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Research Article

Comparative study of adenosine deaminase activity, insulin resistance and lipoprotein(a) among smokers and healthy non-smokers

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ABSTRACT

Background: Adenosine deaminase also known as adenosine aminohydrolase involved in purine metabolism. Its primary function is development and maintenance of immune system. The main objective of the study was to estimate adenosine deaminase (ADA) enzyme and find its correlation with lipoprotein(a) and insulin resistance among smokers and healthy non-smokers.
Methods: Fifty smokers and fifty healthy non-smokers were selected based on WHO definition. ADA, lipid profile and glucose was estimated on a fully automated analyser by IFCC approved methods and lipoprotein(a) was done by latex enhanced immune-turbidimetric assay method respectively.
Results: After appropriate screening ADA activity and insulin was significantly elevated among smokers when compared with healthy non-smokers. A positive correlation was found between pack size of cigarette and ADA activity and also with Lp(a) respectively. In addition, there was no correlation between serum lipid profile and ADA activity.
Conclusions: Adenosine deaminase activity was increased in patients in response to nicotine which is the key component of cigarette smoke. These findings indicate that nicotine and carbon monoxide can alter lipoprotein synthesis and also modify LDL to oxidized form which can lead to ischemic heart disease.

Keywords: Adenosine deaminase, Smokers, Lipoprotein(a), Insulin resistance

INTRODUCTION

Adenosine deaminase also known as adenosine aminohydrolase involved in purine metabolism. Its primary function is development and maintenance of immune system.[1] It has two isoenzymes ADA1 and ADA2 which is encoded by different genes.[2] In humans ADA1 is called as single-chain Zn binding protein and it is found in lymphocytes and macrophages.

Physiological role of ADA2 was not clearly known and it might be produced by monocytes in negligible quantities.[3,4] The activity of ADA2 was drastically increased in patient's plasma of liver diseases like chronic hepatitis and cirrhosis and also in diseases like AIDS, Tuberculosis, Diabetes mellitus, adult T-cell leukemia and acute lymphoblastic leukemia.[5-8]

Cigarette smoke has various toxic substances and it promotes oxidative damage and also initiates platelet aggregation.[9]

Number of cigarette smoked per day is directly related to cardiovascular morbidity and mortality. Nicotine a major component of cigarette smoke leads to the production of free radicals and increases the oxidative stress. It stimulates lipolysis thus increasing the flux of free fatty acids to liver and re-esterification of free fatty acid in the liver leads to the enhanced VLDL secretion, thus explaining the atherogenic effect of smoking.[10]

Cigarette smoking is associated with insulin resistance and also has equal effect on the progression of complications of Diabetes mellitus. At present only few studies are reported regarding ADA activity among smokers.[11] So our study was undertaken to explore any difference in ADA activity among smokers when compared to non-smoking healthy individuals and also to establish any correlation between ADA levels and dyslipidaemia among smokers which can help to substantiate the role of ADA in modulating some of the smoking induced health hazards.

METHODS

This study was conducted at Mahatma Gandhi Medical College and Research Institute a tertiary health care institution at Puducherry after obtaining clearance from institute ethics committee. 50 non-smoking healthy control and 50 smokers based on WHO definition i.e. who had smoked more than 100 cigarettes in their lifetime were included as cases in the study. Smokers with Diabetes, hypertension, coronary heart disease, tuberculosis, rheumatoid arthritis, chronic liver and kidney disorders were excluded from the study.

ADA, lipid profile and glucose was estimated on a fully automated autoanalyzer by using IFCC approved methods. Fasting insulin was estimated by chemiluminescence assay and Lp(a) by latex enhanced immunoturbidimetric assay. Data were expressed as mean±SD. Unpaired t test was used for comparing the means between the two groups. Pearson's correlation was used to find the association between pack size and other biochemical parameters.

RESULTS

After appropriate screening totally 50 healthy non-smokers and 50 smokers were included in this study. The mean and standard deviation of biochemical parameters of the control and cases are shown in table 1.

Table 1: The mean and standard deviation of biochemical parameters of the control and cases.

