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ORIGINAL ARTICLE

A STUDY OF LIPID PROFILE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT: BACKGROUND: Chronic obstructive pulmonary disease (COPD) the third leading cause of death in the world, represents an important public health challenge that is both preventable and treatable. According to Global Initiative for Chronic Obstructive Lung Disease (GOLD), Spirometric tests, Forced Expiratory Volume in first second (FEV1) less than 80% of the expected value and forced expiratory volume in first second to the forced vital capacity ratio (FEV1/FVC) less than 70% is the diagnostic criteria for COPD. In COPD smoking is the major risk factor and smoking affects the lipid profile of COPD patients. **MATERIALS AND METHODS:** Spirometric parameters including FEV1, FEV1/FVC ratio and lipid profile was studied in 100 cases of COPD patients admitted Government Fever Hospital, Guntur and 40 nonsmoker healthy subjects were selected as a control group. They were correlated using Pearson's correlation coefficient "r". **RESULTS:** Majority of the cases are males belonging to 50-60years age group and all of them are smokers. Majority of the patients had moderate airflow limitation (GOLD Stages II and III). The lipid profile in COPD patients showed significant elevation of LDL cholesterol levels when compared to controls ($P<0.005$). **CONCLUSION:** Spirometric parameters FEV1, FEV1/FVC ratio is important to diagnose as well as to assess the severity of the disease. Smoking is an important risk factor for COPD and smoking effects the lipid profile of COPD patients. There was no correlation between lipid profile and severity of COPD.
KEYWORDS: COPD, GOLD, lipid profile, Spirometric parameters.

INTRODUCTION: According to WHO, Chronic obstructive pulmonary disease (COPD) will be the third leading cause of death in the world by 2030, represents an important public health challenge that is both preventable and treatable. Globally the COPD burden is projected increase in coming decades because of continued exposure to COPD risk factors and aging of the population.(1) Inhaled cigarette smoke and noxious particles such as smoke from biomass fuels cause lung inflammation, a normal response that appears to be modified in patients who develop COPD. In COPD, smoking is the major risk factor and smoking affects the lipid profile of COPD patients. The plasma β-lipoprotein, cholesterol and triglycerides concentration are higher and HDL cholesterol is lower in smoker than in nonsmokers.(2)

The clinical importance of hyperlipoproteinemia derives chiefly from the role of lipoproteins in atherogenesis. Abundant epidemiological evidence establishes the multi-factorial character of this disease and indicates that the effects of the multiple risk factors are at least additive. Hence an attempt was made to investigate the levels of total cholesterol (TCH), triglycerides (TG), low density lipoproteins (LDL), very low density lipoproteins (VLDL), high density lipoproteins (HDL) in COPD patients. Moreover, correlation of Forced Expiratory Volume in first second (FEV1) and FEV1/FVC ratio with lipid profile were carried out.

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AIMS AND OBJECTIVES: To study the levels of TCH, TG, LDL, VLDL, HDL and correlating FEV1 and FEV1/FVC ratio with lipid profile in patients with COPD.

MATERIALS AND METHODS:

Study Design: Descriptive prospective study.

Study Population: Patients admitted in Government Fever Hospital/Guntur medical college Guntur with COPD.

Study Period; From June 2013 to March 2015.

Inclusion Criteria: As per GOLD guidelines [3], any patient who has symptoms of chronic cough, sputum production, or dyspnoea, and/or a history of exposure to risk factors for the disease are considered and is confirmed by spirometry. The values of FEV1 less than 80% of the expected value and FEV1/FVC ratio less than 0.7 (70%) after post bronchodilator inhalation were included in this study, 40 nonsmoker healthy subjects were selected as a control group.

Exclusion Criteria: Patients with Bronchial asthma, Pulmonary tuberculosis, Bronchiectasis, Known congenital or acquired heart diseases, Diabetes mellitus and Hypertension, Patients on hypolipidemic drugs and steroids, Patients with history of familial hyperlipidemias were excluded from the study.

