Evaluation of Serum Amylase, Lipase, Lipid Profile and Insulin levels in Smokers

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Abstract

Introduction: *Cigarette smoking is known to cause damage to many organs in the body. Nicotine may cause alterations in the exocrine and endocrine function of pancreas. This study aims to assess the effect of smoking on amylase, insulin levels and lipid profile in healthy smokers.*

Materials and Methods: *27 healthy smokers and 27 non- smokers were recruited as cases and controls. Fasting blood samples were collected for estimating the glucose, lipid profile, amylase, lipase and insulin levels.*

Results: *There is a significant dyslipidemia, higher insulin levels and HOMA-IR and decrease in amylase and lipase levels in smokers compared to non- smokers.*

Conclusion: *Smoking is associated with risk of development of diabetes mellitus and cardiovascular disease.*

Key words: *Smoking, Diabetes mellitus, cardiovascular disease, HOMA-IR, Insulin resistance.*

Introduction

Smoking is one of the avoidable causes of mortality[1]. There are approximately 120 million smokers in India. According to the World Health Organization (WHO), India is home to 12% of the world's smokers. More than 10 million die each year due to tobacco in India[2].

Smoking contains greater than 2000 chemicals including nicotine, polycyclic aromatic hydrocarbons, trace metal ions, pro-oxidants and reactive oxygen species[3]. Globally, smoking alone causes about 71% of lung cancer, 42% of chronic respiratory diseases, and about 10% of cardiovascular disease[4]. Cigarette is considered as a toxic due to its various other components but the nicotine is responsible for its addiction[5].

Cigarette smoking is a major risk factor for cardiovascular disease, cancer, chronic obstructive pulmonary disease and cerebrovascular disease[5] and pancreas is one of the first organs pathologically affected by the tobacco smoking.

In addition, smoking is associated with differences in lifestyle, such as diet and degree of physical activity, which could contribute to the increased risk of cardiovascular disease (CVD).

An acute effect of cigarette smoking is to increase the activity of the sympathetic nervous system and the levels of circulating catecholamines. Since catecholamines are powerful antagonists to insulin action, smoking may be linked to insulin resistance. Smoking can acutely impair insulin action.

Of all the chemicals present in cigarette smoke, nicotine and NK are the most studied in pancreatic disease. Nicotine is the major biological active substance in cigarette smoke[9]. These metabolites are capable of penetrating pancreas tissue. Cotinine, the predominant metabolite of nicotine, and NNK were detected in 100% and 83% of pancreatic juice samples from smokers that were analyzed by gas chromatography with mass spectrometric detection using a selected ion monitoring technique (GC- SIM-MS)[8-10]. Alteration in glucose metabolism may be reflected by changes in amylase, lipase levels.

According to Hayden et al, dysfunction of insulin- acinar ductal- incretin-gut hormone is evident in young animal models and humans due to disruption in cellular communication and extracellular matrix remodeling fibrosis.

This study aims at studying the serum levels of amylase, insulin and lipid profile in smokers.

Materials and methods

This study is a cross-sectional study involving patients attending Master Health checkup department at SRMMCH&RC. All the procedures were approved by the institutional ethical committee 1871(A)/ IEC/2019.

Study population

The study was carried out on 27 smokers and 27 non-smokers (Age: 20-55 yrs.). All the participants were explained about the procedures and recruited, after obtaining informed consent. We excluded from the study all subjects with alcoholism, liver diseases, chronic renal failure, hypothyroidism and diabetes mellitus and also with other chronic illness.

Biochemical assays

Whole blood samples were collected from the antecubital vein after 10-12 hours of overnight fasting. Amylase (Kinetic colour test), fasting plasma glucose, total cholesterol, triglycerides (TGL), high-density lipoprotein (HDL-C) and low-density lipoprotein (LDL-C), measured by enzymatic procedure (on Beckman coulter AU 480 fully auto analyser). Insulin levels are estimated (Chemiluminescence immuno assay method in Vitros ECi immuno analyzer). The HOMA-IR index was calculated by using the formula: fasting insulin level (U/ml) × fasting plasma glucose (mmol/L)/ 22.5

Statistical analysis

Data was analysed using statistical package for scientific studies (SPSS) version 25. The results were represented as mean ± Standard deviation (SD). Student t- test was used to analyze the difference between levels of various parameters. Correlation between various variable was assessed using Pearson's correlation. The p- value < 0.05 was considered statistically significant.

