

Comparing lipid profile in healthy participants with and without smoking: A prospective study

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ABSTRACT

Background: Smoking can induce cancer, stroke, and heart disease and also has been reported to cause periodontal disease, gastric ulcer, sudden infant death syndrome, and metabolic syndrome. **Objectives:** The objective is to study and compare alterations of lipid profile among smokers and non-smokers in different age groups. **Materials and Methods:** One hundred participants were studied at JA Group of Hospitals and GR Medical College, Gwalior, from June 2014 to October 2015 after dividing them into Group A ($n = 50$, non-smokers) and Group B ($n = 50$, smokers). Lipid profile including total cholesterol (TC), triglycerides (TGs), low-density lipoproteins (LDLs), high-density lipoproteins (HDLs), and HDL/TC ratio was estimated. **Results:** The mean age of participants in Group A and Group B was 34.56 ± 12.12 and 36.45 ± 62.22 years, respectively. TC, TG, and LDL were higher in Group B compared to Group A, whereas HDL and HDL/TC ratio were lower in Group B compared to Group A across all age groups. **Conclusion:** Significantly higher serum level of TC, TG, LDL, and very LDL with lower level of HDL suggests that smoking is associated with dyslipidemia. Dyslipidemia is an independent risk factor for vascular diseases, so it can be used as a biomarker to identify smokers at risk.

KEY WORDS: High-density Lipoprotein; Low-density Lipoprotein; Smoking; Total Cholesterol; Triglycerides

INTRODUCTION

In Indian population, cigarette smoking occurs in later age as compared to the western population. Reports have shown that the third decade of life has the maximum number of Indian smokers. Globally, for premature deaths, tobacco smoking is reported to be the most common preventable cause.^[1]


As per the recent reports in the year 2010, around 120 million Indian people smoked, which made India at the second place after China in the most number of smokers.^[2,3]

The most common form of tobacco smoking among Indian population is in the form of bidis which is made from tendu leaf.^[2]

Reports from 2010 showed that, in the same year, 1 million deaths were due to smoking only which comprises 10% of the death in India. Of this, 70% death occurred at the age of 30–69 years.^[4]

Smoking cigarette is considered as the risk factor for coronary heart disease (CHD) as compared to non-smokers. The possible reason for this association is may be due to altered blood coagulability, decreased fibrinolysis, impaired integrity of the arterial wall, and changes in the blood lipid and lipoprotein concentrations.^[5]

In India, female smokers are few, and hence, this study comprises only adult male smokers. In the present study, we tried to study and compare alterations of lipid profile among smokers and non-smokers in different age groups.

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MATERIALS AND METHODS

The present study was performed in 100 participants attending JA Group of Hospitals and GR Medical College, Gwalior, from June 2014 to October 2015. Participants were divided into Group A (*n* = 50, non-smokers) and Group B (*n* = 50, smokers).

Ethical clearance was obtained from the Institutional Ethical Committee before starting the study.

Those participants who never smoked and were non-obese and healthy were included in Group A, whereas those on diet restriction and on any drugs known to alter lipid profile, for example, beta-blockers, thiazides, and statins and those with ischemic heart disease (IHD), hypertension, or any systemic disease which may alter lipid metabolism were excluded from Group A. In Group B, participants who were non-obese healthy and were present smoker (1 to >20 bidis or cigarette/day) were included in the study.

Both cigarette smokers and bidi smokers were included in the study. The variables including total cholesterol (TC), triglycerides (TGs), low-density lipoproteins (LDLs), high-density lipoproteins (HDLs), and HDL/TC ratio were estimated.

The blood samples for analysis were taken at least after minimum of 12 h of complete fasting. The participants were asked to have a light, fat-free diet on the day before sampling. The venipuncture was done in the cubital fossa, and tourniquet was used but was released just before sampling to avoid artifactual increase in the concentration of serum lipids.

About 10 ml blood was drawn using perfectly dry and sterile disposable syringes. The serum was separated within 2 h of collection to prevent artifactual changes in the concentration of HDL. The samples were analyzed the same day or within 48 h.

All the data analysis was performed using IBM SPSS ver. 20 software. The mean levels of various variables were correlated with basal reference for normal individuals. Relevant statistical methods such as Student’s *t*-test and whenever required Mann–Whitney test were used to see the significance of difference in mean values between groups and to know their correlation between inter- and intra-group variations.

