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Effects of Cigarette Smoking and Alcohol Consumption on Hepatorenal Indices

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Abstract

Alcohol consumption and cigarette smoking aside has been documented to cause various harm to various organs, like the brain, heart, liver, and pancreas, it is also a known major risk factor for developing chronic diseases such as chronic obstructive pulmonary disease, cardiovascular disease and several malignancies. However, their effects on renal and hepatic functions remain unclear. The aim of this study was to evaluate the effect of cigarette smoking and alcohol consumption on hepatorenal indices such as liver enzymes [Alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine transaminase (ALT), gamma-glutamyltransferase (GGT)] and renal indices (Urea, Creatinine). 160 subjects within Ilorin metropolis comprising of 40 smokers, 40 alcoholics, 40 alcoholic smokers and 40 controls were recruited for this cross-sectional study. A structured questionnaire was used to obtain information on their clinical history, and anthropometric data after the consent form was signed. Liver enzymes (ALP, AST, ALT, GGT) and renal indices (Urea, Creatinine) were determined spectrophotometrically using commercially prepared kits. However, the values for glomerular filtration rate (eGFR) were calculated using

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Modification of Diet in Renal Disease (MDRD) study equations. A one-way ANOVA p-value of <0.05 was considered using SPSS 23.0 statistical package. ALP, ALT, AST, GGT, Urea and Creatinine were significantly elevated in smokers when compared with control ($p<0.05$), however, the eGFR level was reduced in smokers, alcoholics and alcoholic smokers when compared with controls ($p<0.05$).These findings shows that cigarette smoking and alcohol consumption have a deleterious effect on both renal and hepatic function indices, and suggest that they do these by promoting lipid peroxidation and thus induces oxidative stress.

Keywords: Cardiovascular diseases, cigarette smoking, alcohol consumption, liver enzymes, renal indices, oxidative stress

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Introduction

Alcohol has historically, and continues to hold an important role in social engagement and bonding for many, however, alcohol consumption, especially in excess has been tagged as a risk factor for diseases and health impacts; crime; road incidents; and dependence causing 2.8 million premature deaths per year (Hannah and Max, 2018). In the general adult population, cigarette smoking has been associated with elevated risk of incident chronic kidney disease (CKD) / end stage renal disease (CKD) (Xia *et al*., 2017).

Cumulatively, alcohol consumption and cigarette smoking aside has been documented to cause various harm to various organs, like the brain, heart, liver, and pancreas, it is also a known major risk factor for developing chronic diseases such as chronic obstructive pulmonary disease, cardiovascular disease and several malignancies. For a fact, we know that both alcohol consumption and cigarette smoking has been implicated in liver diseases (Hong *et al*., 2019).

Some of the hepatorenal biomarkers that may be affected by cigarette smoking and alcohol consumption include liver enzymes Alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine transaminase (ALT), gamma-glutamyl transferase (GGT)] and renal indices (Urea, Creatinine) (Fredrik *et al*., 2023). There are also limited research data on effects of consumption of alcohol and cigarette smoking on hepatorenal function especially in the light of understanding the mechanism involved. Understanding the mechanism involved in the successive effect of cigarette smoking and alcohol consumption on these organs liver and kidney allows for further risk stratification of patients who may be at an intermediate risk of disease and thus may encourage more aggressive therapy,

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(Rutledge and Asgharpour, 2020) but, there is still a paucity of data on the effect ofcigarette smoking and alcohol consumption on hepatorenal indices and relatively no study has been conducted on to evaluate the impact of these factors in Nigeria as of now.

Thus, we evaluate the effect of cigarette smoking and alcohol consumption on hepatorenal function indices which includes ALP, ALT, AST, GGT, Urea, Creatinine and eGFR.

Materials and Methods

This was a population based cross–sectional study carried out in Ilorin and surrounding cities within north central part of Nigeria. The survey was conducted from June 2022 to November 2022. The participants include adult males who were living in the location of the study. The following assumptions were used to calculate the sample size for the study subjects. The desired level of significance was 0.05 (5%), with a standard deviation of 1.96 at 95% confidence interval, and a prevalence rate of 8.5% obtained from the 2018 World Health Organization Global Status Report on Alcohol and Health (2018) (WHO, 2018). The sample size of 120 subjects was calculated using the formula of Fisher *et al.*, (1998). Hence, a total of one hundred and twenty (120) adult subjects with not less than five (5) years of cigarette smoking and alcohol drinking were recruited from Ilorin Metropolis, Kwara State, Nigeria containing Forty (40) adult subjects who smoke cigarette alone, Forty (40) adult subjects who drink alcohol alone, Forty (40) adult subjects who are alcoholic smokers. Forty (40) apparently healthy adult were also recruited from the same metropolitan area to serve as study control. The participants were

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informed about the study by trained field workers, who also lived in the same areas.

