EVALUATION OF OXIDATIVE STRESS & TOTAL ANTIOXIDANT CAPACITY, OXIDIZED LDL IN ADULT SMOKERS

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ABSTRACT

Introduction: Tobacco is very harmful to our body. It contains five thousand of injurious chemicals injurious for our body and heart and cause various diseases like lung, oral cancers and obstructive pulmonary diseases. It is a serious threat for health. Smoking causes deaths. India ranks second as a cause of death in the world.

Objectives of the study: to determine the levels of MDA as marker of oxidative stress, total antioxidant capacity and oxidized LDL in adult smokers.

Methodology: Fasting Venous blood (5 ml) was collected under taking aseptic precautions and the serum was separated used for the estimation of Oxidised LDL (Ox-LDL), total antioxidant capacity (TAOC) and malondialdehyde (MDA) .

Results: In the present study, Ox-LDL and MDA were significantly increased & TAOC (Total antioxidant capacity) was significantly decreased ($p < 0.005$) in smokers compared to non-smokers.

Conclusion: Our study provides the evidence that total antioxidant capacity is decreased in smokers as compared to healthy controls. Levels of MDA and Ox-LDL is increased in smokers as compared to healthy controls.

Key-words: oxidised low density lipoprotein, total antioxidant capacity malondialdehyde and smokers.

INTRODUCTION

Tobacco is very harmful to our body. It contains five thousand of injurious chemicals injurious for our body [1] and heart and cause various diseases like lung, oral cancers and obstructive pulmonary diseases [2]. It is a serious threat for health. Smoking causes deaths. India ranks second as a cause of death in the world [3,4].

The World Health Organization (WHO) has entitled tobacco as one of the greatest public health threats of the twenty-first century. Tobacco smoking is an increasing public health problem especially in a developing countries like India. Prevalence of smoking in India varies from 15% to 50% among men with physical or psychological dependence [5].

India is the biggest manufacture and exporter of tobacco with inside the world [6]. Half of its amount is consumed in chewing, smoking [7]. Hence smoking is done with beeds, hookah, pipe, cigarette, cigar etc [8]. Tobacco kills 8 lakh to 10 lakh people every year in India [9]. Death rate is increasing day by day every year in youngsters also. It has more percentage in India than other countries [10]. Significant changes in the lipid profile is encountered in smokers, the Stimulation of sympatho-adrenal system by Nicotine leads to lipolysis and it increases the serum free fatty acid levels which leads to the synthesis of VLDL from liver. It increases Triglycerides and the repessive action of smoking reduces the levels of HDL cholesterol. Smoking causes various diseases like cancer, coronary heart disease, peptic ulcer etc. With Tobacco smoking, a person exhales burnt tobacco in the form of fumes [11]. In pregnancy, it has adverse effect on the Foetus [12]. Tobacco smoke contains abundant reactive oxygen species which damage tissue with cigarettes [13]. Many harmful chemicals enter to the body through our lungs and damage tissues [14].

Total Antioxidant Capacity: It is an analytic measuring the antioxidant amount. Antioxidant or free radical scavenger [15] is the compound binding chemically to free oxygen radicals and hence, preventing healthy cells from getting damaged by these radicals thus, decreasing the lipid oxidation reaction rate [16]. Various agents gets included in the antioxidants like enzymes (superoxide dismutase, catalase, and glutathione peroxidase), small molecules (Vitamin C, Vitamin E, uric acid, bilirubin), large molecules (ferritin, albumin) etc [17].

Antioxidants prevents or delays the oxidation of the cell thus, preventing its damage caused due to oxidative agents. Antioxidants have reducing capability thus causes reduction of oxidative agents such as free radicals, reactive oxygen species and reactive nitrogen species and hence neutralize their effect [18, 19].

Free radicals are produced due to smoking. It leads to imbalancing of antioxidant protective mechanism due to excessive free radical production in the body which is known as Oxidative stress [20, 21]. To counteract the adverse effects produced due to oxidative stress, the cells tries to maintain the redox balance leading to activation of encoding genes. These genes leads to the formation of defensive enzymes, transcription factors and whereas structural proteins gets formed [22]. Malondialdehyde is formed as result of degradation of cell membranous lipids i.e. polyunsaturated fatty acids by Reactive oxygen species. Thus as a biomarker, MDA is used for the measurement of oxidative stress levels in several organisms. The major presentations of oxidative stress is lipid peroxidation and thus it is considered as a good oxidative stress marker.

AIM AND OBJECTIVES

Aim: Evaluation of oxidative stress & total antioxidant capacity, oxidized LDL in adult smokers.

Objectives: To determine the levels of MDA as a marker of oxidative stress, total antioxidant capacity and oxidized LDL in adult smokers.

MATERIALS AND METHODS

Study design: Hospital based observational study Duration: 6 months Inclusion criteria: At least 3 cigarettes / bidis/ day for ≥ 2 years.

Exclusion Criteria

1. Any history of chronic illness (cardiovascular diseases, lung diseases, diabetes)
2. History of drug intake such as statins, hypoglycemic and antihypertensives
3. Chronic alcoholism

4. Pregnancy & lactation
5. Obese persons (BMI ≥ 30)

Methodology: Venous blood (5 ml) was be taken from all the subjects included in the study after 12-14 hours of overnight fasting and taking all aseptic precautions. Serum will be separated by centrifuging for 15 minutes at 3500 rpm. The following parameters were analyzed in serum by fully auto-analyzer EM- 360(ERBA): Apolipoprotein A1, Apolipoprotein B. The following parameters were done by ELISA method by commercially provided kits by- Ox-LDL. The following parameters were analysed in serum by colorimetry: TAOC, MDA

RESULTS

The present study was done to compare the serum levels of MDA, TAOC & Ox-LDL in smokers and non- smokers. This comparative study of extended lipid profile, antioxidant capacity and healthy controls were done by student's t-Test.

