Original Research Paper

Volume - 7 | Issue - 6 | June - 2017 | ISSN - 2249-555X | IF : 4.894 | IC Value : 79.96

Physiology

EFFECT OF SMOKING ON SERUM LIPID PROFILE AND BLOOD PRESSURE

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ABSTRACT Smoking is the most common method of consuming tobacco and tobacco is the most common substance smoked. Smoking is an addiction as well as a habit. A large number of risk factors which predispose to atherosclerosis and hence coronary heart disease have been identified. These include modifiable ones like hypertension, dyslipidemia, smoking, diabetes mellitus, changing life style and non modifiable ones like age and sex. Hence, the present study has been taken up to find out the alteration of serum lipid profile and changes in blood pressure if any between smoker and non-smokers and also to see any dose related changes in lipid profile and blood pressure in Kurnool city. cigarette smoking is associated with an increased risk of developing hypertension and dyslipidemia in a dose response manner, which in turn are the risk factors for coronary artery disease. A significant decrease in serum HDL-C was seen in smokers, and this decrease was more with the increase in the number of cigarettes per day. The serum LDL-C, VLDL-C were also increased in smokers in a dose response manner.

KEYWORDS : serum lipid profile, blood pressure, cigarette smokers.

Introduction

Tobacco has been smoked for centuries and possibly used for millennia. The name Nicotine is derived from Nicitina tobaccum, the tobacco plant.

In 2000, Khurana M et al in their study on lipid profile in smokers and tobacco chewers a comparative study found higher values of total cholesterol, VLDL and TG and lower levels of HDL cholesterol in both smokers and tobacco chewers.

In 2001, Hiroyuki Imamura et al in their study on "Cigarette smoking, blood pressure and serum lipids and lipoproteins in middle aged women" showed that there was a negative relationship between cigarette smoking and HDL-C and positive relationships for cigarette smoking and TC/HDL-C, TG and low density lipoprotein cholesterol and a negative association of smoking with SBP and DBP.

In 2002, NS Neki in "A clinical study on lipid profile in chronic smokers" showed a direct dose response relationship between smoking and TG, LDL-C, VLDL & total cholesterol and inverse relationship with HDL-C[56].

In 2002, Bulliyya G in a clinical study on "Blood pressure and serum lipid profile in smokers and non smokers" a comparative study showed that smokers had a significantly higher mean levels of systolic blood pressure, pulse rate, serum total cholesterol, triglycerides, LDL cholesterol, where as HDL cholesterol levels were significantly lower in smoking group.

In 2003, Azra Mahmud, John Feely in their study on "Effect of smoking on arterial stiffness and pulse pressure amplification" showed adverse hemodynamic effects in chronic young smokers.

In 2005, Jin Won Kim et al in their study on "Acute and chronic effects of cigarette smoking on arterial stiffness" showed an acute increase in arterial stiffness in chronic smokers which was surprisingly greater than that in non-smokers.

In 2006, A. Venkatesan et al in their study on "Effect of smoking on lipid profile and lipid peroxidation in normal subjects" showed association between smoking and the alteration in plasma concentration of lipid profile and lipid peroxides where in there was significantly elevated levels of total cholesterol, LDL-cholesterol, and MDA when compared with smokers[1].

In 2007, Thomas S. Bowman et al in their study "A prospective study of cigarette smoking and risk of incident hypertension in women" showed that cigarette smoking was modestly associated with an increased risk of developing hypertension.

In 2009, H Mahesar et al in their study on "Effect of smoking on blood pressure of inhabitants of Hyderabad vicinity" showed the prevalence of hypertension in smokers to be higher than in non-smokers.

Materials and Methods

Source of data:

Fifty healthy male smokers and fifty healthy male non smokers were selected.

Method of collection of data:

Consent from ethical committee was obtained. After taking informed consent of the subjects, detailed history and physical examination was done in all subjects as per the proforma.

In the present study 100 healthy male volunteers were taken. 50 smokers and 50 non smokers between the ages of 30-50 years of age (average age of 39.6 years).