PROCEDURE: After applying above inclusion and exclusion criteria, the 100 patients were selected on the basis of simple random sampling method, and detailed history and clinical examination was done. Both cases and controls were subjected for investigations as described below. Chest X-ray Postero Anterior view was obtained to detect features of the emphysema and chronic bronchitis as per Simon's criteria.[4] Radiological evidence for right heart enlargement was also looked for. Spirometry was done on computerized spirometer (NDD Easyone). Spirometry was performed when the patients were clinically stable and as per ATS/ERS guidelines.[5] Written informed consent was taken from both controls and subjects.

The computerized spirometry gives age, sex, race, weight and height matched predicted and test values. The best of the three attempts was selected. Sixteen parameters were available in graphic recording of spirometry. Among these FEV1 and FEV1/FVC ratio were analyzed and categorized as per GOLD staging. The subject was asked to abstain overnight from oral and inhaled bronchodilator. After a baseline spirometry, 200µg of inhaled salbutamol was administered via a metered dose inhaler and the test was repeated after 15 minutes.

As per GOLD guidelines an increase in FEV1 that is both greater than 200 ml and12% above the pre-bronchodilator FEV1 were not taken to the study. After 12 hour of overnight fast, 5 ml blood samples were drawn in the morning before breakfast from the subjects, total cholesterol, HDL and triglycerides were directly analyzed by using standard enzymatic techniques. LDL cholesterol was calculated by using Friedewalds equation i.e: LDL cholesterol=(Total cholesterol)–(HDL cholesterol)–(Triglycerides/5). VLDL cholesterol was calculated by using the equation, VLDL cholesterol =Triglyceride/5.[6] Hb%, TC, DC, urine routine, ESR, RBS, blood urea, serum creatinine, done to all patients. Sputum for gram stain was done to patients in whom secondary infection was suspected. Sputum for Acid Fast Bacilli (AFB) was done to patients in whom clinically and radiologically pulmonary tuberculosis was suspected. Nonsmoker healthy persons were taken as control group. Spirometry was done for all control and study group.

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Statistical Analysis: Comparison of various parameters among male and female subjects with COPD were performed by "t" test, correlation between two variables were performed by Pearson's correlation coefficient "r", analysis was done by using SPSS+ 10.0computer package for statistics.

RESULTS: Hundred cases of COPD were studied and the following observations were made Out of 100 cases studied males were 88 and female patients were 12 in number, data of subjects are shown in table1. Mean age of the male patients among cases was 63.32±10.73 years and that of the female in the study was 63±10.18 years. Majority of the patients in the cases studied were males constituting 88%. Among controls males were 32 and females were 8 in number. The mean age of the males among controls was 57.87±10.20 and that of females was 57.50±8.20.

In the present study the duration of illness was ranged from 2-20 years with majority belonged to 6-10years of duration of illness, shown in table 2. The mean duration of illness in males was 9.02±4.41 years and in females was 8.83±3.76 years respectively with $p>0.005$ which was statistically not significant.

In the present study smoking was the major risk factor. All the 88 male patients were smokers. All female cases were non-smokers in our study, history of exposure to smoke of burnt fuels was present in all the female cases. $p <0.005$ which was statistically significant. There is a dose response relationship between the number of pack years of smoking and decline in lung function. In the present study duration of smoking range from 20 to 60 pack years. Majority were in 30-50 pack years of exposure.

Clinical features of subjects at the time of study were shown in table 3. As all the control group were healthy nonsmokers none of the subjects had respiratory symptoms or signs.

All the patients had normal hemoglobin levels, sputum gram staining showed gram positive and/or gram negative cocci in 14(14%) of the patients who presented with fever. Out of these 10 patients sputum culture yielded organisms such as streptococcus viridans, hemophilus influenzae, and klebsiella group. Sputum for AFB was done in 20 patients in whom clinical features and radiological features were doubtful for pulmonary tuberculosis, all patients were negative for sputum for AFB.RBS, Blood Urea and serum creatinine were normal in all the patients. 56% of patients had chronic bronchitis with emphysema on chest x-ray. 22% had chronic bronchitis, 22% had normal chest x-ray, results are shown in table 4. Chest x ray of all the control group were normal.

