Effect of Cigarette Smoking on Fasting Serum Glucose in Healthy Male Population

Naznin R[1], Nazrina S[2], Parveen S[3], Ansary NB[4], Nasrin S[5], Khanom A[6]
Taskia T[7], Muktadira[8], Hussain M[9]

Abstract

Acute effects of cigarette smoking include impaired insulin action that leads to abnormal glucose metabolism. Smoking is an independent risk factor for type 2 diabetes which is one of the global health crises and insulin resistance is one of the main risk factors for cardiovascular disease. This cross-sectional study was done to examine whether fasting serum glucose differs between cigarette smokers compared to non-smokers and to investigate the association of cigarette smoking with the development of impaired fasting glucose and type 2 diabetes. This study was done in the Physiology department of Mymensingh Medical College, an outpatient department of Mymensingh Medical College Hospital, for over a period of one year from July 2014 to June 2015. Fasting serum blood glucose was done by enzymetric colorometric GOD-PAP method in a total of 150 subjects, in which non-smokers were 50 and numbers of smokers were 100 with the duration of smoking 5 - 10 years and >10 years. A questionnaire including data was completed in all cases. The data were checked, coded, and entered into an SPSS 11.5. Statistical significance of difference among the groups was calculated by Students unpaired t' test. P-value <0.05 was considered as a level of significance. Fasting Serum Glucose level was increased gradually with the duration of smoking in smokers than the non-smokers. Results were statistically highly significant. Fasting Serum Glucose gradually increase with the duration of smoking and results were within the physiological limit in two study groups (Group-II A with 5-10 years duration of smoking and Group- II B with more than 10 years duration of smoking) but not statistically significant. This study showed cigarette smoking has deleterious effects on insulin metabolism causing an increase in serum glucose with duration of smoking period.

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Introduction

Cigarette smoking is one of the major sources of toxic chemical exposure to humans and is the greatest cause of preventable illness and premature death.[1] Deaths from smoking are projected to increase to more than 10 million a year by 2030.[2] Unfortunately, 73 % of these smokers live in developing country.[3] Having 21.9 million adult smokers, Bangladesh ranks among the top ten heaviest smoking countries in the world.[4] Current cigarette smokers were defined adults aged > 18 years who reported having smoked >100 cigarettes during their lifetime and who now smoke every day or some days.[5] Cigarette smoke contains about 4000 compounds belonging to a variety of chemical classes known to be toxic, including polycyclic aromatic hydrocarbons (eg: benzopyrene, acenapthelene, phenanthrene, pyrene and chrysene),

1. *Dr. Rubiat Naznin, Assistant Professor, Department of Physiology. Community Based Medical College, Mymensingh*
2. *Lt Col Sayeda Nazrina, Associate Professor, Department of Pharmacology, AFMC, Dhaka*
3. *Professor Dr. Shahanaz Parveen, Professor, Head of the Department of Physiology. Community Based Medical College, Mymensingh*
4. *Professor Dr. Nahid Bintay Ansary, Professor, Department of Physiology. Community Based Medical College, Mymensingh*
5. *Dr. Shamima Nasrin, Assistant professor, Department of Physiology, TMCC Medical College, Bogura*
6. *Dr. Aklima Khanom, Assistant professor, Department of Physiology. Gazipur City Medical College*
7. *Dr. Tanzina Taskia, Assistant professor, Department of Physiology. President Abdul Hamid Medical College, Kishorgonj.*
8. *Dr. Muktadira, Department of Radiology and imaging, CBMCB.*
9. *Dr. Maria Hussain, Assistant professor, Department of Physiology. CBMCB.*

Address of correspondence:
Email: rubiatnaznin@gmail.com
Mobile: 01718355113

nitrosamines, heavy metals (eg: cadmium, lead and cobalt), alkaloids (nicotine), aromatic amines and so forth.[6] The effect of nicotine on cortisol secretion becomes demonstrable as early as six months of mild persistent exposure and perpetuates with a duration of exposure even a low dose.[7]

During the past decade, accumulating evidence of cigarette smoking constituting a strong and independent risk factor of the development of type 2 diabetes has appeared.[8] In healthy people, glucose levels are maintained within normal range through proper insulin secretion by bcell.[9] Smoking reduces insulin-mediated glucose uptake by 10-40% in men who smoked compared with non-smoking men.[10] Nicotine absorbed during smoking increase the discharge of catecholamines from the adrenal medulla and from extra-adrenal chromaffin tissue which causes hyper-insulinemia and it is more likely that adrenergic activity induces by smoking is responsible for insulin resistance.[11]

This study aimed to clarify the association of cigarette smoking with the development of impaired fasting glucose and type 2 diabetes and differ fasting serum glucose (FSG) between cigarette smokers compared with non-smokers.

