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Original Research Article

A comparative study of lipid profile in smokers and non smokers between 30 to 40 years and prediction of 10 years risk of cardiovascular disease based on Framingham scores

Mithun M.[1], Dhandapani[1]*, Venkatraman[1], Arun Daniel J.[2]

[1]Department of General Medicine, Sri Venkateshwaraa Medical College Hospital and Research Centre, Pondicherry, India
[2]Department of Community Medicine, Aarupadai Veedu Medical College, Pondicherry, India

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***Correspondence:**
Dr. Dhandapani,
E-mail: drdanfi@yahoo.co.in

ABSTRACT

Background: Atherosclerosis has been described as a lipid driven inflammatory disorder of the arterial wall. Smoking is one of the most common modifiable risk factors for atherosclerosis and is the major epidemiological factor in increasing morbidity and mortality of chronic heart diseases (CHD). The objectives of this study were based on to estimate the status of lipid profile in both smokers and non-smokers and compare with each other, to predict the 10 years risk of cardiovascular risk based on Framingham score in smokers and non-smokers.
Methods: This retrospective, case-control study was conducted among 50 smokers (cases) and 50 normal individuals (controls) attending to the department of medicine during the period between December 2016 and May 2018. The socio-demographic data and clinical history was obtained using a semi-structured questionnaire and then patients were subjected to blood investigations including estimation of lipid profile by CHOD/PAP method.
Results: The mean age of the study participants was 34.7±2.9 years. The duration of smoking among the smokers was 5.4±2.9 years on an average. There was a significant increase in serum cholesterol levels (245.6±39.8 versus 155.8±15.2 mg/dl), serum triglycerides (217.3±42.2mg/dl versus 127.4±10.6), LDL (171.1±35.2 versus 85.7±15.1 mg/dl) and VLDL (43.5±10.5 versus 15.3±5.5mg/dl) among the smokers versus non-smokers. There was a significant ($p<0.001$) decrease in HDL levels among the smokers (30.8±3.4 mg/dl) when compared with the non-smokers (44.8±5.3 mg/dl). There was a highly significant difference between Framingham risk scores of smokers and non-smokers.
Conclusions: The study established that the lipid profile was deranged towards atherogenesis among the smokers when compared to the non-smokers which was reflected in the significant increase in risk as calculated by Framingham risk score.

Keywords: Framingham risk scores, Lipid profile, Smokers and non-smokers

INTRODUCTION

Smoking is one of the most common modifiable risk factors for atherosclerosis especially when started at a much younger age group and is the major epidemiological factor in increasing morbidity and mortality of chronic heart diseases (CHD).[1] Cigarette toxins raise the plasma catecholamine levels which in turn inflict lipolytic changes and release of free fatty acid taken up by the liver.[2] Atherosclerosis has been described

as a lipid driven inflammatory disorder of the arterial wall.[3] Previous research workers have reported that tobacco smoking is associated with increased levels of total cholesterol, triglyceride, LDL-C, VLDL and decreased level of HDL-C.[4] Lipid oxidation mechanisms operate as one of the important pathways of modifying LDL, which was understood by the fact that a wide variety of structurally different antioxidants in the animal models blocked the progression of atherosclerosis.[5,6] Cigarette smoke contains an array of oxidizing agents which are included in the list of more than 4000 identified constituents and enumerable carcinogens. Several potent antioxidants such as polyphenols also have been associated in lipid oxidation pathways.[7] The previous studies also determined a dose-effect relationship between cigarette smoking and the severity of cardio-vascular diseases. The various toxins seem to accumulate lipid changes over years, and it was faster in the patients who were heavy smokers compared to the light smokers.[8]

However, in spite of all this information, there is still much controversy about which part or parts in the lipid profile are mainly altered in response to cigarette smoking. The target lipid damage if identified would be a better area to concentrate the interventions which help in averting the untoward complications related to cardio-vascular events.

