Scholars International Journal of Biochemistry

Abbreviated Key Title: Sch Int J Biochem ISSN 2616-8650 (Print) | ISSN 2617-3476 (Online) Scholars Middle East Publishers, Dubai, United Arab Emirates Journal homepage: <u>https://saudijournals.com</u>

**Original Research Article** 

# Assessment of Biochemical Parameters in Sudanese Male Cigarette Smoker in Khartoum State in Omdurman

Mohammed Abdalsalam Ahmed Ali<sup>1\*</sup>, Salman Taha Ahmed Elmukashfi<sup>1</sup>, Gad Allah Osman H. Modawe<sup>2</sup>

<sup>1</sup>Department of Clinical Chemistry, Faculty of Medical Laboratory Science, University of Dongola, Al Dabbah, Sudan <sup>2</sup>Department of Biochemistry, Faculty of Medicine, Omdurman Islamic university, Khartoum, Sudan

DOI: 10.36348/sijb.2023.v06i02.001

| Received: 06.01.2023 | Accepted: 13.02.2023 | Published: 17.02.2023

\*Corresponding author: Mohammed Abdelsalam Ahmed Ali Department of Clinical Chemistry, Faculty of Medical Laboratory Science, University of Dongola, Al Dabbah, Sudan

#### Abstract

Background: Hundreds of thousands around the world die from a disease caused by smoking cigarettes. Cigarette smoking (CS) is considered a worldwide major cause of preventable morbidity and mortality. The main clinical consequences of prolonged exposure to CS are chronic respiratory diseases, increased incidence of a variety of cancers, cigarette smoking had a dangerous effect on the essential biochemical mechanisms on the human body. Objective: This study aimed to assess the liver function in Sudanese male cigarettes smoker to identify the influence of cigarettes smoking on the level of their parameters. *Material and methods*: The study was designed as case control and include 81 samples, which is divided into case (51 samples) and control group (30 samples). The data collected by the use of questionnaire and blood specimens, and the levels of the parameters is measured by A15 automation spectrophotometer. Then the collected data is analyzed by the use of SPSS. *Results*: The results of the study showed statistically significant increase in total bilirubin and liver enzymes in case group when compared with control group. The mean of plasma total bilirubin, GOT (AST), GPT (ALT), and ALP levels in case group is (0.663, 26.49, 18.82 and 159.27) and in control group is (0.543, 23.00, 14.30 and 146.63) with p. value (0.001, 0.041, 0.004 and 0.000) respectively. And showed statistically insignificant decrease in total protein and albumin in case group when compared with control group, the means of total protein and albumin in case group is (66.47 and 33.73) and in control group is (68.30 and 34.43) with p. value (0.322 and 0.403) respectively. Conclusion: The study concluded that there was significant increase in liver enzymes (GOT, GPT, and ALP) and total bilirubin in cigarette smoking. And significant decrease in total protein and albumin, in cigarette smoking when compared to nonsmoking.

Keywords: Male Cigarette Smoking, Liver Enzymes, Total Bilirubin, Total Protein, Albumin, Sudanese.

Copyright © 2023 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

### **1. INTRODUCTION**

Cigarette smoking (CS) is considered a worldwide major cause of preventable morbidity and mortality [1]. The main clinical consequences of prolonged exposure to CS are chronic respiratory diseases, increased incidence of a variety of cancers, and increased risk of atherothrombotic clinical events such as myocardial infarction [2]. Hepatologists have traditionally paid scant attention to the deleterious effects of CS. This reflects the fact that smoking does not appear to cause liver injury and therefore is not considered a causative agent for chronic liver diseases [3]. However, there is increasing evidence that CS may negatively impact the incidence, severity, and clinical course of many types of chronic liver diseases [4, 5]. In fact, epidemiologic studies suggest that CS could accelerate the progression of a variety of liver diseases such as hepatitis CS [6, 7] and primary biliary cirrhosis [8, 9] and could represent a risk factor for hepatocellular carcinoma [10, 11]. It is unknown whether CS also influences the severity of nonalcoholic fatty liver disease (NAFLD), the main cause of chronic liver injury in Western countries. Because the prevalence of CS is increased in obese people, who are at a risk of developing NAFLD, it is likely that CS may affect the clinical course of this entity. Nonalcoholic steatohepatitis (NASH) is a severe form of NAFLD characterized by inflammatory infiltrate and hepatocellular damage, with or without fibrosis. In a minority of patients this condition progresses to cirrhosis and end stage liver disease [12]. The objective

**Citation:** Mohammed Abdalsalam Ahmed Ali, Salman Taha Ahmed Elmukashfi, Gad Allah Osman H. Modawe (2023). Assessment of Biochemical Parameters in Sudanese Male Cigarette Smoker in Khartoum State in Omdurman. *Sch Int J Biochem*, *6*(2): 13-16.

of this study was to investigate the liver function in Sudanese cigarettes smoker.

