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Comparison of Lipid Profile in Smokers and Non Smokers in and around Nalgonda, Andhra Pradesh

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ABSTRACT

A large number of risk factors which predispose to atherosclerosis and coronary artery diseases have been identified. These include modifiable ones like hypertension, dyslipidemia, smoking, diabetes mellitus, changing lifestyle and non-modifiable ones like age and sex. As the number of risk factors in an individual increases, so does the risk of developing atherosclerosis and its complications mainly as coronary artery diseases (CAD). In subject more than one of these risk factors the risk is more than additive.

Although smoking has been established as an independent risk factor for coronary heart disease, the mechanism by which it increases the risk of coronary heart disease is unclear. However, studies to date have revealed incomplete, inconclusive or conflicting results about the association of smoking on the plasma lipid and lipoprotein levels. In some studies, smokers had increased plasma cholesterol levels, in others plasma cholesterol level have actually been lower. Only a few studies have specifically examined the plasma lipoprotein according to smoking status or no. of cigarettes (dosage). Smokers are reported to have higher LDL and lower HDL cholesterol levels than non-smokers. A total number of 200 age and sex matched subjects comprising of 100 healthy non smokers as controls and 100 healthy smokers as cases were included in the study. Lipid profile was studied in both the groups. However in the present study, the levels total cholesterol, triglycerides, VLDL and LDL cholesterol very significantly increased and decrease in HDL cholesterol in smokers when compared to non smokers. This can be attributed to the risk of cardiovascular diseases and atherosclerotic changes in smokers.

Keywords: *Lipid profile. Total cholesterol, HDL cholesterol, Triglycerides, VLDL, LDL cholesterol*

INTRODUCTION

A large number of risk factors which predispose to atherosclerosis and coronary artery diseases have been identified. These include modifiable ones like hypertension, dyslipidemia, smoking, diabetes mellitus, changing lifestyle and non-modifiable ones like age and sex. As the number of risk factors in an individual increases, so does the risk of developing atherosclerosis and its complications mainly as coronary artery diseases (CAD) [1]. In subject more than one of these risk factors the risk is more than additive.[2]

Although smoking has been established as an independent risk factor[3] for coronary heart disease, the mechanism by which it increases the risk of coronary heart disease are unclear. Four explanations have been postulated.

i. The increased carbonmonoxide[4] in the blood of cigarette smokers may damage the endothelium and accelerate the entry of cholesterol into the wall of the artery promoting the development of atherosclerosis, thrombosis[5],

ii. The formation of carboxyhemoglobin creates relative anoxemia in the tissue, including the myocardium, Smoking enhances the platelet aggregation[6], and

iii. The nicotine absorbed from cigarette smoke may induce cardiac arrhythmias through its

pharmacologic action.

iv. An additional mechanism has been recently suggested that smoking adversely affects the concentration of the plasma lipids and lipoproteins.

However, studies to date have revealed incomplete, inconclusive or conflicting results about the association of smoking on the plasma lipid and lipoprotein levels. In some studies, smokers had increased plasma cholesterol levels, in others plasma cholesterol level have actually been lower[7]. 0nly a few studies have specifically examined the plasma lipoprotein according to smoking status or no. of cigarettes (dosage) [8]. Smokers are reported to have higher LDL and lower HDL cholesterol levels than non-smokers[9].

There is inadequate data on the association of smoking and dyslipidemia in India. Also there is wide spread habit of smoking cigarette and beedis and also increased prevalence of coronary artery disease among rural population of Nalgonda district and surrounding area.

The present study provides a detailed prolife of the plasma lipid and lipoprotein levels according to cigarette smoking status (smoker, ex-smoker and non-smoker) and dosage (number of cigarette smoked per day) in this part of India

AIMS AND OBJECTIVES

The present study is done to

1. Compare lipid profile values of smokers with non smokers.
2. To know if smoking has dyslipidemic potential.

MATERIALS AND METHOD

The study was carried out in 100 healthy male smokers and 100 healthy male non smokers from January 2007 to December 2008, selected from volunteers from general public, patient attendants and hospital staff of KIMS Narketpally, Nalgonda, Andhra Pradesh, India.