Though there are many spirometric parameters, the FEV1 and FEV1/FVC ratio are often considered as indices of pulmonary function in chronic obstructive pulmonary disease. FEV1– reflects the degree of airway obstruction. The mean expected FEV1among the cases studied was 2.30±0.35 litres. However the actual mean FEV1was 1.16±0.33 litres. The mean FEV1% of expected value in this study was 50.64±13.86%. FVC–reflects the change in vital capacity. The mean expected FVC was 2.92±0.43 litres. The actual mean FVC was 2.25±0.49 litres. All the control group had normal spirometric parameters.

Maximum number of patients in the present study were in stage II with 50% of the patients showing moderate air flow obstruction with a mean FEV1 of 64.08±6.64, 42% of patients had severe obstruction with a mean FEV1 of 38.17±7.12 and8% of patients had very severe obstruction of FEV1 27.78±2.01. None of the patients in the present study had mild airflow obstruction as per GOLD staging. Out of 100 cases studied, 60% of patients were present in FEV1/FVC ratio of 51-70%, 34% of patients were present in 31-50%group. 6% of patients were present in 21-30%group, results are shown in table 5.

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It was observed that mean LDL concentration among cases was 115.96±16.82 as against control group who had mean LDL concentration of 94.48±20.06 (mg%), which was statistically significant (p value<0.005). All other lipid parameters such as Total cholesterol, triglycerides, HDL cholesterol, and VLDL cholesterol were within normal range when compared to controls, shown in table 6. In the present study patients with moderate airflow obstruction had mean total cholesterol, HDL, LDL, and triglyceride concentrations are 182.64±18.80 (mg%), 38.68±13.76 (mg%), 124.44±20.96 (mg%), 138.80±48.68 (mg%) respectively. Relative risk of raised LDL in COPD patients is 6.83. The patients with severe airflow obstruction had mean total cholesterol, HDL, LDL, and triglyceride concentrations are 177.18±16.68, 40.38±7.92, 124.93±15.90, 138.68±42.56 respectively, shown in table 7. There are no significant differences in lipid profile as the severity of airflow obstruction increases. Lipid profile of smokers and nonsmokers of our study are shown in table 8.

DISCUSSION: In the present study 100 cases were selected on the basis of simple random sampling method from the chest diseases wards of Government Fever Hospital, Guntur Medical College, Guntur. COPD is the leading cause of morbidity and mortality worldwide and results in economic and social burden that is both substantial and increasing. COPD prevalence, morbidity and mortality vary across countries and across different groups within countries. COPD is the result of cumulative exposures over decades. Often the prevalence of COPD is directly related to prevalence of tobacco smoking, although in many countries outdoor, occupational and indoor air pollution (resulting from burning of wood and other biomass fuel) are major COPD risk factors. Cigarette smoking is clearly the single most important identifiable etiological factor in COPD. About 85% people with COPD develop the disease because of cigarette smoking.[7]

Age is often listed as a risk factor for COPD. As the age advances FEV1 declines and other risk factors add to the disease process.[8] It is unclear if healthy aging as such leads to COPD or if age reflects sum of cumulative effects throughout life. In the present study mean age was 63.16±10.45, which is comparable to Albert D et al of 64.0±19.00. Thiruvengadam KV et al, Kamat SR et al and VK singh et al. study group who had mean age of 55.0±18.00, 50.0±20.20, 52.0±20.00respectively which is not statistically significant when compared with present study COPD is a male dominant disease, the high prevalence in males which is due to higher prevalence of smoking in this gender, and also males are more susceptible to smoking than females.[9,10,11]

The results of our study can be compared with the similar results of studies conducted by and VK singh et al. Thus all the studies indicated a higher incidence of COPD in males. Most of the previous studies suggest that COPD prevalence and mortality 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.[12] Duration of illness in the present study ranged from 2 to 20 years with majority of patients belonging to the group of 6-10 years. These results can be compared with similar results of Thiruvengadam KV et al and Kamat SR et al study groups.

