IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 4 Ver. 8 (April. 2018), PP 36-39 www.iosrjournals.org

Impact of Cigarette Smoking On Lipid Profile among South Indian Smokers.

Nagendra Prasad Naik Dugavanth[1] ,Dwarakanath Nallapoola [2],

1. Assistant Professor, Department of Pathology, Sri Venkateswara Medical College, Tirupati.
2. Associatet Professor, Department of Physiology, Sri Venkateswara Medical College, Tirupati. Corresponding author: Dwarakanath Nallapoola

Abstract: *Tobacco smoking is one of the well known modifiable risk factors for atherosclerosis, coronary heart diseases, lung & oral cancers and chronic obstructive pulmonary diseases. Tobacco is consumed in many ways such as chewing, smoking. In India, tobacco kills 8–10 lakh people each year and majority of these deaths occur in young age. An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked. This study was designed to demonstrate the effects of smoking on lipid profile among south Indian smokers and to identify the differences between the lipid profiles of smokers. The study group comprised of 25 males with age range between 40 and 55years and 25 age matched males as controls. The smokers in this study were those smoking 11-20 cigarette/day for last 15 years. There were no significant differences in age, height, weight and BMI between the groups. But, Heart rate ($p<0.001$), systolic blood pressure ($p<0.001$) and diastolic blood pressure ($p<0.001$) were significantly high in smokers when compared to controls. The total cholesterol ($p<0.001$), low density lipoproteins ($p<0.001$), very low density lipoproteins ($p<0.001$) and triglycerides ($p<0.001$) were significantly high and high density lipoproteins ($p<0.001$) were significantly low in smokers when compare to non smokers. The results of this study show that smokers are at much greater risk of developing atherosclerotic plaques and different heart diseases than non-smokers.*

Key Words: *Tobacco smoking, lipid profile, high density lipoproteins*.

Date of Submission: 30-03-2018 Date of acceptance: 16-04-2018

I. Introduction

Smoking of tobacco is done in various forms like cigar, cigarette, beedi, hukka, pipe, etc. Beedi and cigarette smoking is highly prevalent in rural as well as urban India (1). Tobacco smoking is one of the well known modifiable risk factor for atherosclerosis, coronary heart diseases, lung & oral cancers, chronic obstructive pulmonary diseases, etc (2). Tobacco is consumed in many ways such as chewing, smoking, etc (3).

In India, tobacco kills 8–10 lakh people each year and majority of these deaths occur in young age. An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked (4). 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 (5). Nicotine is one of the toxins present in tobacco smoke (6). It is found to have effect on person's catecholamine & cortisol secretion (7), (8).

Elevated catecholamine and cortisol can alter carbohydrate and lipid metabolism in such person (9). 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 (10).

Cigarette smoking is one of the most important modifiable risk factor for atherosclerosis and increasing morbidity and mortality of Chronic Heart Diseases (CHD) (11). Although the precise mechanism of tobacco smoke role in the atherosclerotic process remains not fully understood, several chemicals among thousands that exist within tobacco smoke produce harmful and toxic effects on health. Many changes which could promote atherosclerosis in chronic tobacco smokers have been reported by many research workers, whereby these changes include; alternation of lipid profile, increased oxidative LDL-C, decreased nitric oxide (NO) availability (12), platelet dysfunction, high blood viscosity, on-going inflammation with increasing inflammatory markers (13), and more recently free radicals-mediated oxidative stress appear to play an important role in mediation of athero-thrombotic disease in chronic smokers (14). Cigarette smoking increases plasma catecholamine which induces lipolysis and release of free fatty acid (8), which will be taken up by the liver.

Although many studies have been done worldwide to identify the lipid profile among smokers, but we have little evidence about the impact of smoking on lipid profile among south Indian smokers. This study was

designed to demonstrate the effects of smoking on lipid profile among south Indian smokers and to identify the differences between the lipid profiles of smokers.

