DOI Number: 10.5958/0976-5506.2018.01944.7

Indirect Effect of Smoking on Liver Function: A Comparative Study among Iraqi Students in Baghdad City

Falah S. Al-Fartusie [1], Hassanain K. Al-Bairmani[1], Emad M. Eltayef [1], Ahmed H Yousif [2]

[1]Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq,
[2]Ministry of Health, Baghdad, Iraq

ABSTRACT

Smoking is a process in which a substance like tobacco is burned to produce smoke that inhales and absorbed into the bloodstream. The cigarette smoking is one of the greatest causes of illness, such as lung cancer, and premature death. The present study was constructed to evaluate the effect of cigarette smoking on the liver biochemical state. The study includes 50 smokers as patient group and 50 (age and sex matched) non-smokers as control group. The liver functions tests; aspartate aminotransferase (AST), alanine aminotransferase (ALT), total protein and alkaline phosphatase (ALP) were estimated spectrophotometrically. The results revealed that there was a significant reduction in the levels of AST, ALT, serum total protein and ALP of smokers in comparison with the healthy control (non-smokers) group. Also the results revealed the presence of a positive correlation between ALT and AST. To conclude, alterations in liver parameters levels could be attached to the oxidative stress created through the accumulative effect of inhaled smoke as well as the interaction of absorbed toxic chemicals like nicotine. Therefore, the association between smoking attitude and liver functions tests should be carefully analyzed.

Keywords: *liver; smoking; ALP; ALT; AST and total protein.*

www.IndianJournals.com
Members Copy, Not for Commercial Sale
Downloaded From IP - 98.82.120.188 on dated 15-Sep-2024

INTRODUCTION

Smoking is a process in which a substance, most commonly is the dried leaves of the tobacco, is burned and the resulting smoke inhales and absorbed into the bloodstream[1]. The use of tobacco cigarette smoking as well as hookahs smoking are become a major aspect of our society. It has been revealed that cigarette smoking is one of the greatest reasons of illnesses and precocious death for human. It is also reported that smoking-related diseases kill about half of long-term and heavy smokers, where it is found that males losing about 13.2 years and females losing about 14.5 years of their life as a result of smoking. In fact, about 4.9 million people per year have been reported to die worldwide as a result of smoking [2-5]. Smoking has been found to harm and injury approximately all parts of the human body. Lung cancer, heart attacks, vascular stenosis and chronic obstructive pulmonary disease are the most common diseases that can occur as a result of smoking [6-8]. Also, smoking has been found to cause harmful effects on the fetus during pregnancy [9]. Alarmingly, cigarette smoke contains at least 200 toxic substances, 73 known carcinogens, and large amounts of oxidants and free radicals that promote oxidative stress in the human body [10-12]. The liver is a large and vital organ of human body. It plays major and substantial roles in the human body, like digestion processes, metabolism, storage of nutrients and detoxification [13]. Therefore, it is extremely important to maintain the health conditions of this vital organ, and everyone should be aware of some things that can cause liver damage. Liver function tests are a set of blood tests that include serum total protein, albumin, total bilirubin (TB), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT) and γ-glutamyl transferase (GGT). They are useful and very important tests in the clinical evaluation of the state of the liver, and to help diagnose and monitor liver disease or liver damage, as well as to monitor

Corresponding author:
Falah S. Al-Fartusie. Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq;
Email: sci.falah.al_fartusie@uomustansiriyah.edu.iq

liver response to treatment. There are many personal and environmental factors that can affect liver function tests including age, sex, drinking alcohol, malnutrition, the suffering of certain diseases (such as heart disease, skeletal muscle, endocrine diseases), as well as liver health [14-16]. In fact, there is a lack of information about the influence of smoking on the function of liver. In contrast, alcohol consumption was found to be the most common cause of liver damage in humans. In drinking, the liver focuses mainly on eliminating the potential damage of alcohol by converting it into a less toxic form, where drinking alcohol leads to significant changes in fat metabolism in the liver [17-19]. These changes eventually lead to inflammation, steatohepatitis, cirrhosis and liver cancer [20,21]. Furthermore, it was found that alcohol consumption leads to increased levels of ALT and AST in humans [22]. Despite that cigarette smoke does not have a direct contact with the liver, it was found that the harmful chemicals in cigarette smoke causes a variety of adverse effects on the liver [23]. It has been reported that cigarette smoking increased serum ALP and GGT levels [24,25]. To date, there is still a delay in the researches that concerned with the full investigation of the influence of cigarette smoking on liver tissue and liver functions. Thus, the present study was constructed on Iraqi students in Baghdad city to evaluate the influence of cigarette smoking on the biochemical state of liver by investigating the most commonly used set of liver function tests.