Materials and Methods

The present study was a cross-sectional comparative study with permission from an institutional ethical committee conducted in the Department of Physiology, Mymensingh Medical College, from July 2014 to June 2015. Subjects were recruited from the Department of Medicine, Mymensingh Medical College Hospital. This entire study was conducted in healthy male smokers and non-smokers in the age group of 25-55 years with matching BMI. Subjects with any sort of medication, family history of DM or with an acute inflammatory response within previous 2 weeks and female person were excluded from this study. Body mass index, systolic, and diastolic blood pressure were recorded. A total of 150 voluntary subjects were selected conveniently based on history and clinical examination. Among them, 50 participants (Group I) had no experience with cigarette smoking or other form of tobacco consumption (betel leaf with Jorda, snuff tobacco). The intensity of cigarette smoking was determined in 100 smokers in which 50 were cigarette smokers for 5-10 years (Group IIA), and 50 were smoking cigarettes more than 10 years (Group II B) who were smoking > 20 cigarettes per day and free from other form of tobacco use except cigarette smoking. All the subjects were selected based on history and clinical examination, written informed consent was obtained. Subjects body weight was measured in kilogram and height in meter. Fasting serum glucose (FSG) level was done by enzymetric colorometric GOD-PAP method. The participants were asked to fast for at least 8 hours and to avoid smoking and heavy physical activity for more than 2 hours before the examination. 5 ml of venous blood were drawn from the antecubital vein in the morning between 8.00 am to 9.00 am. It was taken as the data for the subject. Statistical analyses were done by using Statistical Package for the Social Sciences (SPSS) for windows version 11.5. Data were expressed as mean ± Standard error (±SE) and statistical difference among the group was calculated by unpaired "t" test. P-value < 0.05 was considered as level of significance.

Results

Table-I and Figure- I shows the results of FSG. According to the result, FSG increased gradually with the duration of smoking in study groups, compared with the control group. All the results were statistically highly significant. FSG gradually increased in two study groups, group IIA and IIB. But results were not statistically significant in comparison with 5-10 years and above 10 years smokers.

Table I: Statistical analysis (unpaired t-test) of mean values of FSG for control and study group.

Biochemical variable	Group I n=50 mean SE	Group IIA n-50 mean SE	Group IIB n=50 mean SE	P-Value
	4.43 ± 0.04	5.25 ± 0.03	-	<0.001**
FSG (mmol/L)	4.43 ± 0.04	-	5.32 ±0.036	<0.001**
	-	5.25 ± 0.03	5.32 ±0.036	0.142

All values are the mean ± SE

NS = Not significant

p<0.05 = Statistically significant.

p>0.05 = Not statistically

= Highly significant difference(P<0.001)

P-Value = Level of significance

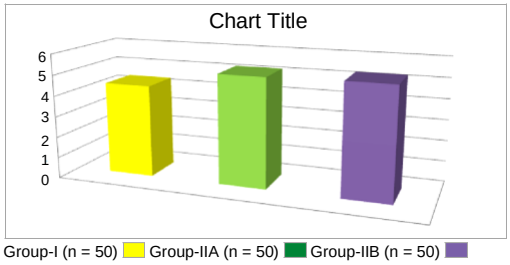

Figure I: Bar Diagram Shows Comparison of Mean Value of Fasting Serum Glucose between Control & Study Groups

Discussion

The present work was carried out to study the effects of cigarette smoking on FSG in male cigarette smokers. The study populations were divided into control group (Group I) and study group (Group II) depending on smoking habits that means non-smoker and the smoker respectively. Again smokers (Group II) were divided into five to ten years smoking duration (Group II and more than ten years smoking duration (Group II B). In this study, two groups of subjects (smokers and non-smokers) were matched for age and BMI. The mean (±SE) FSG of the control group & study group (duration of smoking 5-10 years) were 4.43±0.04mmol/l & 5.25±0.03 mmol/l respectively. In the study group, mean (±SE) FSG was increased. The result was statistically highly significant (p >0.001).The mean (±SE) FSG of the control group & study group (duration of smoking >10 years) were 4.43±0.04 mmol/l & 5.32±0.036 mmol/l respectively. In the study group, mean (±SE) FSG was increased. Result was highly Significant (p > 0.001). The mean (±SE) FSG was 5.25±0.03 mmol/l & 5.32±0.036 mmol/l in the study group of 5-10 years & > 10 years smokers respectively. The mean (±SE) FSG levels were increased with the duration of smoking. But the result was not statistically significant (p >0.05). According to the above discussion, FSG increased gradually with the duration of smoking and results were within a physiological limit in the study group and not statistically significant. Sairenchi et al. 2004[12] in their study found an increased fasting serum glucose level in smokers. Nakanishi et al. 2000[13] also reported in their study that cigarette smoking elevated fasting plasma glucose levels. It is consistent with that of the result Narawiw at. 2003.[14] Kume A et al. 2009[15] and Parchwani et al. 2013[16] also conclude that Fasting Blood Sugar levels increase in smokers compared with non-smokers.

Smoking induced oxidative stress might have some effect on blood glucose, either directly alter blood glucose homeostasis and cause insulin resistance and also it is suspected high concentration of circulating epinephrine and norepinephrine due to smoking may contribute to hyperglycemia by increasing the rate of hepatic gluconeogenesis and glycogenolysis.[17] The presence of neuronal nicotinic acetylcholine receptors (nAch Rs) was discovered in β cells of pancreatic islets, there are subunits of nAch Rs, such as α2, α3, α4, α7 and β2. Both in the case of acute (60 min) and chronic (48hr) exposure to nicotine, decrease β cell of insulin secretion was observed. It was further found that exposure to nicotine concentration above 1 mol/L inhibits insulin secretion in isolated human islets cell.[18] These results were inconsistent with the result of Onyesom et al. 2012[19] reported that- blood sugar level for non-smokers was significantly higher than that of smokers observed and this could be due to poor feeding habits of most smokes in

this locality

Conclusion

According to this study, along with the other numerous threats to public health, cigarette smoking has deleterious effects is associated with the increased risk of incidence of diabetes. Smoking aggravates glucose homeostasis, increases diabetic incidence and also increases chronic diabetes complications.

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