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# Effect of Tobacco Smoking on Lipid Profile

KEYWORDS: Lipid profile, Smoking, LDL, HDL


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**ABSTRACT** *Background & objective: Tobacco smoking is an independent & modifiable risk factor for cardiovascular disease. Tobacco smoking has been found to alter lipid metabolism by stimulating sympathetic adrenal system leading to increased secretion of catecholamines. The proposed study was undertaken to elucidate the effect of tobacco smoking on lipid profile.*

*Materials & methods: 45 healthy smokers and 45 non smokers in the age group of 25 to 55 years were included in this study. Lipid profile was estimated by using enzymatic end point kit method.*

*Results: The total serum cholesterol, Triglyceride, LDL & VLDL were higher in smokers as compared to Non-smokers ($p<0.001$). Serum levels of HDL are lower in smokers as compared to in non-smokers ($p<0.001$).*

*Conclusion: The results shows that smoking influences the lipid profile adversely, resulting in the premature development of atherosclerosis, and also raising the cardiovascular disease risk.*


**Introduction:**

Tobacco, especially cigarette smoking is a major cause of CVD, responsible for about one third of CVD deaths. Tobacco smoking has been known to be an independent & modifiable risk factor for cardiovascular disease [CVD] (1, 2). Cardiovascular disease (CVD) is the leading cause of mortality and morbidity worldwide (1). Smoking is one of the modifiable risk factor for CVD. The risk of CVD deaths increases with increasing exposure to cigarette smoke, as measured by number of cigarette smoked daily, the duration of smoking & the degree of inhalation & the age of initiation. The relative risk for CVD is substantially greater in early adult life than in old age & is associated more strongly with the cigarette smoke than other forms of tobacco. Smoking also causes cancer, stroke and also have close relationship with gastric ulcer, periodontal disease, sudden infant death syndrome, and metabolic syndrome.

In addition it is also known to affect lipid profile adversely. Many studies have shown that tobacco smoking for long periods of time increases serum concentration of total cholesterol, triglycerides, LDL-Cholesterol, VLDL-Cholesterol and fall in the level of anti atherogenic HDL-Cholesterol (4). Tobacco smoking has been found to alter lipid metabolism by stimulating sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids [FFA] which further result in increased secretion of hepatic FFA and hepatic triglyceride along with VLDL-C in the blood stream (6, 7). Smoking prevents the intravascular remodelling of HDL particles (8). There are certain confounding factors which results in increase in serum lipids and cholesterol levels in addition to smoking habits these include hypertension, hyperlipidemia, obesity and physical inactivity (9, 10). The proposed study is being undertaken to elucidate the effect of tobacco smoking [cigarette / beedis] on lipid profile in otherwise healthy persons and to compare with non smokers.

**Materials & methods:**

The study involved forty five non smokers (control group) & forty five smokers (study group) within the age group of 25-55 years selected from the Department of Medicine, Vinayaka Mission's Hospital, Salem, Tamil Nadu, India. Subjects were selected based on inclusion and exclusion criteria.

**Inclusion criteria [Control group]:**

Forty five healthy non smokers between 25 and 55 years.

**Exclusion criteria [Control group]:**

- History of Hypertension
- History of Diabetes mellitus
- History of smoking
- Persons abusing alcohol, ex-smokers.
- History of renal disease, hepatic impairment, endocrine disorders

**History of obesity**

- History of lipid lowering drugs, b-blockers, thiazide diuretics, oral antidiabetic drugs and hormonal preparations.
- Inclusion criteria [Study group]:
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History of lipid lowering drugs, b-blockers, thiazide diuretics, oral antidiabetic drugs and hormonal preparations.

**Methodology:**

Forty five apparently healthy male subjects in the age

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group of 25-55yrs were included for the study. The subjects were selected by a detailed history & thorough physical examination. They were asked to fill a questionnaire to assess their smoking habits. The experimental protocol was fully explained to the participants to allay apprehension. They refrained from any smoking for 12hours before the test. Informed consent was taken from all the subjects. The study was approved by Institutional Ethical Committee, Vinayaka Mission's Kirupananda Variyar Medical College & Hospitals, Salem, Tamil Nadu, India.

**Experimental design:**
Lipid profile was estimated by drawing 5ml of fasting blood sample. Serum lipids and lipoproteins cholesterol fractions were measured on fasting state. Total cholesterol and triglyceride level estimations are carried out using enzymatic end point kit method. HDL cholesterol was estimated by precipitation of non HDL lipoproteins and estimations done using superannuant. Both VLDL and LDL are estimated by calculation.

