Original Article

Comparative Impact of Chronic Consumption of Burukutu and Beer on Liver Biomarkers in Male Volunteers in Akwanga Lga Narasawa State

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Abstract

Background/Aim: Alcohol is implicated as one of the major risk factor in the development of liver diseases. Its consumption has a long time history in human existence. The comparative impact of burukutu, a sorghum-based alcoholic beverage and commercial beer on the liver biomarkers of consumers was investigated. **Method and Design:** Eighty (80) volunteer male subjects in Akanga LGA, Nasarawa state, between the ages of 20-60 yrs, were recruited for the study. Thirty (30) subjects were chronic burukutu consumers, another thirty (30) were chronic beer consumers and the remaining twenty (20) were control subjects who neither consume burukutu nor beer or any other alcoholic drink. All the subjects were apparently healthy and free from liver disease. The liver function status (LFT) was determined by analyzing the liver function biomarkers with chemistry auto analyzer (CHEMWELL 2910) using 5ml of blood sample collected from each subject. **Results:** Results showed a statistically significant increase in ALP level ($p<0.001$) and AST level ($P<0.05$) in chronic burukutu consumers when compared with control subjects; while chronic consumers of beer had statistically significant increase in ALP levels ($P<0.05$) only as against the control subjects. Data comparison according to age bracket revealed a statistically significant difference in ALP level ($P<0.05$) between 20-29 yrs and 40-49 yrs in chronic burukutu consumers. Furthermore, ALP was significantly decreased ($P<0.001$) and ($P<0.05$) in groups, 6-10 yrs and >10 yrs respectively when data was compared with 1-5 yrs group according to period of exposure to chronic burukutu consumption. Albumin also had a statistically significant decreased level ($P<0.05$) in chronic burukutu consumers with >10 yrs of exposure to chronic drinking when compared with 1-5 yrs. AST/ALT ratio in chronic burukutu consumers was suggestive of alcohol abuse. Other liver biomarkers such as total protein (TP), total bilirubin (TB), direct bilirubin (DB) as well as ALT showed slight increase in levels, but the increase were however not significant ($P>0.05$) in both chronic burukutu or beer consumers when compared with control subjects. **Conclusion:** Burukutu seems to have more statistically significant impact on the liver biomarkers than commercial beer perhaps, due to its relatively high alcoholic content obtained by local and unstandardized brewing process.

Keywords: Liver; Alcohol; Liver biomarkers; Burukutu; Beer

Introduction

Consumption of alcohol is an aged long tradition. It is the third most popular drink overall, after water and tea. [1] Here in Nigeria, alcohol consumption is a common decimal among adult (i.e., age 18 and above) and plays a role in social, religious, political and economic relationships. [2] Alcohol is both locally and industrially brewed.

Burukutu is an indigenous locally brewed alcoholic beverage of vinegar-like flavor prepared from sorghum grains. [3] Burukutu production involves 6 stages namely steeping, malting, mashing, cooking, fermentation and maturation. It is widely consumed as food (because it is heavy and thick) in the rural population of northern and central Nigeria as well as poor urban centers due to its affordability compared to commercially brewed Beer. [4] The alcohol concentration or content of Burukutu is said to be 4-10%. [5] Nutritionally, Burukutu has been reported to contain vitamins, iron, magnesium, manganese, phosphorus, calcium, 26.7% starch and 5.9% proteins per liter. [4] While beer (commercially brewed alcohol) is said to have alcohol percentage concentration of less than 3-6% alcohol by volume (abv) to about 14%; though this strength can be increased to around 20% by re-pitching with champagne yeast and to 55% by freeze distillation process. The pale lagers that most consumers are familiar with fall in the

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range of 4-6% with typical alcohol above volume (abv) of 5%. Both Burukutu and Beer are metabolized in the liver.

