

Comparison of Fasting Plasma Glucose among Smoking and Non Smoking Medical Student Population

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Abstract: *Diabetes mellitus is an established widespread morbidity factor across the globe. Smoking is a major risk factor for developing stress. Smoking is shown to elevate cortisol level. Smoking also contributes to development of insulin resistance and hence type 2 diabetes mellitus. The purpose of this study was to find the association between cigarette smoking and diabetes mellitus. This study included 30 students (15 smokers, 15 non smokers) from 1st year and similar group was chosen from 3rd year medical students. Serum cortisol level was estimated by Electrochemiluminescence immunoassay and Plasma glucose was estimated by glucose oxidase method on a HITACHI 917 analyzer. The data was statistically analyzed using students t test and chi-square test. P value <0.05 was considered as statistical significance. This study found a positive association between smoking and diabetes. It showed that with advancing age and longer duration of exposure to nicotine and prolonged activation of HPA axis, the chances of developing frank hyperglycemia increasing in due course.*

Keywords: fasting plasma glucose, cigarette smoking, serum cortisol, nicotine, hyperglycemia

1. Introduction

Diabetes mellitus (DM) is a major morbidity factor across the globe. Screening for diabetes mellitus thus is essential as early measures on appropriate prevention can be instituted. It is important to be aware of the pausable risk factors associated with screen detected diabetes to interpret its prevalence. Heavy alcohol consumption, obesity, sedentary lifestyle, cigarette smoking are major risk factor associated with diabetes mellitus. [1,2,3,4]. Cigarette smoking may increase the incidence of non insulin dependent DM [5,6] and is epidemiologically related to factors such as serum lipid and insulin resistance.[7,8,9]. Cigarette smoking induces stress response in the body leading to elevated cortisol levels [10]. This study examines the impact of cigarette smoking on glucose level among healthy young population. Also it aims to find a pausable association between cortisol rise and hyperglycemia.

2. Materials and Methods

This study was carried out among students studying in 1st year and 3rd year in Kasturba Medical College, Manipal University, Mangalore. 15 students from 1st year with a history of smoking for 6-12 months and 15 students from 3rd year with smoking duration of at least 3-4 yrs were selected. 15 students in each group who were ages matched and non smokers served as controls. All the subjects were from the hostel. Students on any sort of medication, family history of DM or with acute inflammatory response within previous 2 weeks were excluded from the study. Body mass index, waist circumference, systolic and diastolic blood pressure were recorded.

5 ml of venous blood was collected from 1st year students, early morning between 7- 7:30 am. Samples were

immediately analyzed in the biochemistry laboratory using HITACHI 917 analyzer for serum cortisol. Serum cortisol was assessed using Electrochemiluminescence immunoassay. 1st incubation: 20 microlitre of sample is incubated with a cortisol specific biotinylated antibody and a ruthenium complex labeled cortisol derivative. Depending on the concentration of the analyte in the sample and the formation of the respective immune complex, the labeled antibody site is occupied in part with the sample analyte and in part with ruthenylated haptens. 2nd incubation: after addition of streptavidin coated micro particles, the complex becomes bound to the solid phase via interaction of biotin and streptavidin. The reaction mixture is aspirated into the measuring cell where the micro particles are magnetically captured onto the surface of electrode. Unbound substances are then removed with procell. Application of the voltage to the electrodes then induces chemiluminescent emissions which is measured by a photomultiplier. Results are determined via a calibration curve which is instrument specifically generated by 2 point calibration and a master curve provided via reagent barcode.

Fasting plasma glucose was estimated using glucose oxidase method which includes enzymatic oxidation of glucose the calorimetric indicator is quinoneimine which is generated from 4-aminoantipyrene and phenol by hydrogen peroxide under the catalytic action of peroxidase.

The results obtained were statistically analyzed using students T test to compare the parametric data among the groups. Chi square test was used to for association of stressors in the stress scale. P value less than 0.05 was considered significant.

3. Results

Table 1: Comparison between non smokers and smokers among the 1st year students (short term exposure)

Parameter	Non Smoker(15)	Smoker(15)	P Value
Fasting glucose	86.66±5.72	83.67±3.98	0.109
Serum cortisol	13.18±3.81	15.53±5.30	0.174

P value<0.05 considered significant

FPG is found to elevate in non smokers when compared to students who had a short duration of exposure to cigarette smoke. Serum cortisol level were elevated among smokers than non smokers.

Table 2: Comparison between non smokers and smokers among the 3rd year students (long term exposure)

Parameter	Non Smoker	Smoker	P Value
Fasting glucose	86.94±6.50	89.43±11.47	0.471
Serum cortisol	13.09±4.90	17.79±3.83	0.007*

P value<0.05 considered significant

FPG was shown to be higher in students with long term exposure to smoking, although not statistically significant. There was a significant difference in SC levels between non smokers and smokers with long term exposure.

