

Research Article

Assessment of Biochemical Markers of Hepatic Synthetic Ability and Anion Transport in Active Smokers

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Abstract: This research assessed the hepatic synthetic capability and anion transport in smokers. Thus, albumin and γ GT levels were determined. Determinations were carried out on two groups of sixty subjects each. They were: active and non smokers respectively. All samples were analyzed and the data generated were subjected to statistical analysis. γ GT was significantly higher while albumin was significantly lower. Smoking is therefore injurious to both the hepatocytes and cells of the biliary system. It could also be a contributing factor to the development of liver diseases.

Keywords: Smoking; Tobacco, Albumin, γ GT.

INTRODUCTION

Smoking is a social habit. Alharbi, 2012[1] described it as a kind of lifestyle factor that affects the health of humans which has been shown to be an important risk factor in a variety of disorders where it is involved in the pathogenetic pathways. The habit of tobacco smoking starts during the period of adolescence or early adulthood as teenagers are attracted more by their peers than by the adults [2]. There are numerous harmful substances found in tobacco and tobacco smoke. The tobacco plant contains over 2,200 compounds of which nitrogenous compounds comprise more than 30 per cent [3]. Nicotine is one of these substances that may be acquired through active and passive smoking[4]. Associated with nicotine exposure is the incidence of occupational influences, Passive smoking, nephrotoxicity and induced nephropathy [5]. Smoking is associated with an acute increase in arterial pressure due to systemic vasoconstriction, decreased skin and coronary blood flow, nicotine being associated with the cardiovascular effect [6]. In addition to nicotine and pyridine, other known toxic substances in tobacco are heavy metals (lead, cadmium) and tar [7].

Albumin and gamma glutamyltranspeptidase (γ GT) are parameters used in the assessment of both the liver's synthetic and biliary function respectively [8,9, 10]. Resting on the background Knowledge that smoking is dangerous to health, in the process inhibiting spontaneous processes, this research work is design to

verify and ascertain the effect of smoking on the synthetic and hepatic anion transport capabilities of the liver.

MATERIALS AND METHODS

Subjects

A total of 120 subjects participated in this study. They were randomly selected. The subjects consists of sixty(60) male tobacco smokers and sixty(60) non tobacco smokers who served as control for this study.

Study Area

The study area was Owo, Ondo state, Nigeria. The coordinates $7^{\circ}11'N$ $5305'E$ / $7.183^{\circ}N$ $5.5830E$. Owo is situated at the southern edge of the Yoruba hills, and at the intersection of roads from Akure, Kabba, Benin city, and siluko[11].

Active Smokers are those who smoke cigarette of various brands, each being a smoker of more than four sticks of cigarette per day.

Nonsmokers are those who have never smoked, neither presently smoke nor endure smoking in their vicinity.

Inclusion and exclusion criteria

The criteria of the selection of subjects (either smoking or non-smoking) was that no one should have any medical complication such as hypertension, heart

disease, stroke, diabetes or any other disorder. Hence all male subjects included in the present study are normal healthy subjects between the ages of 18 to 50 years.

Blood collection

Five milliliters (5mls) of each blood sample was collected by venous puncture of the cubital fossa from each subject, using 22G needle and syringes. The 5mls of whole blood was dispensed into lithium-heparinized bottles, mixed and centrifuged at 2000 rpm for 10 minutes. The supernatant plasma was then removed using automatic pipettes and placed into a plain bottle. The plasma samples were then refrigerated at 4°C until the samples were analyzed within 24hours for renal and hepatic profiles.

Analytes estimated

Gamma glutamyltranspeptidase (γGT) estimation was used in the assessment of hepatic anion transport while albumin was used to assess the hepatic synthetic function. All estimations were done in duplicates to ensure accuracy and precision.

Plasma Albumin determination

The colorimetric method as explained by Doumas *et al.* [12] was employed. The measurement of serum albumin is based on its quantitative binding to the indicator 3, 3',5, 5'-tetrabromo-m-cresol sulphonephthalein (bromocresol green, BCG). The albumin-BCG-complex absorbs maximally at 630nm, the absorbance being directly proportional to the concentration of albumin in the sample.

