Lipid Profile in Patients with Chronic Obstructive Pulmonary Disease

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Abstract

The aim of this study was to evaluate the serum level of total cholesterol, triglycerides (TG), low density lipoproteins (LDL), very low density lipoproteins (VLDL), high density lipoproteins (HDL), apolipoprotein A [Apo(a)], apoprotein B [Apo(b)] and lipoprotein [Lp(a)] in chronic obstructive pulmonary disease (COPD) patients. Twenty patients with COPD and 20 healthy controls were recruited to the study. Total cholesterol, HDL and TG levels were determined with ILAB 1800 Chemistry Analyzer using ILAB test Reagents. LDL concentration was calculated using the Friedewald Equation. VLDL concentration was calculated using VLDL-cholesterol=triglyceride/5 formula. Apo(a), Apo(b), Lp(a) concentrations were measured with the Beckman-Array 360 Nephelometers using Beckman Array® system reagents. HDL level was 53.45±16.39 mg/dl in COPD patients and 36.65±5.33mg/dl in controls (p=0.00). No relationship was observed between the increased HDL levels of the patients, the blood gas and respiratory function tests (RFT), or other parameters.

It was 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.

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Key words: *chronic obstructive pulmonary disease, plasma cholesterol, lipoprotein A, lipoprotein B, apolipoprotein A, triglycerides*

Introduction

Chronic obstructive pulmonary disease (COPD) is a state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases (1).

The clinical importance of hyperlipoproteinemia derives chiefly from the role of lipoproteins in atherogenesis. Abundant epidemiological evidence establishes the multifactorial character of this disease and indicates that the effects of the multiple risk factors are at least additive.

Risk factors that have been convincingly identified for arteriosclerosis of the coronary arteries are hyperlipidemia, arterial hypertension, cigarette smoking, diabetes mellitus, physical inactivity and a decrease in plasma HDL level (2).

The aim of this present study was to investigate the levels of total cholesterol (TCH), triglycerides (TG), low density

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Table 1. Age, smoking habits and results of RFT in the patients and controls

	Patient	Control
Age	60±11.15	53.75±11.81
FEV_1 %	38.75±24.40	87.12±44.3
FVC %	62.45±24.97	80.3±20.1
FEV_1/FVC %	71.15±19.77	83.12±32.5
FEF_{25-75} %	27.60±21.33	75.33±12.11
Smoking (p/y)	14.15±27.98	19.1±17.335
Ph	7.38±4.1	7.40±15.21
PO_2	59.64±15.59	89.11±10.7
PCO_2	48.05±10.7	45.02±22.12

FEV_1; forced expiratory volume during the first second,
FVC; forced vital capacity,
FEF_{25-75}; forced expiratory flow rate

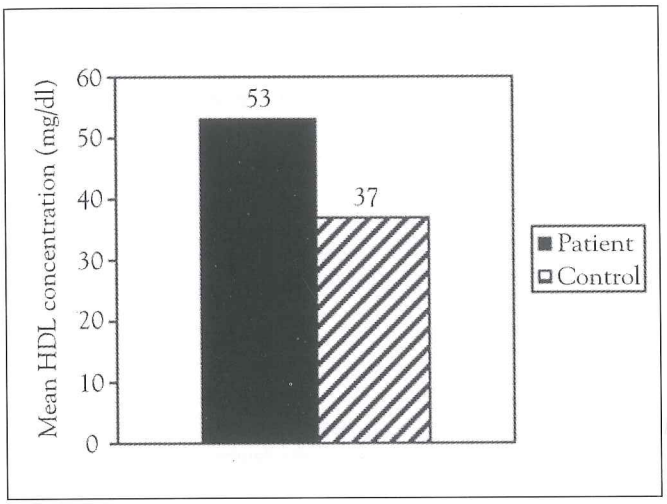

Figure 1. *HDL levels of the patients and controls.*

lipoproteins (LDL), very low density lipoproteins (VLDL), high density lipoproteins (HDL), apolipoprotein A [Apo(a)], apoprotein B [Apo(b)] and lipoprotein [Lp(a)] and to reveal the relationship between respiratory function tests (RFT) and blood gas analyses in COPD patients.

MATERIALS AND METHOD

Twenty male patients with stable moderate COPD without cardiac or other systemic diseases and 20 age and sex matched healthy controls were recruited for the study. The diagnosis of the patients were made according to the criteria of the American Thoracic Society (3). A skin prick test, total IgE, and blood eosinophil count were performed on all COPD patients to distinguish COPD from asthma. A marked decline in pulmonary function tests and a poor response to administration of inhaler bronchodilators were other features used as criteria to discard a diagnosis of asthma. All patients continued to receive their treatment ($beta_2$-adrenergic agonist, anticholinergics and their combination, steroids and theophylline) during the study.