METHODS

This retrospective, case-control study was conducted among 50 patients with history of smoking for at least one year (cases) and 50 normal individuals opting for master general health check-up or for any ailment not affecting the lipid levels or chronic diseases (controls) attending to the department of medicine, at the tertiary care center, Puducherry, India during the period between December 2016 and May 2018. The patients as they come to the hospital were asked for their opinion about participating in the study. A written informed consent was obtained from them after explaining the nature of the study using a participant information sheet and also verbally in their own vernacular language. The patients who did not satisfy the inclusion criteria were excluded from the study.

Matching

The patients were selected based on age 30-40 years and absence of chronic diseases. Hence most of the confounders were removed at the stage of selection. During analysis, the categorization of age, gender and other confounding variables were matched to obtain the true association between smoking and lipid profile.

History taking

The patients were enquired about the socio-demographic variables, starting, frequency and intensity of smoking, diet history and other relevant morbidities. The cardio-vascular status of the patients was also enquired.

Clinical examination

The patients were then examined clinically for any signs pertaining to impaired cardio-vascular status including body mass index, waist circumference, pulse quality and rate, pedal edema, xanthelasma, xanthomas, blood pressure, apex beat, murmurs and any signs suggestive of cardiac diseases.

Blood investigations

Blood samples for lipid profile were taken in morning with minimum of 8 hours over night fasting. Venous sample were collected in plain vacutainer and transported and processed in biochemistry lab on the same day. The samples were centrifuged (2500×g10 minutes at 4°C) and the plasma thus obtained were used for the estimation of lipid profile. The estimation of lipid profile parameters like the total cholesterol levels, serum triglycerides and HDL were done using 'CHOD/ PAP method' in vitro.

LDL cholesterol was estimated using the formula

Serum LDL = Total cholesterol (mg/dl) -(HDL (mg/dl) + TGL /5(mg/dl))

Non-HDL cholesterol is defined as the difference between total cholesterol and HDL cholesterol and includes all the cholesterol present in lipoprotein particles considered to be atherogenic. It was calculated as total cholesterol minus HDL cholesterol. Risk ratios were calculated as total cholesterol/ HDL cholesterol, LDL cholesterol/HDL cholesterol, non-HDL cholesterol/HDL cholesterol.

Statistical analysis

The data entry and analysis were done using SPSS version 21.0 (statistical software for social sciences). The statistical significance between two means was tested using independent student t-test and between categorical variables was tested using chi-square test. Pearson correlation test (r value) was used to correlate between two continuous variables. For all statistical tests of significance, a p-value of less than 0.05 was considered significant within 95% confidence limits.

RESULTS

The mean age of the study participants was 34.7±2.9 years. The mean age between the smokers (mean age= 34.7±2.9 years) and non-smokers (mean age=34.7±3.0 years) was not significantly different as tested by Student t-test. The smokers included in the study were habitual smokers for 5.4±2.9 years on an average. The number of cigarettes smoked per day was over a pack estimating 11.6±4.5 cigarettes per day. The magnitude of overweight

(32%) was higher among smokers when compared with the non-smokers (22%). The lipid profile differences between smokers and non-smokers are given in (Table 1).

Table 1: Lipid profile differences among smokers and non-smokers.

Lipid profile (Mean± SD)	Smokers (n=50)	Non-smokers (n=50)	t-test value	p-value
Cholesterol	245.6±39.8	155.8±15.2	14.9	<0.001
Triglycerides	217.3±42.2	127.4±10.6	14.6	<0.001
LDL	171.1±35.2	85.7± 15.1	15.8	<0.001
HDL	30.8±3.4	44.8 ± 5.3	15.7	<0.001
VLDL	43.5±10.5	15.3 ± 5.5	16.8	<0.001

The above table shows that there was a highly significant difference (p<0.001) in the various lipid profile parameters among the smokers and non-smokers. The mean HDL level was significantly lower among the smokers when compared with the non-smokers.

The Framingham risk score

The mean Framingham risk score of the study participants was 5.97±7.9% (minimum 0.1% and maximum 37.8%). There was a highly significant difference between Framingham risk scores of smokers and non-smokers. All the non-smokers had a low risk score of 0.1 to 1% and all smokers had high scores above 1% (Table 2).

