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TO COMPARE THE LIPID PROFILE IN SMOKERS AND NON SMOKERS

Sushitha E S[1], Sujesh Shanker[2]
Ph.D Scholar[1], MSc MLT[2]
Srinivas Institute Of Medical Sciences & Research Center, Srinivas Nagar, Mukka, Surathkal, Mangalore-574146, India

Abstract: **Background**
Smoking is an escalating public health problem especially in a developing country like India. Active smoking has been associated with endothelial dysfunction an high lipid profile including lower levels of high-density lipoprotein (HDL). Thus lipid profile is a simple investigation which helps estimate future cardiovascular morbidity and mortality among smokers

Method
Laboratory investigations are done in 50 subjects and their Total Cholesterol, Triglyceride, and HDL-C are estimated. LDL-C and VLDL are calculated by using friedewald formula This is a retrospective study done in biochemistry department of Lourdes Hospital Ernakulam, Kerala during three month period.

Result
In the present study, the results showed that the serum level of total cholesterol, triglyceride, LDL-C, and VLDL-C were significantly higher in smokers as compared to non-smokers.

Conclusion
India is one of the largest consumers of tobacco in the world. Addition of tobacco smoking is related with many health hazards. Nicotine of tobacco can be attributed to changes in lipid profile and atherogenic complications due to increasing the atherogenic lipoprotein(LDL-c) with a further decrease in antiatherogenic lipoprotein(HDL), such changes are associated to occurrence of cardiovascular disease with high risk of morbidity even mortality. So smokers should be counseled about health hazards of smoking and encouraged to quit smoking and adapt healthy lifestyle to improve the life.

I. INTRODUCTION

Smoking is an escalating public health problem especially in a developing country like India[1]. Cigarette smoke is a dominant risk factor for premature or accelerated peripheral, coronary, and cerebral atherosclerotic vascular diseases[2]. A one to threefold increase in risk of myocardial infraction (MI) has generally been noted among current cigarette smokers. Oxidative damage to unsaturated lipids is a well established general mechanism for oxidant mediated cellular injury. Cigarette smoke contains oxidizing substances among its > 4000 identified constituents. oxidative stress as a probable clinically relevant factor in cigarette smoke-related atherogenesis and cancer[1]. Smoking is considered to cause heart disease, cancer, stroke and also have close relationship with gastric ulcer, periodontal disease, sudden infant death syndrome, and metabolic syndrome. Smoking is considered to cause heart disease, cancer, stroke and also have close relationship with gastric ulcer, periodontal disease, sudden infant death syndrome[2]. Sudden death is 2- 4 times more in heavy smokers than in non smokers. It has been suggested that cigarette smoking when it is consumed more than 10/day on regular constitute a major risk factor for chronic heart disease[3]. Other pro inflammatory markers, including homocysteine, fibrinogen and C-reactive protein also appear to be adversely affected
chronic cigerete smoking[4]. Cigarette smokers have a higher risk of coronary artery disease than non smokers. Several possible explanations have been offered for this association, including altered blood coagulation,' impaired integrity of the arterial wall, and changes in blood lipid and lipoprotein