After obtaining written consent, detailed history and physical examination was done in all subjects.

Inclusion criteria for smokers and non-smokers

1. The subjects were divided into 4 groups

a) Non- smokers : subjects who have never smoked or those who left smoking atleast 5 yrs before in the present study

b) Mild smokers: 1-10 cigarettes or 1-15 beedis / day for atleast 5 yrs or more

c) Moderate smokers: 11-20 cigarettes or 16- 30 beedis / day for atleast 5 yrs or more.

d) Heavy smokers: more than 20 cigarettes or 30 beedis / day for atleast 5 yrs or more.

2. The subject's were chosen in age groups of 20 – 50 yrs of age
3. The subject's BMI were less than 28
4. The subjects were taking average Indian diet.

Exclusion criteria for smokers and non- smokers

1. Subjects having diseases mentioned below known to influence blood lipids were excluded from the study

- Diabetes mellitus
- Nephrotic syndrome
- Alcoholism
- Hypertension

2. Subjects who were on following drugs:

- HMG Co A Reductase inhibitors
- Nicotinic acid
- Beta blockers
- Diuretics

3. Subjects who were on diet restriction

After overnight fasting following laboratory investigations were done in all subjects

- Serum total cholesterol
- Serum high density lipoprotein (HDL)

- Serum low density lipoprotein (LDL)
- Serum very low density lipoprotein (VLDL)
- Serum triglyceride (TGL)

Collection of blood sample for analysis:

5ml of fasting venous blood was drawn from the subjects after overnight fasting in to a sterile disposable syringe which was transferred into plain tubes. The samples were centrifuged at 3000 rotations per minute for 10 min and serum was collected. The serum was processed within one hour of collection.

Investigations performed

1) Total Cholesterol and HDL cholesterol

Method: CHOD-POD.

Principle: Cholesterol is determined after enzymatic hydrolysis and oxidation, Cholesterol esters are hydrolyzed by the enzyme cholesterol esterase to give free cholesterol and fatty acid molecules. The free cholesterol gets oxidized in the presence of cholesterol oxidase to liberate cholest-4-ene-3one and peroxide. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.

The intensity of this colored complex is measured at 505nm and is directly proportional to the cholesterol concentration present in the sample.

Total cholesterol = Absorbance of T_1/Absorbance of Standard x 200 mg/dl.

HDL cholesterol = Absorbance of T_2/ Absorbance of Standard x 50 mg/dl.

2) Triglycerides:

Method: Glycerol phosphate oxidase (GPO) and peroxidase (POD)

Principle: Triglycerides are determined after enzymatic hydrolyzed with lipases, Serum triglycerides are hydrolyzed to glycerol and free fatty acid by lipases. In the presence of ATP and glycerol kinase. Glycerol is converted to glycerol 3 phosphate which is then oxidized by GPO to yield hydrogen peroxide. Peroxide catalyses the conversion of hydrogen peroxide, 4-aminoantipyrine and ESPAS to a colored quinoneimine complex measured at 546nm.

3. **LDL & VLDL** were calculated as follows: Friedwald formula (NCEP 2001)

(1) VLDL = TGL ÷ 5

(2) LDL = Total Cholesterol – (VLDL + HDL).

Concentration represented in mg/dl.

4. **Atherogenic index:** AIP was computed for each patient according to the following equation[11]:

$$AIP = \log (TGL/HDL)$$

With units for TGL and HDL in mmol/L.

Statistical Analysis

The results were statistically analyzed by the student's t-test by using sigma stat software. P-value of < 0.05 was considered as statistically significant.

RESULTS

In the present study 100 smokers and 100 non-smoker subjects were studied for their lipid profile. The smokers were further divided into three groups based on number of cigarettes/beedis smoked per day which is shown in table 1.

Table 1: Distribution of smokers based on no. of cigarettes/beedis smoked per day.

