Original article

Effect of Tobacco Consumption on Serum Alanine Aminotransferase and Alkaline Phosphatase Levels in Smoking

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Abstract

Background:
The use of tobacco has been significantly increased globally in recent decades. Easy availability and the low price gives rise to high consumption of tobacco smoking. Tobacco use is a leading preventable cause of premature mortality and morbidity.. Previous studies described the detrimental effects of tobacco smoking on liver function.

Objectives:
To observe the effects of tobacco consumption on the levels of serum alanine aminotransferase and alkaline phosphatase levels in smokers.

Methods:
The cross-sectional analytical study was conducted from January 2017 to January 2018 in the department of physiology, Rangpur Medical College, Rangpur. A total number of 60 subjects were selected, among them 30 were apparently healthy non-tobacco chewer non-smoker subjects as control group (group A) and 30 were apparently healthy smoker non-tobacco chewer subjects (group B). The subjects were selected from different area of Rangpur city. The effects of cigarette smoking on liver function were studied by measuring the levels of serum alanine aminotransferase and alkaline phosphatase levels. For statistical analysis independent sample "t" test was performed by computer based software SPSS-17.0 version for windows.

Results:
Serum alanine aminotransferase and alkaline phosphatase levels were significantly higher ($p<0.001$) in smoker non-tobacco chewer subjects as compared with the healthy control subjects.

Conclusion:
The increased serum alanine aminotransferase and alkaline phosphatase levels in smoker non-tobacco chewer subjects were evidence of development of liver function impairment due to tobacco smoking and this might offer a new preventive approach to liver function impairment in population with tobacco smoking.

Keywords: Tobacco smoker, Serum alanine aminotransferase and alkaline phosphatase

Introduction:

Tobacco is a plant product prepared from dried tobacco leaves, containing the alkaloid nicotine, which is an extremely addictive drug.[1,2] In addiction to nicotine, tobacco contains thousands of other chemicals such as cresol, pyrene, DDT, carbon monoxide, ammonia, hydrogen cyanide, acetone, methanol, formaldehyde, arsenic, cadmium etc.[3,4] Dried tobacco leaves are mainly used for smoking in cigarettes, cigars, pipe tobacco and flavored shisha tobacco. They can be also consumed as snuff, chewing tobacco and dipping tobacco.[1] There are two kinds of commonly used tobacco products in Bangladesh i.e. smoking and smokeless tobacco products.[4] Women usually used smokeless tobacco but rarely smoke or both.[4,5]

In Bangladesh 43.3% of adults (41.3 million) use tobacco in smoking and or smokeless tobacco form. More than five million people die globally each year due to tobacco related illness, the figure expected to increase to 8.3 million by 2030.[6]

Cigarette smoke also contains large numbers of free radicals that can trigger or promote oxidative damage. Cigarette smokers are at greater risk of developing cardiovascular diseases, respiratory disorders, blindness, bone matrix loss, peptic ulcers, gastroesophageal reflux disease and hepatotoxicity and cancer comparing with

non-smokers.[7] Now a days the adverse effects of smoking on liver function have gained more attention, because it is a major cause of preventable morbidity and mortality. Tobacco chewing and cigarette smoking is considered to be the most common particular risk factor for the liver function disorder.[8]

Smoking induces three major adverse effects on the liver- these are direct or indirect toxic effects, immunological effects and oncogenic effects. The chemicals in cigarette smoke cause oxidative stress in the liver. It leads to damage to the liver cells and fibrosis. In addition, smoking increases the production of pro-inflammatory cytokines (IL-1, IL-6 and TNF-α) which are involved in liver cell injury. Smoking contributes to the development of secondary polycythemia and so red cell mass increased and turnover. As a result iron overload occur which promote oxidative stress of hepatocytes. Smoking yields chemicals with oncogenic potentials that increase the risk of hepatocellular carcinoma (HCC).[9]

Consumption of tobacco is now increasing rapidly throughout the developing world and is one of the biggest threat to current and future world health. Bangladesh has been implementing surveys under GTSS since 2004 regularly at periodical intervals.[6] Global Adult tobacco surveys uses the global standard protocol for systemically monitoring adult tobacco use (smoking and smokeless) and tracking key tobacco control indicators. The GATS also provides a key to overseeing the WHO MPOWER Policy Suite, which includes: tobacco monitoring and prevention policies, protecting people from tobacco smoke, providing smoking cessation assistance, warning about the dangers of tobacco, enforcing advertising bans, sales promotions and sponsorships, increasing taxes on tobacco products.[6]

According to the official Agriculutural statistics three varieties of tobacco – Jati, Motihari and Virginia – are grown in different districts of Bangladesh. Rangpur still remains highest with 40,345 acres during 2008-2009.[10]

In smokers alteration of the relevant biochemical parameters may be indicators of onset of liver failure. Consumption of tobacco and smoking may be toxic and can impair liver function.[11,12] The liver functions are affected adversely in smoker non tobacco chewer group indicated by the higher level of serum serum alanine aminotransferase and alkaline phosphatase.[13]

The purpose of this study was to assess the effect of tobacco on some hepatic parameters of smoker non tobacco chewer subjects of northern region because the rate of tobacco use is more among the people of this region. As far as our knowledge, this kind of study is not previously done in our country. This study would increase awareness about the adverse effects of nonjudicial tobacco use on hepatic system.