www.IndianJournals.com
Members Copy, Not for Commercial Sale
Downloaded From IP - 98.82.120.188 on dated 15-Sep-2024

MATERIALS AND METHOD

Subjects and study design:

This study was conducted in the department of chemistry, college of sciences, Mustansiriyah University, Baghdad, Iraq, between October, 2016 and April, 2017. The study includes 50 male smokers as patients group with age ranging from 19-23 years, and 50 non-smokers male volunteers with age ranging from 18-22 years as control group.

Sample Collection

From each individual patients and control, 5 ml of blood were drawn by vein puncture and placed in a disposable plastic tube. The sample left to clot for about 20 minutes at room temperature and then centrifuged at 3000 g for 10 minutes. The obtained serum was carefully transferred into another tube and then frozen at – 20 °C for subsequent analysis.

Evaluation of Liver Function Tests

Serum levels of AST and ALT were determined by colorimetric method (Reitman and Frankel) by following the protocol of the commercially available *Randox* kits supplied by Randox Laboratories Ltd. (UK). Serum total protein levels was measured by spectrophotometry according to the colorimetric method protocol of the commercial *BioSystems* kit supplied by Biosystems SA, Barcelona, Spain. In addition, the colorimetric determination of serum ALP activity was carried out according to the protocol of the commercially available *bioMerieux* kit supplied by bioMerieux SA Company, Marcy-l'Etoile. France.

Statistical analysis

Data analysis was performed by Independent-Samples Student *t* test and Pearson's correlation analysis for assessment of mean differences between smokers and non-smokers control groups using the SPSS statistical software, version 20.0.

RESULTS AND DISCUSSION

The collective results for quantitative analysis of the liver function tests (AST, ALT, total protein and ALP) among two study groups are summarized in Table 1. These results indicated that the mean serum levels of AST, ALT, total protein and ALP (13.21 IU/L, 7.62 IU/L, 17.46 g/L and 13.26 IU/L) measured in smokers group were lower when compared to that of the control group (19.55 IU/L, 17.53 IU/L, 75.41 g/L and 59.61 IU/L). This decrease was found to be statistically highly significant ($p<0.001$). The relationship between all parameters included in this work was tested using Pearson's correlation analysis. Data analysis revealed a positive correlation between ALT and ALP levels only, while no association was found between the other study parameters, see Table 2. We found that the liver enzymes levels in serum of smoker patients differ significantly compared with their levels in non-smoker controls. İn fact, ALT and AST have important roles in amino acid metabolism. It catalyzes interconversion of L-alanine and α-ketoglutarate to pyruvate and L-glutamate. While AST catalyzes reversible transamination reaction of aspartate and α-ketoglutarate to oxaloacetate and glutamate. ALP is a hydrolytic enzyme that catalyzes removing of phosphate groups from nucleotides, proteins and alkaloids[26]. Cigarette smoke contains a wide range of hazard substances that can harm every part of the body either

directly nor indirectly[10]. It has been proved that smokers are more likely to be under risk of a lot of deadly diseases such as cardiovascular diseases, respiratory disorders, lung cancer and pancreas cancer [27]. Indirectly, smoking induces large adverse effects on the liver, however these toxic effects still need to clarify very well. Therefore, and in order to clarify the association between smoking and its effect on the liver, this work is studied the serum levels of AST, ALT, total protein and ALP in smokers and non-smoker persons. The results of the current work showed a significant decrease in the serum activity of AST, ALT, total protein and ALP in cigarette smokers when compared to non-smokers control group. It has been reported previously that there are significant increases in the levels of aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase activities in smokers compared to non-smokers [28]. These results are inconsistent with the results of the current work. Although cigarette smoke has no direct contact with the liver, however, the indirect effect of certain chemicals in cigarettes may occur over time. Where, these chemicals in cigarette smoke may prevent the liver from functioning normally. Over time, the liver is become less efficient in detoxifying the body. The results obtained in this study give an impression of dysfunction in liver functions led to a decrease in the ALP, ALT and AST enzymes levels. Or perhaps due to an inhibition of enzyme activity as a result of the presence of nicotine in tobacco smoke. There is an imperious requirement to increase research and investigate the reasons for this decline in enzymes activity. The results obtained in this study on total protein are consistent well with the data in the previous study [28], which found that the serum level of total protein in smokers was lower than the levels of healthy control group (non-smokers). Cigarette smoking is associated with increased excessive production of free radicals leading to oxidative stress [29]. Albumin, bigger part of total protein, has antioxidant properties [30]. The lowe level in serum total protein revealed in this study may be due to the decrease in albumin level that involves in the antioxidant defense since the smokers are under higher oxidative stress. Furtheremore, the decline in protein levels may be due to increased proteolytic activity or reduce protein synthesis as a result of exposure to cigarette smoke.