## 2. MATERIALS AND METHODS

**2.1 Study design:** This study was designed as case control study.

**2.2 Study area:** This study is carried out in Khartoum state, in Omdurman.

**2.3 Study period:** The study was carried out during the period from March to May 2012.

**2.4 Study population:** Male cigarette smoker, especially heavy smoker.

**2.5 Data collection:** By questionnaire including data concerning the aim of study (such as smoker history, hypertension, and number of cigarettes per day), and blood specimen for measurement the parameters.

**2.6 Sample size:** The study included 81 samples, 51 samples collected from cigarette smoker people (case group) and 30 samples from nonsmoker people (control group).

**2.7 Inclusion criteria:** Male with heavy cigarette smoking only (case group), and healthy nonsmoker people (control group).

**2.8 Exclusion criteria:** Male with heavy cigarette smoking and suffering from disease or condition can affect the results, like hepatitis and alcoholic consumption.

**2.9 Blood specimen collection:** Before collection, a local antiseptic (70% alcohol) was used to clean the skin, venous blood (about 3 ml) was taken from each participant (case and control group), using standard venipuncture technique. Plasma specimens were collected as heparinized container after centrifugation at 3000 rpm for 5 minutes. The specimen stored at breezed until analysis.

**2.10 Instrument:** By A15 automation spectrophotometer.

**2.11 Method:** The estimation of liver function testes was carried by A15 automation spectrophotometer device using analytical method for A15 automation spectrophotometer that principally based on the technique makes use of absorption spectrometry to assess the concentration of an analyte in a sample. It requires standards with known analyte content to establish the relation between the measured absorbance

and the analyte concentration and relies therefore on Beer's-Lambert law. In short, the electrons of the atoms in the atomizer can be promoted to higher orbital's (excited state) for a short period of time (nanoseconds) by absorbing a defined quantity of energy (radiation of a given wavelength). This amount of energy, i.e., wavelength, is specific to a particular electron transition in a particular element. In general, each wavelength corresponds to only one element, and the width of an absorption line is only of the order of a few picometers (pm), which gives the technique its elemental selectivity. The radiation flux without a sample and with a sample in the atomizer is measured using a detector, and the ratio between the two values (the absorbance) is converted to analyte concentration or mass using Beer's-Lambert law.

**2.12** Advantages of A15 automation spectrophotometer: The advantage of A15 automation spectrophotometer include: quick, proper, low sample throughput, and high precision.

**2.13 Statistical analysis:** Statistical analysis were performed using statistical package for social sciences (SPSS). Statistical significance and differences from control and test values were evaluated by student T.test, at which P. value is less than 0.05 (p. value  $\leq 0.05$ ).

# **3. RESULTS**

This study include 81 samples, which is divided into case (51 samples with 63%) and control group (30 samples with 37%), to identify the effect of cigarettes smoking on liver functions. The data collected by the use of questionnaire and blood specimens, and the levels of the parameters is measured by A15 automation spectrophotometer. Then the collected data is analyzed by the use of SPSS.

The results of the study showed statistically significant increase in the plasma levels of total bilirubin, GOT, GPT and ALP in case group when compared with control group, the means of total bilirubin, GOT, GPT and ALP in case group is (0.663, 26.49, 18.82 and 159.27) and in control group is (0.543, 23.00, 14.30 and 146.63) with p. value (0.001, 0.041, 0.004 and 0.000) respectively.

The study showed statistically insignificant decrease in total protein and albumin in case group when compared with control group, the means of total protein and albumin in case group is (66.47 and 33.73) and in control group is (68.30 and 34.43) with p. value (0.322 and 0.403) respectively.

