Original article

Effect of Tobacco Consumption on Serum Albumin and Bilirubin Levels Among the Smoker

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Abstract

Background:
The recent decades there has been a massive global increase in tobacco use. Tobacco consumption is a major preventable cause of premature mortality and morbidity. Immediate availability and the low price give rise to high consumption of tobacco smoking and causes alteration of liver function.

Objective:
To observe the effects of tobacco consumption on liver functions 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 the control group (group A) and 30 were apparently healthy smoker non-tobacco chewer subjects (group B). The subjects were selected from a different area of Rangpur city. The effects of cigarette smoking on liver function were studied by measuring the levels of serum albumin and bilirubin. For statistical analysis independent sample "t" test was performed by computer-based software SPSS-17.0 version for windows.

Results:
Serum albumin and bilirubin levels were significantly lower ($p< 0.001$) in smoker non-tobacco chewer subjects as compared with the healthy control subjects.

Conclusion:
The decreased serum albumin and bilirubin levels in smoker non-tobacco chewer subjects were evidence of the development of liver function impairment due to tobacco smoking and this might offer a new approach to liver function impairment prevention in population with tobacco smoking.

Keywords: Tobacco smoker, Albumin, Bilirubin

Introduction:

Tobacco is a product prepared from the cured leaves of these plants. Tobacco contains the alkaloid nicotine, which is an extremely addictive drug.[1,2] In addition 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 in cigarettes, cigars, pipe tobacco, and flavored shisha tobacco for smoking. They can be also consumed as snuff, chewing tobacco, and dipping tobacco[1].

Smoking and smokeless tobacco items are the most commonly used tobacco products in Bangladesh.[4] Women usually used smokeless tobacco but rarely smoked 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]

Nowadays 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 are considered to be the most common particular risk factor for liver function disorder.[7,8]

Tobacco smoke contains over 4000 different chemicals, including nicotine, tar, and a gaseous compound including carbon monoxide which are highly cytotoxic potentials. It also contains various monoxide oxidants such as oxygen free radicals and volatile aldehydes which are probably the major causes of damage to biomolecules. Large numbers of free radicals are abundant in cigarette smoke that is capable of initiating or promoting oxidative injury. Cigarettes smokers are at greater risk for cardiovascular diseases, respiratory disorders, blindness, bone matrix loss, peptic ulcers, gastroesophageal reflux disease and hepatotoxicity, and cancer compared with non-smokers.[9]

Consumption of tobacco is now rapidly increasing all over the developing world and is one of the biggest threats to current and future world health. Bangladesh has been implementing surveys under Global Adult Tobacco Survey (GATS) since 2004 regularly at periodical intervals.[6] GATS uses the global standard protocol for systemically monitoring adult tobacco use (smoking and smokeless) and tracking key tobacco control indicators. GATS also provides the key for monitoring the MPOWER policy package developed by WHO which includes: Monitor tobacco use and prevention policies, protecting people from tobacco smoke, offering help to quit tobacco use, warning about the dangers of tobacco, enforcing bans on tobacco advertising, promotion and sponsorship and raise taxes on tobacco.[6]

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

In smokers alteration of the relevant biochemical parameters may be an indicator of the 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 the smoker non-tobacco chewer group, indicated by the lower level of serum albumin, bilirubin.[13]

The purpose of this study was to assess the effect of tobacco on some hepatic parameters of smoker non-tobacco chewer subjects of the northern region because the rate of tobacco use is more among the people of this region. Adequate data on this kind of study is not available in our country. This study would increase awareness about the adverse effects of unjudicial tobacco use on the 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. A total number of 60 apparently healthy subjects of both sexes with age 30-45 years were divided into the following groups: Group A–30 apparently healthy non-tobacco chewer non-smoker subjects as the control group 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 a history of liver, heart, lung, and other chronic systemic diseases, obesity and diabetes mellitus, hypertension, pregnancy, and lactating mother. After the selection of subjects, the objectives, and the procedure of the study were explained written consent was taken. A standard questionnaire was filled out after taking history and thorough clinical examinations. On the first day, all the study procedures were maintained and advised the subjects were to be in an overnight (8-10 hrs) fasting state. Then attended the next day at 8.00 A.M. at the Department of Physiology, Rangpur Medical College, Rangpur. A 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 a standing position till the formation of a 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 methods 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 albumin was 4.8433 ± 0.33701 gm/dl in group A and 3.6267 ± 0.38411 gm/dl in group B. The mean ± SD of serum bilirubin was 0.6233 ± 0.14547 mg/dl in group A and 0.4433 ± 0.18511 mg/dl in group B. The mean serum albumin and bilirubin levels are significantly (p <0.01) lower in group B than A (Table-I).

Table-I: Showing mean ± SD serum albumin and bilirubin levels of the study subjects in group A and group B

Variable	Group A (n=30)	Group B (n=30)	p value
Serum albumin (gm/dl)	4.83 ± 0.33	3.62 ± 0.38	.000*
Serum bilirubin (mg/dl)	0.62 ± 0.14	0.44 ± 0.18	.000*

*= p<0.001
Normal range of serum albumin level is 3.4-5.0 gm/dl.[14]
Normal range of serum bilirubin level is 0.2-1.00 mg/dl.[15]

Discussion:
In this cross-sectional study, serum albumin and bilirubin levels were significantly lower in smoker non-tobacco chewer subjects than those of healthy control subjects which is comparable to others.[16,17] The literature review suggested several mechanisms for these changes of serum albumin and bilirubin levels in smoker non-tobacco chewer subjects. Lower levels of serum albumin and bilirubin in smoker non-tobacco chewer subjects might be due to tobacco-containing a hepatotoxic agent which causes intoxication of liver might be lead to impairment of liver functions. As a result, decreased synthesis of albumin in tobacco users. They also suggested that tobacco smoking and chewing are associated with increased oxidative stress. Albumin has antioxidant properties, through binding to copper ions and scavenging HOCL, then the oxidized albumin may be cleared rapidly from the circulation and degraded. So there is decreased albumin level in serum.[16] They suggested that the decrease in plasma albumin level might be due to reducing protein synthesis or increased proteolytic activity due to cigarette smoke exposure or via release of high levels of cellular oxidative free radicals could result in increased proteolytic activity.[13] Chronic smokers manifest evidence of increased oxidative stress and inflammation. They also showed several metabolites reflecting antioxidant capacity in chronic smokers. In one antioxidant system, an increase in heme degradation by haem oxygenase to biliverdin and bilirubin is a protective anti-inflammatory response to increased oxidative stress. Exposure to cigarette smoke is known to increase the expression of HO-1. Bilirubin might be acting as an antioxidant and reduce free radicals produced by cigarette smoking. Plasma metabolomic profiles lower serum albumin and bilirubin levels in smoker non-tobacco chewer subjects might be to tobacco smoking for a prolonged period of time which induced sustained rise of blood nicotine and also carboxyhemoglobin levels. Increased concentration of nicotine, carboxyhemoglobin, and free radicals might be because of impaired liver functions in tobacco smokers.

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

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