ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 01 | January - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

"STUDY TO COMPARE THE EFFECT OF CIGARETTE SMOKING ON LIVER FUNCTION BETWEEN SMOKER NON-SMOKER MALES IN DEHRADUN CITY, UTTARAKHAND".

Clinical Biochemistry

Jyoti Sharma PhD scholar, Department of biochemistry, Ras Bihari Bose subharti University Dehradun Uttarakhand

KEYWORDS

INTRODUCTION

One of the main causes of avoidable morbidity and mortality is cigarette smoking [1]. Nowadays, smoking-related deaths from cigarettes claim the lives of almost 3 million people annually worldwide [2]. Approximately 22% of cancer deaths worldwide are caused by smoking tobacco, making it the primary risk factor for cancer and the largest cause of preventable death in wealthy countries [3]. Despite the numerous health hazards associated with tobacco use, its usage is nonetheless very common. Tobacco usage has become widespread in today's society [4].

Numerous infections, cancers, heart conditions, and respirator issues have identified it as a potential cause of their emergence [5]. In clinical practice, liver function tests are helpful instruments for evaluating possible liver illnesses, tracking therapy responses, and estimating the prognosis of patients with liver diseases. Serum total protein, albumin, alkaline phosphatase (ALP), total bilirubin (TB), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) are the most often measured components of liver functions tests. Although albumin is a well-known negative acute phase protein with antioxidant and inflammatory marker qualities [6]., the underlying biochemical mechanisms of many smoking-related clinical diseases remain unclear. Thus, the purpose of this study was to look at how smoking affected other biochemical parameters, including total protein, and inflammatory markers. Nonetheless, thorough and cautious interpretation of the liver function test is necessary because these parameters.

MATERIAL AND METHOD

Hundred male non-smokers (control) and hundred male Dehradun smokers (test) who were between the ages of 35 and 55 participated in the study. The Ras Bihari Bose Subharti University Dehradun ethical review committee gave its approval to the research undertaking. For statistical comparison, two groups (smokers and non-smokers) were gathered with similar age ranges. For at least 15 years, the smokers consistently smoked 12 cigarettes a day. The smokers were gathered in the city of Dehradun between February 2023 and October 2023. The enrolled patients had no significant medical conditions, no drug use history, and no blood transfusions or receipts within the previous six months. Personal interviews were used to obtain clinical data, medical histories, and other pertinent information from the individuals.

Determination of liver function test

The Development of Liver Function Exams Each participant had a venipuncture to extract 5 ml of whole blood, which was then put in a heparinized tube and centrifuged for 15 minutes at 3000 rpm. All subjects had their plasma levels of total bilirubin, albumin, triglycerides, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total cholesterol, and triglycerides measured using an automated analyzer (Beckman BUN analyzer, USA) using a kit approach. The study employed test kits that were commercially available and manufactured by Analytic on Biotechnologies, Germany. The manufacturer's instructions were closely followed. The difference between plasma total protein and albumin was used to calculate plasma globulin.

Determination of plasma malondialdehyde

The assessment of the lipid peroxidation process is achieved via determining the end product MDA. The level of serum MDA was determined spectrophotometrically with a thiobarbituric acid (TBA) solution. In brief, to150 µl serum sample the following were added: 1ml (17.5%) trichloroacetic acid (TCA) and 1ml of 0.66% TBA, mixed well by vortex, incubated in boiling water for 15 minutes, and then allowed to cool. One ml of 70% TCA was added and the mixture allowed to stand at room temperature for 20 minutes, centrifuged at 2000rpm for 15 minutes, the supernatant was taken out for spectrophotometer assay at 532nm [7].

Statistical analysis

All data are expressed as mean ± standard division (M±SD), and statistical analysis was carried out using statistically available software (SPSS) version 22.0. Unpaired t-test were applied to test the significance of variance ($p<0.05$) of the parameters under study between control and smokers groups.