Parameter	Healthy non-smokers	Smokers	'p' value
ADA	25.55±4.40	39.18±12.97	0.001*†
Insulin	12.43±5.66	28.03±12.64	0.001*†
Glucose	90.64±10.52	94.37±11.33	0.026*
Total cholesterol	148.64±28.79	202.69±40.12	0.001*†
Triacylglycerols(TAG)	152.59±2.37	167.68±52.78	0.026*
HDL	42.80±9.93	36.81±9.27	0.001*†
LDL	87.10±25.31	128.79±32.16	0.001*†
VLDL	30.59±8.47	37.72±10.55	0.021*
Lp(a)	17.95±9.13	35.26±20.85	0.001*†

Data expressed as mean ± SD. *p<0.05, *†p<0.005; ADA Adenosine deaminase activity; TAGs, Triacylglycerols; HDL High Density Lipoprotein; LDL Low Density Lipoprotein; Lp(a) Lipoprotein(a)

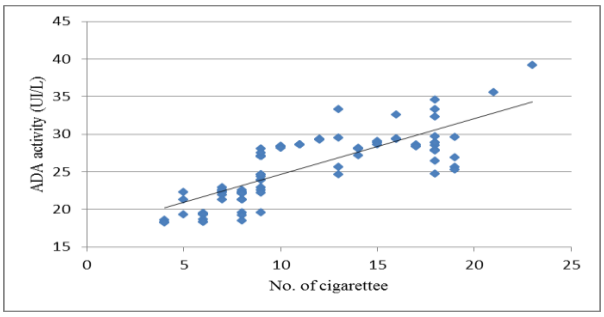

Figure 1: Pearson's correlation between pack sizes versus ADA activity.

In our study we found that ADA activity and insulin was significantly elevated among smokers. Total cholesterol, LDL and Lp(a) also showed significant difference between smokers and healthy non-smokers with the p value of 0.001.

We found that there was a strong correlation between pack size of cigarette and ADA activity with the r value of 0.83 and weak correlation between pack size and Lp(a) with the r value of 0.75 are shown in Figure 1 and 2.

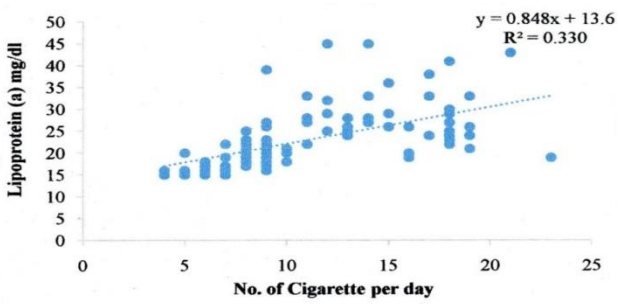

Figure 2: Correlation analysis between pack size and Lp(a).

DISCUSSION

Cigarette smoking is considered as leading cause of mortality worldwide and both active and passive smoking contributing to it. It has a direct effect on diseases like ischemic heart disease and lung cancer and also involved in the pathogenesis of diseases like rheumatoid arthritis, diabetes mellitus and inflammatory bowel disease and immune function.[12-14]

Studies demonstrated change in ADA activities in pleural fluid of patients with COPD which are more commonly associated with smoking.[15]

Our study also shows that an increased ADA activity among smokers when compared to healthy non-smokers which can explain a role from ADA in mediating some of the chronic health hazards of smoking like COPD and TB which are associated with an increased levels of ADA and also reported positive correlation between pack size and ADA activity.

Nicotine stimulates the release of catecholamine's from peripheral nerve endings which decreases the synthesis of glucose transporter.[15]

Decrease in adenosine level due to increase ADA activity leads to down regulation of GLUT 4 receptors. Which could be attributed for insulin resistance and increased HOMA IR in smokers.[16] Though certain studies have reported significant correlation between serum ADA levels and HbA1c among T2DM patient.[17] Our study does not show any such correlation among smokers.

Components of cigarette smoke like nicotine and carbon monoxide can alter lipoprotein synthesis and also modify LDL to oxidized form which can lead to ischemic heart disease. Reports available for low levels of HDL-C among smokers.[18]

A limitation of the study is that due to limited sample size in this study correlation between lipid profile and ADA activity is not significant.

CONCLUSION

Adenosine deaminase activity was increased in patients in response to nicotine which is the key component of cigarette smoke. These findings indicate that nicotine and carbon monoxide can alter lipoprotein synthesis and also modify LDL to oxidized form which can lead to ischemic heart disease.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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