RESULTS

The mean age of participants in Group A and Group B was 34.56 ± 12.12 and 36.45 ± 62.22 years, respectively. Maximum patients in Group A belong to 21-30 (*n*=17) years whereas in Group B 31-50 (*n*=30) was the most common age group. The mean TC, TG, LDL, and HDL in smokers

and non-smokers were 202.2 ± 26.7 versus 162.2 ± 22.2 (*P* < 0.001), 195.2 ± 46.3 versus 145.1 ± 24.1 (*P* < 0.001), 128.0 ± 28.3 versus 92.5 ± 20.9 (*P* < 0.001), and 34.2 ± 5 versus 42.2 ± 4.6 (*P* < 0.001) mg/dl, respectively [Table 1 and Figure 1].

DISCUSSION

Tobacco smoke contains many toxic compounds and free radicals which can alter body metabolic pathways significantly.^[6] Inhalation of such smoke regularly for long time can aggravate the situation and lead to serious health hazards.^[7] The mean TC value in smokers was 202.2 ± 26.7 mg/dL, whereas in non-smokers, it was 162.2 ± 22.2 mg/dL (*P* < 0.001). In smokers of age group 31–40 years, the mean TC was 189.2 mg/dL, while in controls, it was 172.4 mg/dL. The mean TC was 214.9 mg/dL in smokers of age group 41–50 years, while it was 161.1 mg/dL in controls. The association of an increased TC value with smoking was noticed when smokers were compared with controls of similar age group.

Table 1: Comparing lipid levels among study cohort

Age group	Groups	TC	TG	LDL	HDL
21–30	A	152.6±11.6	142.3±29.5	80.3±9.7	41.9±4.6
	B	179.2±17.2	186.6±44.2	103.8±23.1	40.6±5.5
31–40	A	146.0±17.6	146.0±17.6	97.6±15.8	45.8±3.3
	B	189.3±27.0	186.4±47.1	119.8±27.2	32.9±3.3
41–50	A	161.1±29.4	138±23.3	100.2±28.0	41.0±4.6
	B	214.9±21.4	202.4±50.2	141.4±24.8	34.1±4.3
>50	A	170.5±22.6	167.5±3.7	98.5±23.8	38.3±3.5
	B	212.3±23.0	204.3±24.0	140.8±23.2	30.8±3.1

Data are expressed as mean±SD, TC: Total cholesterol, TG: Triglyceride, LDL: Low-density lipoprotein, HDL TC: High-density lipoprotein-to-total cholesterol ratio, SD: Standard deviation

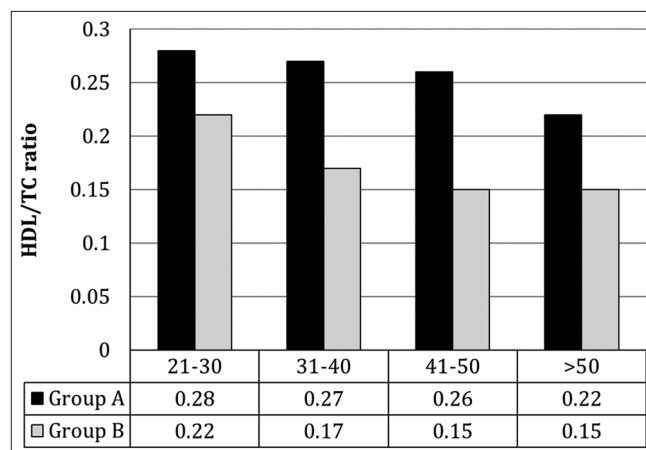


Figure 1: High-density lipoprotein cholesterol/total cholesterol ratio among study cohort