concentrations[5]. India is one of largest producer and exporter of tobacco in the world. Tons of tobacco is grown every year in India. Approximately half of it is released for local consumption . Tobacco is consumed in many ways such as chewing, smoking, etc Tobacco smoke is a complex, dynamic and reactive mixture containing an estimated 5,000 chemicals. Many of them can harm our body in various aspects An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked .Nicotine is one of the toxins present in tobacco smoke . Cigerette smoking is found to have effect on person's catecholamine & cortisol secretion . Elevated catecholamine and cortisol can alter carbohydrate and lipid metabolism in such person . Alteration in lipid metabolism may lead to dyslipidemic changes which may become a predisposing factor for atherosclerosis and ischemic heart disease leading to increased morbidity and mortality in smokers[7]. The possible mechanisms of tobacco consumption in the pathogenesis of coronary heart disease is atherogenesis. 1. Nicotine stimulation of the
adrenergic drive, thus raising the blood pressure and the myocardial oxygen demand. 2. Lipid metabolism[11]. Among the components of the gaseous phase are carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, dinitrotrioside, ammonia, hydrogen cyanide, volatile sulphur containing compounds, volatile aldehydes (formaldehyde, acetaldehyde and acrolein) alcohols and ketones. Tobacco smoke also contains various types of nitrosamines. These nitrosamines are potential carcinogenic substances and they are capable of alkylating the DNA[8]. Smoking appears to positively contribute to glucolipotoxicity and insulin resistance, which are the hallmarks of diabetes. Nicotine and the free radicals in cigarettes have been linked to accelerated β-cell apoptosis and impedance of intracellular GLUT-4 mobilisation, which may feed into hyperglycaemia associated with diabetes[9]. Active smoking has been associated with endothelial dysfunction an high lipid profile including lower levels of high-density lipoprotein (HDL) and with adverse effects on blood coagulability, including increased fibrinogen levels and platelet aggregation[10]. Thus lipid profile is a simple investigation which helps estimate future cardiovascular morbidity and mortality among smokers[6].

AIM AND OBJECTIVES

AIM

To compare the lipid profile in smokers and non smokers.

OBJECTIVE

- To detect the lipid profile in smokers.
- To detect the lipid profile in non smokers.
- To compare lipid profile between smokers and non smokers.

MATERIALS AND METHOD

The present study was conducted in last 3 month

INCLUSION CRITERIA

- The subjects who have smoking habit for atleast 5 years.
- Healthy non- smokers.
- The subjects are chosen in age groups of 20 - 60 years of age.

EXCLUSION CRITERIA

1. Subjects having diseases mentioned below known to influence blood lipids are excluded from the study

- Diabetes mellitus
- Nephritic syndrome
- Alcoholism
- Hypertension

2. History of Alcohol Intake/Drug abuse.

Investigations done are:

- Serum total cholesterol (T C)
- Serum high density lipoprotein (HDL)
- Serum low density lipoprotein (LDL)
- Serum very low density lipoprotein (VLDL)
- Serum triglyceride (TGL)

SPECIMEN COLLECTION

Collection of blood samples for biochemical assays was done after fasting for at least 12 hours. 5 ml of blood sample collected from anterior-cubital vein from each subject and serum is separated. Estimations are done in ERBA TRANSASIA.

ESTIMATION OF TOTAL CHOLESTEROL BY CHOD -POD PRINCIPLE

Cholesterol Esters $\xrightarrow{\text{Cholesterol Esterase}}$ Cholesterol +Fatty acid

Cholesterol + O_2 $\xrightarrow{\text{Cholesterol Oxidase}}$ Cholest-4-en-3-one+H202

2 H2O2 + Phenol + 4-Aminoantipyrine $\xrightarrow{\text{Peroxidase}}$ Quinoneimine dye +4H2O

REFERENCE RANGE

Blood cholesterol - <200mg\dl

DETERMINATION OF TRIGLYCERIDES BY GPO- TRINDERS METHOD, (END POINT)

PRINCIPLE Lipoprotein Lipase

Triglycerides + H_2O $\xrightarrow{\text{Glycerol Kinase}}$ Glycerol + Free Fatty Acids

Glycerol + ATP $\xrightarrow{\text{Glycerol Phosphate Oxide}}$ Glycerol -3- Phosphate + ADP

Glycerol - 3^- Phosphate + O_2 $\xrightarrow{\text{Peroxidase}}$ Dihdroxyacetone Phosphate + H_2O_2

H_2O_2+ 4 Aminoantipyrine + 3 , 5 Dichloro-2-hydroxybenzene Sulfonate $\longrightarrow$ Quinonemine dye + $2H_2O$

REFERENCE RANGE :- Triglyceride: 70-150mg/dl

DETERMINATION OF VLDL **:-** $\text{VLDL} = \frac{\text{Try glycerides (mg/dL)}}{5}$

REFERENCE RANGE VLDL: 10-130 mg\dl

DETERMINATION OF HDL DIRECT

PRINCPLE

HDL + LDL +VLDL+ Chylomicrons $\xrightarrow{\text{Polyvinyl Sulfonic Acid}}$ HDL+(LDL+VLDL+CM)