RESULT AND DISCUSSION

Age of non-smokers was 45.97 ± 4.83 years. Subjects who did not smoke weighed 70.13 ± 13.16 kg, while those who did smoke weighed 68.72 ± 12.20 kg. The MDA level in the group of smokers increased significantly, with a mean value of the general features of the male smokers and non-smokers in Dehradun are presented in Table 1. The average age of male smokers was 47.11 ± 5.53 years, while the average f 3.756 µmol/l ± 0.113.had greater levels of plasma total cholesterol, triglycerides, ALT, AST, and ALP than did never-smokers and current smokers. On the other hand, compared to never-smokers, smokers had lower plasma levels of albumin, bilirubin, and total protein (Table 2). $P < 0.05$ indicated that each of these differences was statistically significant.

According to Table 2, our research showed that among healthy Dehradun men, smoking cigarettes is linked to significantly decreased ($P<0.05$) concentrations of plasma total protein, albumin, and bilirubin levels. Additionally, as demonstrated by the rise in the activities of plasma ALT, AST, and ALP in this study, the decrease in smokers' plasma protein may also be partially explained by the detrimental effects of toxic chemicals from cigarettes on liver cells (Table 2).Our findings showed that in healthy Dehradun men, smoking cigarettes is linked to reduced plasma bilirubin concentrations (Table 2).

Table 1: Average mean of Demographic characteristics in control and smokers.

Characteristics	Control Non-smoker (n=100)	smoker (n=100)
Age(years)	45.11 ± 5.53	47.97 ± 4.83
Weight(kg)	70.11 ± 13.16	66.12 ± 13.16

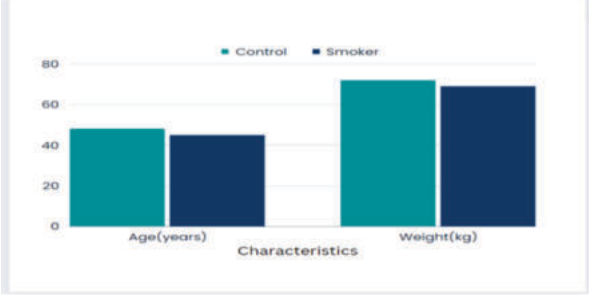

Figure1: Mean of demographic characteristics in control and smokers.

Table 2:Average mean of biochemical parameters in control and smoker.

Parameters	Control (n=30)	Smoker (n=30)
ALT	23.47±3.33	35.22±7.55
AST	24.65±2.22	37.01±8.66

ASP	144.0±9.80	245.55±66.75
Total protein (g/l)	7.71±0.35	5.02±1.2
Albumin	4.19±0.38	3.68±0.67
Globulin (g/l)	2.55±0.71	2.10±0.61
Triglycerides (mg/dl)	99±3.43	205±10.40
Cholesterol (mg/dl)	217±4.34	360±55.13
Total bilirubin (mg/dl)	0.67±0.7	0.40±0.05

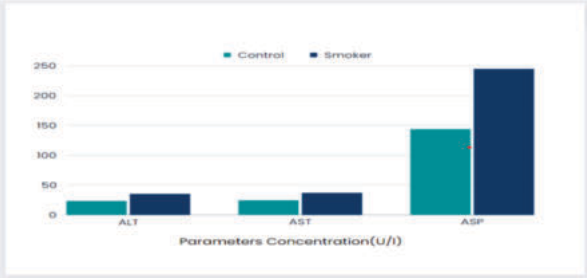

Figure 2: Graphical representation of average mean ALT, AST and ASP in control and smokers.

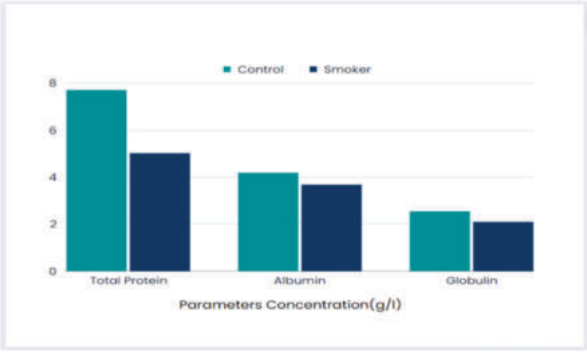

Figure 3: Average mean of total protein, albumin, globulin in control and smokers.

