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Assessment of Biochemical profile in smokers

[1] Dr. Pasupurekula Laxmi, [2] Dr. Srirekha P

[1] Senior Residenr, Department Of Biochemistry, Svims, Tirupathi, Andhra Pradesh, India
[2] Consultant Biochemist, Medall Diagnostics, Kurnool, Andhra Pradesh, India

Abstract
Today 80% of the world's 1 billion smokers Iive in low- and middle-income countries. If trends continue tobacco-related deaths will increase to 8million per year by 2030 with 80% of those deaths in low- and middle income countries. The present study aims to evaluate biochemical oxidative stress markers, serum lipid profile and liver function test in smokers and non-smokers patients. The biochemical oxidative stress markers estimated were malondialdehyde, vitamin C and serum uric acid levels, plasma lipid profile and liver function test Plasma levels of total bilirubin, alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein and albumin were estimated in all subjects The results of present study demonstrate that smoking significantly increases oxidative stress. In our study we found that level of cholesterol, triglyceride, LDL and VLDL is significantly increases in smoker as compared to nonsmoker while HDL decreases significantly in smoker, level of liver enzymes (AST, ALT, ALP) were significantly increased in smokers while level of serum bilirubin, total protein and albumin decreases in smokers as compared to nonsmoker.so we conclude that chronic smoking leads to oxidative stress and These effects could lead to alteration in liver function test. Increased plasma lipid profile shows increased cardiovascular risk.

Keywords: Biochemical profile, smokers, cholesterol, triglyceride

Introduction
Smoking is one of the most common forms of recreational drug use [1]. Diseases related to tobacco smoking have been shown to kill approximately half of long term smoker when compared to average mortality rates faced by non smokers. More than 5 million of those premature deaths are the result of direct tobacco use while more than 600 000 are the result of non-smokers being exposed to second-hand smoke [1].
Smokers are at greater risk for cardiovascular diseases, respiratory disorders, peripheral vascular disease, cancer, peptic ulcers and gastro esophageal reflux disease, blindness, bone matrix loss, and hepatotoxicity [2].
Long delay between smoking and onset of smoking related diseases resulted in the ignorance of ill effects of smoking [3].
Cigarette smoke contains 1017 oxidant molecules per puff [4].
The oxidants in cigarette smoke cause lung injury by a number of mechanisms including the depletion of glutathione and other antioxidants, the initiation of redox cycling mechanisms, enhancement of the respiratory burst in neutrophils and macrophages, inactivation of protease inhibitor, and direct damage to lipids, nucleic acids and proteins [5].
Lipid hydroperoxides formed, are unstable compound that tend to decompose rapidly forming secondary products, which are responsible for deleterious effects of lipid peroxidation. these include alkanes, like pentane, ethane, and aldehydes, such as malondialdehyde [6]. Production of malondialdehyde can be used as a biomarker to measure the level of oxidative stress [7].
Antioxidants were define as substances that are at relatively low concentration; able to compete with other oxidizable substrates and thus to significantly delay or inhibit the oxidation of these substrates [8]. The above definition include the enzymes superoxide dismutase, glutathione peroxidase and catalase as well as non enzymes such as Vitamin E, Beta Carotene, Ascorbate and Glutathione.
Glutathione is the reducing agent that recycles ascorbic acid from its oxidized to its reduced form by the enzyme Dehydroascorbate Reductase [9].
Uric acid scavenges free radicals and it is formed from xanthine by the enzyme xanthine oxidase and it is converted to allontoin during the reaction.
Among the strongest determinants of plasma total antioxidant defence, vitamin C is one of the most effective free radical scavengers [10].
Recently it has been suggested that smoking adversely affects the concentration of plasma lipids and lipoprotein levels. It has been estimated that 1% increase in plasma concentration is associated with a 2.7% increase in risk [11].
Smoking may affect the liver through inflammatory pathways and may aggravate the pathogenic effects of alcohol on the liver. Smoking cigarettes can also severely affect your liver, the numerous toxins found in cigarette tobacco lead to chronic inflammation and scarring in the liver, which in turn, increases your risk for liver damage including diseases such as Hepatitis B, and C, liver cancer and liver fibrosis [12].
The present study was aimed to evaluate the, plasma malondialdehyde and serum uric acid, plasma ascorbate (vitamin C), plasma lipid profile, liver function tests in smokers and non smokers.

Materials and methods
Study design and subjects
Cross- sectional study was done in smokers with long period from June 2015-Dec 2016. This study was carried out in Government General Hospital, Department of Biochemistry, Sri Venkateswara medical college, Tirupati. The cases were selected from the outpatient department of medicine and surgery. The subjects were male, aged between 25-50 years.

Control group consists of 40 healthy individual non smokers and case group consists of 160 cigarette smokers.

Inclusion criteria

In this study, we included > 18 years of age, smoking more than five cigarettes per day (smokers only), normal weight (20 kg/m2 < BMI <30 kg/m2).

Exclusion criteria

We excluded smokers with history of chronic diseases, regular use of medicine, any use of vitamin or other dietary supplements after pre-screening, and smoking within the last 2 years (non-smokers only).

Ethical consideration

The objectives of the study were explained to all individuals participating in this study. All participants who agreed were given written acceptance form.