HDL $\xrightarrow{\text{Cholesterol Oxidase,Cholesterol esterase}}$ fatty acid + H_2O_2

$2H_20_2$ + 4-Amino antipyrine + N,N-Bis(4-sulfobutyl)-3-methyl anililine disodium salt $\xrightarrow{\text{Peroxidase}}$ Quinone $+5H_2O$

REFERENCE RANGE:- HDL: 30-70mg/dL

DETERMINATION OF LDL CHOLESTEROL BY FRIEDEWALD EQUATION

LDL = Total cholesterol - (HDL— TG/5)
REFERENCE RANGE :- LDL=90-130Mg\dl

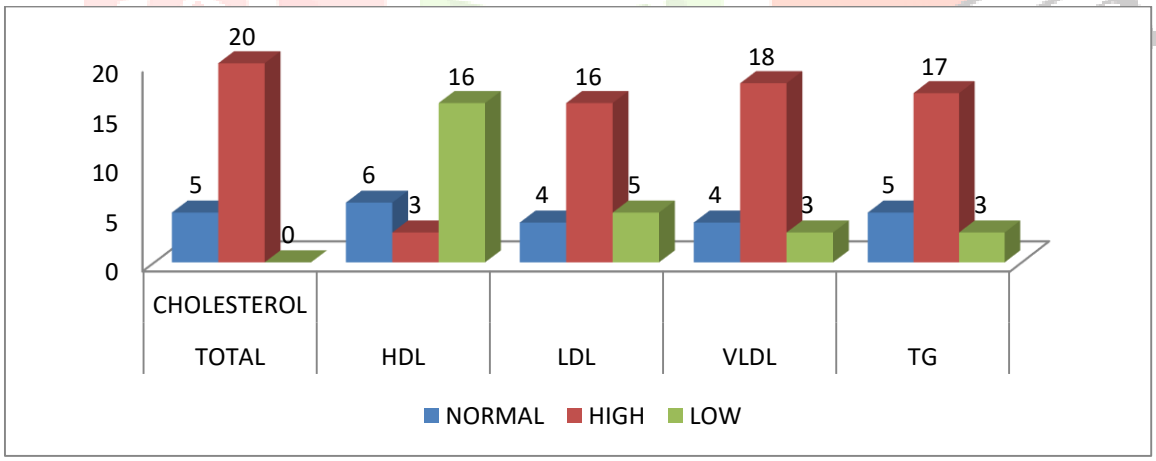
RESULT

Laboratory investigations are done in 50 subjects and their Total Cholesterol, Triglyceride, and HDL-C are estimated. LDL-C and VLDL are calculated by using friedewald formula. Results obtained are given below

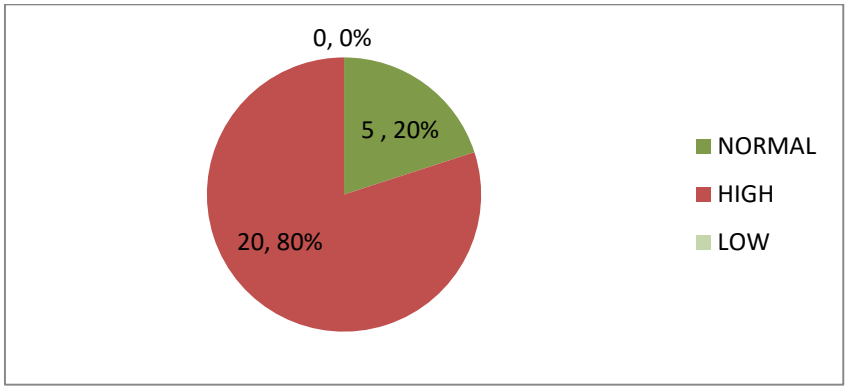
Table 1: Summary of lipid profile in smokers

