A Comparative Study on the Effect of Tobacco Smoke and Tobacco Salivary Extract on Serum Lipid Profile

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Abstract
Background: Which form of tobacco use has more adverse effects on lipid profile?
Objective: To identify the effect of tobacco use as smoking or chewing is associated with greater adverse effects on various components of serum lipid profile.
Materials and Methods: The study was conducted on 400 subjects (200 tobacco smoker & 200 tobacco chewer) admitted in wards and attending OPD under department of medicine, NIMS medical college & hospital Jaipur and 400 controls. S. Cholesterol by enzymatic end point CHOD/PAP method. S. Triglyceride by enzymatic glycerol phosphate oxidase/peroxidase method S. HDL by homogenous enzyme direct assay. Statistical analysis was done using SPSS Ver. 20 (IBM SPSS Statistical Inc., Chicago, Illinois, USA). The independent t test and ANOVA with post hoc Bonferroni test were used for comparison of all clinical indicators. Chi – square test used for qualitative data whenever two or more than two groups were used to compare. Level of significance was set at P≤0.05.
Results: Mean value of TC, TG, LDL, VLDL, of tobacco smokers and tobacco chewers were found significantly higher (P<0.001) as compared to mean values of these parameters amongst non smokers, non chewers. The mean HDL amongst tobacco smokers was significantly lower as compared to controls.
Conclusion: Tobacco is the major preventable risk factor for atherosclerosis related clinical events like coronary artery disease. Cigarette smoking had greater adverse effects on the serum lipid profile as compared to bidi smoking and tobacco chewing thereby increasing the cardiovascular risk.
Keywords: Tobacco, Lipid profile, Cigarette smoke, Tobacco Chewing.

Introduction
Tobacco was introduced to India in the 17th century[1]. Moreover, India is third largest tobacco producer and its product consumer, after China and U.S.A. in the world. The main form of tobacco use in developed countries is high grade smoking (Cigarette), while low grade sucking (Bidi), chewing and snuffing are common in developing countries[2]. There is a wide variety of tobacco products found in India, consumption of tobacco in its various forms is the major and single most preventable risk factor for atherosclerosis related clinical events[3]. All forms of tobacco use are reported to carry serious health
consequences, an important one being dyslipidemia. According to National Cholesterol Education Programme: Adult Treatment Panel-III (NCEP: ATP-III)\(^4\) dyslipidemia is defined as follows:

- Hypercholesterolemia: Serum cholesterol levels >200 mg/dl
- Hypertriglyceridemia: Serum triglyceride levels >150 mg/dl
- Low HDL cholesterol: HDL cholesterol levels <40 mg/dl for men and <50 mg/dl for women.
- High LDL cholesterol: High LDL cholesterol LDL cholesterol levels >130 mg/dl (Friedewald equation)
- Isolated hypercholesterolemia: Serum cholesterol ≥200 mg/dl and triglyceride <150 mg/dl
- Isolated hypertriglyceridemia: Serum triglycerides ≥150 mg/dl and cholesterol <200 mg/dl

Several studies provide the evidence that tobacco is strongly associated with altering the normal status of lipid profile. There is still much controversy about which parts of lipid are mainly altered in response to tobacco use. In a worldwide report, it was found that male smoking far exceeds female smoking, while a smaller gender difference occur in high-income countries.

**Material and Methods**

A cross sectional study was carried out in the department of medicine, NIMS medical college and hospital, Jaipur (INDIA). Subjects were both male and female of age more than 18 years and less than 60 years. All were admitted in wards or attending OPD under the department of medicine. Total 400 subjects were taken in two groups 200 tobacco chewing and 200 tobacco smoking. Blood sample was collected by venipuncture in standard aseptic condition. Serum was separated and subjected to estimation biochemical parameters on fully automatic analyzer (Trivitron Nano lab 150). Lipid profile estimation was done using following methods:

- S. Cholestrol by enzymatic end point CHOD/PAP method \(^5\).
- S. Triglyceride by enzymatic glycerol phosphate oxidase / peroxidase method \(^6\).
- S. HDLc by homogenous enzyme direct assay \(^7\).
- S. VLDL=TG/5 (Freidwald’s formula)\(^8\).
- S.LDL=Total Cholestrol-(HDL+VLDL) (Freidwald’s formula)\(^8\).

The data was analyzed using SPSS version 20 (IBM SPSS Statistics Inc., Chicago, Illinois, USA) windows software program. The variable was assessed for normally using the Kolmogorov Smirmov test. Descriptive statistics included computation of percentages, means and standard deviation. The independent (unpaired) t test ( for quantitative data within three groups) and analysis of variance (ANOVA) (for quantitative data within three groups) with post hoc Bonferroni test ( to make more intra – group comparison) were used for comparison of all clinical indicators Chi square test used for qualitative data whenever two or more than two groups were used to compare. Level of significance was set at P<0.05.