www.IndianJournals.com
Members Copy, Not for Commercial Sale
Downloaded From IP - 98.82.120.188 on dated 15-Sep-2024

Table 1: Serum levels of the liver function tests (AST, ALT, total protein and ALP) in smokers and control groups.

Element	Control group N= 50				Smokers group N= 50				
	Mean	SD	Upper limit	Lower limit	Mean	SD	Upper limit	Lower limit	*p* value
AST (IU/L)	19.55	4.95	21.93	17.16	13.21	2.81	14.57	11.85	< 0.001
ALT (IU/L)	17.53	2.11	18.54	16.52	7.62	1.62	8.41	6.84	< 0.001
Total Protein (g/L)	75.41	13.06	80.71	68.11	17.46	5.39	19.06	13.86	< 0.001
ALP (IU/L)	59.61	10.08	63.47	53.75	13.26	3.59	13.98	10.53	< 0.001

Table 2. Correlations between variables in patients group (r value).

Parameter	AST	ALT	Total Protein	ALP
AST	1	0.127	-0.105	0.023
ALT	0.127	1	-0.049	0.748**
Total Protein	-0.105	-0.049	1	-0.182
ALP	0.023	0.748**	-0.182	1

CONCLUSION

Data obtained in this work indicate that exposure to cigarette smoke leads to an indirect disturbance in some parameters (e.g. enzymes and total protein) in the liver. Alterations in the concentrations of these parameters may be related to the oxidative stress resulting from the accumulative effect of inhaled smoke as well as the interaction of absorbed toxic chemicals like nicotine. AST, ALT and ALP enzymes and total protein are significantly lower in smoker patients compared to non-smoker controls. According to the results of this study, we can conclude that smokers are more likely to have liver problems than non-smokers. Therfore, the association between smoking habits and liver functions must be carefully analyzed. Further works to understand the mechanisms of these associations are needed to clarify the harmful effects of smoking on the liver. In fact, smoking habit in Iraq among young people is growing strongly. Therefore, smoking in Iraq must be regulated by strict law, as in developed countries. Smoking habits that have spread widely in primary and secondary schools, public transportation, health institutions and cafes should be banned.

Financial Disclosure: There is no financial disclosure.

Conflict of Interest: None to declare.

Ethical Clearance: All experimental protocols were approved under the Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq and all experiments were carried out in accordance with approved guidelines.

www.IndianJournals.com
Members Copy, Not for Commercial Sale
Downloaded From IP - 98.82.120.188 on dated 15-Sep-2024

REFERENCES

1. Turner JA, Sillett RW, McNicol MW. Effect of cigar smoking on carboxyhaemoglobin and plasma nicotine concentrations in primary pipe and cigar smokers and ex-cigarette smokers. British Medical Journal. 1977; 2(6099): 1387–1389.
2. Doll R, Peto R, Boreham J, Sutherland I. Mortality from cancer in relation to smoking: 50 years observations on British doctors. Br J Cancer. 2005; 92(3): 426-429.
3. Thun MJ, Day-Lally CA, Calle EE, Flanders WD and Heath CW. Excess mortality among cigarette smokers: changes in a 20-year interval. American Journal of *Public Health*. 1995; 85 (9): 1223–1230.
4. Centers for Disease Control and Prevention. Annual smoking-attributable mortality, years of potential life lost, and economic costs--United States, 1995-1999. MMWR Morb. Mortal. Wkly. Rep. 2002; 51(14): 300–3033.
5. West R, Shiffman S. Fast Facts: Smoking Cessation. Health Press Ltd. 2007; 28.
6. Nyboe J, Jensen G, Appleyard M, Schnohr P. Risk factors for acute myocardial infarction in Copenhagen. I: Hereditary, educational and socioeconomic factors. Copenhagen City Heart Study. Eur Heart J. 1989; 10(10): 910–916.
7. Al-Fartusie FS, Hafudh A, Mustafa N. Levels of Some Trace Elements in Sera of Patients with Lung Cancer and in Smokers. Indian journal of advances in chemical science. 2017; 5(4): 344-352.
8. Devereux G. ABC of chronic obstructive pulmonary disease. Definition, epidemiology, and risk factors. BMJ. 2006; 332(7550): 1142–1144.
9. Braun JM, Kahn RS, Froehlich T, Auinger P. Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. Environ. Health Perspect. 2006; 114 (12): 1904–1909.
10. Rubin H. Synergistic mechanisms in carcinogenesis by polycyclic aromatic hydrocarbons and by tobacco smoke: a bio-historical perspective with updates, Carcinogenesis. 2001; 22: 1903-1930.
11. U.S. Department of Health and Human Services, (2014). The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General, Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. 2014.
12. National Toxicology Program. Report on Carcinogens. Thirteenth Edition. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program. 2014.
13. Yu MW, Hsu FC, Sheen IS, Chu CM. Prospective study of hepatocellular carcinoma and liver cirrhosis in as-ymptomatic chronic hepatitis B virus carriers. Am J Epidemiol. 1997; 145: 1039-47.
14. Rahmioglu N, Andrew T, Cherkas L, Surdulescu G.