Inclusion criteria:

1. The subjects were divided into four groups:
a. Non smokers: who had never smoked.
b. Smokers who smoke less than 10 cigarettes per day for at least 5 years or more.
c. Smokers who smoke 10-20 cigarettes per day for at least 5years or more.
d. Smokers who smoke more than 20 cigarettes per day for at least 5 years or more

2. The subjects were chosen in age group of 30-50 years.
3. Information on anthropometric measurements like height and weight were taken.
4. BMI was calculated. The subjects with BMI > 20-25 kg/m^2 were taken.
5. The subjects were on average Indian diet.

Estimation of serum lipid profile

Serum cholesterol estimation:

Reagents:

Pipes buffer	35 mmol/L
Na cholate	0.5 mmol/L
Phenol	28 mmol/L
Cholesterol esterase	• 0.2 U/ml
Cholesterol oxidase	• 0.1 U/ml
Peroxidase	• 0.8 U/ml

Aminoantipyrine	0.5 mmol/L
pH	7.0
Standard	5ml

Procedure:
As a manual procedure on a suitable photometer

Reaction conditions:

Wavelength	500 ± 10nm
Light path	1cm
Temperature	37°c
Sample fraction volume	1/101

The Reagent is brought to room temperature and pipette into test tubes and mixed well.

	Blank	Standard (ST)	Sample (S)
Distilled H20	10 µl	_	_
Standard	_	10 µl	_
Sample	_	_	10 µl
Reagent	1.0ml	1.0 ml	1.0 ml

Test tubes are incubated for 5 minutes in a water bath at 37ºc.

After incubation, the photometer is brought to zero and the absorbance of the standard and the sample are read. The color remains stable for at least 2 hours.

According to NECP guidelines for classification of lipid profile:

Lipid constituents	Desirable mg/dl	Borderline to high mg/dl	High mg/dl
Total cholesterol	<200	200-239	>240
LDL cholesterol	<130	130-159	>160
Triglycerides	<150	150-499	>500
HDL cholesterol	>60	40-59	<40

Data Analysis:
All the data is expressed as mean ± standard derivation.
Probability values ('p' values) are derived from analysis of variants.
'p' values less than 0.05 is considered statistically significant.
All the data was tabulated and statistical analysis was done.

RESULTS

Distribution of smokers depending on the number of cigarettes smoked per day

Comparison of mean SBP (mmHg) between smokers and non smokers

Group	MEAN	SD
Controls(non smokers)	116.28	4.517720212
Smokers	137.76	9.960247518
P value	<0.0001	

Table.1

Table. 1 shows that, the non smokers have a mean SBP of 116.28 ± 4.51, compared with that of smokers which is 137.76 ± 9.96 mm Hg.

Statistical study shows a highly significant elevation (p value <0.0001) of SBP in smokers when compared with that of non smokers.

Comparison Of Mean Diastolic Blood Pressure Between Smokers And Nonsmokers.

GROUP	MEAN	SD
Controls (Non smokers)	77.04	4.125381862
Smokers	87.04	3.859575952
P value	< 0.0001	

Table.2

Table.2shows that the mean DBP in non smokers is 77.04±4.12, when compared with that of smokers, which is 87.04±3.85.
Statistical analysis shows a highly significant (p value 0.0001) elevation of DBP in smokers.

Comparison of mean serum LDL in non smokers and smokers

GROUP	MEAN	SD
Controls(non smokers)	115.04	11.27207132
Smokers	128.58	34.01205669
P value	<0.0001	

Table.3

Table.3 shows that the mean serum LDL levels when compared between smokers and non smokers is highly significantly (p value < 0.0001), increased in smokers with a mean value of 128.58±34.01 in smokers and 115.04±11.27 in non smokers.

DISCUSSION

Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease. There is a dose response relationship between the number of cigarettes (or) beedies smoked and cardiovascular morbidity and mortality.

In the present study there is a significant increase in (P value <0.0001) SBP and DBP among smokers compared with that of non smokers. The mean SBP value in subjects smoking more than 20 cigarettes per day was 144.09 ±7.91 mm of hg as compared with that of those smoking 10-20 cigarettes per day and less than 10 cigarettes per day was 136.25±5.74 mm of hg and 127.07±6.09 mm of Hg respectively. That is the number of cigarettes smoked per day increased the higher was the blood pressure.