Chronic consumption of alcohol posts a major risk factor for the development of liver fibrosis, alcohol liver disease (ALD), and hepatocellular carcinoma (HCC). [6-8] Alcohol-dependent induction of cytochrome P 450 2E1 (CyP2E1) leads to formation of acetaldehyde. [9] CYPE1-dependent alcohol metabolism leads to increased hepatic oxidative stress due to the production of reactive oxygen species (ROS) including hydroxyethyl radicals. [10] Excess (ROS) inflict injury on the parenchyma cells of the liver, leading to release of enzymes and other biomarkers which can be detected in body fluid especially serum.

Despite available literatures on the impact of chronic consumption of Burukutu and Beer on the liver biomarkers, information on which affect the liver biomarkers more seems obscured, hence the need for this study. The aims of this study were to investigate the impact of chronic consumption of Burukutu and Beer on the liver biomarkers (serum albumin, total protein, and bilirubin, ALT, AST, ALP and AST/ALT) in consumers and to assess the relationship between age, period of exposure to chronic drinking and volume of consumption with the impact on liver biomarkers.

Materials and Methods

Location

This study was carried out in Akwanga Local Government Area of Nasarawa state involving the towns and the villages.

Subjects

Eighty (80) apparently healthy volunteer male subjects between the ages of (20-60) yrs were recruited for this study. Questionnaires were applied to collect their bio- data and the history of drinking habit.

Inclusion criteria

These were based on the following:

• Those who consume Burukutu only

• Those who consume beer only

• Those who neither consume burukutu nor beer (controls)

Exclusion criteria

• Those who are consumers of burukutu and beer.

• Those that have known liver disease for example hepatitis B virus (HBV), Hepatitis C virus (HCV) or cirrhosis.

• Those who consume other alcoholic beverages.

Ethical approval

The ethical clearance for the study was granted by the Health Research Ethics Committee, State Ministry of Health, Lafia, Nasarawa State. Because the study was purely voluntary, informed consent form was applied to secure the agreement of the volunteer subjects.

Study design

The eighty subjects were divided into three groups namely A, B and C.

Group A: Contained 30 subjects who are chronic consumers of burukutu.

Group B: Contained 30 subjects who are chronic consumers of beer (commercially brewed).

Group C: Contained 20 control subjects who are neither consumers of burukutu nor beer nor any other alcoholic beverage.

Group A and B were further regrouped according to age bracket, average volume of consumption per day and the average period they have been drinking in years. The age bracket regrouping was thus: (20-29) years, (30-39) years, (40-49) years and (50-59) years.

The average volume of consumption per day was categorized into: Light drinkers (1-2L), Moderate drinkers (3-4L), Heavy drinkers (5 or more litres) per day for burukutu; while that of beer was categorized into: Light drinkers (1-2 bottles), Moderate drinkers (3-4 bottles), Heavy drinkers (5 or more bottles) per day.

Sample collection

Vacutainer closed system of venous blood collection was used. Five (5ml) of blood was carefully collected from each of the subjects at the forearm into a plain tube avoiding venoustasis and allowed to clot at room temperature. Samples were then placed in the cooler and transported to the hospital. These were spurn at 3500 rpm for 10 minutes within one hour of sample collection. The serum was aspirated using pasteur pipettes into micro vials for the analysis of the analytes of interest

Biochemical analysis

The samples were analysed using an autoanalyser, CHEMWELL 2910; methods were according to the followings:

Serum total protein

Serum total protein was estimated using the Biuret method.

Measurement of serum albumin

Method was by Automated Dye-binding in 1978.

Measurement of bilirubin (Total and direct)

Colorimetric method based on Mordified dimethyl sulphoxide (DMSO)

Measurement of liver enzymes

Measurement of ALP: DGKC-SE recommended technique in 1972.

Measurement of ALT and AST: Determination of ALT and AST were by IFCC recommended technique in 1987 and 1985 respectively.

De Ritis ratio (Calculated): AST/ALT.