II. Materials And Methods

This study was carried out in the department of Physiology, Sri Venkateswara medical college, Tirupati, Andhra Pradesh, India. Informed consent was obtained from all the participants. The study group comprised of 25 males with age range between 40 and 55years and 25 age matched males as controls. The smokers in this study were those smoking 11-20 cigarette/day for last 15 years. Before the collection of blood samples from smokers and non-smokers, questionnaires were administered to provide the details about their smoking habits. The age, body weight, height and other physical measurements were obtained. All the recruited subjects were neither alcoholic nor having any form of diseases or ailment. Hence, all male subjects included in the present study were apparently normal healthy individual. About 5ml of venous blood samples was collected from each of the subjects into lithium heparin bottle after 8 - 12 hrs overnight fast with individuals being on their normal diet prior to the test. The samples were spurned and the plasma separated within 2hrs of collection and analyzed immediately using standard enzymatic methods for each of the parameters investigated. The LDL was calculated from the values of triglyceride and cholesterol using the Friedewalds (15).

III. Results

The baseline characteristics of smokers and non smokers are given in Table 1. There were no significant differences in age, height, weight and BMI between the groups. But, Heart rate ($p<0.001$), systolic blood pressure ($p<0.001$) and diastolic blood pressure ($p<0.001$) were significantly high in smokers when compared to controls.

Table 2 shows the comparison of lipid profile between smokers and non smokers. The total cholesterol ($p<0.001$), low density lipoproteins ($p<0.001$), very low density lipoproteins ($p<0.001$) and triglycerides ($p<0.001$) were significantly high and high density lipoproteins ($p<0.001$) were significantly low in smokers when compare to non smokers.

Table 1. Baseline characteristics of smokers and non smokers.

Sl.No	Parameter	Smokers	Non smokers	P value
1	Age (years)	46.0 ± 3.76	45.56 ± 3.45	0.66
2	Height (cms)	168.68 ± 6.66	166.68 ± 5.83	0.82
3	Weight (kg)	61.16 ± 4.17	59.64 ± 4.42	0.21
4	BMI (kg/m^2)	21.56 ± 2.14	21.56 ± 2.14	0.96
5	HR (bpm)	79.24 ± 2.04	70.24 ± 2.57	0.001
6	SBP (mmhg)	125.52 ± 7.61	114.82 ± 6.30	0.001
7	DBP (mmhg)	82.96 ± 3.06	76.05 ± 4.40	0.001

Data expressed as mean and standard deviation.
BMI: Body mass index, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure.

Table 2. Lipid profile paramerters of smokers and non smokers.

Sl.no	Parameter	Smokers	Non smokers	P value
1	TC	190.48 ± 9.61	163.76 ± 7.57	0.001
2	LDL	114.92 ± 10.43	83.88 ± 4.03	0.001
3	VLDL	23.4 ±2.51	18.20 ± 1.35	0.001
4	HDL	53.44 ± 6.57	61.68 ± 7.21	0.001
5	TRIG	117.00 ± 12.58	91.00 ± 6.77	0.001

Data expressed as mean and standard deviation.
TC: Total cholesterol, LDL: Low density lipoproteins, VLDL: Very low density lipoproteins, HDL: High density lipoproteins, TRIG: Triglycerides.

IV. Discussion

In the present study of 50 subjects, 25 non - smokers and 25 smokers, who smoke 11 - 25 cigarettes per day for the past 15 years. There were no significant difference in the baseline parameters like age, height, weight, and body mass index, on calculating the mean and the standard deviation.

It hasbeen established that one of the major constituents of tobacco i.e nicotine has a considerable influence in increasing the lipid levels in blood. Lipid have important roles In all aspect of life, serving as hormones or hormones precursors, aiding in digestion, providing energy storage metabolic fuel, acting as functional and structural component in cell membranes and forming insulation to allow nerve

conduction or to prevent heat lost (16), but their excessive concentrations are associated with various metabolic disorders.

