ORIGINAL RESEARCH

A Study to Compare the Biochemical Markers Among Smokers and Non Smokers

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ABSTRACT

Introduction: Out of biochemical antioxidant glutathione appears to be pivotal glutathione is part of the antioxidant enzyme glutathione peroxides and the major liver antioxidant. It is basic tenet of natural medicine that health can not exist if liver is toxic. Hence low levels of glutathione seen in patients with AIDS, Cancer, Parkinson's disease, COPD in free radical can have asthma, emphysema, chronic pulmonary disease in lung. Objective of the study was to compare the biochemical markers among smokers and non smokers.

Material and Methods: A hospital based cross sectional study was carried out among smokers and non smokers. Institutional Ethics committee permission was obtained prior the study. Informed consent was obtained from each and every study and control group patients. The study was conducted over a period of one year. The study included 56 patients in study group out of which emphysema 9 patients, COPD 12 patients, bronchiectasis 14 patients, and bronchial asthma 21 patients. Control group included 25 patients with no respiratory complaints and normal PFT. All patients who satisfied inclusion criteria are included in the study.

Results: There were no smokers with bronchial asthma whereas there were 21 non smokers with bronchial asthma. For alpha 2 MG the value was more among non smokers. There is no significant difference in the mean values of antioxidant levels among smokers and non smokers. Non smokers have better PFT values compared to smokers.

Conclusion: Non smokers had better pulmonary function test values than smokers.

Key words: Biochemical markers, smokers, non smokers

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INTRODUCTION

Air pollutants significantly increase the amount of free radicals present within the linings of the lung and these free radicals have been related to chronic obstructive pulmonary disease (COPD). So oxidative stress is more in acute phase of COPD.

Free radicals are also released in the body from the detoxification of drugs, artirficial food coloring and flavoring agents, smog, preservation and processed foods, alcohol, cigarette smoke, chlorinated drinking water, pesticides, radiation, cleaning fluids, heavy metals such as cadmium and lead and assorted chemicals such as solvent traces found in processed foods and aromatic hydrocarbons such as benzene and naphthalene.

Balance of oxidant and anti-oxidant is very important in view of maintaing healthy lung. Vitamin C, vitamin E and reduced glutathione are important antioxidants. Vitamin C is water soluble present in aqueous fluid within the lung tissue. It acts as antioxidant along with vitamin E.

Out of biochemical antioxidant glutathione appears to be pivotal glutathione is part of the antioxidant enzyme glutathione peroxides and the major liver antioxidant. It is basic tenet of natural medicine that health can not exist if liver is toxic. Hence low levels of glutathione seen in patients with AIDS, Cancer, Parkinson's disease, COPD in free radical can have asthma, emphysema, chronic pulmonary disease in lung.[1]

Therefore it is decided to study the biochemical markers among smokers and non smokers.

MATERIAL AND METHODS

A hospital based cross sectional study was carried out among smokers and non smokers. Institutional Ethics committee permission was obtained prior the study. Informed consent was obtained from each and every study and control group patients. The study was conducted over a period of one year. The study included 56 patients in study group out of which emphysema 9 patients, COPD 12 patients, bronchiectasis 14 patients, and bronchial asthma 21 patients.

Control group included 25 patients with no respiratory complaints and normal PFT. All patients who satisfied inclusion

criteria are included in the study.
Data was recorded by taking history from each and every patient. All study subjects have undergone the complete clinical examination.
Investigations like serum alpha 1 AT alpha 2 MG were done by byimmunoturbidimetric method. Levels of vitamin C and vitamin E were measured by titration. Activity of alpha 1 AT was assessed by using enzyme trypsin, enzyme elastase. [2]

RESULTS

The study included 56 patients in study group out of which emphysema 9 patients, COPD 12 patients, bronchiectasis 14 patients, and bronchial asthma 21 patients.
Control group included 25 patients with no respiratory complaints and normal PFT. All patients who satisfied inclusion criteria are included in the study. All patients had undergone pulmonary function tests and their biochemical parameters (alpha 1 AT, antitrypsin activity, anti elastase activity, alpha 2 MG, Vitamin E, Vitamin C, reduced glutathione) had been studied. Complete data was available and these subjects formed population for analysis.
In our study, male and female are equal in control group and males were predominant in study group.
Table 1 shows smoking wise distribution of patients. There were no smokers with bronchial asthma whereas there were 21 non smokers with bronchial asthma. There was one smoker and 13 non smokers with bronchiectasis. There were 7 smokers and 5 non smokers with COPD. There were 7 smokers and 2 non smokers with emphysema.
Table 2 shows comparison of protease inhibitors among smokers and non smokers. It is seen that for alpha 1 AT there was no difference between smokers and non smokers. For alpha 2 MG the value was more among non smokers. But for anti trypsin and anti elastase activity also there was no difference in the mean values among smokers and non smokers.
Table 3 shows comparison of antioxidant levels among smokers and non smokers. It can be seen that there is no significant difference in the mean values of antioxidant levels among smokers and non smokers.
Table 4 shows pulmonary function test (PFT) results in smokers and non smokers. It is seen that non smokers have better PFT values compared to smokers. All non smokers have normal PFT. But among smokers, only 7 have normal PFT and 18 have Severely abnormal PFT.