Parameters	Smoker (N=51)	Nonsmokers (N=30)	p. value
Total protein	66.47±9.100	68.30±5.522	0.322
Albumin	33.73±4.070	$34.43 \pm 2.800$	0.403
Total bilirubin	0.663±.1442	0.543±.1633	*0.001
GOT	26.49±8.427	23.00±4.712	*0.041
GPT	18.82±8.890	14.30±4.684	*0.004
ALP	159.27±13.217	146.63±8.672	*0.000
* Significant			

 Table 3.1: Show the Mean ± SD of parameters in smoker and nonsmoker

## 4. DISCUSSION

In this study we found statistically a significant increase in concentration of liver enzymes (GOT (AST), GPT (ALT) and ALP) and total bilirubin and insignificant decrease in total protein and albumin, in cigarette smoking when compared to nonsmoker individuals. In a study conducted with hamsters, (Dontenwill, W, *et al.*, 1974) [13]. Stated that long-term exposure to cigarette smoke did not affect serum enzyme activities and concentrations of serum total protein. In contrast, an elevation in serum AST, g-GPT, total bilirubin values in rats due to smoke have recently been reported (Watanabe, K, *et al.*, 1995) [14]. In addition, (Chan-Yeung, M, *et al.*, 1981) [15] suggested that smoking was associated with lower total bilirubin, AST, total protein values in human serum.

In this study we suggests that cigarettes smoking mode exerted significant effects primarily on the liver enzyme activities and on some other organs and systems, as well. Thus our study did not confirm the findings of (Dontenwill, W, *et al.*, 1974) [13], but had some similarities with results from rats (Watanabe, K, *et al.*, 1995 and Chan-Yeung, M, *et al.*, 1981) [14, 15].

Therefore, these results support the well accepted views related to the multidirectional effects of cigarettes on certain organs and systems (Sullivan, F. M., 1993, Oduola, T, *et al.*, 2005, Seitz, H. K, *et al.*, 1992, Mützell, S., 1988 & Bassi, J. A, *et al.*, 1984) [16-20].

In this study, the effects of smoking on serum bilirubin concentrations were studied and serum bilirubin was found to be higher in individuals who smoke than in those who do not. These studies confirm the in vitro studies showing that cigarette smoke decreases serum bilirubin concentrations (Frei, B, et al., 1991 & Frei, B, 1994) [21, 22]. Further studies will have to be performed to determine if other antioxidants are also decreased with cigarette smoking, if serum bilirubin is an effective measure of oxidative stress and if it can be used to monitor antioxidant therapy. Chronic smoking may lead to a deficiency of both bilirubin and other antioxidants and these deficiencies could lead to oxidant injury, higher concentrations of oxidized LDL, increased plaque formation and increases in DNA oxidation productions.

In this study, we shown that serum total bilirubin concentrations are inversely related to cigarette smoking. We have previously found that serum bilirubin is decreased in individuals with coronary artery disease (Schwertner, H. A., *et al.*, 1994) [23]. These findings have been recently confirmed in studies of subjects with early familial coronary artery disease (Hopkins, P. N., *et al.*, 1996) [24].

# **5. CONCLUSION**

The study concluded that there was significant increase in liver enzymes (GOT, GPT, and ALP) and total bilirubin in cigarette smoking. And significant decrease in total protein and albumin, in cigarette smoking when compared to nonsmoking.

**Competing Interests:** Authors have declared that no competing interests exist.

#### REFERENCE

- He, J., Gu, D., Wu, X., Reynolds, K., Duan, X., Yao, C., Wang, J., Chen, C. S., Chen, J., Wildman, R. P., & Klag, M. J., (2005). Major causes of death among men and women in China. New England journal of medicine, 353(11), 1124-1134.
- Pham, T. M., Fujino, Y., Ide, R., Shirane, K., Tokui, N., Kubo, T., Mizoue, T., Ogimoto, I., & Yoshimura, T. (2007). Mortality attributable to cigarette smoking in a cohort study in Japan. *European journal of epidemiology*, 22, 599-605.
- Whitehead, T. P., Robinson, D., & Allaway, S. L. (1996). The effects of cigarette smoking and alcohol consumption on serum liver enzyme activities: a dose-related study in men. *Annals of clinical biochemistry*, 33(6), 530-535.
- Michael, L. B., Edward, P. F., & Larry, E. S. (2005). Clinical chemistry: principles, procedures, correlations.
- Bataller, R. (2006). Time to ban smoking in patients with chronic liver diseases. *Hepatology*, 44(6), 1394-1396.
- Pessione, F., Ramond, M. J., Njapoum, C., Duchatelle, V., Degott, C., Erlinger, S., Rueff, B., Valla, D. C., & Degos, F. (2001). Cigarette smoking and hepatic lesions in patients with chronic hepatitis C. *Hepatology*, 34(1), 121-125.
- Hezode, C., Lonjon, I., Roudot-Thoraval, F., Mavier, J. P., Pawlotsky, J. M., Zafrani, E. S., &

Dhumeaux, D. (2003). Impact of smoking on histological liver lesions in chronic hepatitis C. Gut, 52(1), 126-129.