Statistical analysis

Data was analyzed using SPSS software version 18. All data were expressed as mean ± SEM. Level Of Significance was determined by the student t-test or by the one way analysis of variance (ANOVA) followed by the Tukey's Post-HOC multiple comparison tests. P<0.05, p<0.01 or P<0.001 was considered significant.

Results

Table 1 show the results of liver biomarkers in eighty (80) subjects who were categorized into three (3) groups of Control subjects (20), Chronic burukutu consumers (30) and Chronic beer consumers (30). Observation made from the results was that chronic burukutu consumers only had a significant increases in ALP and AST levels when compared with control subjects (168.90 ± 14.27 vs. 91.15 ± 5.06, p< 0.001) and (53.03 ± 5.31 vs. 36.32 ± 2.51, p< 0.05) respectively. Furthermore, it is worthy of note that chronic beer consumer subjects also had a significant increase in ALP level when compared with control subjects (135.20 ± 8.52 vs. 91.15 ± 5.06, p<0.05). However, there was no significant difference in the levels of other liver biochemical parameters in chronic burukutu or beer consumers when compared with control subjects (p>0.05).

Table 2 shows the results of liver biomarkers in thirty (30) chronic burukutu consumer subjects who were categorized according to age brackets into four (4) groups of 20-29 (years), 30-39 (years), 40-49 (years) and 50-59 (years). Observation made from the results was that chronic burukutu consumers belonging to the age group 20-29 (years) only had a significant increase in ALP level when compared with age group 40-49 (years) subjects (202.80 ± 23.82 vs. 126.00 ± 14.37, p< 0.05). However, it is also worthy of note that there were no statistically significant differences when other liver biomarkers were compared among the age groups (p>0.05).

Table 3 shows the results of liver biomarkers in thirty (30) chronic burukutu consumer subjects who were categorized according to average period of consumption of burukutu into three (3) groups of 1-5 (years), 6-10 (years), and over 10 (years). Observations made from the results were these: chronic burukutu consumers belonging to the consumption period >10 (years) had a significant decrease in albumin level when compared with consumption period group 1-5 (years) [33.50 ± 0.94 vs. 38.13 ± 1.34, p< 0.05]. Also, chronic burukutu consumers belonging to the consumption periods 6-10 (years) and >10 (years) had a significant decrease in ALP level when compared with consumption period group 1-5 (years) subjects (158.90 ± 25.03 vs. 247.40 ± 27.46, p< 0.05) and (128.10 ± 12.93 vs. 247.40 ± 27.46, p< 0.001) respectively. However, it is also worthy of note that there were no statistically significant differences when other liver biochemical parameters were compared among the consumption period groups (p>0.05).

Table 1: Comparison of liver biomarkers in consumer subjects with control subjects (non-consumers).

Variables	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ ALT
Control Subjects	63.05 ± 1.12	35.85 ± 0.73	11.60 ± 0.74	3.66 ± 0.40	91.15 ± 5.06	24.06 ± 2.20	36.32 ± 2.51	1.76 ± 0.20
Chronic Burukutu Consumers	63.90 ± 1.12	35.47 ± 0.72	13.31 ± 0.69	3.89 ± 0 .26	168.90 ± 14.27***	29.48 ± 3.51	53.03 ± 5.31*	2.05 ± 0.21
Chronic Beer Consumers	61.87 ± 1.16	37.30 ± 0.70	14.55 ± 0.94	4.20 ± 0.27	135.20 ± 8.52*	29.32 ± 3.06	42.25 ± 3.77	1.73 ± 0.16

Values are given as Mean ± SEM. ***P□0.001 or *P□0.05 is significant when Chronic burukutu consumers or Chronic beer consumers group is compared with control subjects (non-consumers). Statistical comparison was done using the Student's t-test.

Table 2: Comparison of liver biomarkers according to age group in subjects who are chronic burukutu consumers.