**Result**

**TC:** Mean TC amongst cigarette smokers was 226.26±59.54mg/dl as compared to mean TC among controls was 155.10±39.28mg/dl. There was a statistically significant difference between mean TC for cigarette smokers and controls as P < 0.001. Mean TC amongst bidi smokers was 190.73±29.59mg/dl as compared to mean TC among controls was 155.10±39.28mg/dl with mean difference of 35.63. There was a statistically significant difference between mean TC for bidi smokers and controls as P < 0.001. Mean TC amongst tobacco chewers was 187.51±40.46mg/dl as compared to mean TC among controls was 155.10±39.28mg/dl with mean difference of 32.41. There was a statistically significant difference between mean TC for tobacco chewers and controls as P < 0.001[Table-1, Fig-1,2,3].

**TG:** Mean TG among cigarette smokers was 198.85±120.84mg/dl as compared to mean TG
against controls was 134.31±61.67mg/dl. Mean TG difference between cigarette smokers and controls was highly significant as P <0.001. Mean TG amongst bidi smokers was 172.56±75.33mg/dl, as compared to mean TC against control was 134.31±61.67. Mean TG difference of 38.25 between bidi smokers and controls was highly significant as P <0.001. Mean TG amongst tobacco chewers was 168.53±80.46mg/dl, as compared to mean TC against control was 134.31±61.67. Mean TG difference of 34.15 between tobacco chewers and controls was highly significant as P <0.001[Table-1, Fig-1,2,3].

**HDL:** Mean HDL amongst cigarette smokers was 36.13±1.08mg/dl as compared to mean HDL amongst controls was 39.89±8.48mg/dl. The mean HDL amongst cigarette smokers was significantly decreased as compared to mean HDL amongst controls as P was 0.002. Mean HDL amongst bidi smokers was 36.13±1.08mg/dl as compared to mean HDL amongst controls was 39.89±8.48mg/dl with mean difference of 3.58 between these two groups. The mean HDL amongst bidi smokers was significantly decreased as compared to mean HDL amongst controls as P < 0.001[Table-1, Fig-1,2,3]. Mean HDL amongst tobacco chewers was 35.95±7.901mg/dl as compared to mean HDL amongst controls was 39.89±8.48mg/dl with mean difference of 3.94 between these two groups. The mean HDL amongst tobacco chewers was significantly decreased as compared to mean HDL amongst controls as P < 0.001.

**LDL:** Mean LDL amongst cigarette smokers was 142.78±53.26mg/dl as compared to mean LDL amongst controls was 89.5±34.33mg/dl. Mean LDL difference between cigarette smokers and controls was highly significant as P was 0.001. Mean LDL amongst bidi smokers was 115.91±26.705mg/dl as compared to mean LDL amongst controls was 89.5±34.33mg/dl. Mean LDL difference between bidi smokers and controls was 26.41 which was highly significant as P < 0.001. Mean LDL amongst tobacco chewers was 112.92±37.19mg/dl as compared to mean LDL amongst controls was 89.5±34.33mg/dl. Mean LDL difference between tobacco chewers and controls was 23.42 which was highly significant as P <0.001[Table-1, Fig-1,2,3].

**VLDL:** Mean VLDL amongst cigarette smokers was 39.89±24.109 mg/dl as compared to mean VLDL amongst controls was 27.09±12.85mg/dl. Mean VLDL difference between two groups was highly significant as P <0.001. Mean VLDL amongst bidi smokers was 34.56±15.03 mg/dl as compared to mean VLDL amongst controls was 27.09±12.85mg/dl. Mean VLDL difference between two groups was 7.46 which was highly significant as P <0.001. Mean VLDL amongst tobacco chewers was 33.78±16.07mg/dl as compared to mean VLDL amongst controls was 27.09±12.85mg/dl. Mean VLDL difference between two groups was 6.69 which was highly significant as P <0.001[Table-1, Fig-1,2,3].

**Table-1:** Comparison of lipid profile between cigarette smokers, bidi smokers and tobacco chewers with respect to control