- Gershwin, M. E., Selmi, C., Worman, H. J., Gold, E. B., Watnik, M., Utts, J., Lindor, K. D., Kaplan, M. M., Vierling, J. M., & USA PBC Epidemiology Group. (2005). Risk factors and comorbidities in primary biliary cirrhosis: a controlled interviewbased study of 1032 patients. *Hepatology*, 42(5), 1194-1202.
- Zein, C. O., Beatty, K., Post, A. B., Logan, L., Debanne, S., & McCullough, A. J. (2006). Smoking and increased severity of hepatic fibrosis in primary biliary cirrhosis: a cross validated retrospective assessment. *Hepatology*, 44(6), 1564-1571.
- Marrero, J. A., Fontana, R. J., Fu, S., Conjeevaram, H. S., Su, G. L., & Lok, A. S. (2005). Alcohol, tobacco and obesity are synergistic risk factors for hepatocellular carcinoma. *Journal of hepatology*, 42(2), 218-224.
- Fujita, Y., Shibata, A., Ogimoto, I., Kurozawa, Y., Nose, T., Yoshimura, T., Suzuki, H., Iwai, N., Sakata, R., Ichikawa, S., & Tamakoshi, A. (2006). The effect of interaction between hepatitis C virus and cigarette smoking on the risk of hepatocellular carcinoma. *British journal of cancer*, 94(5), 737-739.
- Wild, S. H., & Byrne, C. D. (2006). Risk factors for diabetes and coronary heart disease. *Bmj*, 333(7576), 1009-1011.
- Dontenwill, W., Chevalier, H. J., Harke, H. P., Lafrenz, U., Reckzeh, G., & Leuschner, F. (1974). Biochemical and haematological investigations in syrian golden hamsters after cigarette smoke inhalation. *Laboratory animals*, 8(2), 217-235.
- Watanabe, K., Eto, K., Furuno, K., Mori, T., Kawasaki, H., & Gomita, Y. (1995). Effect of cigarette smoke on lipid peroxidation and liver function tests in rats. *Acta Medica Okayama*, 49(5), 271-274.
- Chan-Yeung, M., Ferreira, P., Frohlich, J., Schulzer, M., & Tan, F. (1981). The effects of age, smoking, and alcohol on routine laboratory tests.

*American journal of clinical pathology*, 75(3), 320-326.

- Sullivan, F. M. (1993). Impact of the environment on reproduction from conception to parturition. *Environmental Health Perspectives*, 101(suppl 2), pp.13-18.
- Oduola, T., Adeosun, O. G., Aduola, T. A., Agbaje, N. R., & Raheem, Z. A. (2005). Drinking patterns: biochemical and haematological findings in alcohol consumers in Ile-Ife, Nigeria. *African Journal of Biotechnology*, 4(11).
- Seitz, H. K., Xu, Y., Simanowski, U. A., & Osswald, B. (1992). Effect of age and gender on in vivo ethanol elimination, hepatic alcohol dehydrogenase activity, and NAD+ availability in F344 rats. *Research in experimental medicine*, 192, pp.205-212.
- 19. Mützell, S. (1988). Cardiovascular and some biochemical effects of high alcohol consumption. *Upsala journal of medical sciences*, 93(3), 277-288.
- Bassi, J.A., Rosso, P., Moessinger, A. C., Blanc, W. A., & Stanley James, L. (1984). Fetal growth retardation due to maternal tobacco smoke exposure in the rat. *Pediatric research*, 18(2), 127-130.
- 21. Frei, B., Forte, T. M., Ames, B. N., & Cross, C. E. (1991). Gas phase oxidants of cigarette smoke induce lipid peroxidation and changes in lipoprotein properties in human blood plasma. *Protective effects of ascorbic acid. Biochemical Journal*, 277(1), 133-138.
- 22. Frei, B. (1994). Reactive oxygen species and antioxidant vitamins: mechanisms of action. *The American journal of medicine*, 97(3), S5-S13.
- Schwertner, H. A., Jackson, W. G., & Tolan, G. (1994). Association of low serum concentration of bilirubin with increased risk of coronary artery disease. *Clinical chemistry*, 40(1), 18-23.
- Hopkins, P. N., Wu, L. L., Hunt, S. C., James, B. C., Vincent, G. M., & Williams, R. R. (1996). Higher serum bilirubin is associated with decreased risk for early familial coronary artery disease. *Arteriosclerosis, thrombosis, and vascular biology*, 16(2), 250-255.