The result of this study showed a statistically significant difference in the total cholesterol level of smokers ($p<0.001$) when compared with non -smokers, this indicate that the cigarette smokers have increased serum concentration of cholesterol than non -smokers. The result of this work is in line with work of Adedeji and Etukudo, where high concentration of cholesterol was recorded in smokers when compared with the nonsmokers (17). The increase in the total cholesterol level seen in the smokers was as a result of increase in the activity of hepatic HMG-CoA reductase (18) reported that hepatic HMG CoA reductase, the main rate limiting enzyme in cholesterol synthesis is subject to induction and repression by several hormones, dietary factors and drugs one of which is nicotine. Increased cholesterol is a causative factor in the etiology of atherosclerotic disease (19).

The values of triglycerides of smokers in this study were significantly increased compared to that of non-smokers. The increase in the value of triglyceride is due to induction of lipogenic enzyme by nicotine as reported by (20) where they established that there is induction of both glycerolkinase and glycerol - 3 – phosphate acyl transferase by nicotine. The result of this study is in line with the work of Odedeji OA (17) where there is an increase in the triglyceride of smokers compared with non-smokers. It has also been documented that 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 (21) as well as hepatic secretion of very low density lipoprotein (VLDL) and hence increased TG (22).

High density lipoprotein of smokers was significant lower when compared with non -smokers' in this work. The low level recorded in this work might not be unconnected to the increase in the hepatic lipase and LCAT activity by nicotine. This result is in line with the report of Robbert KM et.al., (19) that HDL concentration varies directly with the activity of hepatic lipase as well as LCAT. A decreased HDL cholesterol concentration is associated with coronary heart disease (23). This shows that smokers are predisposed to developing coronary heart disease earlier than their non -smoking counterpart.

Carl and Edward reported that clinically increase in LDL cholesterol is associated with increased risk of coronary heart disease (19). The findings of this present work revealed high level of LDL cholesterol in smokers when compared with non -smokers. This finding is in consonance with the work of Khurana Et.al., (20) where it was reported that increase in LDL level in cigarette smokers was due to the down regulation of LDL receptors and failure of receptor mediated endocytosis by metabolite of cigarette (24) specifically attributed the down regulation of LDL receptor to inhibiting action of smoke allylamine and nicotine (17) also reported high level of LDL in smokers, suggesting that there is increased LDL-Cholesterol synthesis in smokers which is dangerous to their health. LDL/HDL ratio was significantly higher in smokers as compared to that of controls. The result agrees with that of [28]. This ratio is an index of possibility of developing coronary heart disease (CHD) in smokers. This finding indicates that smoking habit predisposes individuals involved to various deleterious effects associated with increased LDL and reduced HDL concentrations. This result is in consonance with that obtained by Adedeji OA et. al., (17). Cigarette smoking, obesity, hypertension and increased cholesterol have been previously implicated as risk factors associated with atherosclerotic plaque formation (25).

V. Conclusion

Cigarette smoking causes an alternation of lipid profile which includes increasing the levels of total cholesterol, triglyceride, LDL-C, VLDL-C and non-HDL-C with a decrease in HDL-C level. The results of this study show that smokers are at much greater risk of developing atherosclerotic plaques and different heart diseases than non-smokers.

References

[1]. Mishra S, Joseph RA, Gupta PC, Pezzack B, Ram F, Sinha DN, et al. Trends in bidi and cigarette smoking in India from 1998 to 2015, by age, gender and education. BMJ Glob Health. 2016; 1(1):e000005.
[2]. Glantz S, Gonzalez M. Effective tobacco control is key to rapid progress in reduction of non-communicable diseases. Lancet. 2012;379(9822):1269–71.
[3]. Gupta PC, Ray CS. Tobacco, education & health. Indian J Med Res. 2007;126(4):289–99.
[4]. Chatterjee T, Haldar D, Mallik S, Sarkar GN, Das S, Lahiri SK. A study on habits of tobacco use among medical and non-medical students of Kolkata. Lung India Off Organ Indian Chest Soc. 2011;28(1):5–10.
[5]. Talhout R, Schulz T, Florek E, van Benthem J, Wester P, Opperhuizen A. Hazardous Compounds in Tobacco Smoke. Int J Environ Res Public Health. 201;8(2):613–28.
[6]. Stedman RL. Chemical composition of tobacco and tobacco smoke. Chem Rev. 1968;68(2):153–207.
[7]. Tweed JO, Hsia SH, Lutfy K, Friedman TC. The endocrine effects of nicotine and cigarette smoke. Trends Endocrinol Metab TEM. 2012;23(7):334–42.
[8]. Mizobe F, Livett BG. Nicotine stimulates secretion of both catecholamines and acetylcholinesterase from cultured adrenal chromaffin cells. J Neurosci Off J Soc Neurosci. 1983;3(4):871–6.

