A COMPARATIVE STUDY OF ANTIOXIDANTS IN SMOKERS & NON-SMOKERS

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ABSTRACT

Smoking is a complex and profound neurochemical and behavior disorder influenced by social, environmental, psychologic, and biologic factors. Cigarette smoking is one of the most extensively used potentially hazardous social habits throughout the world. Smoking induces oxidative stress. The aim of this study was to compare uric acid, ascorbic acid & ceruloplasmin levels as antioxidants in smokers and non-smokers. This was a prospective study and controls were selected from the workers in the medical college and hospital in the age group of 25 to 50 years of males. Blood levels of uric acid, ascorbic acid & ceruloplasmin levels were measured using standard methods. It was found that uric acid, ascorbic acid & ceruloplasmin levels were significantly lower in smokers than in non-smoker group. Significantly decreased plasma levels of antioxidants like uric acid, ascorbic acid & ceruloplasmin levels suggests an increased oxidative stress in smokers group. Therefore, quitting smoking represents an irreplaceable preventive strategy against tobacco-induced oxidative stress.

Introduction:

Smoking is a complex and profound neurochemical and behavior disorder influenced by social, environmental, psychologic, and biologic factors. Smoking has a particularly large impact in the developing world & accounts for 1.17 million deaths per year. The first Global Adult Tobacco Survey of 2010 reports that currently 34.6% of adults (47.9% males & 20.3% females) in India are users of tobacco products.

According to WHO approximately one third of world population older than 15 years, are consuming tobacco. Cigarette smoking is one of the most extensively used potentially hazardous social habits throughout the world but more extensively prevalent in South East Asia.

Today tobacco consumption has been established as a number one preventable cause of death and disease worldwide. Tobacco use has become widespread in many societies for religious, medicinal and recreational purposes.

Addicted smokers regulate their nicotine intake by adjusting the frequency and intensity of their tobacco use, both to obtain the desired psychoactive effect and also to avoid withdrawal. Along with nicotine other toxic
and carcinogenic compounds are inhaled, deposited in the airway and alveoli and absorbed into the body and hence the use of tobacco as cigarettes is more hazardous.

Nicotine in the particulate phase in fresh smoke volatilizes into the gaseous phase as the smoke ages and is a major vapor phase constituent in environmental tobacco smoke. Biologic markers of extent of smoke inhalation includes carbon monoxide bound to hemoglobin in the blood, nicotine, cotinine in the blood, saliva and urine, hydrogen cyanide in the blood, saliva and urine, globin adducts of nitrogen oxides, ethylene and tobacco specific nitrosamines etc. 8,9,10

Smoking has long been being associated with an increased risk on developing several chronic diseases including the atherosclerosis, which is believed to be initiated by lipid peroxidation. The chronic smoking which is defined as more than 10 years which creates more free radicals in human system by which all of the major classes of biomolecules may be attacked by the free radicals; but lipids are probably the most susceptible.11,12,13

Cigarette smoke contains numerous radicals and radical generating compounds14; the action these oxidative agents and other mutagens present in cigarette smoke are accompanied by DNA damage, mutations in oncogene’s activation and tumor suppressor gene inactivation.

Smoking enhances oxidative stress not only through the production of ROS in cigarette tar and smoke but also through weakening of the antioxidant defense systems.15

However, smokers have also consistently being shown to have a lower intake of fruits and vegetables in the diet which result in a lower plasma antioxidant concentration. Vitamin C being one of the important plasma antioxidant; the lowered plasma Vitamin C levels in smokers could also be either due to either of its impaired absorption or increased turnover.16

High serum uric acid concentrations might be protective in situations characterized by increased cardiovascular risk and oxidative stress as smoking and by reducing its level the susceptibility to oxidative damage increases and accounts for the excessive free radical production1, since uric acid act as an endogenous antioxidant

Ceruloplasmin is a copper containing enzyme. It oxidizes a variety of amines including epinephrine, melatonin, serotonin, para-phenylene diamine etc. Ceruloplasmin concentration was decreased in the advanced stage of oxidative stress during smoking.17,18

Methodology:
The present study was conducted at Vinayaka Mission’s Medical College & Hospital, Karaikal. This was a prospective study and controls were selected from the workers in the medical college and hospital in the age group of 25 to 50 years of males. The cases were selected from the outpatient department of medicine and surgery. The subjects were male, aged between 25-50 years, non-smokers and smokers. Subjects with history of Diabetes mellitus, Hypertension, Hepatic disorders,Renal disorders, On medications like beta blockers, steroids & multi- vitamins were excluded from the study

After recording and receiving the completed questionnaire, the controls and cases were selected. Under aseptic precautions the venous blood samples were collected from the subjects, after getting their consent. Each participant gave an informed consent and this study was approved by the Ethical committee of Vinayaka Mission’s Medical College, Karaikal. Plasma Ascorbic acid was estimated using Roe & Kuether; 1943; Roe,1961 method, Serum uric acid was estimated usingUricase/ POD method and Serum ceruloplasmin was estimated using Nephelometric Immunoassay.