In the present study, cyanosis was present in 14 cases, pedal oedema and pursed lip breathing in 12 cases and indrawing of intercostal muscles in 10 cases and raised JVP in 10 cases and clubbing in one case. In the present study majority of patients had signs suggestive of both chronic bronchitis with emphysema and34% of cases had barrel shaped chest. Present study consists of mean value of FEV1, FVC and FEV1/FVC% are 1.16±0.33, 2.25±0.49 and 52.81±13.92 respectively.

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These results are comparable with Kamat SR et al. study group in which FEV1, FVC and FEV1/FVC% are 1.28±0.06, 1.97±0.66 and 54.9±13.9 respectively. According to GOLD criteria majority of the patients in the present study belong to moderate to severe airflow obstruction. Present study shows 60% of patients had FEV1/FVC ratio between 50-70%,

As the majority of the patient in present study belong to moderate degree of airflow obstruction, which was comparable with that of V.K. Singh et al study group in which majority consists FEV1/FVC ratio of 48.45%. Now COPD is considered a systemic inflammatory condition, possible mechanisms of systemic inflammation are: genetic and constitutional factors, spillage of respiratory inflammatory mediators into systemic circulation, systemic inflammation involving the lungs, and smoking.[13] In relation to systemic inflammatory condition in COPD, important co-morbidities are highlighted in process of the disease. One of the main co-morbidities in COPD is cardiovascular disease. Previous studies have shown that more than 50% of COPD patients have cardiovascular disease.[14]

The identified risk factors for accelerating atherosclerosis in cardiovascular system are: hypertension, diabetes mellitus, hyperlipidemia especially high LDL and low HDL, and smoking. One of the most important risk factors in cardiovascular disease is dyslipidemia [15]. The prevalence of lipid profile abnormalities in COPD are different in studies and range of 9-50%have been reported. Smoking affects the lipid profile in the following ways. The plasma β-lipoprotein, cholesterol and triglycerides concentration are higher and HDL cholesterol is lower in smoker than in nonsmokers. [16]Free fatty acid concentration tends to be variable, but inhalation during smoking produces an immediate increase of free fatty acids of about 30% through stimulation of the adrenal medulla, by nicotine which increases the concentration of epinephrine in the plasma and the urinary excretion of catecholamine and their metabolites [17] In addition, the plasma cortisol concentration may increase by as much as 40% within 5 minutes of the start of smoking, although the normal diurnal rhythmicity of cortisol is unaffected.

Smokers excrete more 5-hydroxyindole acetic acid than do nonsmokers. In this context, the mechanisms for the altered lipid profile among COPD smokers were recalled [18].Nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol as well as hepatic secretion of very low density lipoprotein (VLDL) and hence increased TGL. Smoking decreases estrogen levels and further leads to decreased HDL cholesterol concentration. Also, HDL concentration was inversely related to VLDL concentration in serum. Smoking increases insulin resistance and thus, causes hyperinsulinemia. LDL, VLDL and TGL are elevated in hyperinsulinemic conditions due to decreased activity of lipoprotein lipase. R. Gupta et al. study shows significantly higher LDL and significantly lower VLDL levels when compared to controls.[19]

Present study also shows elevated LDL significantly when compared with controls but VLDL is normal. This may be due to present study group had 88% of smokers, which increases VLDL levels also. We found a statistically significant difference in mean serum levels of LDL between the patients and the controls. Although 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.[20]

The Trigleceride levels in the patients of our study group was not elevated significantly compared to controls. In 1987, Fekete T, Mosler R. studied plasma lipoprotein fractions in 29 patients with COPD and compared with non-COPD subjects and found triglycerides were significantly lower in