Blood sample

An overnight fasting blood sample was collected from each subject by using EDTA as an anticoagulant. The blood samples were immediately centrifuged at 2000 g for
5 min and plasma was separated.

Biochemical Analysis

Serum MDA level was measured as per thiobarbituric Acid (TBA) method. Serum vitamin C was estimated by 2, 4 – dinitrophenyl hydrazine method. Serum uric acid estimated by uricase/ peroxidase method the liver function test, total cholesterol, HDL and triglycerides were estimated by enzymatic method in a fully automated analalyser A25. Low Density lipoprotein (LDL) and (Very Low Density lipoprotein) VLDL were calculated by Friedwald's Equation.

Statistical analysis

The statistical package Graph pad Prism 6 for windows was used for data analysis. Variables distributed normally are presented as mean and standard deviation were done

Results

Table 1: Comparison of serum malondialdehyde (MDA), serum vitamin C, serum uric acid in smoker and non-smoker group:

Parameters	Smoker (n=160)	Non-smoker (n=40)	P Value
Serum MDA (n mol/ml)	5.27±0.75	2.84±0.81	<0.01
serum vitamin C(mg/dl)	0.80±0.16	1.22±0.29	<0.01
serum uric acid	3.42 ±0.72	6.15±0.49	<0.01

Table 2: Comparison of liver function test in smoker and non-smoker group

Parameters	Smoker (n=160)	Non-smoker (n=40)	P Value
Total Bilirubin(mg/dl)	0.74±0.11	0.35±0.10	<0.01
AST(IU/L)	59.30±2.35	34.37±2.68	<0.01
ALT (IU/L)	42.29±4.28	30.23±3.65	<0.01
ALP(IU/L)	156.14±9.78	83.20±14.94	<0.01
γGT(U/L)	23.11±2.65	18.11±3.98	=0.05
Total Protein (g/dl)	7.58±0.32	5.43±0.78	<0.01
Albumin(g/dl)	4.31±0.11	2.78±0.43	<0.01

Table 3: Comparison of plasma lipid profile in non-smoker and smoker group.

Parameters	Non-smoker (n=40)	Smoker (n=160)	P Value
Cholesterol (mg/dl)	165.30±19.42	278.56±17.36	<0.01
Triglyceride(mg/dl)	132.85±9.36	183.46±15.65	<0.01
HDL(mg/dl)	48.78±7.42	24.20±2.15	<0.01
LDL(mg/dl)	86.16±28.21	212.18±16.80	<0.01
VLDL(mg/dl)	23.58±3.10	31.54±4.36	<0.01

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. The smokers have higher plasma concentrations of MDA compared with non-smokers. This suggests that the observed increase in plasma MDA was induced by smoking per se. While poor antioxidant status presumably also affects lipid peroxidation as shown in several previous studies of Birgul Isik *et al*. Bulent Ozbay *et al* and Kushdeep Singh Arora *et al* [17].

In the present study, it was found that uric acid, ascorbic acid levels were significantly lower in smokers than in non-smoker group. These findings are in accordance with the study of Mukuddar colikoglu *et al* and Gordon *et al* [13].

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 [14].

In the present study, the serum uric acid level is significantly altered in the smokers. These findings are in accordance with the previous study by Hanna *et al* and Tahini *et al*

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 [15]. 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 [16].

Cigarette smoke contains a large number of chemical substances with hepatotoxic potential including nicotine. Cigarette smoke also induces oxidative stress by stimulating NADPH oxidase and decreasing antioxidant defenses, leading to lipid peroxidation. These effects could lead to increased

hepatocellular damage and subsequent activation of resident hepatic stellate cells, a major fibrogenic cell type. Other fibrogenic cell-types such as mesangial cells are stimulated by products from cigarette smoke, such as nicotine to proliferate and produce increased amounts of extracellular matrix proteins [17].

Total Bilirubin, Total Protein, Albumin and Globulin decreases significantly in smoker as compared to nonsmoker. Free radicals released as result of oxidative stress causes proteolysis or due to loss of protein [18, 19].

These findings are in accordance with the previous study by Sudeep Kumar *et al*. [20]

Alteration in lipid profile may be due to nicotine in smokers. Nicotine stimulates catecholamine secretion which inturn causes lipolysis. and release of free fatty acids [21].

These findings are in accordance with the previous study by Dr. Hari Shankar *et al* 2016 Sathish Kumar M *et al* 2015 and Dr. ketan patel etal 2014.

The results of present study demonstrate that smoking significantly increases Malondialdehyde, decreases Vitamin C, Uric acid levels, increased AST, ALT, ALP, increased total cholesterol, LDL,VLDL and decreased HDL.

Conclusion

1. The present study showed that smokers have higher levels malondialdehyde. Indicating increased oxidative stress, as a marker for lipid peroxidation in smokers.
2. The present study showed that smokers have lower values for serum vitamin C and uric acid, reflects antioxidant capacity in smokers.
3. The present study showed that smokers have abnormal liver function test indicates nicotine have hepatotoxic potential.
4. The present study showed that smokers have increased total cholesterol, LDL, VLDL and decreased HDL, indicates nicotonine have atherogenic effect and increases risk of coronary heart disease.

Thus our study concludes that smokers have higher risk than that of non-smokers.

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