

Original Research Article

A comparative study of lipid profile among smokers and non-smokers

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Abstract

Background: Cigarette smoking leads to the uptake of many hazardous compounds and their metabolites extracted from burning tobacco. The substances may be electrophilic and react with biological molecules giving rise to oxidative stress through the formation of reactive species or the initiation of lipid peroxidation chains in the membranes. Cigarette smoking has been found to alter lipoprotein levels.

Aim of the study: A prospective study was carried out to find the variations in lipid profile in smokers when compared to non-smokers and to study the alterations in lipid profile in terms of severity of smoking.

Materials and methods: This study was carried out among 200 patients who attended Government Thiruvarur Medical College in 2019 for various ailments. The population was divided into 100 non-smokers and 100 smokers. The smokers were further divided into three groups depending on the intensity of smoking. Serum lipid profile was analyzed in all subjects.

Results: The association of an increased total cholesterol value with smoking was noticed when smokers were compared with non-smokers of a similar age group. The mean total cholesterol value was 191.96 in smokers (100 persons) and 161.18 in non-smokers (100 persons), P-value <0.001. In the age group of 41-50 years, the smokers had a mean HDL value of 47.33 mg/dl (21 persons) while the non-smokers of the same age group had a mean HDL value of 49.48 (21 persons). The mean HDL in smokers was 44.72 mg/dl (100 persons) while the mean of non-smokers was 49.58 mg/dl (100 persons) with P-value =0.002. The mean LDL value in smokers was 103.08 mg/dl when compared with non-smokers which were 82.34 mg/dl with P-value (<0.001). The mean value of TG in smokers was 164.29 mg/dl when compared to non-smokers, which was 103.58 mg/dl (p-value <0.001). The triglycerides show a steady increase with the increase in the severity of smoking. The

mean value in nonsmokers was 21.69 mg/dl while that of smokers was 29.02 mg/dl with P-value (<0.001). VLDL values showed a steady increase with the severity of smoking. Dyslipidemia was directly proportional to the intensity of smoking. Conclusion: Increase in total Cholesterol, Triglycerides, LDL and VLDL were found in smokers of all age groups whereas HDL values showed an inverse relationship. These changes were directly proportional to the severity of smoking. So, Tobacco smoking is associated with atherogenic dyslipidemia.

Key words

Cigarette Smoking, Total Cholesterol, Triglycerides, Low-density lipoprotein, High-density Lipoprotein, Very lowdensity Lipoprotein.

Introduction

Tobacco is one of the most potent and prevalent addictives, influencing the behavior of human beings for over 4 centuries. Smoking is now rapidly increasing through the developing world and is one of the biggest threats to current and future health [1]. Cigarette smoking is the most common type of tobacco use. Tobacco continues to be the second major cause of death in the world [2]. Smoking is an important risk factor for atherosclerosis, peripheral vascular disease and stroke, and cancers. It also has a strong relationship with gastric ulcers, periodontal disease, and metabolic syndrome [3]. A one to three-fold increase in the risk of myocardial infarction has generally been noted among smokers. Plasma lipoprotein abnormalities are said to be the major underlying risk factor for the common occurrence of atherosclerotic vascular disease. Most of the studies indicate a definite correlation between smoking and lipid profile alteration in which there is a definite dose-response relationship between the number of cigarette smoking as well as the duration of smoking and changes in the lipid profile noted [4]. However, despite 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. In the present study, an attempt has been made to find out the effect of smoking on the lipid in the health smokers are compared with that of same age group healthy non-smokers [5].

Materials and methods

This study was carried out among 200 patients who attended Government Thiruvarur Medical College in 2019 for various ailments. The population was divided into 100 non-smokers and 100 smokers. The smokers were further divided into three groups depending on the intensity of smoking. Serum lipid profile was analyzed in all subjects.

Inclusion criteria

- Age: 20-50 years, 100 males who never smoked.
- 100 males smoking for 5 or more years, divided into three groups depending on the intensity of smoking as follows, Mild Smokers (1 – 10 Cigarettes/ Bidis per day), Moderate Smokers (11 – 20 Cigarettes/ Bidis per day) Severe Smokers (> 20 Cigarettes/ Bidis per day)

Exclusion criteria

- Obese
- Those on diet restriction.
- Those on drugs were known to alter lipid profiles.
- Those with a history of alcohol intake.
- Those with diseases known to alter lipid profile like diabetes mellitus, Hypothyroidism, renal failure.
- Those with hypertension and coronary artery disease.
- Those with a family history of dyslipidemia.