Group	No. of subjects	Percentage
Mild smokers: 1-10 cigarettes or 1-15 beedis/day	42	42%
Moderate smokers: 11-20 cigarettes or 16-30 beedis/day	44	44%
Heavy smokers: >20 cigarettes or >30 beedis/day	14	14%
Total	100	100%

According to this table 1, in the present study the number of subjects in mild smokers group were 42 (42%), the number of subjects in moderate smokers group were 44 (44%), the number of subjects in heavy smokers were 14 (14%) of the total 100 smokers.

Table 2: Lipid profile in non-smokers and smokers.

Lipid profile	Non-smokers n = 100	Smokersn = 100	P value
Total cholesterol	140.12 ± 28.58	175.63 ±28.46	<0.05
Serum triglycerides	101.84 ± 24.23	129.76± 38.62	<0.05
Serum LDL	73.93 ± 28.35	118.11 ±29.08	<0.05
Serum VLDL	20.36 ± 4.84	29.95 ± 7.72	<0.05
Serum HDL	45.82 ±7.65	31.56 ± 5.91	< 0.05

According to table 2, smokers had higher total cholesterol levels compared to non-smokers (175.63 versus 140.12) this difference was statistically significant. Smokers had higher plasma triglyceride level compared to non-smokers (129.76 versus 101.8) this difference was statistically significant. Smokers had higher serum LDL levels compared to non-smokers, (118.11 versus 73.93) this difference was statistically significant. Smokers had higher VLDL levels compared to non-smokers (29.95 versus 20.36) this difference was statistically significant. Smokers had lower levels of serum HDL compared to non-smokers (31.56 versus 45.82) and this difference was statistically significant.

Table 3: Lipid profile in relation to number of cigarette/beedis smoked per day in smokers as compared to non-smokers.

Lipid profile (mg/dl)	Non-smokers (n=100)	Mildsmokers (n=42)	PValue	Moderate smokers (n=44)	PValue	HeavySmokers (n=14)	PValue
Total cholesterol	140.12	149.85 ±12.32	<0.05	187.79 ±14.04	<0.05	214.71 ±27.30	<0.05
Serum triglycerides	101.84	117.78 ±28.71	<0.05	129.04 ±37.77	<0.05	167.92 ±44.77	<0.05
Serum LDL	73.93	91.60 ±12.61	<0.05	131.39 ± 14.84	<0.05	155.91 ±28.37	<0.05
Serum VLDL	20.36	23.55 ±5.74	<0.05	25.80± 7.55	<0.05	33.58± 8.95	<0.05
Serum HDL	45.82	34.69 ±4.40	<0.05	30.59 ± 5.29	<0.05	25.21 ± 5.87	<0.05

In table 3, smokers were further subdivided into mild, moderate, heavy group based on number of cigarette/beedis smoked and the values of each group were compared with non-smokers.

Total cholesterol value was highest in heavy smokers (214.71), less in moderate smokers (187.79) and least in mild smokers (149.85). The difference of these values with non-smokers was statistically significant.

The triglyceride levels were highest in heavy smokers (167.92), less in moderate smokers (129.04) and least in mild smokers (117.78). The difference of these values with non-smokers was statistically significant.

The serum LDL level were highest in heavy smokers (155.91), less in moderate smokers (131.39) and least in least in mild smokers (91.60).the difference of these values with non-smoker was statistically significant.

The serum VLDL level was highest in heavy smokers (33.58), less in moderate smokers (25.80) and least in mild smokers (23.55). The difference of these values with non-smokers was statistically significant.

The serum HDL level was lowest in heavy smoker group (25.21), higher in moderate smoker group (30.59) and highest in mild smoker group (34.69). The difference of these values compared to non-smoker group was statistically significant.

DISCUSSION

Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease. In the present study 100 smokers and 100 nonsmokers were studied for their lipid profile. Age, sex, obesity, alcohol, diet-these parameters were matched in smokers and non smokers.

It is revealed that triglycerides, LDL, VLDL, HDL, and TC were significantly higher in smokers as

compared to non smokers. The mean serum total cholesterol in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum triglycerides were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum VLDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum LDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum HDL were higher in non smokers when compared to smokers, which was statistically significant ($p<0.05$).