These findings are in accordance with those of Muscat *et al.*^[8] and Neeki *et al.*^[9] Increased cholesterol levels and CHD are observed in cigarette smokers.^[10] According to Zamir *et al.*,^[11] nicotine causes increase in cholesterol levels. Our findings are in accordance with the work of Rustogi *et al.*^[12] As serum HDL level decreases, reverse cholesterol transport also decreases. Due to this, cholesterol deposition in peripheral tissue increases.^[13] The mean HDL value in smokers was 34.2 ± 5.1 mg/dL, whereas in non-smokers, it was 42.2 ± 4.6 mg/dL ($P < 0.001$). A similar study by Joshi *et al.* who studied 50 smokers and compared their lipid profile with 50 healthy participants reported that the mean HDL-C in non-smokers and smokers was 53.58 ± 9.87 and 47.32 ± 6.90 , respectively ($P < 0.002$).^[14] This finding is similar to study done by Neki who reported that there is a fall in HDL-C level by 3–5 mg/dl in smokers. The fall in estrogen level that occurs due to smoking further results in decreased HDL-C.^[9] In the present study, 9 smokers in the age group of 21–30 years had mean HDL-C of 39.9 mg/dL while 17 controls showed a value of 41.9 mg/dL. Age-related decrease was observed with advancing age group probably due to increase in the duration of smoking. Smokers in 31–40 years of age had mean HDL value of 32.8 mg/dL, which was low compared to controls of same age group 45.8 mg/dL. In the age group of 41–50 and >50 years, the smokers had mean HDL-C of 34.0 mg/dL and 30.9 mg/dL (12 persons), respectively, while in controls of the same age group, it was 41.0 mg/dL (14 persons) and 38.2 mg/dL, respectively. These findings are in conformity with the work of Zamir *et al.*^[11] Direct relationship of smoking toward CHD has been mentioned by Padmavathi *et al.*,^[15] who described that increase in HDL level by 1 mg/dL was associated with decrease in the risk of CHD by 3%. In the present study, LDL levels were also increased in smokers than non-smokers and are in agreement with results of Kesaneimi and Grundy.^[16] The mean LDL levels in smokers were 128.0 ± 28.3 mg/dL when compared to non-smokers 92.5 ± 20.9 mg/dL ($P < 0.001$). These observations are also similar to those of Rustogi *et al.*^[12] It has been described that nicotine contained in cigarette increased the circulatory pool of atherogenic LDL through accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment, and hence, LDL-C in the arterial wall increased.^[17,18] The results show that smoking influences the lipid profile negatively, hence causing dyslipidemia in smokers. Smoking results in increase in oxidized LDL-C level which plays a key role in the development of atherosclerosis and also raising the cardiovascular disease risk.^[9] Therefore, it could be probably deduced that smoking is very dangerous to health and should be discouraged. LDL-C level in this study showed increasing trend with increasing age; however, LDL values in 40–50 and >50 age group were almost same. In the present study, mean TG levels in smokers were 195.2 ± 46.3 mg/dL when compared to non-smokers 145.1 ± 24.1 mg/dL ($P < 0.001$). These findings are similar to those observed by Wynder *et al.*^[19] and Rustogi *et al.*^[12] Recent studies have suggested that triglyceride levels are the most important factor leading

to CHD.^[13] The reduced lipoprotein lipase activity in smokers as observed by Freeman *et al.*^[20] may explain impaired triglyceride metabolism and higher triglyceride levels. The present study findings are also in accordance with studies done by Sharma *et al.*,^[21] Mouhamed *et al.*,^[5] and Fariduddin *et al.*^[22] Several studies which have evaluated the risk factor for IHD have showed that reduced HDL-C-to-TC ratio is a very important risk factor. The present study showed a significant reduction in the HDL/TC ratio compared to those of control. Sonagra *et al.* studied 70 participants for lipid profile and reported that mean serum TC, serum TG, serum LDL, and serum very LDL were significantly higher while serum HDL-C was significantly lower in cases when compared with controls ($P < 0.05$).^[13] Smoking is also an independent and modifiable risk factor for the development of pulmonary diseases, cancers, cerebrovascular diseases, peripheral vascular diseases, etc. Risk of occurrence of these complications is directly proportional to the amount of smoking.^[23] Therefore, smokers should be counseled regarding health hazard to them as well as to people around them who become victims due to passive smoking. They should be encouraged to quit smoking and adopt healthy lifestyle to reduce the risk of developing health-related problems.^[24]

In limitations, small sample size and unblinding nature of the study were important; an element of observer bias may have occurred. Large population studies should be conducted to confirm the results.

CONCLUSION

The increase in TC, TG, and LDL was noted in all age groups with a history of smoking, whereas HDL showed an inverse relationship. Healthy smokers were found to be at risk of IHD due to these above changes. The observed values were in concordance with studies done in India and abroad. The alteration of lipid profile in smokers has raised serious medical concern with respect to atherogenic risk and recommendation for counseling the smokers to quit smoking, and routine evaluation of serum lipid profile has been suggested.

