WORLD JOURNAL OF PHARMACY AND PHARMACEUTICAL SCIENCES
SJIF Impact Factor 6.647
Volume 6, Issue 12, 189-199 Research Article ISSN 2278 – 4357

THE EFFECT OF BEER BRANDS ON SOME RENAL INDICES IN MALE ADULT CONSUMERS IN PORT HARCOURT, NIGERIA

Ebirien-Agana Samuel Bartimaeus*, Stella Urekweru Ken-Ezihuo and Avidi Ereseiye

Department of Medical Laboratory Science, Rivers State University, P.M.B. 5080, Nkpolu Oroworukwo, Port Harcour3, Nigeria.

Article Received on
12 October 2017,
Revised on 03 Nov. 2017,
Accepted on 24 Nov. 2017,
DOI: 10.20959/wjpps201712-10602

***Corresponding Author**
Dr. Ebirien-Agana Samuel Bartimaeus
Department of Medical Laboratory Science, Rivers State University, P.M.B. 5080, Nkpolu Oroworukwo, Port Harcour3, Nigeria.

ABSTRACT

Background: It is thought that immoderate consumption of beer among consumers could have adverse effects on the kidney. **Objectives of the study:** The effect of beer brands on some renal function indices was investigated in consumers in Port Harcourt, Rivers State. **Materials and Methods:** Sixty male subjects, aged (18-45) years participated in the study. Heineken, Hero, Star and '33' lager beer brands were used. Baseline blood samples were collected from the subjects before beer intake. Subjects were divided into 4 groups of 15. Each group was given a bottle of each beer brand and blood samples were collected from the subjects after I hr. The subjects were further offered 2-3 more bottles of the beer brand and blood samples were collected 1hr after. Serum creatinine, urea and uric acid levels were determined from the samples by standard procedures. **Results:** Significant increase ($p<0.05$) were observed in the level of the parameters in subjects who consumed one bottle and two or more bottles of beer when compared with the baseline results. **Conclusion:** The beer brands studied causes increase in the levels of serum creatinine, urea and uric acid which implied that immoderate drinking could cause renal damage and impairment.

KEYWORDS: Beer, creatinine, urea, uric acid, renal impairment.

INTRODUCTION

The consumption of alcohol particularly in moderate quantities has consistently been reported to lower risk of cardiovascular disease.[1] This effect occurs because alcohol has been observed to intervene in the atherogenetic process and I n the regulation of the secretion of vasoactive peptide[2] but not much data on the association of alcohol at low concentration

with the risk of chronic kidney disease (CKD) exist so far. However, in some longitudinal cohort studies, an inverse associations of the effect of alcohol consumption on the development of CKD has been reported.[3,4] Some authors, however, have reported inconsistent findings.[5,6] Evidence also exist showing that chronic alcohol consumption may possibly cause direct and severe kidney damage [7,8] and induce hyperuricaemia.[9] Bartimaeus and Eno-Eno[10] also reported significant increase in uric acid levels in Nigerian adult male alcohol consumers.

The metropolitan and peaceful nature of Port Harcourt City, Nigeria has witnessed rapid increase in the number of restaurants and bars and at most evenings hundreds of folks are usually seen hanging out and drinking severally bottles of assorted beer brands which are popularly sold in Port Harcourt. To date, however, little is known about any association between the different beer brands sold and consumed in the Nigeria markets and renal function indices and kidney dysfunction. Thus, this study was designed to investigate the effect of some beer brands on some renal indices in male adult consumers in Port Harcourt, Nigeria.

MATERIALS AND METHODS

Subjects' selection and preparation

For the purpose of this study, blood samples were randomly collected from sixty persons between the ages of eighteen to forty five years (18- 45yrs) within Port- Harcourt environs who have been drinking for at least two years. The subjects were all male residents in Port Harcourt and admitted to have been drinking various brands of beer products for at least two years. The subjects were adviced to avoid red meat 24 hours to the time of the test. They also abstained from drinking alcohol 2 days before the test. Three restaurants/bars were used as focal point for collection of samples. The beer brand were purchased from vendors in these restaurants/bars.