	TOTAL CHOLESTEROL	HDL	LDL	VLDL	TG
NORMAL	5	6	4	4	5
HIGH	20	3	16	18	17
LOW	0	16	5	3	3

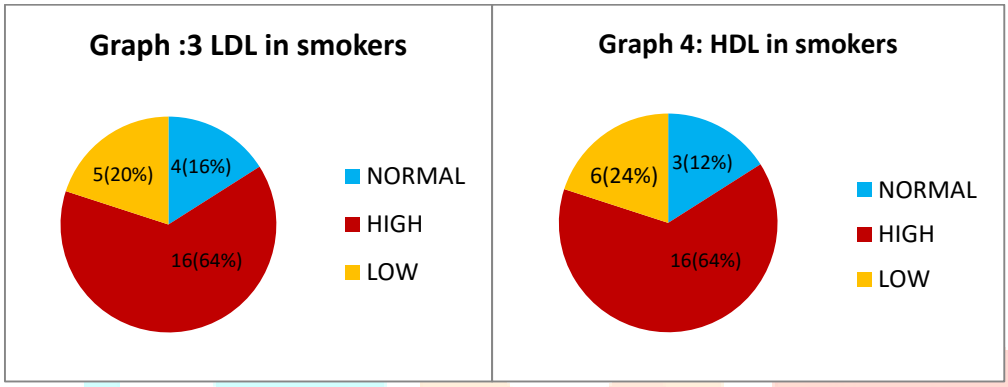
Graph 1: Summary of lipid profile in smokers



Graph2: Total Cholesterol in smokers

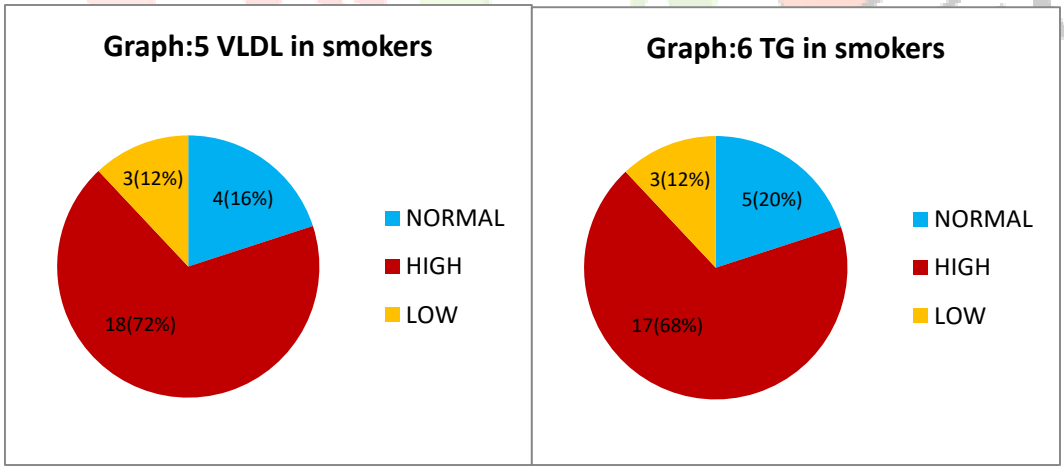


20 out of subject shows high total cholesterol level (80%) and 5 out of 25 subject show normal total cholesterol (20%).



16 out of 25 subject shows low HDL level(64%), 6 Out of 25 shows normal (24%) and 3 out of 25 subject show high HDL (12%).

In case of LDL 5 out of 25 subject shows low LDL level (20%), 4 out of 25 normal (16%) and 16 out of 25 shows high LDL level (64%).