COPD females only, the other parameters being almost identical. In the present study, though the HDL levels of the cases were decreased compared to controls, it was not significant.[21] These results are in contrast to the studies conducted by Floren CH et al. and Bahar et al.[22,23] In 1997, Floren CH et al. studied lipid profile in patients with COPD and found increased HDL cholesterol was due to drugs like Bambuterol (Inhaled β_2-adrenergicagonist) and concluded that Bambuterol increases HDL synthesis in the liver and intestinal wall. In 2003, Bahar Ulubap et al. studied lipid profile in 20 patients with COPD and 20 healthy controls and concluded that COPD patients do not show an atherogenetic lipid pattern and that the increased HDL levels might be related to the drugs used by these patients.[24]

The difference may be due to large number of patients compared to Bahar et al. study group and more number smokers in the present study. Don D. and S.F. Paul[25] study shows no significant differences in the LDL or HDL concentrations and however with severe airflow obstruction had slightly lower serum concentrations of triglycerides. Present study shows no significant difference in LDL, HDL and also in triglycerides levels. This is may be due to the study population studied was large (N=6629) in Don D. and S.F. Paul and the present study has done on only small (N=100) population, and Don D. and S.F. Paul study group had 73.7% of severe form of COPD patients but present study has only 50%.

LIMITATIONS OF THE STUDY: Since all the male patients in the present study were smokers it is difficult to analyze the lipid profile abnormality between smokers and nonsmokers when compared with severity of airflow obstruction. Even though the present had 12% of female patients who were not tobacco smokers, the lipid abnormality could not be compared with male patients as gender is a confounding factor.

CONCLUSIONS: Since the presence of significant co-morbidities in COPD, in this study we evaluated the serum levels of TG, TCH, HDL, LDL and VLDL in patients with COPD. Smoking is an important risk factor for COPD and smoking effects the lipid profile of COPD patients. The serum levels of LDL was significantly raised inpatients of COPD compared to healthy controls. The serum lipid profile did not correlate with the severity of air flow obstruction in COPD patients.

REFERENCES:

1. COPD predicted to be third leading cause of death in 2030; http://www.who.int/respiratory/copd/World_Health_Statistics_2008/en/; accessed on 28-04-2015.
2. Burtis A Carl, Ashwood R Edward. Lipids lipoproteins and apolipoproteins. In: Burtis A Carl, Ashwood R Edward editors, Tietz Textbook of Clinical Chemistry, 3rd edition. Philadelphia: WB Saunders Company 1993; 61-62.
3. Global Initiative for Chronic Obstructive Lung Disease. Global Strategy for Diagnosis, Management, and Prevention of COPD; http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html; accessed on 29-04-2015.
4. Simon George. High density shadows & hypertransradiances. Chapter 5.In: Simon George Editor Principles of chest X-ray diagnosis, 4th edition. Butterworths; Jaypee brothers New Delhi 1990; p. 147-172.