[9]. Arner P. Catecholamine-induced lipolysis in obesity. Int J Obes Relat Metab Disord J Int Assoc Study Obes. 1999;23 Suppl 1:10–3.
[10]. Nelson RH. Hyperlipidemia as a Risk Factor for Cardiovascular Disease. Prim Care. 2013 ;40(1):195–211.
[11]. Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, et al. Cigarette smoking and progression of atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study. JAMA. 1998;279(2):119–24.
[12]. Mayhan WG, Patel KP. Effect of nicotine on endothelium-dependent arteriolar dilatation in vivo. Am J Physiol-Heart Circ Physiol. 1997;272(5):H2337–42.
[13]. Tracy RP, Psaty BM, Macy E, Bovill EG, Cushman M, Cornell ES, et al. Lifetime Smoking Exposure Affects the Association of C-Reactive Protein with Cardiovascular Disease Risk Factors and Subclinical Disease in Healthy Elderly Subjects. Arterioscler Thromb Vasc Biol. 1997;17(10):2167–76.
[14]. Nedeljkovic ZS, Gokce N, Loscalzo J. Mechanisms of oxidative stress and vascular dysfunction. Postgrad Med J. 2003;79(930):195–200.
[15]. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, Without Use of the Preparative Ultracentrifuge. Clin Chem. 1972;18(6):499–502.
[16]. Samuel O, Ibrahim S, Olusegun Taiwo O, Oluyombo R, Dorcas Yetunde O. Lipid Profile of Cigarette Smokers in an Ancient City. Sch J Appl Med Sci. 2013;1:447–51.
[17]. Lipid Profile of Cigarette Smokers in Calabar Municipality [Internet]. [cited 2018 Apr 5]. Available from: https://scialert.net/abstract/?doi=pjn.2006.237.238
[18]. Sinha AK, Misra GC, Patel DK. Effect of cigarette smoking on lipid profile in the young. J Assoc Physicians India. 1995;43(3):185–8.
[19]. Carl AB, Edward RB; Lipid and Lipoprotein in clinical chemistry, 3 rd edition, W.B. Sauders Philadelphia, 1999: 809-862. In.
[20]. Khurana M, Sharma D, Khandelwal PD. Lipid profile in smokers and tobacco chewers--a comparative study. J Assoc Physicians India. 2000;48(9):895–7.
[21]. Goh EH, Heimberg M. Stimulation of hepatic cholesterol biosynthesis by oleic acid. Biochem Biophys Res Commun. 1973;55(2):382–8.
[22]. Muscat JE, Harris RE, Haley NJ, Wynder EL. Cigarette smoking and plasma cholesterol. Am Heart J. 1991;121(1 Pt 1):141–7.
[23]. Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, et al. HDL cholesterol and other lipids in coronary heart disease. The cooperative lipoprotein phenotyping study. Circulation. 1977;55(5):767–72.
[24]. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. BMJ. 1989;298(6676):784–8.
[25]. Brown MS, Goldstein JL. Lipoprotein metabolism in the macrophage: implications for cholesterol deposition in atherosclerosis. Annu Rev Biochem. 1983;52:223–61.

Dwarakanath Nallapoola.“ Impact of Cigarette Smoking On Lipid Profile among South Indian Smokers.."IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 4, 2018, pp 36-39.