Methods:

The Cross-sectional analytical study was conducted in the Department of physiology, Rangpur Medical College, Rangpur from January 2017 to December 2017. The Rangpur Medical college ethical committee and thesis protocol review committee approved the study protocol. Total number of 60 apparently healthy subjects of both sexes with age 30-45 years were divided into following groups: Group A–30 apparently healthy smokers and Group B–30 apparently healthy smokers non tobacco chewers. The subjects included in each group matched in their age and socio-economic condition. The duration of smoking is more than three years. All the subjects were free from history of liver, heart, lung and other chronic systemic diseases, obesity and diabetes mellitus, hypertension, pregnancy and lactating mother. After selection of subjects, the objectives and the procedure of the study were explained written consent were taken. A standard questionnaire was filled after taking history and through clinical examinations. At first day all the study procedures were maintained and advised the subjects to be overnight (8-10 hrs) fasting state. Then attended next day at 8.00A.M. at the department of Physiology, Rangpur Medical College, Rangpur. Fasting venous blood sample was collected from the antecubital vein from each subject under all aseptic precaution by a disposable syringe. The test tube containing blood was kept in standing position till formation of clot. Serum was separated by centrifuging the blood at 3000 rpm for 5 minutes. The clear supernatant was taken and kept in ependroffs. All biochemical tests were carried out as early as possible and done by enzymatic colorimetric method at the Department of Biochemistry, Rangpur Medical College,

Rangpur. For statistical analysis independent sample "t'' test was performed by computer based software spss-17.0 version for windows.

Results :

The mean±SD of serum alanine aminotransferase were 19.566±7.4124 U/L in group A and 37.200±12.2091 U/L in group B. The mean±SD of serum alkaline phoshatase were 56.0333±21.3242 U/L in group A and 93.5667±18.0013 U/L in group B. The mean serum alanine aminotransferase and alkaline phosphatase levels are significantly (p <0.001) higher in group B than A (Table–I).

Table-I: The mean±SD serum alanine aminotransferase and alkaline phosphatase levels of the study subjects in group A and group B.

Variables	Group A (n=30)	Group B (n=30)	p-value
Serum ALT U/L	19.56±7.41	3.62±12.20	.000*
Serum ALP U/L	56.03±21.32	93.56±18.00	.000*

n= Number of subjects.
*= p<0.001
Normal range of serum alanine aminotransferase level is 14-63 U/L.[14]
Normal range of serum alkaline phosphatase level is 25-90 U/L.[15]

Discussion:

In this cross-sectional study, serum alanine aminotransferase and alkaline phosphatase levels were significantly higher in smoker non tobacco chewer subjects than those of healthy control subjects which is comparable to others.[16,17]

Literature review suggested several mechanisms for these changes of serum alanine aminotransferase and alkaline phosphatase levels in smoker non tobacco chewer subjects. Higher levels of serum alanine aminotransferase and alkaline phosphatase in smoker non tobacco chewer subjects might be due to increased oxidative stress and increased lipid peroxidation lead to damage of liver tissue.[16] Cigarette smoke contains high concentration of the gaseous compounds like carbon monoxide, nitric oxide and other substances in cigarettes such as aldehydes, hydrogen cyanide, lead cadmium etc. In addition, it contains considerable number of free radicals that can be detected. Free radicals are highly reactive atom, which damage the biological cell membrane through lipid peroxidation. The heavy metal ions lead and cadmium present in smoke is also capable of inducing lipid peroxidation and hence provokes damage of organ propagating lipid peroxidation. Cigarette smoke propagates the lipid peroxidation, which damage the biological cell membrane of the liver and serum aminotransferases are enzymes that act as sensitive indicators of hepatocellular damage. The enzymes are leaked out into blood and increase the level of alanine aminotransferase level in smokers when compared with control. They also showed increased serum alkaline phosphatase level in smoker might be due to osteoporosis have documented serum alkaline phosphatase levels in smokers as a mainly marker of the liver and bones turnover.[17]

From the results of the present study, higher serum alanine aminotransferase and alkaline phosphatase levels in smoker non tobacco chewer subjects might be due to tobacco smoking for a prolong period of time which induced sustained rise of blood nicotine and also increased oxidative stress. Increased oxidative stress and increased lipid peroxidation lead to damage of liver tissue might be cause impaired liver functions in tobacco smokers.

Conclusion:

In this present study, it has been concluded that higher serum alanine aminotransferase and alkaline phosphatase levels in smoker non tobacco chewer subjects might be due to liver functions impairment in tobacco smoking for a prolong period of time which induced sustained rise of blood nicotine level. But exact mechanism of higher serum alanine aminotransferase and alkaline phosphatase in this group of people could not be demonstrated. Estimation of serum nicotine level, in same study population with large sample size which may help us to know the mechanism of increase serum alanine aminotransferase and alkaline phosphatase levels due to tobacco smoking. Reduction in tobacco consumption improves hepatic disorders and reduce mortality and morbidity in tobacco users.

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