RESEARCH PAPER | Medical Science | Volume : 5 | Issue : 6 | June 2015 | ISSN - 2249-555X

Study of Lipid profile and Glycosylated Hemoglobin in Smokers

KEYWORDS: TG- Triglycerides,HDL-C-High density lipoprotein cholesterol, LDL-C-Low density lipoprotein cholesterol,HbA1c-Glycosylated hemoglobin

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ABSTRACT *Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease.The present study was undertaken to evaluate lipid profile and glycosylated hemoglobin in smokers and compare it with non-smokers.The study was conducted by taking 45 healthy cigarette smokers and age ,sex matched 30 healthy volunteers with no history of smoking taken as controls.The lipid profile and glycosylated hemoglobin were measured in both both groups.It was revealed that mean serum total Cholesterol,Triglycerides,LDL-C,VLDL-C, were found to be increased in smokers as compared to non-smokers. Mean serum HDL-C was found to be significantly lower in smokers as compared to non- smokers.Mean blood glycosylated hemoglobin was significantly higher in smokers as compared to non-smokers,which is a risk factor for type-2 Diabetes mellitus. So by avoiding smoking we can prevent Diabetes mellitus and cardiovascular disease risk.*

Introduction

Cigarette smoking is a serious health problem and most important avoidable cause of death in world wide. Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease and peripheral vascular disorders.[1] Cigarette smoking may be a risk factor for type-II Diabetes mellitus in men and women.[2]

Aim and Objectives

- To study the alteration in lipid profile in healthy smokers and compare the same with non-smokers.
- To study the alteration in glycosylated Hemoglobin level in smokers and non-smokers.

Materials and Methods

The study was conducted by taking 45 healthy individuals (males) who were smokers with no history of Diabetes mellitus, hypertension, coronary artery disease attended the OPD of Department of medicine for routine health check up. Age and sex matched 30 healthy volunteers with no history of smoking were taken as controls. The informed consent was taken from all of them.

After 12 hours fasting blood samples were collected from both test and control group. The lipid profile was done for both the groups. Serum lipids were estimated as follows:

- Serum Total Cholesterol- By cholesterol oxidase/peroxidase method
- Serum Triglycerides (TG) –By GPO/POD Method
- High density lipoprotein cholesterol (HDL-C)-By cholesterol oxidase/peroxidase method
- Low density lipoprotein cholesterol (LDL-C)- By Friedewald's formula[3]
- LDL cholesterol=Total cholesterol-(HDL-C+TG/5)
- Very low density lipoprotein cholesterol (VLDL-C)=TG/5

The glycosylated Hemoglobin (HbA1c) was estimated in both the study groups.

Result

Statistical analysis was done by student t test. The degree of significance was found out by p value.

Table-1: Comparison of lipid profile and HbA1c in Non-smokers and Smokers

Parameters	Non-Smokers (mean± SD)	Smokers (mean± SD)	p Value
Total cholesterol (mg%)	164.7±16.98	184.56±11.34	<0.0001
Triglycerides(mg%)	183.30±17.77	266.51±15.85	0.0001
HDL cholesterol (mg%)	46.70±5.07	42.47±4.99	0.0006
LDL cholesterol (mg%)	81.34±18.071	88.79±13.77	0.0468
VLDL cholesterol (mg%)	36.66±3.55	53.30±3.17	<0.0001
HbA1c (%)	5.62±0.48	6.81±1.06	<0.001

P< 0.05- significant

From Table 1, it was observed that serum total cholesterol, TG, LDL-C, VLDL-C were significantly increased in smokers as compared to non-smokers. Serum HDL-C was significantly decreased in smokers as compared to non-smokers. HbA1c level in blood was significantly increased in smokers as compared to non-smokers.

Discussion

The present study revealed that cigarette smoking leads to significant increase in the concentration of serum total ch olesterol(p<0.0001),triglycerides(p=0.0001),LDL-cholesterol(p=0.0468),VLDL-cholesterol (p<0.0001)and significant fall in the level of antiatherogenic HDL-cholesterol (p=0.0006). The same finding was reported from various workers.[4-10]

Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system

Volume : 5 | Issue : 6 | June 2015 | ISSN - 2249-555X

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 FFA and hepatic triglycerides along with VLDL-C in the blood stream.[5,11] (b) Fall in oestrogen level occurs due to smoking which further leads to decrease HDL-cholesterol.[12] (c) Presence of hyperinsulinaemia in smokers leads to increase cholesterol, LDL-C, VLDL-C and TG due to decrease activity of lipoprotein lipase.[13,14]

From the study it was observed that glycosylated Hemoglobin was significantly higher($p<0.001$) in smokers as compared to non-smokers. HbA1c is a marker of long term glucose homeostasis reflecting average blood glucose concentration in the past two to three months. Increase HbA1c level in smokers suggest higher transient increase in blood glucose concentration after an oral glucose challenge test and higher insulin resistance than non-smokers; suggesting potential risk of Diabetes mellitus.

Studies showed that nicotine not only has a direct toxic effect on pancreatic beta cells but also associated with increased insulin resistance leading to impaired glucose tolerance.[15] Our study correlated with studies from other workers.[16-17]

Conclusion

Our study showed a strong relationship between alteration of lipid profile and HbA1c with cigarette smoking. Increased LDL-C and decreased HDL-C are major causes of coronary artery disease. Increase HbA1c level in smokers suggesting risk of Diabetes mellitus. By avoiding smoking we can prevent Diabetes mellitus and cardiovascular disease risk.

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