Ethical clearance

Ethical clearance was obtained from the the Rivers State Health Management Board, Port Harcourt, Nigeria. In addition, informed consent were provided by all of the participants included in the present study after full explanation of the purpose of the study to them.

Beer Brands

The beer brands considered and used in this study are Star Cold Filtered lager beer, Heinekens lager beer, "33" Export Premium lager beer and Hero Premium lager beer. The chemical content of the beers include but not limited to water, malted barley, malted sorghum and hops. The alcoholic content of the beer brand is as follows: Star Cold Filtered lager beer (5.1% vol), Heineken lager beer (5% vol.), "33" Export Premium lager beer (5% vol) and Hero Premium lager beer (5.2% vol.). Each of the beer brand was delivered in 600 mL bottle. The beer brands are brewed and bottled by Nigerian Breweries PLC, Lagos, Nigeria.

Experimental design

On the day of the test, baseline blood samples were collected from all the subjects. The blood samples were taken to the Chemical Pathology laboratory of the Braithwaite Memorial Specialist Hospital (BMSH), Port Harcourt, Nigeria for analyses. Subjects were divided into 4 groups of 15. Each group was given a bottle of each beer brand and blood samples were collected from the subjects after I hr. The subjects were further offered 2-3 more bottles of the beer brand and blood samples were collected 1hr after. All the blood samples were analysed at the Chemical Pathology Laboratory of the Braithwaite Memorial Specialist Hospital (BMSH), Port Harcourt, Nigeria. Adequate precautionary measures were taken to ensure the cooperation of the subjects throughout the testing period.

Biochemical determinations

Determination of Serum Creatinine

Serum creatinine level was determined by the alkaline Jaffe's Picrate method.[11] The Jaffe's method for serum creatinine determination is based on the principle that picric acid in an alkaline medium reacts with creatinine to form an orange coloured complex with the alkaline picrate. The absorbance of the complex formed whose intensity is directly proportional to the amount of creatinine present in the sample is read in a spectrophotomer at 490nm.

Determination of Serum urea

The serum urea level was determined by the diacetyl monoxime method as reported by Cheesbrough.[12] In the diacetyl monoxime method, urea reacts with diacetyl monoxime at high temperature in an acid medium in the presence of cadmium ions and thiosemicarbazide to produce a red coloured solution whose absorbance is read at 530 nm in spectrophotomer.

Determination of serum uric acid

Serum uric acid was estimated by using MERK Kit with the help of semi-automated analyzer (Microlab 300, Merck) on the same day of sample collection.

Statistical analysis

The data was analyzed using SPSS version 11 and data are presented as means and standardard deviation. Variation in means was tested using analysis of variance (ANOVA) and the Tukey multiple comparison test was used to compare differences between groups. The level of significance was set at $p< 0.05$.

Quality control

Adequate quality control measures including use of serum controls with known normal and abnormal values were run to monitor the validity of the reaction to ensure that the results of analyses were reliable, accurate and reproducible.

RESULTS

The baseline uric acid, creatinine and urea concentrations in the subjects were 309.7+37.42 µmol/L, 69.73+15.10 µmol/L and 2.51+0.59 mmol/L respectively. The results showing the concentrations of uric acid, creatinine and urea in the subjects administered with Star Lager beer is shown in table 1. The table shows that the concentration of uric acid in the subjects 1 hour after consumption of one bottle of Star lager and 2 or 3 bottles of the beer were 346.4+38.27 µmol/L and 459.7+15.29 µmol/L respectively. Comparison of the means using analysis of variance (ANOVA) showed significant difference ($p<0.05$, F=). Tukey multiple test of comparison also showed that the concentrations of uric acid 1 hr after consumption of 1 bottle of the beer and 1 hour after consumption of 2 or 3 bottles of the beer were significantly different ($p<0.05$). The creatinine and urea concentration in the subjects' afer consumption of one bottle and 2 or 3 bottles of the beer were 64.07+12.37 µmol/L and 3.02+0.59 mmol/L respectively. No significant difference ($p>0.05$) in mean was observed in the means of creatinine between the baseline sample and the samples collected after consumption of 1 and 2 or 3 bottles of Star beer respectively.