5. M.R. Miller, R. Crapo, J. Hankinson, V. Brusasco, F. Burgos, R. Casaburi et al; SERIES ‘‘ATS/ERS TASK FORCE: STANDARDISATION OF LUNG FUNCTION TESTING’’ Edited by V. Brusasco, R. Crapo and G. Viegi; Eur Respir J 2005; 26: 153–161.
6. Seth S. Martin, MD, Michael J. Blaha, MD, MPH, Mohamed B. Elshazly, MD, Peter P. Toth, MD, PhD, Peter O. Kwiterovich, MD, Roger S. Blumenthal, MD, and Steven R. Jones, MD; Comparison of a Novel Method vs the Friedewald Equation for Estimating Low-Density Lipoprotein Cholesterol Levels From the Standard Lipid Profile; JAMA. 2013 November 20; 310(19): 2061–2068
7. Susan Lang. Good cholesterol may be healthy for the lungs, too. Am J Epidemiol2002; 155(9):842-48.
8. Postma S Dirkje and Kerstjens AM Huib. Epidemiology and natural history of chronic obstructive pulmonary disease. In: Gibson G John, Geddes M Duncan, Costabel Ulrich, Sterk J Peter, Corrin Bryan, editors. Respiratory Medicine. 3rd ed. Saunders 2003; 2:1109-20.
9. Thiruvengadam KV, Raghavan TP, Bhardwaj KV. Survey of prevalence of chronic bronchitis in madras city. In: Viswanathan R, Jaggi OP editors. Advances in chronic obstructive lung disease. Delhi; Asthma and Bronchitis Foundation of India 1977; 59-69.
10. Kamat SR, Sama BS, Rajiv BRK et al. Indian Norms of Pulmonary Function observed values –Predictions, equation and inter correlations. JAPI 1977; 25: 531-540.
11. Singh VK and Jain SK. Effects of airflow limitation on the electrocardiogram in chronic obstructive pulmonary disease (COPD). Indian J Chest Dis & All Sci1989; 31(1):1-8.
12. Sara C Hitchman, Geoffrey T Fong. Gender empowerment and female to male smoking prevalence ratios. Bulletin of the World Health Organization 2011;89:195202.
13. Evrim Eylem AKPINAR, Serdar AKPINAR, Sibel ERTEK, Esen SAYIN, Meral GÜLHAN. Systemic inflammation and metabolic syndrome in stable COPD patients. Tuberk Toraks. 2012; 60 (3): 230-7.
14. Jurczak I, Jurczak K.Chronic obstructive pulmonary disease as a risk factor for coronary heart disease. Pol Merkur Lekarski. 2015 Jan; 38 (223): 39-41.
15. Sonestedt E, Hellstrand S, Schulz C-A, Wallström P, Drake I, et al. (2015) The Association between Carbohydrate-Rich Foods and Risk of Cardiovascular Disease Is Not Modified by Genetic Susceptibility to Dyslipidemia as Determined by 80 Validated Variants. PLoS ONE 10(4): e0126104.
16. Ľ CIBIČKOVÁ, D KARÁSEK, K LANGOVÁ, H VAVERKOVÁ, J ORSÁG, J LUKEŠ, D NOVOTNÝ. Correlation of Lipid Parameters and Markers of Insulin Resistance: Does Smoking Make a Difference?. Physiol. Res. 63 (Suppl. 3): S387-S393, 2014
17. YU Jian-Ping, HE Jin, LIU Dan, DENG Chun-Yu, ZHU Xiao-Nan, WANG Xue-Lan et al. Noradrenaline release by activation of κ-bungarotoxin-sensitive nicotinic acetylcholine receptors participates in long-term potentiation-like response induced by nicotine. Acta Physiologica Sinica, December 25, 2007, 59 (6): 814-820
18. Burtis A Carl, Ashwood R Edward. Lipids, lipoproteins and a polipoproteins. In: Burtis A Carl, Ashwood R Edward editors, Tietz Textbook of Clinical Chemistry, 3rd Edition. Philadelphia: WB Saunders Company 1993; 61-62.
19. Gupta R, Bhadoria DP, Mittal A, Bhandoria P, Gupta S. Lipid profile in obstructive airway disorders. J Assoc Physicians India 2002; 50:186-87.

20. K Begum, MK Begum, ZH Sarker, MRK Dewan, MJH Siddique. Lipid Profile Status of Chronic Obstructive Pulmonary Disease in Hospitalized Patients. Bangladesh J Med Biochem 2010; 3(2): 42-45
21. Fekete T, Mosler R. Plasma lipoproteins in chronic obstructive pulmonary disease. HormMetab Res 1987; 19(12): 661-62.
22. Floren CH, Kjellstrom T, Bauer CA. Bambuterol raises high-density lipoprotein levels in patients with hyperlipidaemia. J Intern Med 1997; 242(2):167-71.
23. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. Chest 1991; 100: 385-88.
24. Ulubab Bahar, Clmen Filiz, Eryyluaz, Buddaycy Resul, Calykodlu Mukadder. Lipid profile in patients with chronic obstructive pulmonary disease. Turkish Respiratory Journal 2003; 4(3):120-22.
25. Sin D Don, Man Paul SF why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular disease? Circulation 2003; 107:1514-19.