Also the mean DBP values in subjects smoking more than 20 cigarettes per day was 89.52± 1.99 when compared with those smoking 10-20 cigarettes/day and less than 10 cigarettes per day was 87.37± 0.50 and 85.84 ± 3.10 respectively.

It is revealed that triglycerides, LDL-cholesterol, VLDL- cholesterol and total cholesterol were significantly higher (p value, 0.001) in smokers as compared to non smokers.

Also the mean total cholesterol in those smoking < 10 cigarettes per day was 199.46±37.61 as compared to those smoking 10-20 cigarettes per day (217.62 ± 23.67) and those smoking > 20 cigarettes per day (234.19 ± 28.42).

The mean serum triglycerides were also significantly elevated in smokers when compared to non smokers. The mean serum triglycerides levels in those smoking more than 20 cigarettes per day were significantly higher (188.33 ± 34.93) as compared with those smoking 10-20 cigarettes per day (183.12 ± 48.19)and with those smoking less than 10 cigarettes per day(182.92±55.10).

The mean LDL-C and VLDL-C values in smokers were significantly higher than in non smokers. But these values were significantly higher in subjects smoking more than 20 cigarettes per day (158.19 ± 31.17, 35.52 ± 9.81) as compared to those smoking 10-20 cigarettes per day (145.81 ± 22.02, 34.25 ± 6.77) and less than 10 cigarettes per day (137.92 ± 40.19, 33.15 ± 9.84).

All the above findings are consistent with the findings of Bulliyya who found that smokers had significantly higher mean level of had systolic blood pressure, pulse rate, serum total cholesterol, triglycerides, LDL-C and LDL-C: HDL-C ratio than nonsmoking counterparts, where as HDL-C level was significantly lower in smoking group.

Also Mahesar et al in their study with 3382 volunteers showed that the prevalence of hypertension was higher (23.5%) in smokers against non smokers (16.4%) with SBP (130.1 ± 16.33) and DBP (87 ± 11.28) compared to 2449 non smokers with SBP (127.6 ± 15.19) and DBP (84.8 ± 9.80).

These findings suggest that a long term cigarette smoking alter the serum lipid profile adversely causing dyslipidemia and is also associated with development of hypertension in a dose response manner, which suggests a gradient of increased absolute risk of coronary artery disease between light and heavy smokers.

Also there is a biological inter relation between blood pressure and atherogenic blood lipid fractions and that pathophysiological factors underlying these interrelation may influence the mechanisms where by hypertension is associated with increased risk of coronary heart disease.

Thus the present study is conducted to evaluate the unfavorable effects of chronic cigarettes smoking on serum lipids and lipoprotein and on blood pressure which lead to dyslipidemia and causes elevation of blood pressure which independently and combinedly are the major risk

factors for the development of coronary heart disease.

SUMMARY AND CONCLUSION

1. A significant decrease in serum HDL-C was seen in smokers, and this decrease was more with the increase in the number of cigarettes per day.
2. The serum LDL-C, VLDL-C were also increased in smokers in a dose response manner.

The total cholesterol and serum triglycerides were also significantly elevated and this increase in the total cholesterol and serum triglycerides was proportionate to the number of cigarettes smoked per day.

Thus a direct relation between the security of smoking with an increase in LDL-C, VLDL-C, total cholesterol and triglycerides and HDL-C showed an inverse relationship.

The SBP and DBP were also elevated significantly in of smokers; it was in proportion with the severity of smoking.

The present results conclude that smoking is a risk factor causing dyslipidemia and elevation of blood pressure. So the doctors should strongly advice smokers to stop smoking and recommend the use of symptom less nicotine replacement therapy.

Also the people should be education on the possible ill effects of smoking. Ban on smoking in public and work places should be announced and strictly implemented by the Government of India, to prevent a large number of premature deaths due to smoking and smoking related diseases.

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