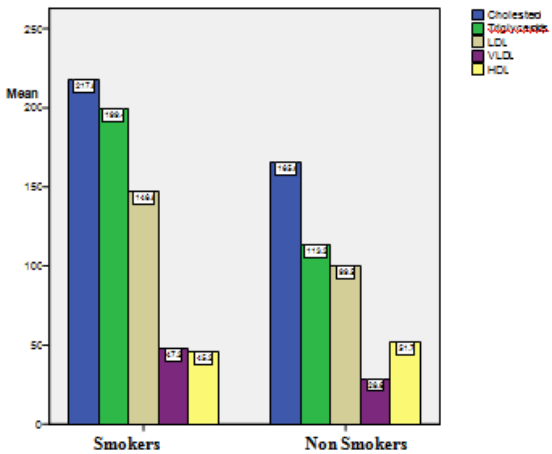
**Statistical analysis:**
The results were expressed as mean ± standard deviation (SD). A p value of <0.05 was considered statistically significant. Statistical analysis was performed using the statistical package for social & sciences. Students unpaired t' test was applied to correlate between the parameters.

**Results:**
Lipid profile was estimated in Forty five healthy non smokers in the age group of 25 and 55 (42.38 ± 8.02) years and Forty five smokers in the age group of 25 and 55 (43.62 ± 6.63) years. Total cholesterol (TC) (217.75 ± 38.05)mg/dl, triglyceride (TG) (199.44 ± 59.80) mg/dl, low-density lipoprotein cholesterol (LDL-C) (146.84 ± 28.64) mg/dl, very-low density lipoprotein cholesterol (VLDL-C) (47.29 ±10.02) mg/dl, and LDL-C/HDL-C (atherogenic index) (4.84 ± 0.90) were significantly higher in smokers as compared with non-smokers TC (165.6 ± 19.39 ) mg/dl, TG (113.22 ± 22.27 ) mg/dl, LDL-C (99.53 ± 13.95)mg/dl, VLDL-C (28.6 ± 6.16) mg/dl, and LDL-C/HDL-C (3.21 ± 0.74) with p<0.001, p<0.001, p=0.001, p<0.001, p=0.001 respectively. While high-density lipoprotein (HDL-C) (45.17 ± 3.36) mg/dl in smoking group was significantly lower as compared with non-smoker (51.69 ± 7.85) mg/dl (p=0.001). The results are shown in the table 1 & figure 1.

**Table 1: Comparison of lipid profile between Smokers & Non-smokers**

| Parameters | Non Smokers | Smokers | 'p' value |
|---|---|---|---|
| TC (mg/dl) (mean ± sd) | 165.6 ± 19.39 | 217.75 ± 38.05 | < 0.001 |
| TG (mg/dl) (mean ± sd) | 113.22 ± 22.27 | 199.44 ± 59.80 | < 0.001 |
| LDL(mg/dl) (mean ± sd) | 99.53 ± 13.95 | 146.84 ± 28.64 | < 0.001 |
| VLDL(mg/dl) (mean ± sd) | 28.6 ± 6.16 | 47.29 ±10.02 | < 0.001 |
| HDL(mg/dl) (mean ± sd) | 51.69 ± 7.85 | 45.17 ± 3.36) | < 0.001 |
| TC/HDL (mean ± sd) | 3.21 ± 0.74 | 4.84 ± 0.90 | < 0.001 |

**Fig 1: Comparison of lipid profile between Smokers & Non-smokers**




**Discussion:**
Epidemiological studies have shown that long-term morbidity and mortality in coronary heart disease (CHD) is directly related to circulating levels of atherogenic lipoproteins, in particular LDL-C (12). Various mechanisms leading to lipid alteration by smoking are (a) nicotine stimulates sympathetic adrenal system leading to increase secretion of catecholamines 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 (13); (b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL – cholesterol(14); (c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL-C, VLDL-C, and TG due to decreased activity of lipoprotein lipase(15).

Willett et al reported that cigarette smoking affects cholesterol metabolism, it lower levels of the protective high-density lipoprotein (HDL) cholesterol (16) and Rabkin et al reported smoking cessation raises HDL cholesterol (17). In animal models, cigarette smoke can damage the inner lining of blood vessels, thus enhancing the transfer of low-density lipoprotein cholesterol particles across the arterial wall and into the developing cholesterol-laden plaque (18). Cigarette smoking also can affect the blood clotting system, including adherence of blood platelets to the lining of arterial blood vessels (19) and the formation of blood clots that block a narrowed artery. Selley et al reported that Acrolein in cigarette smoke may be partly responsible for its platelet-adhering effects (20). Cigarette smoke also causes spasm of the coronary arteries (21), many chemical components of cigarette smoke have been found to accelerate the development of atherosclerotic disease. Nicotine, the major psychoactive component of smoke, causes powerful changes in heart rate and blood circulation. Nicotine appears to cause injury to the arterial lining (18). Sheps et al reported that Carbon monoxide in cigarette smoke binds to the hemoglobin in red blood cells, thereby reducing the oxygen-carrying capacity of the blood (22).

Despite the damaging effects of tobacco use, quitting smoking has immediate and long term effects such as improved circulation, drop in heart rate. Smoking cessation is an important lifestyle measure for the prevention of cardiovascular disease, and in patients with myocardial infarction there is 50% reduction in risk of re-infarction, sudden

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cardiac death (11). Quitting in late in life also has positive effects.