REFERENCES

1. Jha P, Peto R. Global effects of smoking, of quitting, and of taxing tobacco. *N Engl J Med* 2014;370:60-8.
2. Shafey O, Eriksen M, Ross H, Mackay J. *The Tobacco Atlas*. 3rd ed. Atlanta, USA: American Cancer Society; 2009.
3. *Global Adult Tobacco Survey: India Report*. Mumbai, India: International Institute for Population Sciences (IIPS), Ministry of Health and Family Welfare GoI; 2010.
4. Jha P, Jacob B, Gajalakshmi V, Gupta PC, Dhingra N, Kumar R, *et al.* A nationally representative case-control study of smoking and death in India. *N Engl J Med* 2008;358:1137-47.
5. Mouhamed DH, Ezzaher A, Neffati F, Gaha L, Douki W, Najjar MF. Association between cigarette smoking and

- dyslipidemia. *Immuno Anal Biol Spéc* 2013;28:195-200.
6. Talhout R, Schulz T, Florek E, van Benthem J, Wester P, Opperhuizen A, *et al.* Hazardous compounds in tobacco smoke. *Int J Environ Res Public Health* 2011;8:613-28.
 7. Jha P, Ramasundarathetige C, Landsman V, Rostron B, Thun M, Anderson RN, *et al* 21st-century hazards of smoking and benefits of cessation in the united states. *N Engl J Med* 2013;368:341-50.
 8. Muscat JE, Harris RE, Haley NJ, Wynder EL. Cigarette smoking and plasma cholesterol. *Am Heart J* 1991;121:141-7.
 9. Neki NS. Lipid profile in chronic smokers. *J Indian Acad Clin Med* 2002;3:51-4.
 10. White PD. Coronary disease and coronary thrombosis in youth. *J Med Soc* 1935;32:596-605.
 11. Zamir MA, Muhammad SB, Muhammad S. Lipid Profile in smoking. *J Appl Math Comput* 2000;12:19-21.
 12. Rastogi R, Shrivastava SS, Mehrotra TN, Singh VS, Gupta MK. Lipid profile in smokers. *J Assoc Physicians India* 1989;37:764-6.
 13. Sonagra AD, Shylaja TV, Makandar A, Deba Z. Study of lipid profile among healthy smokers and non-smokers. *Int J Biotechnol Biochem* 2017;13:87-94.
 14. Joshi N, Shah C, Mehta HB, Gokhle PA. Comparative study of lipid profile on healthy smoker and non-smokers. *Int J Med Sci Public Health* 2013;2:622-6.
 15. Padmavathi P, Reddy VD, Varadacharyulu N. Influence of chronic cigarette smoking on serum biochemical profile in male human volunteers. *J Health Sci* 2009;55:265-70.
 16. Kesaniemi YA, Grundy SM. Significance of low density lipoprotein production in the regulation of plasma cholesterol level in men. *J Clin Invest* 1982;70:13-22.
 17. Terry PD, Rohan TE, Franceschi S, Weiderpass E. Cigarette smoking and the risk of endometrial cancer. *Lancet Oncol* 2002;3:470-80.
 18. Carlson LA, Böttiger LE. Ischaemic heart-disease in relation to fasting values of plasma triglycerides and cholesterol. Stockholm prospective study. *Lancet* 1972;1:865-8.
 19. Wynder EL, Harris RE, Haley NJ. Population screening for plasma cholesterol: Community-based results from connecticut. *Am Heart J* 1989;117:649-56.
 20. Freeman DJ, Griffin BA, Murray E, Lindsay GM, Gaffney D, Packard CJ, *et al.* Smoking and plasma lipoproteins in man: Effects on low density lipoprotein cholesterol levels and high density lipoprotein subfraction distribution. *Eur J Clin Invest* 1993;23:630-40.
 21. Sharma P, Kumar P, Sharma R, Gupta G. Dyslipidemia among smokers. *Asian J Pharm Clin Res* 2016;9:137-8.
 22. Fariduddin JM, Masih SS. A comparative study on lipid profile and uric acid among smokers and non-smokers. *Int J Clin Biochem Res* 2016;3:469-71.
 23. Peto J. That the effects of smoking should be measured in pack-years: Misconceptions 4. *Br J Cancer* 2012;107:406-7.
 24. Wu J, Sin DD. Improved patient outcome with smoking cessation: When is it too late? *Int J Chron Obstruct Pulmon Dis* 2011;6:259-67.

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