipoprotein disorders and also with coronary heart disease. Increase in level of cholesterol cause atherosclerosis which may lead to CHD. Other disorders such as genetic disorder of lipoprotein metabolism leads to dyslipidemia, familial combined hyperlipidemia, hyper- apobetalipoproteinemia, familial hypertriglyceridemia, type V hyperlipoproteniemia, familial hypercholestero- lemia, familial defective apolipoprotein B100 and hypo- poalphalipoproteinemia. Dyslipoproteinemia and determination of best treatment is dependent on evaluation of total cholesterol, triglyceride, HDL cholesterol, LDL cholesterol, collectively it is referred as a lipid panel. Normal values of LDL cholesterol less than 100 mg/dl is optimum, TCH less than 200 mg/dl is desirable, HDL cholesterol 40- 60 mg/dl and VLDL is 2-30 mg/dl. [7]

The physiological and clinical diagnosis, four different group of lipoprotein have been identified, they are namely chylomicrons, which are generated from absorption of triacylglycerols from the intestine, very low density lipoprotein (VLDL), produced in liver it helps in export of triacylglycerol, low density lipoprotein (LDL) which helps in VLDL catabolism; and high density lipoprotein (HDL) that helps in cholesterol transport, metabolism of VLDL and chylomicrons. Condition such as hypo- lipoproteinemias and hyper-liproteinemias results if there is derangement of lipoproteins. In chylomicrons and VLDL triacylglycerol is predominantly present, but in LDL and HDL phospholipids are predominantly present. Lipoproteins are involved in transportation of lipids from intestines as chylomicrons and from liver as VLDL, to most of the tissues for its oxidation and for storage in adipose tissue. [8]

Materials and Method

The study was conducted in Department of Biochemistry in collaboration with Department of Respiratory Medicine at GMCH, Purnea.

Thirty-two patients diagnosed with COPD were selected from OPD and ward of Respiratory Medicine and thirty- two age and sex matched controls were included. Study duration is One year. Group 1(cases): 32 COPD diagnosed subjects Group 2(controls): 32 age sex matched , Thirty-two patients diagnosed with COPD were selected from OPD and ward of Respiratory Medicine and thirty-two age and sex matched controls were taken. Patients were categorized on the basis of GOLD criteria into various stages by Department of Respiratory Medicine. GOLD criterion to diagnose and categorize COPD. [9]

GOLD stage	Severity	Spirometry
0	At risk	Normal
1	Mild	FEV/FVC < 0.7 & FEV1 <=80% predicted
2	Moderate	FEV/FVC< 0.7 & FEV1 =50-80% predicted
3	Severe	FEV/FVC < 0.7 & FEV1 =30-50% predicted
4	Very severe	FEV/FVC < 0.7 & FEV1 <30% predicted or FEV1 < 50% predicted with respiratory failure or signs of right heart failure

Inclusion Criteria

COPD diagnosed subjects, having smoking history of >=10 packets, cooperative and willing to participate subjects, patients with 12 hours of fasting and patients over age of 20 were included in the study.

Exclusion Criteria

Patients with autoimmune disease, bronchial asthma or cancer in last 5 year, diabetes, hypertension, Illness other than COPD; and patients with congenital cardiomyopathy were excluded.

After overnight fasting five ml of venous blood was drawn from the subject and control after written and informed consent, in dry disposable syringe under aseptic conditions and was transferred to a sterile, dry and acid washed vial for biochemical analysis. Sample was allowed to clot at room temperature, centrifugation at 2500 -3000 rpm for 15 minutes and serum was then processed for estimation of total cholesterol by CHOD-PAP Enzymatic method. [10] Triglycerides TG by glycerol phosphate oxidase. (GPO)- tinder method. [11,12,13] HDL by precipitation method. [14] LDL and VLDL was calculated by Friedewalds equation. [1]

Results

The present study was conducted in Department of Biochemistry, in collaboration with Department of Respiratory medicine, on 64 subjects among them 32 cases (Group I) and 32 controls (Group II) between the age group of 30-90 years. Each group was divided on the basis of inclusion and exclusion criteria. COPD diagnosed Cases were taken from the OPD/IPD of Department of Respiratory Medicine, and random control were taken from the healthy subject, among cases, subjects were categorized into moderate and severe category on the basis of GOLD guidelines by department of Respiratory Medicine.