Since smoking is less prevalent among women in our country this study was conducted only in men. The blood samples for analysis were taken at least after a minimum of 12 hours of complete fasting. The subject was asked to have a light, fat-free diet on the day before sampling. The venepuncture was done in the cubital fossa. Tourniquet was used but was released just before sampling to avoid an artifactual increase in the concentration of serum lipids. About 10 ml of blood was drawn using perfectly dry and sterile disposable syringes. The serum was separated within 2 hours of collection to prevent artifactual changes in the concentration of HDL. The sample was analyzed the same day or within 48 hours. The lipid and lipoprotein assay was done using the Dr. Lange LP 700 equipment.

Statistical analysis

The mean levels of various variables were correlated with basal reference for normal individuals. Relevant statistical methods like the student 't' test and whenever required Mann-Whitney test was used to see the

significance of the difference in mean values between groups and to know their correlation between inter and intragroup variation. Ethical clearance was obtained from the institution

Results

In the age group of 21 to 30 (30 persons) the mean total cholesterol value was 197.17 in smokers, while in non-smokers it was 156.64 (36 persons). In smokers of age group, 31 to 40 (49 persons) the mean total cholesterol was 191.45 while in non-smokers it was 163.84 (43 persons). The mean total cholesterol was 185.71 in smokers (21 persons) of age group 41 to 50 years while it was 163.52 in non-smokers (21 persons). The association of an increased total cholesterol value with smoking was noticed when smokers were compared with non-smokers of a similar age group. The mean total cholesterol value was 191.96 in smokers (100 persons) and 161.18 in non-smokers (100 persons), P-value <0.001 (**Table – 1, 2**).

Table – 1: Comparison of total cholesterol among smokers and non-smokers (age wise).

Age in Years	Non-Smokers		Smokers	
	No. of Persons	TC	No. of Persons	TC
21-30	36	156.64±22.58	30	197.17±32.59
31-40	43	163.84±29.21	49	191.45±29.72
41-50	21	163.52±27.11	21	185.71±32.08

Table – 2: Comparison of TG among non-smokers and smokers (age wise).

Age in Years	Non-Smokers		Smokers	
	No. of Persons	TG	No. of Persons	TG
21-30	36	101.75±21.51	30	171.33±29.16
31-40	43	103.65±29.53	49	163.08±25.88
41-50	21	106.57±26.3	21	157.05±32.97

The mean value of HDL cholesterol stood 42.27mg/dl while the non-smokers showed a mean value of 50.47. The age-related decrease was observed in the advancing age group probably due to an increase in the duration of smoking. Smokers in the age group 31-40 years had a mean HDL value of 44.98 mg/dl (49 persons), which is low compared to non-smokers

of the same age group 48.88 mg/dl (43 persons). In the age group of 41-50 years, the smokers had a mean HDL value of 47.33mg/dl (21 persons) while the non-smokers of the same age group had a mean HDL value of 49.48 (21 persons). The mean HDL in smokers was 44.72 mg/dl (100 persons) while the mean of non-

smokers was 49.58 mg/dl (100 persons) with P-value = 0.002 (**Table – 3**).

The mean LDL value of smokers in the age group 31-40 (49 persons) was 103.18 mg/dl and that of non-smokers of the same age group (43 persons) was 83.84 mg/dl. The mean LDL values of smokers of the age group 41-50 were 96.9

mg/dl and that of nonsmokers was 82.52 mg/dl. The LDL values were high in the smokers compared with non-smokers of the same age group. The mean LDL value in smokers was 103.08mg/dl when compared with nonsmokers which were 82.34mg/dl with P-value (<0.001) as per **Table - 4**.

Table – 3: Comparison of HDL among non-smokers and smokers(age wise).

Age in Years	Non-Smokers		Smokers	
	No. of Persons	HDL	No. of Persons	HDL
21-30	36	50.47±7.84	30	42.47±10.68
31-40	43	48.88±9.34	49	44.98±8.96
41-50	21	49.48±7.96	21	47.33±10.38

Table – 4: Comparison of LDL among non-smokers and smokers(age wise).