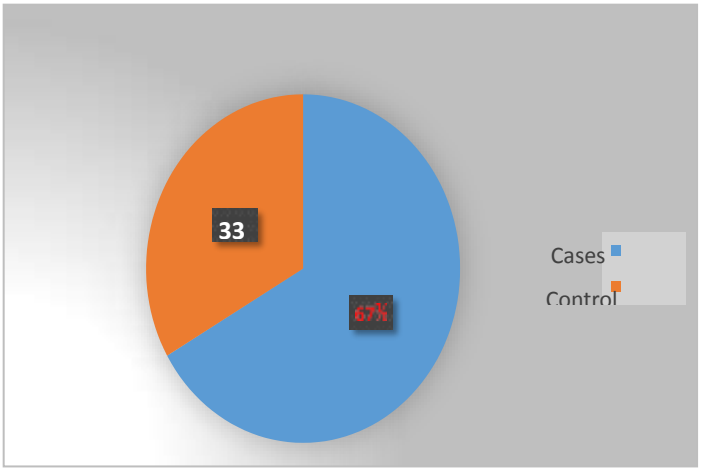

Figure 1: Shows the distribution of cases and controls

Table 1: Shows the comparison of parameters between cases and controls				
Parameters	Cases (n = 80) (Mean ± SD)	Control (n = 40) (Mean ± SD)	t value	p value
Ox-LDL	1.49 ± 0.63	0.38 ± 0.29	10.48	**<.001****
MDA	6.93 ± 1.09	2.80 ± 1.15	19.07	**<.001****
TAOC	0.31 ± 0.11	1.28 ± 0.39	-20.01	**<.001****

It is evident from the table 1 that the parameters taken in our study such as Apo B, Ox-LDL (Oxidized Lowdensity lipid), MDA (Malondialdehyde), were significantly increased in the smokers ($p < 0.001$) when compared to non-smokers and TAOC (Total antioxidant capacity) was significantly decreased ($p < 0.001$) as compared to non- smoker.

DISCUSSION

Smoking is a known modifiable risk factor of pulmonary disease, cancer, arthrosclerosis etc [2]. Tobacco is consumed in many ways like smoking, chewing etc [7]. According to the WHO (world health organization) tobacco is one of the greatest public threat and smoking escalate public health

problems in developing country [5].

Tobacco kills 8 lacks to 10 lakhs people every year in India, which involve mostly young age person[9]. According to the WHO projection, India will have highest rate of deaths related to tobacco as compared to other countries in future [10].

Smoking cigarette causes generation of free radicals and an imbalance between antioxidant preventive mechanism and free radicals called as oxidative stress [20, 21]. Smokers are classified on the basis of packs of cigarette they smoke per year. This is calculated by multiplying quantity of cigarette smoke per day to number of years the person is smoking.

We studied 40 normal nonsmokers healthy individuals male between age group of 20 to 40 years and compared them with smokers (n=80) of the same age group. Studies have shown that cigarette smoking causes significant high levels of MDA & Ox-LDL chronic smokers compared to non-smokers.

In our study the mean and standard deviation of MDA in cases is 6.93±1.09 and in control is 2.8±1.15 with t value 19.07 & $p < 0.001$. Epithelium tissue is more sensitive to cigarette smoke, which leads to the formation of a higher number of free radicals. These free radicals starts oxidizing the phospholipids of the plasma membrane. Polyunsaturated fatty acids are present in the cell membrane which are attacked by free radicals and thereby lead to the formation of lipid peroxidation products. Then it was seen that these products were more in smokers.

In our study the mean and standard deviation of Ox-LDL in cases is 1.49±0.63 and in controls 0.38±0.29 with t value 10.48 & $p < 0.001$. Chronic smoking can leads to increased level of triglyceride, total cholesterol due to decrease in lipoprotein lipase levels. This is being expected to increase the synthesis of VLDL which is consistent with the changes reported in lipid profile of cigarette smokers. Serum HDL levels became susceptible to oxidative modification by cigarette smoking and loses its atheroprotective properties. Previous studies showed that smoking cessation leads to normal levels of HDL in blood as in non-smokers. Non HDL fractions which are strong predictor for risk of coronary heart disease has increased in smokersas compared to non-smokers.

In the present study the mean and standard deviation of TAOC in cases is 0.31±0.11 and in controls 1.28±0.39 with t value -20.1 and $p < 0.001$. Increase oxidative stress in smokers as compared to non-smokers is a potential reason for this finding which is related to the stationary lifestyle. Lower aerobic fitness level is also associated with a high oxidative stress level in smokers.The findings of our study were similar to the studies conducted by others [11-22.

CONCLUSION

In this study we found significantly increased levels of Ox-LDL, Apo B, MDA with p value (<0.001) and significantly decreased levels of total antioxidant capacity, with p value (0.001). Our study provides the evidence that total antioxidant capacity is decreased in smokers as compared to healthy controls. Levels of MDA and Ox-LDL is increased in smokers as compared to healthy controls. More number of studies should be conducted for this.

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