Determination of plasma activity of γGT

It was done using the method described by Szasz [13]. The substrate L-γ- glutamyl- 3-carboxy-4-nitronilide, in the presence of glycylglycine is converted by γ-GT in the sample to 5-amino-2-nitrobenzoate which can be measured at 405nm

Statistical analysis:

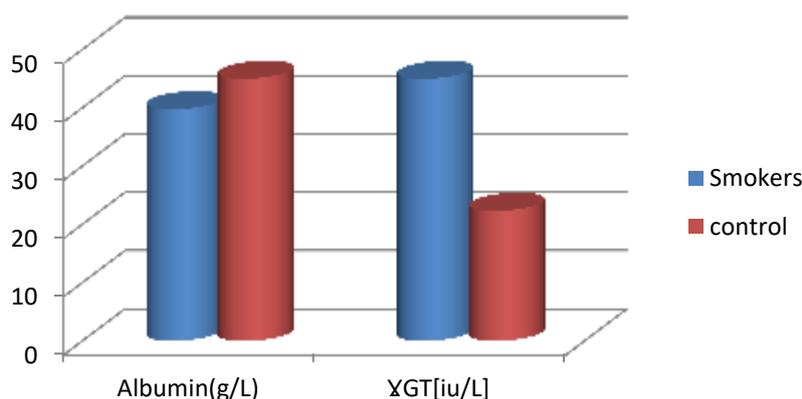
All values were expressed as Mean ± S.D. significance was tested using the t test and post test analysis. All values were found to be significant or otherwise at P<0.05.

RESULTS

Table 1:Plasma Renal and liver indices in active cigarette smokers and non smokers

Variables Mean±SD	Active cigarette smokers(n=60)	Non smokers(n=60)	t .value	P .value
Liver indices				
Plasma Albumin[mmol/L]	39.74 ± 4.24	44.98 ± 3.64	7.089	0.000**
Max.value	47.0	51.9		
Min .value	26.0	37.4		
Plasma γGT[iu/L]	46.41 ± 19.51	22.32 ± 8.23	9.531	0.000**
Max.value	119.3	43.70		
Min .value	11.10	11.60		

Key: ** = t test significant at P<0.0001



DISCUSSION

Albumin is a protein synthesized by the parenchymal cells of the liver and its concentration in blood is used to assess hepatic synthetic function [8, 14].The significant statistical difference seen when both groups were compared with non smokers brings a cause

for concern (table 1), though the variations are not pathologically significant it was not that smoking is not deleterious to the liver, it is just that the liver is an organ with a high functional reserve [15].

γGT is an enzyme used in the assessment of hepatic anion transport. Unlike the transaminases, it is not intracellular but membrane bound [8,9,10]. Its variation in activity, as seen in table 1, was significantly higher when both groups were compared one with the other. A rise in γGT whatever the cause may be indicates that the hepatobiliary system has been affected. Though the observed increase in its activity may not be solely from the biliary tract as it is also produced by some organs other than the liver, a significant portion may have been contributed by it, the rationale being that plasma albumin level is significantly affected. This finding agrees the works of Osifo *et al.* [16] and Whitehead *et al.* [17] where increase in plasma enzyme levels was associated with smoking. As γGT activity in plasma increases in any and all forms of liver disease, making it a very sensitive hepatic profile parameter [18], the damage that would have been done (as will be manifested by a low Prothrombin time or a low albumin level) due to chronic exposure to both active and second hand tobacco smoke, had the liver not been a regenerative and a high functional reserve organ, is better imagined than experienced. More recently, slightly elevated serum γGT have been correlated with cardiovascular and is under investigation as a cardiovascular risk marker. The rationale being that γGT accumulates in atherosclerotic plaques [19], suggesting a role in cardiovascular diseases [20]. Cigarette smokers are contaminated with heavy metals such as Lead and cadmium [5, 21]. Upon contamination with any of the heavy metals mentioned above, they are likely to replace Cu, Mn and Zn as the metal constituents of SOD [22]. When this happens the enzyme activity is diminished, therefore the level of the superoxide, which should have been scavenged increases, leading to, among other conditions, accelerated lipid peroxidation. This research work ventured into and discovered that smoking was associated with significant variations in markers of hepatic synthetic function and hepatic anion transport.

CONCLUSION

This research work goes a long way to corroborate the findings of others that smoking is a form of exposure harmful substances accumulation in the human system. This project work has also attested to the fact that tobacco smoke is also a means of exposure, to harmful substances, thereby capable of causing the same set of clinical features that pertains to the organ or system that has been affected. This research work ventured into and found out that both hepatic synthetic capability and hepatic anion transport is adversely affected by cigarette smokers.

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