Age in Years	Non-Smokers		Smokers	
	No. of Persons	LDL	No. of Persons	LDL
21-30	36	80.44±16.55	30	107.23±19.95
31-40	43	83.84±15.67	49	103.18±18.59
41-50	21	82.52±17.99	21	96.9±14.88

Table – 5: Comparison of VLDL among non-smokers and smokers(age wise).

Age in Years	Non-Smokers		Smokers	
	No. of Persons	VLDL	No. of Persons	VLDL
21-30	36	20.44±6.2	30	30.7±10.12
31-40	43	23.21±6.39	49	28.73±8.56
41-50	21	20.71±6.21	21	27.29±7.73

Table – 6: Comparison of lipid profile among smokers and non-smokers.

Lipid Profile	Non-Smokers(Mean ± Std)	Smokers (Mean ± Std)	P-Value
TC	161.18±26.77	191.96±31.37	< 0.001
TG	103.58±26.26	164.29±28.95	< 0.001
HDL	49.58±8.57	44.72±9.96	= 0.002
LDL	82.34±16.57	103.08±18.66	< 0.001
VLDL	21.69±6.42	29.02±8.98	< 0.001

In the age group of 21-30 years smokers (30 persons) showed a mean value of 171.33 mg/dl while the mean value of triglyceride in non-smokers was 101.75mg/dl in the same age group. In the 31-40 years age group mean value among smokers was 163.08mg/dl (49 persons): non-smokers showed 103.65mg/dl (43 persons). The mean triglyceride value was 157.05mg/dl in smokers of age group 41-50 years (21 persons)

and non-smokers, it was 106.57mg/dl (21 persons). The mean value of TG in smokers was 164.29mg/dl when compared to non-smokers, which was 103.58mg/dl (p-value <0.001). The triglycerides show a steady increase with the increase in the severity of smoking (**Table – 5, 6**).

Discussion

Cigarette smoking is found to occur mostly in later age groups in the Indian population when compared to western society. The third decade of life accounts for a maximum number of smokers [6]. In late adolescent periods and during college life they get captivated by the “Kick of smoke”. Some start smoking to show off their macho image. The majority gets addicted to smoke but some people quit [7]. Some go for other addictive habits like cannabis. Smoking beedi is found mostly among the labor class in India. Smoking filtered tip cigarettes is found mostly among the elite population [8]. Several studies have been conducted both in India and abroad among smokers and they analyzed their clinical, biochemical, and pathological parameters [9]. The present study was conducted to compare the lipid profile among smokers and non-smokers and to study changes in lipid profile depending on the intensity of smoking. This study was done as a part of the postgraduate training program [10]. Smokers are at a higher risk of CAD when compared to non-smokers. The various explanations given for this association include alterations in blood coagulation, reduced fibrinolysis, impaired arterial integrity, and altered lipid profile and lipoprotein levels. The last explanation is examined in this study. Since in India smoking is less prevalent among females this study was done only among male smokers. This discussion is planned to evaluate each variables separately [11]. The association of an increased total cholesterol value with smoking was noticed when smokers were compared with non-smokers of a similar age group. The mean total cholesterol value was 191.96 in smokers (100 persons) and 161.18 in non-smokers (100 persons), P-value <0.001 [12]. A study of this kind comprising a very large number (that is more than 50,000 participants) was done in 1988 by the American Health Foundation of USA. Cholesterol screening was made across the country. The data collected from 10 screening centers were combined. The mean cholesterol values and standard errors were calculated for

both sexes and various age groups [13]. It was noted that with an increase in the severity of smoking there was a statistically significant increase in mean total cholesterol values. Among men in the age group of 20-60, they found a 0.34 mg/dl increase in total cholesterol for every cigarette smoked. In women strongest association was observed in age intervals between 30 to 50 years, where serum cholesterol increased by 0.5 mg/dl for each cigarette smoked [14]. These results indicated that heavy smokers of both sexes had considerably higher levels of plasma cholesterol than non-smokers, with the trend among women smokers aged 30-50 years greater than that for men and other women. For example, men under 50 years of age, who smoked 30 cigarettes or more per day had a predicted level of approximately 10 mg/dl higher than those who never smoked; and women in the 30-50 age group had 15mg/dl values higher than nonsmokers [15]. Of the smokers in the 21-30 years of age group, the mean value of HDL cholesterol stood 42.27mg/dl while the non-smokers showed a mean value of 50.47. The age-related decrease was observed in the advancing age group probably due to an increase in the duration of smoking. Smokers in the age group 31-40 years had a mean HDL value of 44.98 mg/dl (49 persons), which is low compared to non-smokers of the same age group 48.88mg/dl (43 persons) [16]. In the age group of 41-50 years, the smokers had a mean HDL value of 47.33mg/dl (21 persons) while the non-smokers of the same age group had a mean HDL value of 49.48 (21 persons). The mean HDL in smokers was 44.72 mg/dl (100 persons) while the mean of non-smokers was 49.58 mg/dl (100 persons). P-value =0.002. The chemical pathology and medicine department of St. Marys hospital, London conducted a study and put forward an explanation to this effect [17]. The action of endothelial lipoprotein lipase on triglyceride-rich lipoprotein reduces the core volume of these particles and generates surface remnants containing unesterified cholesterol, phospholipid, and apoprotein, which join the HDL3 pool. These surface remnants are thought to constitute the major source of HDL precursors and