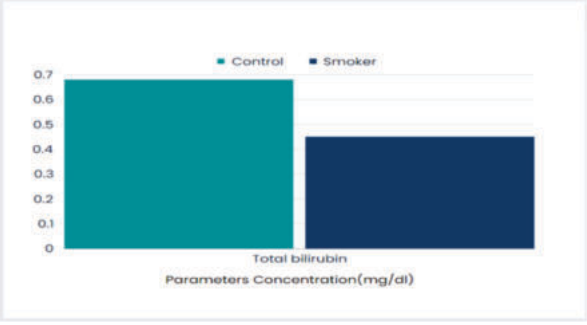

Figure 4: Average mean of triglycerides and cholesterol in control and smokers.

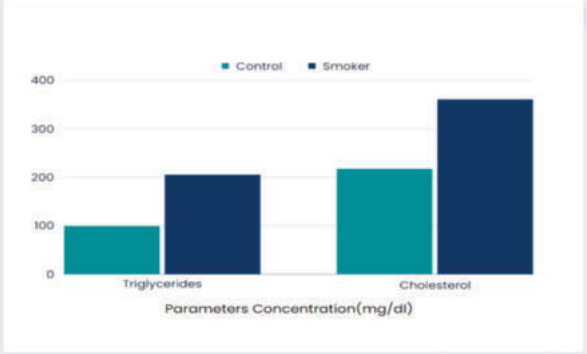

Figure:5 Average mean of total bilirubin in control smokers.

CONCLUSION

To sum up, this study demonstrates that smokers' plasma concentrations of MDA are higher than those of non-smokers. Elevated levels of AST, ALT, and ALP were linked to heavy smoking, along with decreased levels of albumin, globulin, and total protein. All smokers had considerably higher levels of triglycerides and cholesterol than non-smokers. It is important to carefully examine and assess the relationship between smoking habits and liver function tests. It is necessary to do more research on the mechanisms behind these relationships. In guys from Dehradun who appear to smoke, cigarette smoking lowers serum bilirubin. In most clinical laboratories, measuring bilirubin is cheap and simple. To find out if measuring bilirubin is a useful way to identify smoking-related free radicals that can lead to serious illnesses, more research is required.

As a result, our research indicates that smokers are more at risk than non-smokers. Our main goal is to prevent smokers from becoming habitual smokers by educating them about the negative consequences of smoking and shielding them from a variety of diseases.

REFERENCES

1. Funck C., Mathilde R., Michel L., Arnould J., Verstuyft C., Martine L., et al. Effects of type of smoking (pipe, cigars or cigarettes) on biological indices of tobacco exposure and toxicity. Lung Cancer. 2006;54: 11–8.
2. Aurelio, L.. Biochemical markers of cardiovascular damage from tobacco smoke. Curr. Pharm. Des. 2005;11: 2190–208.
3. Grassi, M. C., Baraldo, M., Chiamulera, C., Culasso, F., Raupach, T., Ferketich, A. K., Patrono, C. & Nencini, P. 2014. Knowledge about health effects of cigarette smoking and quitting among Italian university students: the importance of teaching nicotine dependence and treatment in the medical curriculum. BioMed Research International, (2014).
4. Benowitz, N. L., Porchet, H., Sheiner, L. & Jacob Iii, P. Nicotine absorption and cardiovascular effects with smokeless tobacco use: comparison with cigarettes and nicotine gum. Clinical Pharmacology & Therapeutics, (1988), vol 44, pp 23-28.
5. Mehta, H., Nazzal, K. & Sadikot, R. Cigarette smoking and innate immunity. Inflammation Research, vol 57, (2008), pp 497-503.
6. Bridges, B. A., Clemmension, J., Suginura, T. (1979). Cigarette smoking, does it carry a genetic risk. Mutation research/reviews in genetic toxicology 65(1): 71-81. [DOI]
7. Brown, K. M., Morrice, P. C., Duthie, G. G. (1994). Vitamin E supplementation suppresses indexes of lipid peroxidation and platelet counts in blood of smokers and non-smokers but plasma lipoprotein concentrations remain unchanged. Am. J. Clin. Nutr. 60(3): 383-387.