DISCUSSION

In our study, total 56 patients had been included in study group which contain emphysema 9 patients, COPD 12 patients, bronchiectasis 14 patients, and bronchial asthma 21 patients.
These patients are classified into mild, moderate and sever airway obstruction, depending upon their FEV1 values in PFT.

Disease	Smokers	Non smokers
Bronchial asthma	0	21
Bronchiectasis	1	13
COPD	7	5
Emphysema	7	2
Normal	6	19
Total	21	59

Table-1: Smoking wise distribution of patients

Protease inhibitors	Smokers (mean±SD)	Non smokers (mean±SD)
Alpha 1 AT (mg/dl)	186.57±29.97	186.61±37.15
Alpha 2 MG (mg/dl)	228.67±63.28	243.63±54.61
Anti trypsin activity	7.12±1.69	7.67±1.99
Anti elastase activity	8.21±3.49	8.28±2.93

Table-2: Comparison of protease inhibitors among smokers and non smokers

Antioxidant levels	Smokers (mean±SD)	Non smokers (mean±SD)
Vitamin C (mg/dl)	0.97±0.26	0.92±0.23
Vitamin E (mg/dl)	1.14±0.43	1.10±0.35
Glutathione (mg/dl)	34.27±7.45	32.06±6.72

Table-3: Comparison of antioxidant levels among smokers and non smokers

PFT	Non Smokers	Smokers	Total
Normal (> 80% of FEV1)	25	7	32
Mild (60 – 80% of FEV1)	0	17	17
Moderate (40 – 59% of FEV1)	0	14	14
Sever (< 40% of FEV1)	0	18	18
TOTAL	25	56	81

Table-4: Pulmonary function test (PFT) results in smokers and non smokers

Our study included a control group of 25 patients who had no respiratory complaint and had normal PFT. We compared levels of alpha 1 AT, Anti trypsin activity, anti elastase activity, alpha 2 MG activity and anti oxidants (vitamin C, vitamin E and reduced glutathione) among smokers and non smokers.
The study shows that the range of alpha 1 AT among control group varies from lowest level of 130 mg/dl to highest level of 287 mg/dl. And among study group it varies from lowest level of 130 mg/dl to highest level of 260 mg/dl.
Out study shows that there is no relation between FEV 1 value and concentration of alpha 1 AT or its activity, in the serum of patients with COPD, emphysema, bronchial asthma and bronchiectasis as compared with the control group.
Earlier studies have shown that aplh 1 AT deficiency in adults and children may not cause lung function abnormality. It had been seen that after the age of 35 years, there is an accelerated fall in FEV 1 in individuals with alph 1 AT deficiency. [3]
In our study among smokers and non smokers, it was shown

that there is no difference in concentration of alpha 1 AT. It has been studied that there is decrease in anti neutrophil elastase activity because the reactive site methionine of alpha 1 AT gets oxidized and becomes inactive because of oxidative stress of smoke. [4]

Electrophoretic study was not available for classification of alpha 1 AT variants which is essential for their activity. Some variants like ZZ are associated with early onset of COPD and emphysema. Incidence of alpha 1 AT deficiency is rare in black and Asian[5], and most commonly seen in Caucasians from northern Europe.[6] Our study shows that no patient from study group having deficient level of alpha 1 AT. alpha 1 AT deficiency result from homozygous inheritance of the Z type alpha 1 AT gene is associated with serum alpha 1 AT levels of < 50 mg/dl and with the development of emphysema in third and fourth decade.[7] none of our patients in study and control group had such low levels of alpha 1 AT. So in Indian scenario, incidence of deificiency of alpha 1 AT is very rare.

CONCLUSION

It has been studied that the concentration of alpha 2 MG increases in pulmonary diseases.Our study also showed that there is a statistically significant rise in serum alpha 2 MG concentration among bronchial asthma.

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