Table 2: Framingham risk score distribution among smokers and non-smokers.

Framingham risk score	Smokers (n=50)	Non-smokers (n=50)	Total
0.1% to 1%	0 (0)	50 (100)	50 (50)
1.1% t0 10%	28 (56)	0 (0)	28 (28)
10.1% to 20%	17 (34)	0 (0)	17 (17)
20.1% to 30%	3 (6)	0 (0)	3 (3)
30.1% to 40%	2 (4)	0 (0)	2 (2)
Total	50 (100)	50 (100)	100 (100)

DISCUSSION

Smoking causes lipid alteration through various known mechanisms studied. Some include (a) nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamine resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL-C in the blood stream.[9,10] (b) fall in oestrogen levels occurs due to smoking which further leads to decreased HDL-cholesterol.[11] (c) Presence of hyper insulin anemia in smokers leads to increased cholesterol, LDL-C,VLDL-C, and TG due to decreased activity of lipoprotein lipase.[12,13] d) Consumption of a diet rich in fat and cholesterol as well as a diet low in fiber and cereal content by smokers as compared to non-smoker.[12]

In the present study, the smokers included in the study were habitual smokers with an average duration of 5.4± 2.9 years of smoking. The number of cigarettes smoked per day was over a pack estimating 11.6±4.5 cigarettes per day. In a similar study by Rastogi et al, the smokers who had a higher intensity of smoking over 10 cigarettes per day had comparatively lower HDL levels compared to those who smoked lesser.[14] There was a dose-response relationship established between the number of cigarettes smoked and LDL, triglycerides and serum cholesterol levels. Smoking is associated with severe endothelial damage as discussed before and the intensity of smoking can directly be related to the effective damage to the vasculature and faster progression of atherosclerosis.

The characteristic documented feature among the lipid profile changes in the present study among smokers was a significant (p<0.001) variation of HDL among the smokers (30.8±3.4 mg/dl) and the non-smokers (44.8±5.3 mg/dl).

Cigarette smoking is associated with reduced HDL-C level by alteration of the critical enzymes of lipid transport lowering lecithin-cholesterol acyl-transferase (LCAT) activity and altering cholesterol ester transfer protein (CETP) and hepatic lipase activity.[15]

The study by Neki NS et al, 1proved that HDL among the smokers (1.04±0.22 mmol/l) was significantly (p<0.001) lower when compared to the non-smokers (1.34±0.37 mmol/L).[16] This was exactly similar to the results documented in the present study. Thus, the level of HDL remains lower in the smoker's group. The reduction in levels of HDL has also been showed to have significant association with coronary heart diseases. Thus, smoking has a higher cardio-vascular risk due to change towards atherogenesis motivating lipid profile.

In the previous study by Neki NS et al, the results reported were similar to the results of the present study showing a significantly lower level (31.8±3.5mg/dl) of

HDL among the smokers in a clinical study of lipid profile.[16]

In the present study, there was highly significant increase in serum cholesterol levels (245.6±39.8 versus 155.8±15.2mg/dl), LDL (171.1±35.2 versus 85.7±15.1 mg/dl) and VLDL (43.5±10.5 versus 15.3±5.5 mg/dl) among the smokers versus non-smokers.

A plethora of evidence suggests that oxidation of low-density lipoprotein generates potent pro-atherogenic mediators which are more progressive in smokers.[17,18] Khurana et al, explained that lipoprotein oxidation is presumed to occur in the artery and the specific cell type(s) or mechanism(s) that may generate superoxide radicals, hydrogen peroxide or lipid peroxides outside the cell may contribute to the oxidation of LDL.[19]

CONCLUSION

The serum levels of cholesterol, triglycerides, LDL, VLDL were significantly higher and serum HDL levels were significantly lower among the smokers when compared with the non-smokers. The 10 years cardiovascular risk as measured by the Framingham risk score was significantly higher among smokers when compared to the non-smokers.

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