Table 2. Serum concentrations of lipid parameters

	Patients (mg/dl)	Control (mg/dl)	P value
TCH	168.90±50.69	185.3±42.83	0.130
HDL	53.45±16.39	36.65±5.33	0.000*
LDL	104.30±52.43	122.2±40.14	0.20
VLDL	21.70±8.08	31.7±16.06	0.21
TG	109.95±43.76	150.7±58.35	0.17
Apo(a)	24.35±21.35	17.59±15.23	0.473
Apo(b)	141.10±48.11	128.15±23.66	0.490
Lp(a)	85.05±31.36	92.88±22.05	0.152

TCH; total cholesterol, TG; triglycerides, LDL; low density lipoproteins, VLDL; very low density lipoproteins, HDL; high density lipoproteins, Apo(a); apolipoprotein A, Apo(b); apoprotein B and Lp(a) lipoprotein A
*significant

After 12 hours of starvation, 5 mL blood samples were drawn from the subjects. The samples were centrifuged at 3500 RPM for 10 minutes. All samples were analyzed on the same day they were taken. Total cholesterol, HDL and TG analysis were performed with ILAB 1800 Chemistry Analyzer using ILAB test reagents. Before any procedure, HDL concentration was measured with the direct method. LDL concentration was calculated using the Friedewald Equation formulated as: LDL-cholesterol=(total cholesterol)-(HDL-cholesterol)-(triglyceride/5) (4). VLDL concentration was calculated using the following equation: VLDL-cholesterol=triglyceride/5. Apo(a), Apo(b), Lp(a) concentrations were measured with Beckman-Array 360 Nephelometers using Beckman Array® system reagents. Arterial blood gas analyses were done using the Nova Biomedical Stat M. Pulmonary function tests were performed using Sensor Medics, Vmax 22, Yorbalinda, CA. Current smokers were categorized according to pack-years (p/y) of consumption (1 pack year=20 cigarettes per day for 1 year). As smoking habits, only smoking status and pack of cigarettes smoked yearly at the beginning of the study were assessed; no information was collected with respect to the age of starting smoking.

Statistical analysis was performed using the SPSS program (Statistical Package for Social Sciences version 9.05 for windows; SPSS; Chicago, IL). Data were expressed as mean±SD and Mann-Withney tests were used for comparison. A p value of <0.05 was considered significant.

Results

The mean age of the COPD patients was 60±11.15 years and that of the controls was 53.75±11.81 years. Smoking habits expressed as p/y were 14.15±27.98 p/y in COPD patients and 19.12±17.33 p/y in controls. The age and smoking habits of our study and control groups and the results of measured parameters are shown in Tables I and II. COPD patients showed higher serum levels of HDL compared to controls (p=0.00) (Fig 1). Serum concentrations of HDL, TG, LDL, VLDL Apo(a), Apo(b), Lp(b) were similar in the patients

and controls. And no significant differences were found between the COPD patients and the controls with respect to studied variables and pulmonary function tests, Ph, PO_2, PCO_2 values, smoking habits.

Discussion

According to the results of this study, HDL concentrations in COPD patients was significantly higher than the controls ($p=0.00$). This result differs from the results of some former studies. Basili et al (5), reported high Apo B and lp(a) hand, Fekete and Mosler (6) found low triglyceride levels in female COPD patients (6). That the results are different might be due to differences in the characteristics of the patient groups. In this present study, all cases were males and their mean age was 60±11.15 years. Their treatment was not interrupted because of the study. These factors may have affected the results obtained in this study.

In former studies, it has been shown that inhaled $beta_2$-adrenergic agonist (Bambuterol) increases HDL synthesis in the liver and intestinal wall (7). Yavuz also has shown that HDL level increases in children with asthma who use inhaled steroids (8). The mechanism of this effect is not clear. In children with asthma, the effect of slow release theophylline on lipid profile has also been studied and it was suggested that the lipid profile changes with extended use of these drugs and that this might cause an increased risk in atherosclerotic coronary heart disease (9). In another study, HDL level was found to be significantly high in asthmatic patients, a finding which led to the conclusion that children with asthma and using slow release theophylline do not have atherosclerotic risk (10).

Asthma and COPD are both classified as obstructive airway diseases but they differ in their pathogenesis, clinical course and treatment. The common characteristic of these diseases is that they require long-term drug use. However, the fact that these drugs change the lipid profile may increase or decrease the atherogenic risk. The results obtained in the current study are in compliance with the results obtained in former studies with $beta_2$-agonists and inhaled steroids but the fact that the patients use more than one drug simultaneously makes it harder to reach an exact decision.

In this present study group, an increase in the HDL level was found and this increase was not related to blood gas and RFT parameters. These results suggest that the increase in HDL might be related to the drugs the patients were taking. However, since many of the patients receive a combination of drugs, a single drug cannot be held responsible for the increase in HDL. In conclusion, there is a need for more detailed studies to understand these relationships.

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