Statistical analysis was done using appropriate statistical tests & p value was calculated.
Results:

Table 1: Comparison of vitamin C in smoker and non-smoker group

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Smoker (n=30)</th>
<th>Non-smoker (n=30)</th>
<th>Z Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin C (mg/dl)</td>
<td>Mean 0.90</td>
<td>SD 0.16</td>
<td>Mean 1.41</td>
<td>SD 0.14</td>
</tr>
</tbody>
</table>

The table 1 showed that there are significant lower levels of vitamin C in the smoker group than non-smoker group.

Table 2: Comparison of Ceruloplasmin in smoker and non-smoker group

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Smoker (n=30)</th>
<th>Non-smoker (n=30)</th>
<th>Z Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ceruloplasmin (mg/l)</td>
<td>Mean 270.02</td>
<td>SD 42.57</td>
<td>Mean 346.94</td>
<td>SD 34.94</td>
</tr>
</tbody>
</table>

The table 2 showed that there is significant lower levels of ceruloplasmin in the smoker group than non-smoker group.

Table 3: Comparison of uric acid in smoker and non-smoker group

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Smoker (n=30)</th>
<th>Non-smoker (n=30)</th>
<th>Z Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric acid (mg/dl)</td>
<td>Mean 4.31</td>
<td>SD 0.70</td>
<td>Mean 6.15</td>
<td>SD 0.50</td>
</tr>
</tbody>
</table>

The table 3 showed that there is significant lower levels of uric acid in the smoker group than non-smoker group.

**DISCUSSION**

Chronic smoking leads to oxidative challenge and leads to formation of many deleterious substances including free radicals, among which plasma Malondialdehyde (MDA), is a commonly used biomarker of lipid peroxidation.

In the present study, it was found that uric acid, ascorbic acid & ceruloplasmin levels were significantly lower in smokers than in non-smoker group.

The antioxidant status, in particular that of Vitamin C, is a major factor in lipid peroxidation is supported by several studies.22
Gordon et al found that mean serum Vitamin C levels in smokers as 0.94 mg/dl, in the study of influence of smoking on Vitamin C in adults.\textsuperscript{23}

Ali Akbar Shah et al, 2015 reported the mean Vitamin C value in smokers as $0.8 \pm 0.16 \text{ mg/dl}$ and $1.22 \pm 0.29 \text{ mg/dl}$ in non-smokers.\textsuperscript{24}

Since free radicals and superoxide production increases in case of oxidative challenge, there is excessive sequestration of ceruloplasmin as the body tries to ward off any potential damage being caused due to excessive smoking. As a result of this serum ceruloplasmin level decreases in chronic smoking.\textsuperscript{25}

In the present study, mean Ceruloplasmin in smokers is $255.6 \pm 35.55 \text{ mg/l}$, when compared to the value of $346.94 \pm 34.64 \text{ mg/l}$ in non-smokers. The concentration of ceruloplasmin is significantly lower in smokers than in non-smokers.

The low concentrations of ceruloplasmin which was reported in the study by Safaa H et al, that demonstrated the extent of ROS-induced oxidative damage can be exacerbated by a decreased efficiency of antioxidant defense mechanisms.\textsuperscript{26}

Uric acid is the most abundant aqueous antioxidant, accounting for up to 60\% of serum free radical scavenging capacity, and is an important intracellular free radical scavenger during metabolic stress including smoking, therefore, measurement of its serum level reflects the antioxidant capacity.\textsuperscript{27}

Hanna et al, 2008, reported that the significant low serum uric acid levels in smokers was attributed to reduced endogenous production as a result of chronic exposure to cigarette smoke that is a significant source of oxidative stress.\textsuperscript{28} In a study by Tahani et al, its reported that the serum uric acid level in smokers is significantly lower than that of the non-smokers.\textsuperscript{29}

In the present study, the serum uric acid level is significantly altered in the smokers as the uric acid level is $3.5 \pm 0.83 \text{ mg/dl}$ in smokers and in non-smokers it is $6.15 \pm 0.49 \text{ mg/dl}$.

The results of present study demonstrate that smoking significantly decreases Vitamin C, Ceruloplasmin and Uric acid levels.

Even though some studies\textsuperscript{30,31} suggest the smokers to take the additional amounts of Vitamin C in order to avoid the deleterious effects of smoking on their health; Khushdeep et al\textsuperscript{21} concluded that cigarette smoking cessation is followed by a marked increase in plasma antioxidant concentrations which substantially improves plasma resistance towards oxidative challenge. Therefore, quitting smoking represents an irreplaceable preventive strategy against tobacco-induced oxidative stress.

**CONCLUSION**

The results of present study demonstrate that smoking significantly decreases Vitamin C, Ceruloplasmin and Uric acid levels. This decreased antioxidant concentrations which substantially reduces the smoker’s resistance towards oxidative challenge, which may lead to many deleterious effects in them. Therefore, adopting a healthy lifestyle by quitting smoking represents an irreplaceable preventive strategy against tobacco-induced oxidative stress.
References:

5. Dr. Ketan Patel, Dr. Paresh prajapati, Dr. Saurin sanghavi, Dr. Vijay goplani. A study on effects of cigarette smoking on blood cholesterol in young population of Ahmedabad: International Journal of basic & Applied physiology, vol 3(1), 2014, page 129.