There was no significant difference ($p>0.05$) between the mean of urea in the baseline sample and that of the sample collected 1 hour after 1 bottle of the beer was consumed but the difference in means was significant ($p<0.05$) between the baseline sample and samples collected after consumption of 2 or 3 bottles of star lager beer.

Table 1: Mean ± SD of uric acid, creatinine and urea levels of subjects administered with Star lager beer.

Parameters	Baseline conc. before administration of beer	Conc. in 1hr after administration of 1-bottle of beer	Conc. in 1hr after administration of 2 or more bottles of beer	p-value
Uric acid (µmol/L)	309.7+37.42[a]	346.4+38.27[a]	459.7+15.29[a]	P<0.05, F=49.08
Creatinine (µmol/L)	69.73+15.10[b]	64.07+12.37[b]	75.80+13.17[b]	p>0.05, F=2.79
Urea (mmol/L)	2.51+0.59[bc]	3.02+0.59[ab]	3.71+0.65[ac]	p< 0.05, F=14.60

Note: a- represents significant difference (p<0.05) between groups, b- no significant difference (p>0.05) between groups and c-represents significant difference (p<0.05) between baseline samples and samples collected after 2 or 3 bottles of beer was consumed.

Table 2 shows mean ±SD of uric acid, creatinine and urea in the subjects who consumed Heinekens lager beer. The mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 1 bottle of Heinekens lager beer was consumed were 416.7 ±59.80 µmol/L, 74.47±18.01 µmol/L and 2.74±0.40 mmol/L respectively. Similarly, the mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 2 or 3 bottles of Heinekens lager beer were consumed were 560.9 ± 97.99 µmol/L, 102.1± 38.96 µmol/L and 4.35±1.70 mmol/L respectively. The means of uric acid in the basal sample when compared with the samples collected 1 hour after consumption of 1 and 2 or 3 bottles of Heinekens lager beer showed significant (p<0.05) variation between all the groups. The concentration of creatinine and urea was significantly increased (p<0.05) between the samples obtained from subjects who consumed 2 or 3 bottles of Heinekens and the baseline samples and samples collected 1 hour after consumption of Heinekens.

Table 2: Mean ± SD of uric acid, creatinine and urea levels of subjects administered with Heinekens lager beer.

Parameters	Baseline conc. before administration of beer	Conc. in 1hr after administration of 1-bottle of beer	Conc. in 1hr after administration of 2 or more bottles of beer	p-value
Uric Acid (µmol/L)	309.7±37.42[a]	416.7 ±59.80[a]	560.9 ± 97.99 [a]	p<0.05, F=49.08
Creatinine (µmol/L)	69.73±15.10[bc]	74.47±18.01[ab]	102.1± 38.96[ac]	p<0.05, F=6.63
Urea (mmol/L)	2.51±0.59[bc]	2.74±0.40[ab]	4.35±1.70[ac]	p<0.05, F=27.50

Note: a- represents significant difference ($p<0.05$) between groups, b- no significant difference ($p>0.05$) between groups and c-represents significant difference ($p<0.05$) between baseline samples and samples collected after 2 or 3 bottles of beer was consumed.

Table 3 represents the mean ±SD of uric acid, creatinine and urea in the subjects who consumed "33" Export Premium lager beer. The mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 1 bottle of "33" Premium lager beer was consumed were 471.7±81.74µmol/L, 71.80 ±7.65µmol/L and 2.99±0.49 mmol/L respectively. Furthermore, the mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 2 or 3 bottles of "33" Premium lager beer were consumed were 558.7±47.46 µmol/L, 124.8± 7.30 µmol/L and 4.07±0.67 mmol/L respectively. Significant differences ($p<0.05$) was observed between the means of these parameters in the various sample groups. Significant variation ($p<0.05$) was seen in the uric acid levels between the sample of subjects who consumed 1 bottle of the beer and 2 or 3 bottles of the beer and the baseline samples. However, in the creatinine and urea levels, significant variation ($p<0.05$) in mean was observed only between the baseline sample and those who consumed 1 bottle and 2 or 3 bottles of the beer.