Table 1: Sex wise distribution of Study subjects in Cases and control

	Cases		Control		Total	
Sex	Numbers	%	Numbers	%	Numbers	%
Females	8	12.5%	8	12.5%	16	25.0%
Males	24	37.5%	24	37.5%	48	75.0%
Total	32	50.0%	32	50.0%	64	100.0%

Table 2: Gender Wise Comparison of TCH, TG, HDL, LDL, VLDL levels in Cases

Parameters	Gender	N	Mean	Standard Deviation	P Value
TCH	Females	8	173.50	9.86	0.67
	Males	24	175.38	12.97	
TG	Females	8	132.17	32.35	0.929
	Males	24	131.00	30.30	
HDL	Females	8	38.79	7.95	
	Males	24	39.38	10.54	0.87
LDL	Females	8	112.15	11.07	0.599
	Males	24	109.80	9.95	
VLDL	Females	8	25.05	6.18	0.65
	Males	24	26.20	6.06	

The table above shows: TCH, TG, LDL and VLDL levels in male was higher as compared to female. This difference is not statistically significant. HDL level in female is lower as compared to male. This difference is not statistically significant.

Table 3: Total Cholesterol Level (TCH), Triglycerides (TG), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and Very Low-Density Lipoprotein (VLDL) Levels in Cases and Control

Parameter	Cases (n=32)		Controls (n=32)		P Value
	Mean	SD	Mean	SD	
TCH	173.97	10.529	156.06	22.552	<0.001*
TG	131.88	31.365	128.16	24.637	0.6
HDL	38.94	8.489	47.46	8.579	<0.001*
LDL	111.563	10.6923	84.163	19.2889	<0.001*
VLDL	25.338	6.0751	25.319	4.7047	0.98

Analysis of the above table shows: TCH, HDL and LDL levels were higher in cases as compared to controls and this difference is statistically very highly significant. TG and VLDL levels were higher in cases as compared to controls and this difference is not statistically significant**.**

Table 4: COPD Stages of Study subjects

	Cases	
COPD Stages	Numbers	%
Moderate	17	53.1%
Severe	15	46.9%
Total	32	100.0%

The analysis of table above shows: Moderate cases are more in number as compared to severe cases.

Table 5: TCH, TG, HDL, LDL and VLDL in Chronic Obstructive Pulmonary Disease (COPD) Stages

Parameter	COPD Stages		P-Value
	Moderate=17 Mean ± SD	Severe= 15 Mean ± SD	
TCH	173.471±10.4050	174.533±11.0056	0.781
TG	135.706 ± 28.2727	127.533±35.0262	0.471
HDL	26.965±5.5051	23.493 ±6.3425	<0.05**
LDL	104.224±4.8311	119.880±9.3249	<0.01*
VLDL	26.96±5.5	23.49±6.34	<0.11

Analysis of the above table shows: TCH level was higher in severe cases as compared moderate and this difference is not statistically significant.TG LDL and VLDL levels were higher in moderate cases as compared to severe cases and this difference is not statistically significant. And HDL level is lower in severe cases of COPD, and this difference is statistically significant.