| Tobacco Users | Lipid Profile | N  | Mean  | SD  | Mean Difference | P Value |
|---------------|---------------|----|-------|-----|----------------|---------|
| **Cigarette Smokers** | TC | Cont. | 400 | 155.10 | 39.28 | 71.16 | 0.001 |
| | | Sub. | 80 | 226.26 | 59.54 | | |
| | TG | Cont. | 400 | 134.31 | 61.67 | 64.53 | 0.001 |
| | | Sub. | 80 | 198.85 | 120.84 | | |
| | HDL | Cont. | 400 | 39.89 | 8.48 | 3.76 | 0.002 |
| | | Sub. | 80 | 36.13 | 1.08 | | |
| | LDL | Cont. | 400 | 89.5 | 34.33 | 53.28 | 0.001 |
| | | Sub. | 80 | 142.78 | 53.26 | | |
| | VLDL | Cont. | 400 | 27.09 | 12.85 | 12.79 | 0.001 |
| | | Sub. | 80 | 39.89 | 24.109 | | |
| Bidi Smokers | TC | Cont. 400 155.10 35.63 0.001 |
|             | TG | Cont. 400 134.31 38.25 0.001 |
|             | HDL| Cont. 400 39.89 3.58 0.001 |
|             | LDL| Cont. 400 89.5 26.41 0.001 |
|             | VLDL| Cont. 400 27.09 7.46 0.001 |
| Chewers     | TC | Cont. 400 155.10 32.41 0.001 |
|             | TG | Cont. 400 134.31 34.21 0.001 |
|             | HDL| Cont. 400 39.89 3.94 0.001 |
|             | LDL| Cont. 400 89.5 23.42 0.001 |
|             | VLDL| Cont. 400 27.09 6.69 0.001 |

**Fig-1:** Comparison of lipid profile between cigarette smokers and control

**Fig-2:** Comparison of lipid profile between bidi smokers and control
Discussion
Awareness of correlation between smoking and cardiovascular disease remains low in many parts of the world. Tobacco smoking or chewing is a major risk factor cardiovascular morbidity and mortality\[^9\]. Smoking is estimated to cause 10% of cardiovascular disease (CVD) and is second leading cause of CVD, after high blood pressure. The risk for coronary heart disease is 25% higher in female smokers than in male smokers\[^10\]. Chewing tobacco more than doubles the risk of heart attack and tobacco smoking and chewing may lead to alteration of the normal plasma lipoprotein pattern. In this study we have found that among tobacco smokers mean TC, TG, LDL, VLDL, were significantly higher as compared to mean values of these parameters amongst non smokers, non chewers. The mean HDL amongst tobacco smokers was significantly lower as compared to controls. In other study it was found TC,TG,LSL, VLDL were higher in smokers as compared to non smokers (P<0.001). HDL was lower in smokers as compared to in non smokers (P<0.001)\[^11\]. Our study was in agreement with studies of Ketan Patel et al\[^12\] and Srinivasa Rao et al\[^13\].

Conclusion
Tobacco is the major preventable risk factor for atherosclerotic related clinical events like...
coronary artery disease. Cigarette smoking had greater adverse effects on the serum lipid profile as compared to bidi smoking and tobacco chewing thereby increasing the cardiovascular risk.

References

1. Gokhle B G. Tobacco in seventeenth – century India. Agricultural history.1974. 48(4):484-492.
2. Mohamed S, Ibrahim A, and Joyce Y.G. Clinical Methods: The History, Physical and Laboratory examination. Chapter 40 : Tobacco use. 3rd edition. Boston: Butterworths:1990.
3. Jha P, Chaloupka F J, Curbing the Epidemic Governments and the Economics of Tobacco control. Washington, D.C. A World Bank Publication;1995.
4. Executive summary of the third report of the National Cholesterol Education Program (NCEP). Expert Panel on detection, evaluation, and treatment of high blood cholesterol in Adults ( Adults treatment panel III) JAMA . 2001; 285 (19):2486-97.
5. Allain C C, Poon L S, Clen C S G, Richmond W, Fu P C. Enzymatic determination of total serum cholesterol. Clin.Chem.1974.20:470-75.
6. Fossati P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem.1982. 28(10):2077-80.
7. Finley P R, Schifman R B, Williams R J, Lichti D A. Cholesterol in high-density lipoprotein: use of Mg+ / dextran surface in its enzymatic measurement. Clin.Chem. 1978. 24(6):931-933.
8. Friedwald W T. Levy R I, Fredrickson D S. Estimation of the concentration of low – density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem.1972. 18:499-509.
9. Teruo I. Cigarette smoking as risk factors of coronary artery disease and effects on platelets function. Tobacco Induced diseases.2004. 2(1):27-33.
10. Huxley R, Woodward M. Cigarette smoking as a risk factors for coronary heart disease in women compared with men : a systemic review and meta-analysis of prospective cohort studies. Lancet. 2011.378(9799):1297-1305.
11. Prabha V, Waheeda S, Meriton S A.. Effect of tobacco smoking on lipid profile. Indian Journal of applied research, 2015. 5(3):562-564.
12. Patel K, Prajapati P, Sanghavi S, Goplani V. A study on effects of cigarette smoking on blood cholesterol in young population of Ahmedabad. IJBAP. 2014.3(1):106-110.
13. Srinivas R and Emmanuel S Y. The effect of chronic Tobacco Smoking and Chewing on the profile. J Clin Diagn Res. 2013. 7(1):31-34.