Epidemiology and genetic epidemiology of the liver function test proteins. PLoS One. 4:e4435.

15. Sharpe PC. Biochemical detection and monitoring of alcohol abuse and abstinence. Ann Clin Biochem. 2001; 38:652–664.
16. Nathwani RA, Pais S, Reynolds TB, Kaplowitz N. Serum alanine aminotransferase in skeletal muscle diseases. Hepatology. 2005; 41: 380–382.
17. Kusunoki J, Kanatani A, Moller DE. Modulation of fatty acid metabolism as a potential approach to the treatment of obesity and the metabolic syndrome. Endocrine. 2006; 29: 91–100.
18. Baillie M. Alcohol and the liver. Gut. 1971; 12: 222–229.
19. Mantena SK, King AL, Andringa KK, Eccleston HB. Mitochondrial dysfunction and oxidative stress in the pathogenesis of alcohol- and obesity-induced fatty liver diseases. Free Radic Biol Med. 2008; 44: 1259–1272.
20. Forgione A, Miele L, Cefalo C, Gasbarrini G, Grieco A. Alcoholic and nonalcoholic forms of fatty liver disease. Minerva Gastroenterol Dietol. 2007; 53: 83–100.
21. Yeh MM, Brunt EM. Pathology of nonalcoholic fatty liver disease. Am J Clin Pathol. 2007; 128: 837-847.
22. Sharpe PC. Biochemical detection and monitoring of alcohol abuse and abstinence. Ann Clin Biochem. 2001; 38: 652–664.
23. Pessione F, Ramond MJ, Njapoum C, Duchatelle V, Degott C, Erlinger S, Rueff B, Valla DC, Degos F. Cigarette smoking and hepatic lesions in patients with chronic hepatitis C. Hepatology. 2001; 34: 121-5.
24. Gordon T. Factors associated with serum alkaline phosphatase level. Arch Pathol Lab Med. 1993; 117: 187–190.
25. Jang ES1, Jeong SH, Hwang SH, Kim HY, Ahn SY, Lee J, Lee SH, Park YS, Hwang JH, Kim JW, Kim N and Lee DH. Effects of coffee, smoking, and alcohol on liver function tests: a comprehensive cross-sectional study. BMC Gastroenterology. 2012; 12: 145.
26. Berg JM, Tymoczko JL, Stryer L. Biochemistry (6th ed.). New York: W.H. Freeman. 2007.
27. Sherman CB. Health effects of cigarette smoking. Clin Chest Med. 1991; 12: 643-658.
28. Sangar N, Bakhtiar M. Effect of cigarette smoking on liver function test and some other related parameters. Zanco J. Med. Sci. 2013; 17(3): 565-562.
29. El-Zayadi AR. Heavy smoking and liver. World J Gastroenterol. 2006; 12(38): 6098-6101.
30. Hu ML, Louie S, Cross CE, Motchnik P, Halliwell B. Antioxidant protection against hypochlorous acid in human plasma. J. Lab. Clin. Med. 1993; 121(2): 257-262.

www.IndianJournals.com
Members Copy, Not for Commercial Sale
Downloaded From IP - 98.82.120.188 on dated 15-Sep-2024