Discussion

The COPD is a sickness which usually affects middle aged or elderly population more than 30 years of Age group. With the progression of age the lung function slowly decline and a variety of other risk factor may lead to development of the disease. In the present study, themajority i.e. 81.3 % of the patients were within the age group of 51-70 years. (table1). [3] In present study, the prevalence rates of COPD in males and females were 75% and 25%, respectively, rendering a male-female ratio of 3:1 (table 2). [15] The COPD is a male governing disease, prevalence of the disease may be high in males because of more smoking in this gender. [16] Prevalence of COPD, in females is undefined. In India, cooking is done by using cow dung, wood and other biomass fuel in inadequately ventilated kitchen, generally in rural areas. Behera and Jindal reported thirteen percent of non-smoking women suffering from COPD due to domestic cooking. [17] Jindal et al study reported, exposure to solid fuel combustion along with smoking of tobacco were more likely to cause COPD. [18] This finding shows domestic environmental factors may be of great importance in the etiology of COPD. This can be a possible factor for the development of COPD among the females. Most of the previous studies suggest that COPD prevalence and mortality is greater among males than females, but data from developed countries shows that prevalence of disease is almost equal in males and females reflecting the changing patterns of tobacco smoking. [18,19] In present study moderate cases were more in number as compared to severe cases of COPD. Smoking is a very important risk factor for COPD, seen in 85% of patients who develop COPD. Smoking affects the lipid profile in the following ways. Cholesterol, triglycerides, and plasma β- lipoprotein concentration are elevated, and HDL cholesterol is lowered in smokers as compared to non- smokers. FFA concentration is variable, but there is immediate increase of FFA, through stimulation of adrenal medulla by nicotine. As it increases concentration of epinephrine in plasma and excretion of catecholamine and their metabolites in urine. In the present study all males and females were smokers, but Thiruvengadam et al (1977) study group, who had smoking history in all male. [21] Smoking leads to the increase of LDL cholesterol, triglycerides, and VLDL. In addition, this practice results in the decrease of HDL. Though, the lipid profile has not been well categorized in the COPD patients yet. It is unidentified if dyslipidemia is an additional independent factor that might explain the increased risk of cardiovascular morbidity and mortality in the COPD patients.[3] Table 3 depict the analysis of total cholesterol, triglycerides, HDL, LDL, VLDL in cases and controls. Result was statistically significant in between cases and controls of total cholesterol ($p<0.001$), HDL ($p<0.001$) and LDL ($p<0.001$), and statistically not significant in between cases and controls of triglycerides ($p=0.6$) and VLDL ($p=0.98$). In Lucas et al study the incidence rates of dyslipidemia were 48.3% and 31.7% in the COPD patients and control group, respectively ($P=0.001$). In a present study very severe COPD group have significantly higher average values of cholesterol ($p = 0.039$) while the values of LDL and HDL were insignificant different in the group with severe and very severe COPD ($p = 0.66$ and $p = 0.11$ respectively). Nirajan et. Al. (2011) study reported no significant differences in the lipid profile concentrations and even with severe airflow obstruction, had a little lower serum concentrations of triglycerides. [6] The recent study showed that current smoking depression and dyslipidemia were more prevalent among the patient with mild to moderate COPD ($p =0.008$) than among the patient with severe to very severe COPD ($p<0.02$).[8] In study conducted in tertiary care hospital LDL level were 114.89±19.61 and 96.22±19.96 in COPD cases and control group respectively, which was significant ($p=0.05$). In Mitra et al study, it was found that lipid profile parameter were significantly positive correlated and HDL is significantly negatively correlated with chemokine IL-8 in COPD. That means there is relationship between inflammation and dyslipidemia on COPD as LDL stimulates smooth muscle cells.In the present study, the lipid profile was performed on all 64 patients, and the variables were correlated with COPD severity. According to the results, the mean total cholesterol levels were 173.471 ± 10.4050 and 174.533 ± 11.0056 mg/dl in moderate and severe stages, respectively, which was indicative of a non-significant difference between these groups in this regard ($P=0.0781$). Consequently, the level of cholesterol was high in the severe stage COPD patients, which may due to their life style modifications, such as restricted diet, poor lung function and ex-smoker status. [4,16] In present study, high triglycerides, low HDL cholesterol, and high LDL cholesterol, and low TCH were observed, especially in severe stage in comparison to moderate stage of COPD patients; however, these values were statistically significant in HDL and LDL parameters among COPD, moderate and severe stages. In Present study LDL level is 111.563 ±10.692 significant, when compared with controls but VLDL is normal. This may be due to present study group had 100% of smokers. We found a statistically significant difference in mean serum levels of LDL between the patients and the controls. Modini Venkat Rao et al study also shows significant elevated level of LDL and normal level of VLDL, as found in our study. Even though Begum K and colleagues showed that all lipid parameters including TG, TC, LDL, and HDL are elevated in COPD patients, there are studies that have shown the serum of lipid parameters are not different from healthy controls. [16] TCH will increase possibly because excess amount of TCH impairs surfactant function which can result

different lung injury. TG and VLDL very significant positive correlation as TG increases there is increase in VLDL. This may be possibly because VLDL contains highest amount of triglycerides, VLDL is solely dependent on triglycerides as suggested by Friedewalds formula.[15] there were various other correlations found to be seen in our study which were unable to clarify, how derangement of lipid profile was correlated with the severity of disease. Furthermore studies are required to find out the role of smoking and derangement of lipid parameters in COPD patients.

