Research Article

Study of Glycated Hemoglobin in Chronic Cigarette Smokers

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ABSTRACT

Cigarette smoking is a serious health problem and most avoidable cause of death in worldwide. The aim of our study is to know the association between cigarette smoking and glycated hemoglobin levels (GHbA1c). This cross sectional study includes 30 people who are smokers but non alcoholics aged between 20-45 years and without any history of diabetes and any other acute or chronic illness as subjects and similar age group men who are not smokers were included as controls. The results of our study shows that there is significant association between smoking and glycated Hb levels (P<0.001). The HbA1c levels in smokers was (5.93±0.485) and in non smokers (5.430±0.234) indicating a statistical association. In conclusion the study shows people who smokes are at risk of developing diabetes mellitus. Cessation of smoking can prevent not only from developing diabetes but also from other serious health hazards.

Keywords: Cigarette smoking, Diabetes mellitus, glycated hemoglobin.

INTRODUCTION

Cigarette smoking is a serious health problem and most important avoidable causes of death in worldwide. Non-insulin dependent diabetes mellitus is partially of genetic etiology but is also strongly influenced by environmental and life style factors (1). Cigarette smoking is known to cause transient elevation in blood glucose concentration and may also influence insulin sensitivity (2,3). Several studies have found that current smokers have higher glycated HB concentration compared to non-smokers (4). Independent of body size, people who smoke have a higher transient increase in blood glucose concentration after an oral glucose challenge test and higher insulin resistance than non-smokers, suggesting a potential risk of diabetes (5-6).

Several data suggest that smoking may be a risk factor for diabetes in both men and women (7-8). A review in ‘Diabetes Care’ concludes that smoking increases the risk of developing type 2 diabetes and increases the risk of cardiovascular disease, neuropathy and nephropathy in type 2 diabetes.

Glycated haemoglobin (HbA1c) is a marker of long term glucose homeostasis reflecting average blood glucose concentration in the past two to three months. Glycated hemoglobin was shown to predict the progression from impaired glucose tolerance to diabetes in pima Indians (Little et al 1994) (9).

Investigating the association between smoking and blood glucose may clarify the role of smoking in the development of diabetes and its complications (10). Smoking was positively associated with the risk of diabetes in the nurses healthy study (11).

Sandberg et al reported a transient increase of blood glucose levels from 4.21 milli mole / litre (75.8 mg/dl) to 4.93 millimole/litre (88.8 mg/dl) within 30 mins of smoking a cigarette (2).

Similar results were reported by Bornemissa and Suciu (12) by Murchison and Fyfe (13) and by Haggard and Green berg (14). Recent reports have suggested that cigarette smoking may be a risk factor for type-II diabetes in men and women (Rimmetal) (9).

A 2001 study led by taura J. Scott of Harvad school of public health discovered that smokers are at a significantly greater risk of hyperglycemia than non-smokers.

The present study aimed to evaluate predictors of glycated hemoglobin as a measure of hyperglycemia and glycation in the general population.

METHODS

The study was done at SRM Hospital. Smokers who were apparently healthy and non diabetic, aged between 20 to 50 years, without any history of chornic illness and who had come for routine screening at master health checkup unit were included as subjects. No attempt was made to quantitate the level of smoking. We did not include the smokers who has the habit of consuming alcohol.

The control group was selected of the same age group as subjects who were not smokers and with no history of diabetes. Persons with known history of hypertension, alcoholism and any other acute or chronic illness were excluded from the study. Informed consent was obtained from all the participants and the study was approved by ethical committee.

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Blood samples were collected after overnight fasting and was analysed for glucose and glycated Hb. Glucose was measured using Glucose Oxidase method. Glycated Hb was measured using latex agglutination inhibition assay, using Beckman coulter AU400.

**RESULTS**

Results of our study shows that there is an increase in glycated hemoglobin levels in smokers ranging from 4.6-6.8% with an average of 5.9 and SD of 0.485. The control group who were non-smokers glycated hemoglobin levels ranges from 4.9-5.9% with an average of 5.41 and SD of 0.25. p value is less than 0.0001 indicating the study is statistically significant and that there is strong association between smoking and glycated hemoglobin.

**DISCUSSION**

The study results indicated a significant difference in glycated hemoglobin levels between smokers and non-smokers (fig.1) suggesting that these smokers have average plasma glucose levels that are high enough to place them at risk for the same complications as those between smoking and glycated hemoglobin.

**STATISTICAL ANALYSIS**

Data analysis was done using student t-test. Probability (p<0.05) were defined as significant.

**RESULTS**

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with the well controlled diabetes including atherosclerotic heart disease. The highest glycated Hb levels were observed in persons who smoke more cigarettes for more years compared to smokers with lesser duration of years (fig.2). The link between cigarette smoking and abnormalities of glucose homeostasis is biologically plausible, as several studies have suggested that smoking may directly impair insulin sensitivity, one of the key determinants of glucose tolerance.

Increase in plasma catecholamine have been known to cause increased hepatic glycolysis and gluconeogenesis and decreased pancreatic insulin secretion in humans, leading to increased plasma glucose. The association we have found between smoking and HbA1C is consistent with other cross sectional studies. Studies have shown that nicotine not only has a direct toxic effect on pancreatic beta cells but also is associated with increased insulin resistance leading to impaired glucose tolerance. Further more, the antiestrogenic effect of nicotine could contribute to an increase in visceral adipose tissue accumulation and via this mechanism, insulin resistance. Finally nicotine increases cortisols level and inflammation and has influence on adiponecin a peptide that regulates food intake and body weight, all of which could contribute to higher HbA1C.

In conclusion, our study suggests that smoking is associated with the increase in HbA1C in a representative sample with normal glucose metabolism as well as in people with impaired fasting glucose. Our study has several limitations the confounding factors like obesity, physical activity, dietary factors has not been investigated. Further study is needed to determine the clinical significance of this finding. These results support the hypothesis that the smoking, leads to an increase in blood glucose levels which may lead to diabetes mellitus, if smoking is not controlled.

REFERENCES

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