In NS Neki et al[10] serum total cholesterol was significantly higher ($p<0.05$) when compared to non smokers. The mean triglycerides levels were higher in smokers than non smokers and this difference was statistically highly significant ($p<0.01$). The mean LDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean VLDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically highly significant ($p<0.01$).

In OA Odedeji et al[9] serum total cholesterol was significantly higher ($p<0.05$) when compared to non smokers. The mean triglycerides levels difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean LDL levels were higher in smokers than non smokers and this difference was statistically significant ($p<0.05$). The mean VLDL difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically highly significant ($p<0.01$).

In Mokoto et al[11] The mean triglycerides levels difference between smokers and non smokers was statistically significant ($p<0.05$). The mean total cholesterol levels difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean VLDL difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean LDL difference between smokers and non smokers was not statistically significant ($p>0.05$). The mean HDL levels were higher in non smokers than smokers and this difference was statistically significant ($p<0.05$).

In Aneela et al[12] the mean serum total cholesterol in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum triglycerides were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum VLDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum LDL were higher in smokers when compared to non smokers, which was statistically significant ($p<0.05$). The mean serum HDL were higher in non smokers when compared to smokers, which was statistically significant ($p<0.05$).

CONCLUSION

The present study provides a detailed profile of the plasma lipid and lipoprotein level according to cigarette/beedis smoking status(smoker, ex-smoker and non-smoker) and dosage (number of cigarettes/ beedis smoked per day)

Cigarette /beedi smoking is associated with significant higher levels of serum HDL and lower levels of serum cholesterol, serum triglycerides, serum LDL levels.

Further this association is dependent on number of cigarette/beedis smoked per day. The greater risk to smokers for the development of coronary heart disease resulting from this HDL lowering effect of smoking.

REFERENCES

1. John A.Ambrose, Rajat S.Barua: The pathophysiology of cigarette smoking and cardiovascular didease. Journal of the American college of cardiology. 2004; 43:1731-1737.
2. O.A.Adedeji and M.H.Etukudo: Lipid profile of cigarette smokers in Calabar municipality. Pakistan journal of nutrition. 2006; 5 (30): 237-238.
3. Impact of tobacco on coronary risk factor profile. Dr.Janardhan V.Bhatt et al. H.L.Mun. Medical college, Ahmedabad
4. Shoshana Zevin,Sandra Saunders : J Am Coll Cardiol, 2001; 38:16331-1638.
5. Rajat S.Baura, John A.Ambrose: Heavy and light cigarette smokers have similar dysfunction of endothelial vasoregulatory activity. 2002; 39: 1758-1763.

6. Sagcan A, S.Nalbantgil: In-vitro-response of platelet aggregation induced by various agonists in chornic smoking coronary artery disease patients. Journal of clinical and basic cardiology 2003; 6, 55-57.
7. Pairat Saengdith: Effects of cigarette smoking on serum lipids among priests in Bangkok. Journal of medicine assoc Thai 2008; vol.91: 41-4
8. Brischetto CS, Connor WE: Plasma lipid and lipoprotein profiles of cigarette smokers. American journal of cardiology 1983; 52 (7): 675-80.
9. O.A.Adedeji, M.H.Etukudo: Lipid profile of smokers in calabar municipality. Pakistan journal of nutrition 2006; 5(3):237-238.
10. Kaori Teshima, Hiroyuki Imamura, Kazuhiro Uchida, Noriko Miyamoto, Youko Masuda, Daikichi Kobata et al: Cigarette smoking, blood pressure and serum lipids in Japanese middle aged men. Journal of physiological anthropology and applied human science.2001; 20(1):43-45.
11. Thomas Neunteufl, Sandra Herher, Karam Kostner, Goran Mitulovic, StephanLehr, Gholamali Khoschsoror, Rainer W.Schmid, Gerald Maurer, Thomas Stefenelli et al: Contribution of nicotine to acute endothelial dysfunction in long-term smokers. Journal of American college of cardiology.2002; 39; 251-256.
12. Reginald V.Fant, Lucy L.Owen, Jack E.Henningfield: Nicotine replacement therapy.MD Consult journal.1999, vol 26: 635-649.