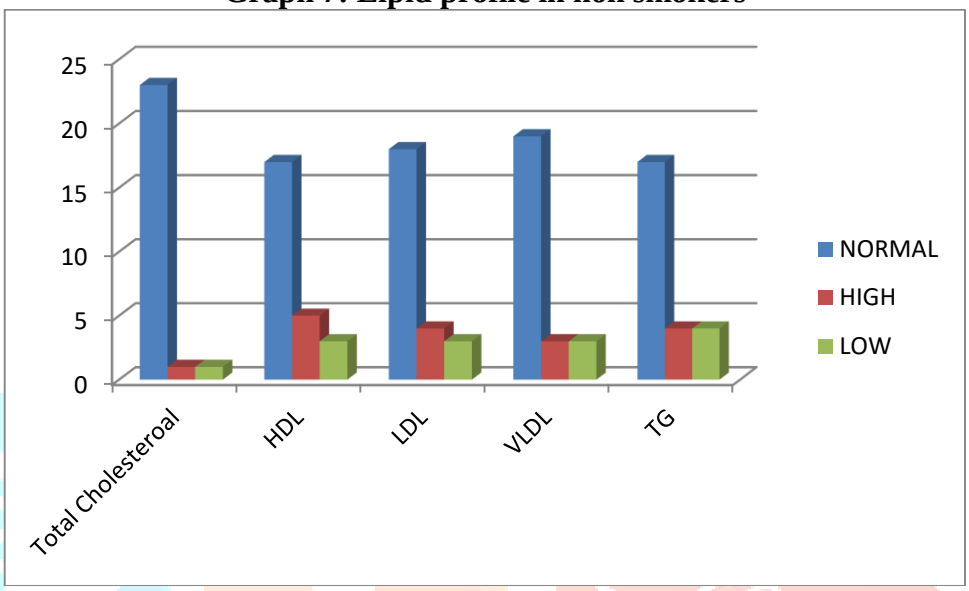


18 out of 25 subject shows highVLDL level(72%), 4 Out of 25 shows normal (16%) and 3 out of 25 subject show low VLDL (12%).**In case of TG 17 out of 25 subject shows high TG level (68%), 5 out of 25 normal (20%) and 3 out of 25 shows low TG level (12%)**

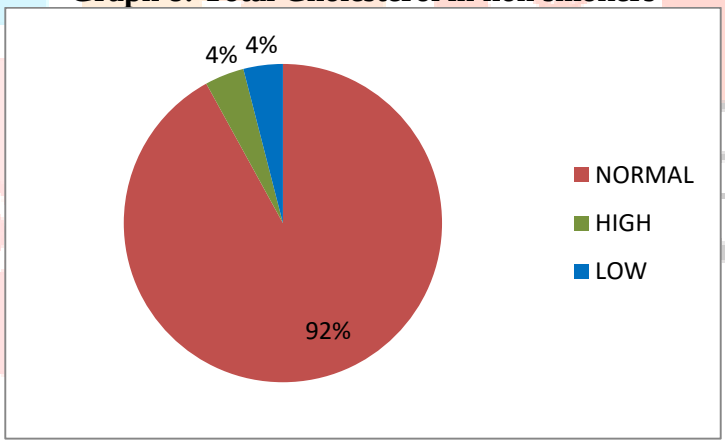
Table :2 **Summary of lipid profile in** non-smoker