Only those who gave written consent were included in the study. The inclusion criteria included men within the age of 18 to 60 years who has either been smoking cigarette, consuming alcohol or doing both for a period of (5) years and above. Individuals with records of diabetes, hypertension, renal and hepatic risk factor or diseases were excluded. Patients with medications which may affect inflammatory biomarkers as well as individuals who do not consent and women were also excluded. The controls were never smokers and must not have been living with a smoker to prevent secondary smoking. The controls were also not taking alcohol and had no history of cardiovascular disease (CVD), diabetes mellitus, and other systemic and metabolic diseases. They also had no history of substance abuse, and no history of the use of the following medications: corticosteroids, nonsteroidalanti inflammatory medication; aspirin, statins, and testosterones replacement.

Information on demographic factors, anthropometric details and lifestyle factors of each study participants was obtained through an accurately structured questionnaire. Other information such as height, weight, smoking habits, alcoholism and other addictions was enquired for and noted.

Clinical examination of the participants that included vital signs was done by a medical practitioner to rule out unstable clinical state. Blood Pressure was measured using Omron Intellisense Automatic Upper Arm Blood Pressure Monitor and the Body Mass Index was calculated from the expression; BMI=Weight (kg)/Height (m^2).

The blood sample from each of the participant was collected after an overnight fast including a light, fat free diet before the day of collection. The fasting blood sample

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was withdrawn from the study participants during the day. The venous puncture was performed on the cubital fossa with tourniquet applied on the upper arm for easy access to the vein. 5.0ml of blood sample was collected following aseptic procedure from each study participant and dispensed into Lithium heparin bottles.

The blood collected was kept after centrifuged at 3,000 r.p.m for 5 minutes. The plasma was separated and refrigerated at -20°C. Analysis of biochemical parameters was thereafter done in the laboratory. The analysis of biochemical parameters was done in 2 weeks. ALP, ALT, AST, GGT, Urea and Creatinine were determined spectrophotometrically using commercially prepared reagent kits (Agappe diagnostics, Switzerland).

Estimated glomerular filtration rate, eGFR, was calculated using Modification of Diet in Renal Disease (MDRD) study equation by Levey*et al.,* (2007) - GFR(ml/min/1.73m²) = 175 x $(Scr)^{-1.154}$ x $(age)^{-0.203}$ x (0.742 if female) x (1.212 if African) (Levey *et al*., 2007).

The data obtained were analyzed using the SPSS statistical package version 23.0 (SPPS Inc., Chicago, IL, USA) after analyzing the data obtained at 0.05 p-values and 95% confidence interval. The analysis of variance (One-way ANOVA) was used to investigate significance among the four groups considered. Values were expressed as values ± standard error of mean.

The permission to conduct research was obtained from the Ethics Committee of Kwara State Ministry of Health before the commencement of the study.

Results

A total of 160 adult males participated in the study including forty smokers (40), forty alcoholics (40), forty alcoholic smokers (40) and forty controls (40). The result of this

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study shows significant difference in the mean age between the groups and control. However, alcoholics and alcoholic smokers have significant higher ($p<0.05$) mean age when compared to individuals that smoke alone. The mean values of body mass index (BMI) were significantly higher ($p<0.05$) in the groups when compared with the control, although there is no significant difference in BMI between smokers, alcoholics and alcoholic smokers (Table 1).

There is significantly elevated ($p<0.05$) serum level of ALP, AST, ALT and GGT in alcoholics and alcoholic smokers when compared with control. However, AST and ALT level in smokers though elevated when compared with control was statistically reduced when compared with individuals that are alcoholics and alcoholic smokers (Table 2).

Also, the result of this study shows elevated level of Urea and Creatinine in smokers, alcoholics and alcoholic smokers ($p<0.05$) when compared with control. In contrast, estimated glomerular filtration rate was reduced in smokers, alcoholics and alcoholic smokers when compared with control (Table 3).

Discussions

The result of this study shows that AST, ALT and GGT activities were significantly increased ($P<0.05$) in smokers, alcoholics and alcoholic smoker's plasma when compared with control. Cigarette smoking have been said to causes a variety of adverse effects on organs that have no direct contact with the smoke itself such as liver and may do these by yielding chemicals with cytotoxic potential which increase necro-inflammation and fibrosis (El-Zayadi, 2006). However, this study is the first to describe a detailed effect of smoking on the individual test of liver functions test and the increased liver enzymes observed may be as a result of

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leakage in liver enzymes into the blood. This is suggestive of the possibility of cigarette smoking causing hepatocellular damage as consistent with the results of Khaled (2015) (El-Zayadi, 2006; Al salhen, 2015). Several previous studies have been controversial on the effect of cigarette smoking on aminotransferase as some investigators claimed AST increased by cigarette smoking (Chan-Yeung, 1981; Awaad *et al*., 2022) as our results support, while other studies argued that smoking did not influence AST or ALT (Suriyaprom *et al*., 2007; Whitehead *et al*., 1996).