lipoprotein lipase is thought to be a major determinant of the plasma concentration of HDL [18]. Esterification of the acquired free cholesterol in HDL3 particles by lecithin cholesterol acyltransferase results in the accumulation of cholesterol ester in the core of the particles and the production of larger, less dense HDL2 particles. A transfer protein mediates the exchange of cholesterol ester in HDL with triglyceride in lipoprotein of a lower density. The magnitude of postprandial lipaemia determines the extent of this exchange and the resultant triglyceride content of HDL2 particles [19]. Furthermore, the triglyceride content of HDL2 particles has been shown to determine, which of the phospholipase or triglyceride lipase activities of hepatic lipase act on them. Triglyceride-rich HDL2 particles are converted to HDL3 particles by removal of the triglyceride from the core of the particles, whereas phospholipid is removed from the surface of triglyceride poor HDL2 particles without any change in size or density. Reduced intravascular lipolysis exists in smokers, hence it was suggested that a consequent increase in the postprandial lipaemia in smokers will result in a greater proportion being converted to HDL3 particles by hepatic lipase [20]. This would explain the lowering of HDL2 and the increase in the concentration of HDL3 cholesterol found in smokers. HDL2 concentration correlates negatively with the severity of angiographically defined atheroma. The change in HDL that is reported in young smokers and the increased exposure of vascular endothelium to the potentially atherogenic lipoproteins as a consequence of impaired clearance of triglyceride-rich lipoproteins is the mechanism whereby smoking predisposes to coronary artery disease [21]. The present study had not focused on HDL values following cessation of smoking. But data published by Kasap, et al. revealed normalization of HDL in smokers who gave up that habit. HDL values increased by 7 mg/dl in 48 days after stopping smoking and the effect disappeared in people who resumed the habit [22]. To establish a causal relationship between exposure to cigarette smoke and changes in

serum lipid and lipoprotein concentrations, dose-response effects were calculated by an analysis of 54 published studies conducted by Anazawa, T et.al and his associates from the foundation of Blood research, Scarborough, USA. They found a higher mean concentration of cholesterol (3%), triglycerides (9.1%), VLDL (10.4%), LDL (1.7%). The dose-response effect on VLDL and LDL among nonsmokers, mild, moderate, and severe smokers were (0, 7.2, 44.4, 39.0%) and (0, -1.1, 1.4, and 11.0%) respectively [23]. Fagerstrom K, et al. study on persons in 8-19 age groups had shown an increase of 11.8% in smokers for the triglyceride value while their analysis of the 54 published data showed an increase in triglyceride value of 9.1% in smokers compared to non-smokers. Sympathy adrenal system stimulation by nicotine leads to lipolysis and increased serum-free fatty acid levels which lead to increased synthesis of VLDL from the liver and hence triglycerides [24]. Several studies which have evaluated the risk factor for ischemic heart disease have shown that reduced HDL-cholesterol to total cholesterol is a very important risk factor [25].

Conclusion

An increase in total cholesterol, triglycerides, low-density lipoproteins, and very-low-density lipoproteins was found in smokers of all age groups, whereas high-density lipoproteins showed an inverse relationship. A direct relationship exists between the severity of smoking and an increase in the total cholesterol, triglycerides, LDL, and VLDL while an inverse relationship is found with HDL. Asymptomatic smokers are at the risk of developing coronary artery disease due to the above changes in lipid profile. The observed values were in concordance with the studies done in India and other countries.

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