**Conclusion:**

Total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), very-low density lipoprotein cholesterol (VLDL-C), LDL-C/HDL-C (atherogenic index), and triglyceride (TG) were significantly higher in smokers as compared with non-smokers.

Smoking has adverse effects on lipid profile, therefore increasing the cardiovascular disease risk.

The high prevalence of an atherogenic lipid profile in smokers makes them prone to develop premature atherosclerosis and the changes become more marked with the number of cigarette/day smoked.

**REFERENCE**

1. Khurana M, Sharma D, Khandelwal PD .Lipid profile in smokers and tobacco smokers-a comparative study. J Assoc Physicians India 2000;48:895-897. | 2. Yambe T, Meng X, Hou X, Wang Q, Sekine K, Shiraishi Y, Watanabe M, Yamaguchi T, Shibata M, Kuwayama T, Murayam M, Konno S, Nitta S. Cardio-ankle vascular index (CAVI) for the monitoring of the atherosclerosis after heart transplantation. Biomed Pharmacother. 2005;59(Suppl 1):S177–S179. [PubMed] | 3. Teo KK, Ounpuu S, Hawken S, Pandey MR, Valentin V, Hunt D, Diaz R, Rashed W, Freeman R, Jiang L, Zhang X, Yusuf S, on behalf of the INTERHEART Study Investigators. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. Lancet. 2006; 368: 647–658. | 4. NS Neki. Lipid profile in chronic smokers-a clinical study. J Academy clinical med 2002;3:51-4. | 5. Sanjay P, Zodpey, Suresh NU. "Tobacco smoking and smoking index" Regional health forum; Vol 3. | 6. Sidhartha das, Mohan V. Disorders of lipid metabolism. Chapter 8, API Text book of medicine, 7TH edition ,API Publishers, 2003; 250 – 256. | 7. Fischer SD, Zarebow, Masti AJ, Marder VJ. Effect of smoking on lipid and thrombogenic factors. Am J Cardiol.2000;86:813-18. | 8. Wu DM, Pai L, Sun PK, Hsu LL, Sun CA .Joint effect of alcohol consumption and cigarette smoking on atherogenic lipids and lipoprotein profiles. Eur J Epid 2001;17:629-35. | 9. Sinha AK, Misra GC, Patel DK. Effect of cigarette smoking on lipid profiles in the young. J Assoc Physcians India.1995;43:185-8. | 10. Adedeji DA, Etukudo MH. Lipid profile of cigarette smokers in calabar municipality. Pak J of Nutrition 2006;5:237-38. | 11. Jatoi NA, Jerrard-Dunne P, Feely J, Mahmud A. Impact of smoking and smoking cessation on arterial stiffness and aortic wave reflection in hypertension. Hypertension 2007;49:981-5. | 12. Sharret AR, Ballantyne CM, Coady SA et al. Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein(a), apolipoproteins A-I and B, and HDL density subfractions: The Atherosclerosis Risk in Communities (ARIC) Study. Circulation 2001;104:1108–1113. | 13. Muscat JE, Harris RE et al. Cigarette smoking and plasma cholesterol. Am Heart J 1991; 121: 141-7. | 14. Benouwitz HL. Pharmacologic aspects of cigarette smoking and nicotine addiction. New Engl J Med 1998; 319: 318-30. | 15. Reaven GM. Role of insulin resistance in human disease. Diabetes 1988; 37: 595-607. | 16. Willett, W., Hennekens, C.H., Castelli, W., Rosner, B., Evans, D., Taylor, J., Kass, E.H. Effects of cigarette smoking on fasting triglyceride, total cholesterol, and HDL-cholesterol in women. American Heart Journal 105(3):417-421, 1983. | 17. Rabkin, S.W. Effect of cigarette smoking cessation on risk factors for coronary atherosclerosis: a control clinical trial. Atherosclerosis 53(2): 173-184, 1984. | 18. Krupski, W.C., Olive, G.C., Weber, C.A., Rapp, J.H. Comparative effects of hypertension and nicotine on injury-induced myointimal thickening. Surgery 102: 409-415, 1987. | 19. Pittilo, R.M., Clarke, J.M., Hams, D., Mackie, I.J., Rowles, P.M., Machin, S.J., Woolf, N. Cigarette smoking and platelet adhesion. British Journal of Haematology 58(4): 627-632, 1984. | 20. Selley, M.L., Bartlett, M.R., McGuiness, J.A., Ardlie, N.G. Effects of acrolein on human platelet aggregation. Chemico-BiologicalInteractions 76(1):101-109, 1990 | 21. Stern S, Bayes de Luna A. coronary artery spasm: a 2009 update. Circulation. 2009 May 12;119(18):2531-4. | 22. Sheps, D.S., Herbst, M.C., Hinderliter, A.L., Adams, K.F., Ekelund, L.G., O'Neil, J.J., Goldstein, G.M., Bromberg, P.A., Dalton, J.L., Pallenger, M.N., Davis, S.M., Koch, G.G. Production of arrythmias by elevated carboxyhemoglobin in patients with coronary artery disease. Annals of Internal Medicine 113(5): 343-351, 1990. |