In table 4, the mean ± SD of uric acid, creatinine and urea levels of subjects administered with Hero lager beer is presented. The mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 1 bottle of Hero Premium lager beer was consumed were 424.1±58.45µmol/L, 76.73±8.46µmol/L and 2.59±0.23 mmol/L respectively. Similarly, the mean ±SD of uric acid, creatinine and urea concentrations in the samples of subjects determined 1 hour after 2 or 3 bottles of Hero Premium lager beer was consumed were 548.7±45.46 µmol/L, 136.7±5.75µmol/L and 3.86±0.65mmol/L respectively. Significant differences ($p<0.05$) was also observed between the means of these parameters in the various sample groups. Significant variation ($p<0.05$) was seen in the uric acid levels between the samples of subjects who consumed 1 bottle of the beer and 2 or 3 bottles of the beer and the baseline samples. However, in the creatinine and urea levels, significant variation ($p<0.05$) in mean was observed only between the baseline samples and those who took 2 or 3 bottles of the beer.

Table 3: Mean ± SD of uric acid, creatinine and urea levels of subjects administered with "33" Export Premium lager beer.

Parameters	Baseline conc. before administration of beer	Conc. in 1hr after administration of 1-bottle of beer	Conc. in 1hr after administration of 2 or more bottles of beer	p-value
Uric Acid (µmol/L)	309.7±37.42[a]	471.7±81.74[a]	558.7±47.46[a]	p<0.05, F=69.54
Creatinine (µmol/L)	69.73±15.10[bc]	71.80 ±7.65[ab]	124.8± 7.30[ac]	p<0.05, F=129.0
Urea (mmol/L)	2.51±0.59[a]	2.99±0.49[a]	4.07±0.67[a]	p<0.05, F=27.59

Note: a- represents significant difference (p<0.05) between groups, b- no significant difference (p>0.05) between groups and c-represents significant difference (p<0.05) between baseline samples and samples collected after 2 or 3 bottles of beer was consumed.

Table 4: Mean ± SD of uric acid, creatinine and urea levels of subjects administered with Hero premium lager beer.

Parameters	Baseline conc. before administration of beer	Conc. in 1hr after administration of 1-bottle of beer	Conc. in 1hr after administration of 2 or more bottles of beer	p-value
Uric acid (µmol/L)	309.7±37.42[a]	424.1±58.45[a]	548.7±45.46[a]	p<0.05, F=98.88
Creatinine (µmol/L)	69.73±15.10[bc]	76.73±8.46[ab]	136.7±5.75[ac]	p<0.05, F=183.1
Urea (mmol/L)	2.51±0.59[bc]	2.59±0.23[ab]	3.86±0.65[ac]	p<0.05, F=0.60

Note: a- represents significant difference (p<0.05) between groups, b- no significant difference (p>0.05) between groups and c-represents significant difference (p<0.05) between baseline samples and samples collected after 2 or 3 bottles of beer was consumed.

DISCUSSION

The study was aimed at investigating the effect of beer brands on some renal function indices in consumers of beer in Port Harcourt, Nigeria. Serum uric acid, creatinine and urea are part of the battery of tests used to assess the glomerular filtration capacity and therefore unctionality of the kidney.[13] Renal insufficiency, the presence of kidney damage or decreased kidney function, is a significant public health problem, due to the increase in the prevalence[14,15] and the economic burden of it.[16] Beer drinking is very popular among Nigerians and excessive alcohol consumption has been linked to increased cardiovascular risk and mortality[17,18] whereas moderate alcohol consumption has been associated with a decreased risk of cardiovascular disease.[1,19] Despite these pathological effects of alcohol,