	TOTAL CHOLESTEROL	HDL	LDL	VLDL	TG
NORMAL	23	17	18	19	17
HIGH	1	5	4	3	4
LOW	1	3	3	3	4

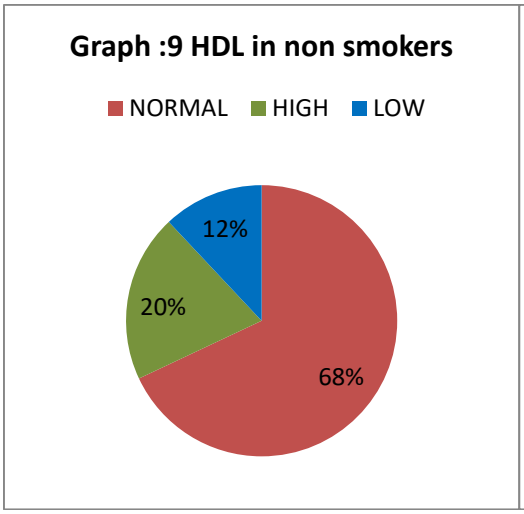
Graph 7: Lipid profile in non smokers



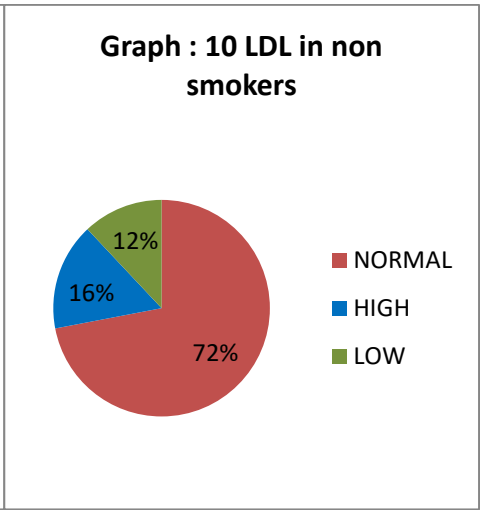
Graph 8: Total Cholesterol in non smokers

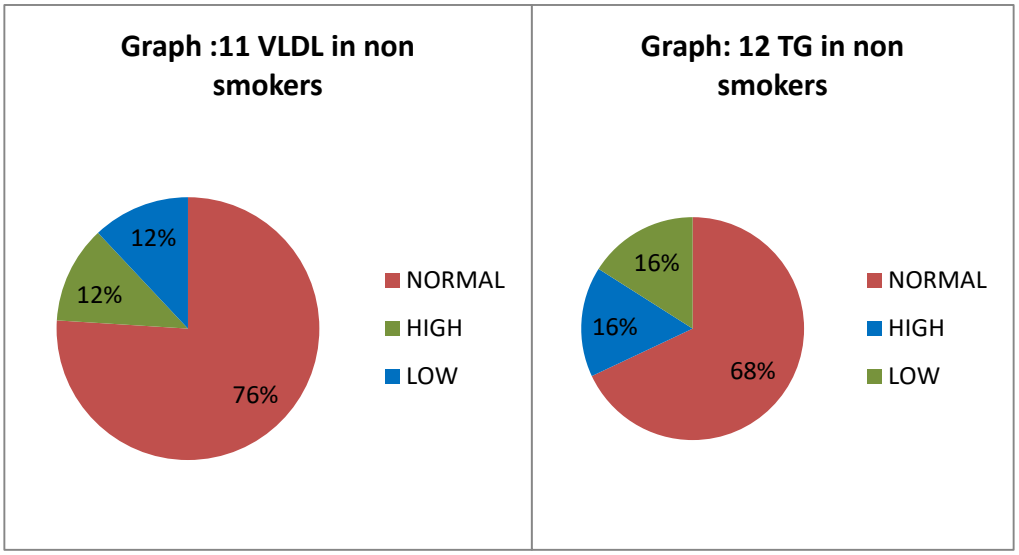


Graph :9 HDL in non smokers



Graph : 10 LDL in non smokers





In majority of non smokers the lipid profiles are shown in normal limits.

DISCUSSION

In the present study, the results showed that the serum level of total cholesterol, triglyceride, LDL-C, and VLDL-C were significantly higher in smokers as compared to non-smokers. The serum level of HDL-C is significantly lower in smokers compared to nonsmokers. Previous studies have reported the same findings that smokers have a higher risk lipid profile than non-smokers .

In 2015, a study by Arjun et al., shows that smokers have higher total cholesterol, triglycerides, and LDL-c, while the HDL-C is lower in smokers as compared to non-smokers[12]. In another study by Devaranavadgi et al., at 2012, reported that a significant increases in total cholesterol, triglycerides, LDL-C ,VLDL and reduction in HDL-c in almost all cigarette smokers as compared to non smokers[13].

CONCLUSION

India is one of the largest consumers of tobacco in the world. Addition of tobacco smoking is related with many health hazards. Nicotine of tobacco can be attributed to changes in lipid profile and atherogenic complications due to increasing the atherogenic lipoprotein(LDL-c) with a further decrease in antiatherogenic lipoprotein(HDL), such changes are associated to occurrence of cardiovascular disease with high risk of morbidity even mortality. So smokers should be counseled about health hazards of smoking and encouraged to quit smoking and adapt healthy lifestyle to improve the life.

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