Table 3: Comparison between non smokers among 1st year and the 3rd yr MBBS students.

Parameter	1 st Year	3 rd Year	P Value
Fasting glucose	86.66±5.72	86.94±6.50	0.901
Serum cortisol	13.18±3.81	13.09±4.90	0.957

P value<0.05 considered significant

Upon comparison of the non smokers among the two groups, there seemed to be no difference in FPG and SC levels.

Table 4: Comparison between smokers among 1st year and the 3rd year MBBS student

Parameter	1 st Year	3 rd Year	P Value
Fasting glucose	83.67±3.98	89.43±11.47	0.071
Serum cortisol	15.53±5.30	17.79±3.83	0.192

P value<0.05 considered significant

Among the smokers, FPG as well as SC was higher among the students with long term exposure to cigarette smoking.

4. Discussion

Several prospective studies have noted a positive relation between cigarette smoking and diabetes mellitus. [6,11] Previous studies reveal that cigarette smoking is an independent and modifiable risk factor for type 2 diabetes mellitus.[12] Cigarette smoking was associated with significant increase in risk of diabetes even after adjusting age, BMI and other potential confounders.[13] This study is in accordance with the previous studies.

In this study we found that longer exposure of cigarette smoking increases the risk of diabetes mellitus. Except for age, which is higher in senior students, no difference was found in BMI, waist circumference, systolic and diastolic blood pressure and fasting plasma levels when inter and

intra group comparisons were made. Thus clinically evident effects of hypercortisolism as obesity, hypertension and hyperglycemia were not demonstrated in the students with 4 to 6 month exposure to nicotine.

Various authors have hypothesized changes in glucose metabolism that develop diabetes. [14,15] Several studies show that cigarette smoking decreases insulin sensitivity[15,16] and this may be due to nicotine which stimulates insulin antagonizing hormone like cortisol, catecholamines and growth hormone which impair the action of insulin.[15]

Pearsons correlation analysis of serum cortisol showed a significant correlation with fasting plasma glucose among smokers($r=-0.405$, $p=0.026$). This is in keeping with the effects of cortisol on plasma glucose levels where cortisol is known to decrease peripheral insulin sensitivity and reduce glucose utilization.[17] Plasma adiponectin concentrations has been reported to be decreased in the smokers with an increase in insulin resistance by Abbasi et al.[18] Although the plasma glucose levels were within the reference range and no significance was found in levels between smokers and non smokers, the significant correlation of cortisol with fasting glucose levels points to the possibility of these students developing hyperglycemia and diabetes mellitus with prolonged exposure and higher dose of nicotine. Serum cortisol remained to be elevated in students who had long term as well as short term exposure to cigarette smoking.

Some reporters have suggested that smoking increases insulin resistance by altering the distribution of body fat or by exerting a direct toxic effect on pancreatic tissue.[6,11] Physical inactivity and obesity may lead to hyperglycemia by impairing the transport of glucose into fat and skeletal muscle cells in case of smoking a chemical component of cigarette may directly alter intracellular glucose transport or may indirectly alter it through changes in serum chemistry or diminished vascular blood flow.[17] As cigarette contain 3500 different compounds in particulate phase and 500 gaseous compounds in volatile phase[18], precisely elucidating such mechanisms may be a formidable task indeed. A study that used an open muscle biopsy procedure found that insulin stimulated glucose transport in skeletal muscle of habitual cigarette smoker was relatively impaired in a comparison with non smokers. [19] Apparently this was more likely due to increased free fatty acids and triglycerides. However the exact pathogenesis of smoking and its association with developing diabetes mellitus is not yet well understood.

5. Conclusion

The effect of nicotine on cortisol secretion becomes demonstrable as early as 6 months of mild persistent exposure and perpetuates with duration of exposure even with low dose. Hyperglycemia was not found in smokers but significant association of cortisol with fasting plasma glucose was found, indicating the ongoing subclinical pathology. With advancing age and longer duration of exposure to nicotine and prolonged activation of HPA

axis, these subjects in due course may develop frank hyperglycemia.

6. Future Scope

Medical student based studies undergo rigorous ethical committee scrutinies by the research & ethical committee of the universities and in this case we were fortunate enough to get permission to conduct this study.

1. This study can serve as a basis for conducting more student related studies in order to evaluate the impact of the Indian medical education system on the students.
2. Extrapolation of evaluation of this nature to final year and graduated interns can give us a meaningful insight into the doctor's profession.
3. This can serve as an indicator for student friendly curriculum changes.
4. This also provides important health information to students and will help them make healthier choices.

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