Results

A total of 54 participants were recruited for this study, among them 27 were smokers and the controls were 27 non-smokers. Table 1 shows the characteristics and biochemical parameters of the study population.

Table-1: Biochemical parameters of smokers and non-smokers

Variables	**Non-Smokers**	**Smokers**	**P value**
Age, years	36.33 ± 12.57	37.63 ± 9.7	0.67
BMI (Kg/m^2)	23.3 ± 3.44	26.23 ± 4.12	0.01*
Fasting glucose (mg/dl)	101.8 ± 9.5	110.89± 18.10	0.01*
Total Cholesterol (mg/dl)	142.56± 20.60	188.59± 36.08	0.001**
Triglycerides (mg/dl)	90.48 ± 33.13	132.3 ± 58.7	0.001**
HDL-C (mg/dl)	45.9 ± 14.92	40.55 ± 9.22	0.13
LDL-C (mg/dl)	105.52 ± 23.32	135.41 ± 32.68	0.01*
VLDL-C (mg/dl)	17.25± 5.7	26.59 ± 11.76	0.01*
Insulin (mIU/ L)	7.06 ± 3.51	20.09 ±11.56	0.001**
HOMA- IR	1.42 ± 0.9	5.2 ± 3.8	0.001**
Amylase(U/L)	61.37 ± 113.29	34.85 ± 11.88	0.01*
Lipase(U/L)	26.71 ± 9.38	10.41 ± 2.3	0.01*

*p < 0.05 was considered as significant, **p < 0.001 was considered highly significant.

Data is expressed as mean ± SD, BMI, body mass index; TC, total cholesterol; TGL, triglycerides; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; VLDL-C, very low-density lipoprotein cholesterol; HOMA-IR, homeostatic model assessment- insulin resistance

We found a significant increase in BMI, fasting plasma glucose, insulin, HOMA-IR, total cholesterol, triglycerides, LDL-c among the smokers compared to non-smokers. There was a significant decrease in serum amylase and lipase levels in smokers.

Table-2: Pearson's correlation of serum amylase & lipase with insulin in smokers.

	Insulin	
Variables	r value	P value
Amylase	-0.44	**0.02**
Lipase	-0.31	0.1

*p < 0.05 is considered as significant

A significant negative correlation was found between serum amylase [r -0.42 (P-0.03)] but negative (non-significant) correlation between lipase [r -0.31 (P-0.1)] levels with insulin in smokers.

Discussion

In our study, we found a significant increase in total cholesterol, triglycerides, LDL, Insulin & HOMA-IR and decrease in amylase and lipase levels in the smokers compared to non-smokers.

There are conflicting reports regarding the amylase levels in smokers. Chowdhary et al. studied the functional changes of rat exocrine pancreas on exposure to nicotine and noted a decreased amylase release in isolated acini[11].

Whereas Dubick et al. found that the basal serum amylase levels were 100% higher in smokers compared to non-smokers[12]. Nourane et al observed decreased amylase and lipase levels in smokers compared to non-smokers with a significant negative correlation between serum lipase levels and pack year index [Number of cigarettes smoked/day x No. of year / 20][4]. Lipase deficiency can result in hypercholesterolemia, hypertriglyceridemia which was also seen in our study.[4]

Impairment of the relationship between the exocrine and endocrine part of pancreas may be reflected by decreased amylase levels. Studies show that binding of insulin to its receptor on acinar cells results in amylase secretion [(13-16)].

Insulin resistance is seen in smokers in our study. Hence, the decrease in amylase secretion could be the result of decreased effect of Insulin on pancreatic acinar cells.

Our study shows an atherogenic lipid profile in smokers. Nicotine has been found to stimulate the catecholamines which results in lipolysis and release of free fatty acids. This also leads to enhanced secretion of hepatic free fatty acids (FFAs) and triglycerides & VLDL in blood [(17-19)]. Hyperinsulinemia in smokers decreases the lipoprotein lipase activity & results in increased cholesterol, triglycerides & LDL-cholesterol.

Conclusion

Our study shows that alteration in the glycemic status, insulin resistance and atherogenic lipid profile are seen in smokers. Insulin resistance may result in a decrease in the serum amylase and lipase levels.

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