Conclusion

From present study we conclude that smoking significantly affect lipid profile in COPD patients, as it increases LDL levels and decreases HDL levels. Hence all patients with COPD patients who are smoker's needs to check their lipid profile. COPD is considered as systemic disease with various co morbidities, which may affect the lipid profile. In present study high TCH, high LDL and low HDL was observed. This may be due to patients having smoking history. However derangement of lipid profile was not highly significantly correlated with severity of disease. Further studies are needed to confirm role of smoking and derangement of lipid parameters in patients with COPD.

References

1. Who.int. (2019). WHO | COPD: Definition.
2. Vijayanan VK. Chronic obstructive pulmonary disease. Ind J Med Res. 2013 Feb;137 (2):251-269.
3. Kumar JB, Pasari N, Songra A, Bajpai A. The Lipid Profile Parameter in Chronic Obstructive Pulmonary disease and its correlation with severity of disease. J Cardiothoracic Med. 2017;5(2):564-68.
4. Zafirova-Ivanovska B, Stojkovikj J, Dokikj D, Anastasova S, Debresliovska A, Zenjel S, et al. The level of cholesterolin COPD patients with severe and very severe stage of disease. OA Maced J Med Sci. 2016;4(2):277-82.
5. Nillawar NA, Joshi BK, Patil BS, Bhadrapurkar SJ, Bhadrapurkar JS. Evaluation of HS-CRP and Lipid Profile in COPD. JCDR. 2013, 7(5):801-3.
6. Nirajan MR, Dadapeer K. Lipoprotein Profile In Patients With Chronic Obstructive Pulmonary Disease in a tertiary care hospital in south India. JCDR. 2011;5(5):990-93.
7. Burtis CA, Ashwood ER, Bruns DE. Tietz fundamentals of clinical chemistry. St. Louis, Missouri: Saunders. 2008, 6th ed. 402-27.
8. Murray RK, Bender DA, Botham KM, Kennelly PJ, Rodwell VW, Weil PA. Harper's illustrated biochemistry. McGraw-Hill Companies, Inc. 2009;28th ed:285-475.
9. Chronic Obstructive Pulmonary Disease. Available at: https://goldcopd.org> uploads> 2016/12(Accessed 28 Jan.2019)
10. Roeschlau P, Brent E, Gruber W. Enzymatic determination of total cholesterol in serum. Clin Chem Clin Biochem. 1974;12(5):226-8.
11. Product data sheet, Triglyceride-G Code No 997- 69801, Wako pure Chemical Industries Ltd. Dallas TX.
12. McGowan MW, Artiss JD, Strandbergh DR, Zak B. A Peroxidase coupled method for the colorimetric determination of serum triglycerides. Clin Chem. 1983;29(3):538-42.
13. Fossati P, Principle L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem. 1982;28(10):2077-80.
14. Burstein M, Scholnick HR, Morfin R. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. J Lipid Res. 1970;11(6):583-95.
15. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low- density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-04.
16. Rao MV, Raghu S, Kiran PS, Rao H. A study of lipid profile in Chronic Obstructive Pulmonary Disease. J Evol Med D Sci. 2015;4(42): 7287-96.
17. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. Chest. 1991;100(2):385-88.
18. Pride NB, Stockley RA. Chronic obstructive pulmonary disease. Oxford Textbook of Medicine. 2nd edition. Oxford. 1996;2766-78.
19. Jindal SK, Aggarwal AN, Chaudhry K, Chhabra SK, D'Souza GA, Gupta D, et al. A multicentric study on epidemiology of chronic obstructive pulmonary disease and its relationship with tobacco smoking and environmental tobacco smoke exposure. Indian J Chest Dis Allied Sci. 2006;48(1):23-27.