Age (years)	Male	Female	Total	Percentage
31-40	2	0	2	2
41-50	12	2	14	14
51-60	24	4	28	28
61-70	30	4	34	34
71-80	18	2	20	20
81-90	2	0	2	2
Total	88	12	100	100

Table 1: Age and Sex Distribution

Duration (years)	Male	Female	Total	Percentage
2-5	22	4	26	26
6-10	38	6	44	44
11-15	20	2	22	22
16-20	4	0	8	8
Total	88	12	100	100

Table 2: Duration of illness in COPD patients

CLINICAL FEATURE	Number of patients (N=100)	Percentage
Cough	100	100
Expectoration	100	100
Breathlessness	90	90
Wheezing	86	86
Fever	14	14
Cyanosis	14	14
Pedal oedema	12	12
Clubbing	2	2
Distention of neck veins	10	10
Pursed lip breathing	12	12
Intercostal indrawing	10	10

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Barrel shaped chest	34	34
Decreased chest movement	70	70
Decreased breath sounds	70	70
Crepitations	92	92
Rhonchi	88	88

Table 3: CLINICAL FEATURES

X-ray Findings	Number of patients (N=100)	Percentage
Chronic bronchitis with emphysema	56	56
Chronic bronchitis	22	22
Normal X-ray	22	22
Total	**100**	**100**

Table 4: Chest X-ray findings

Test	Range	Mean	Standard deviation
FVC (lt)	1.05-3.24	2.25	0.49
FEV1 (lt)	0.63-1.87	1.16	0.33
FEV1%	26.20-75.0	50.64	13.86
FEV1/FVC%	26.50-69.0	52.81	13.92

Table 5: Range and mean values of spirometric parameters

Lipid profile	Cases (mg%) (N=100)	Controls (mg%) (N=40)	P value
Total cholesterol	166.36±16.55	181.25±18.92	0.156(NS)
HDL	38.70±13.38	46.75±7.68	0.115(NS)
Triglycerides	140.38±43.18	134.86±34.26	0.363(NS)
VLDL	35.42±23.02	31.05±17.63	0.825(NS)
LDL	115.96±16.82	94.48±20.06	0.002(S)
Total Cholesterol/HDL	3.96±1.46	3.642±1.61	0.241(NS)

Table 6: Showing serum mean concentration of lipid parameters

Lipid profile	Mild (FEV1>80%)	Moderate (FEV1 50-80%)	Severe (FEV1<50)	P value
Total cholesterol	-	182.64±18.80	177.18±16.68	0.796(NS)
HDL	-	38.68±13.76	40.38±7.92	0.754(NS)
LDL	-	124.44±20.96	124.93±15.90	0.892(NS)
Triglycerides	-	138.80±48.68	138.68±42.56	0.324(NS)

Table 7: Showing correlation of lipid profile with severity of COPD

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Lipid profile	Smokers (n=88)	Non-smokers (mg%) (N=52) (12 subjects +40 controls)	P value
Total cholesterol	167.64±14.57	179.58±12.28	0.176(NS)
HDL	37.64±12.82	47.58±8.92	0.155(NS)
Triglycerides	146.84±44.86	132.69±36.36	0.336(NS)
VLDL	36.68±25.20	29.58±19.38	0.856(NS)
LDL	116.68±17.02	93.98±19.96	0.002(S)
Total Cholesterol/HDL	4.08±1.68	3.762±2.06	0.216(NS)

Table 8: Mean concentration of lipid parameters among smokers and non-smokers

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