Although our results showed elevated ALP levels in alcoholics and alcoholic smokers compared with control. Alcohol intake has been established to have deleterious effects on the liver (Roerecke *et al*., 2019; Hagström, 2017). Chronis alcoholic liver damage is attributed to alcohol metabolism, liver injury may be caused by direct toxicity of metabolic by product of alcohol as well as by inflammation induced by these by product. This results in elevated AST, ALT, ALP and GGT as reported in this study.

Cigarette smoking has been reported by some studies to cause renal damages. Cigarette smoking induced renal damage is due in part to the activation of the sympathetic nervous system resulting in elevation of blood pressure. In addition, cigarette smoking increase lipid peroxidation in the kidney with its attendant effects (Cooper, 2006).

Cigarette smoking has also been associated with elevated CRP levels, suggesting that inflammation may contribute to renal dysfunction (Nowak and Chonchol, 2018; Imig and Ryan, 2013). Furthermore, cigarette smoking promotes increased glomerular permeability with increase proteinuria and loss of glomerular filtration function (Maeda *et al*., 2011; Noborisaka *et al*., 2012). This will result in reduced estimated glomerular filtration rate as reported in this study.

The reduced estimated glomerular filtration rate and increased creatinine level observed in the alcoholics suggests that alcohol intake may cause renal injury. Another study has shown that alcohol intake can induce oxidative stress and stimulate inflammatory

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processes that are harmful to the kidney (Varga *et al*., 2017).

Limitation and Strength

The limitation of the study is that thenon-compliance of the subject with the diet prescription may affect the analysis and that a substantial part of the subject were from the Fulani ethnic group, however, the strength of the study is sample size of the study.

Conclusion

We conclude, based on the data from this study that cigarette smoking and alcohol intake have a deleterious effect on both renal and hepatic function indices. It also promotes lipid peroxidation and thus induces oxidative stress, hence, we recommend regular screening of renal and liver function for cigarette smokers and alcoholics as they may pose as a risk factor for cardiovascular disease.

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Tables.

Table 1: Socio Demographic Characteristics of the Studied Population

Subjects	**Age (years)**	**BMI (kg/m2)**
Smokers (n=40)	28.65 ± 1.44[a]	22.95 ± 0.37[a]
Alcoholics (n = 40)	32.75 ± 1.43[b]	23.28 ± 0.45[a]
Smokers and Alcoholics (n = 40)	33.83 ± 1.98[b]	23.05 ± 0.56[a]
Control (n = 40)	23.98 ± 0.25[c]	21.58 ± 0.30[b]
P value	0.001	0.026

Statistically significant at $p<0.05$

Values expressed as mean ± standard error

BMI= Body Mass Index

Table 2: Effect of Cigarette Smoking and Alcohol Consumption on Liver Function Indices

Subjects	**ALP (IU/L)**	**AST (IU/L)**	**ALT (IU/L)**	**GGT (IU/L)**
Smokers (n=40)	67.33 ± 2.09[a]	25.73 ± 1.08[a]	22.80 ± 1.17[a]	25.65 ± 1.21[a]
Alcoholics (n = 40)	87.65 ± 3.66[b]	40.00 ± 1.97[b]	29.80 ± 1.16[b]	37.05 ± 1.58[b]

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Smokers and Alcoholics (n = 40)	88.73 ± 3.01[b]	34.13 ± 1.40[c]	28.03 ± 1.29[c]	33.0 ± 1.40[b]
Control (n = 40)	69.33 ± 1.76[a]	16.00 ± 0.82[d]	13.58 ± 0.73[d]	20.23 ± 1.08[c]
P value	0.000	0.000	0.000	0.000

Statistically significant at P<0.05

Values are mean ± standard error of mean

ALP= Alkaline Phosphatase

AST= Aspartate Aminotransferase

ALT= Alanine Aminotransferase

GGT= Gamma-glutamylTransferase

Table 3: Effect of Cigarette Smoking and Alcohol Consumption on Renal Indices

Subjects	**Urea (mg/dL)**	**Creatinine (mg/dL)**	**eGFR (ml min-1)**
Smokers (n=40)	5.26 ± 0.15[a]	92.45 ± 2.46[a]	101.18 ± 2.93[a]
Alcoholics (n = 40)	4.30 ± 0.17[b]	90.38 ± 2.47[a]	100.45 ± 2.67[a]
Smokers and Alcoholics (n = 40)	4.89 ± 0.11[a,c]	89.85 ± 2.90[a]	100.40 ± 3.30[a]
Control (n = 40)	4.71 ± 0.11[c]	74.30 ± 2.07[b]	121.78 ± 2.13[b]
P value	0.000	0.000	0.000

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Statistically significant at $P<0.05$

Values are mean ± standard error of mean

eGFR= Estimated Glomerular Filtration rate.

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