studies into the effects of exceesive beer drinking on the health of consumers in Nigeria has not been reported. This study is, therefore, the first of its kind to address the issues of the toxic effects of beer on the renal indices of consumers. The study revealed a consistent increase in the concentrations of uric acid, creatinine and urea which becomes very significant especially in consumers who had consumed at least 2 or 3 bottles of the different beer brands. This finding is in agreement with the work of Epstein.[20] It is common to find subjects who consistently consume as much as 6 bottles (3,600 mL) or more of beer per day in our locality. The study was designed to consider a one-time exposure effect of the beer brands within 1 hour exposure time. The findings obtained in the study is a pointer to the fact that more serious effects on the kidney could occur upon continued exposure at higher volume of concentration. Puddey et al.[21] reported that alcohol not only disrupts homeostasis but causes the body to react abnormally to restore it. They reported that alcohol-induced urination reduced the subjects' plasma volume, which will result in an increased concentration of plasma sodium. In addition, the subjects' blood pressure and plasma potassium concentration would be decreased. These changes in fluid volume, electrolyte balance, and blood pressure may stimulate the activity of hormones to return body fluid volume and composition back to normal, which occur soon after consumption. Though these effects were not investigated in this study, they offer credible background to the assertion that excessive alcohol consumption is toxic to the kidney.

The alcoholic content of the beer brands used in our study is as follows: Star Cold Filtered lager beer (5.1% vol), Heinekens lager beer (5% vol.), "33" Export Premium lager beer (5% vol) and Hero Premium lager beer (5.2% vol.). Each of the beer brand was delivered in 600 mL bottle. The results summarily shows that increase in uric acid level following consumption of 2 or 3 bottles of the beer brands is in the ratio of Heinekens lager beer > "33" Premium Lager beer > Hero Premium lager > Star lager beer. The ratio of increase in creatinine concentration is in the ratio of Hero Premium lager beer > "33" Premium Lager beer > Heinekens lager beer >Star lager beer while the ratio of increase in urea concentration is Heineken lager beer > "33" Premium Lager beer > Hero Premium lager beer > Star lager beer. While Heinekens lager and Hero Premium lager beer caused significant increase in the concentration of these renal indices in the consumers following consumption of 2 or 3 bottles, Star lager beer does this least.

The recommended reference range for creatinine in an adult male is < 115µmol/L, uric acid- 202- 357 µmol/L and urea 1.8- 6.5 mmol/L.[13] Star lager beer which contains only (5.0% vol.) even at 2 or 3 bottles did cause significant variation in these parametes while Heinekens lager beer only caused an increase in uric acid concentration from 309.7±37.42 µmol/L in the baseline sample to 416.70 ± 59.80 µmol/L and 560.90 ± 97.99 µmol/L in the subjects who consumed 1 bottle and 2 or 3 bottles respectively. "33" Export Premium lager beer consumption resulted in an increase in the concentrations of uric acid and creatinine. The uric acid concentration in the baseline sample was 309.7±37.42 µmol/L and it increased to 471.70 ± 81.74 µmol/L in the sample from subjects who took 1 bottle to 558.70 ± 47.46 µmol/L in the sample of subjects who consume 2 or 3 bottles while creatinine levels increased from baseline value of Hero Premium lager beer had the 69.73±15.10 µmol/L to 124.80 µmol/L in the subjects who took 2 or 3 bottles of the beer. The beer brands that affect creatinine grossly is Hero Premium lager beer. With the increase in the number of bottles to 2 or 3, the creatinine level increased from the baseline value of 69.73±15.10 µmol/L to 136.70 µmol/L. Similar increase was obtained in the uric acid concentration. Hero Premium lager beer incidentally contains 5.2% vol of alcoholas against 5.1% vol. found in Heinekens and "33" Export Premium lager beer.

The strength of this study is that the subjects fully complied with all instructions required as part of patient preparation to participate in the study and since this is the first study, to our knowledge, to investigate the effect of beer brands on the renal function indices, the results obtained could act as a guide towards discouraging people from excessive beer consumption which has the potential of causing renal dysfunction. However, this study has some limitations. First, this study was conducted with few number of participants and the number of renal function parameters measured are limited. Second, there was the probability that the subjects may not have fully abstained from meat and alcohol before the date of test.

CONCLUSION

The beer brands in the Nigerian markets if excessively consumed could have profound negative effects on the kidneys and their function in maintaining the body's fluid, electrolyte, and acid-base balance, leaving people who drink beer vulnerable to a host of kidney related health problems. Despite the clinical importance of alcohol's effects on the kidney, however, relatively few or no recent studies have been conducted to characterize them or elucidate their

pathophysiology. It is hoped that future investigations will focus on this important subject area.

REFERENCES

1. Ronksley P E, Brien S E, Turner B J. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. BMJ, 2011: 342: 671.
2. Van Tol A, Hendriks H F. Moderate alcohol consumption: effects on lipids and cardiovascular disease risk. Curr Opin Lipidol, 2001; 12: 19–23.
3. Schaeffner E S, Kurth T, de Jong P E. Alcohol consumption and the risk of renal dysfunction in apparently healthy men. Arch Intern Med, 2005; 165: 1048–1053.
4. Yamagata K, Ishida K, Sairenchi T. Risk factors for chronic kidney disease in a community-based population: a 10-year follow-up study. Kidney Int, 2007; 71: 159–166.
5. White S L, Polkinghorne K R, Cass A. Alcohol consumption and 5-year onset of chronic kidney disease: the AusDiab study. Nephrol Dial Transplant, 2009; 24: 2464–2472.
6. Shankar A, Klein R, Klein BE. The association among smoking, heavy drinking, and chronic kidney disease. Am J Epidemiol, 2006; 164: 263–271.
7. Heidland A, Horl WH, Schaefer RM, Teschner M, Weipert J, Heidbreder E. Role of alcohol in clinical nephrology. Klin Wochenschr, 1985; 63: 948–958.
8. Vamvakas S, Teschner M, Bahner U, Heidland A. Alcohol abuse: potential role in electrolyte disturbances and kidney diseases. Clin Nephrol, 1998; 49: 205–213.
9. Yamanaka H. Alcohol ingestion and hyperuricemia. Nippon Rinsho, 1996; 54: 3369–3373.
10. Bartimaeus, E. S. and Eno, M The effect of alcohol on Uric acid level in consumers. J App.Env. Manag, 2000; 4: 39-41.
11. Owen, A; Iggo, B; Scandrett, F J; Stewart, C P The determination of creatinine in plasma or serum and in urine. A critical examination. *Biochem. J*., 1954; 58: 426.
12. Cheesbrough, M. Clinial chemistry tests. In: District Laboratory Practice in Tropical Countries Part 1 (2edn.). Sheck Wah Tong Printing Press Ltd, Hong Kong, 2005; 310.
13. Sood R. Clinical haematology. In Medical Laboratory Technology: Methods and Interpretations. Jaypee Brothers Medical Publishers Ltd. New Delhi 5th edn., 2002; 167-71.

14. Castro AF, Coresh J: CKD surveillance using laboratory data from the population-based national health and nutrition examination survey (NHANES). Am J Kidney Dis, 2009; 53: S46-55.
15. Imai E, Horio M, Watanabe T, Iseki K, Yamagata K, Hara S, Ura N, Kiyohara Y, Moriyama T, Ando Y, Fujimoto S, Konta T, Yokoyama H, Makino H, Hishida A, Matsuo S: Prevalence of chronic kidney disease in the Japanese general population. Clin Exp Nephrol, 2009; 13: 621-630.
16. Smith DH, Gullion CM, Nichols G, Keith DS, Brown JB: Cost of medical care for chronic kidney disease and comorbidity among enrollees in a large HMO population. J Am Soc Nephrol, 2004; 15: 1300-1306.
17. Bagnardi V, Zatonski W, Scotti L, La Vecchia C, Corrao G: Does drinking pattern modify the effect of alcohol on the risk of coronary heart disease? Evidence from a meta-analysis. J Epidemiol Community Health, 2008; 62: 615-619.
18. Parry CD, Patra J, Rehm J: Alcohol consumption and non-communicable diseases: Epidemiology and policy implications. Addiction, 2011; 106: 1718-1724.
19. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G: Alcohol dosing and total mortality in men and women: An updated meta-analysis of 34 prospective studies. Arch Intern Med, 2006; 166: 2437-2445.
20. Epstein, M. Alcohol's impact on kidney function. Alcohol Health and Research World, 1997; 22(1): 84-93.
21. Puddey, I.B.; Vandongen, R.; Beilin, L. J.; and Rouse, I.L. Alcohol stimulation of renin release in man: Its relation to the hemodynamic, electrolyte and sympathoadrenal responses to drinking. J Clin Endocrin Metab, 1985; 61(1): 37–42.