Age Group (Years)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ALT
20-29	63.70 ± 1.70	37.20 ± 1.02	12.80 ± 0.79	3.77 ± 0.28	202.80 ± 23.82*	30.50 ± 7.30	46.92 ± 6.97	1.80 ± 0.23
30-39	59.70 ± 2.69	33.50 ± 1.28	15.50 ± 0.69	4.52 ± 0.57	172.70 ± 30.65	34.50 ± 4.75	75.37 ± 17.14	2.20 ± 0.41
40-49	66.90 ± 1.78	35.40 ± 1.82	13.70 ± 1.53	3.37 ± 0.57	126.00 ± 14.37	28.10 ± 4,56	49.87 ± 8.87	1.90 ± 0.34
50-59	65.80 ± 3.01	33.00 ± 1.08	10.10 ± 1.73	3.83 ± 1.21	128.30 ± 37.51	21.20 ± 5.90	40.05 ± 4.70	2.50 ± 1.20

Values are given as Mean ± SEM. *P□0.05 is significant when Chronic burukutu consumers belonging to the age group 20-29(years) is compared with the age group 40-49(years) subjects. Statistical comparison was done using the Student's t-test.

Table 3: Comparison of liver biomarkers in chronic burukutu consumers according to average period of consumption.

Period of onsumption (Years)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ALT
1-5	60.88 ± 2.20	38.13 ± 1.34	11.73 ± 0.64	4.25 ± 0.30	247.40 ± 27.46	32.35 ± 11.93	35.26 ± 9.31	1.35 ± 0.23
6-10	64.25 ± 2.33	35.50 ± 1.04	14.14 ± 1.21	3.76 ± 0.56	158.90 ± 25.03*	27.71 ± 3.08	58.75 ± 9.70	2.29 ± 0.35
□10	65.14 ± 1.48	33.50 ± 0.94*	13.41 ± 1.27	3.46 ± 0.41	128.10 ± 12.93***	29.24 ± 3.40	61.62 ± 7.99	2.34 ± 0.35

Values are given as Mean ± SEM. *P□0.05 and ***P □0.001 are significant when consumption period 6-10yrs and 10+yrs groups are respectively compared with 1-5yrs group. Statistical comparison was done using the Student's t-test.

Table 4 shows the results of liver biomarkers in thirty (30) chronic burukutu consumer subjects who were categorized according to average volume per day of consumption of burukutu into three (3) groups of light drinkers (1-2L), moderate drinkers (3-4L) and heavy drinkers 5L or more litres. Observation made from the results was that there were no statistically significant differences when all the liver biomarkers analyzed were compared among all the groups ($p>0.05$).

Table 5 shows the results of liver biomarkers in thirty (30) chronic beer consumer subjects who were categorized according to age brackets into three (3) groups of 20-29 (years), 30-39 (years) and 50-59 (years). Observation made from the results was that there were no statistically significant differences when all the liver biomarkers analyzed were compared among all the groups ($p>0.05$).

Table 6 shows the results of liver biochemical parameters in thirty (30) chronic beer consumer subjects who were categorized according to average period of consumption of beer into three (3) groups of 1-5 (years), 6-10 (years), and over 10 (years). Observation made from the results was that there were no statistically significant differences when all the liver biochemical parameters analyzed were compared among all the groups ($p>0.05$).

Table 7 shows the results of liver biomarkers in thirty (30) chronic beer consumer subjects who were categorized according to average number of bottles consumed per day into three (3) groups of light drinkers (1-2 bottles), moderate drinkers (3-4 bottles) and heavy drinkers (5 bottles) or more. Observation made from the results was that there were no statistically significant differences when all the liver biochemical parameters analyzed were compared among all the groups ($p>0.05$).

Discussion

The comparative impact of chronic consumption of burukutu; a sorghum- based alcoholic beverage [3] and commercial beer on the liver biomarkers of 30 chronic burukutu consumers and 30 chronic commercial beer consumers as well as 20 control subjects was investigated. The investigation revealed a significant increase in ALP ($P<0.001$) and AST ($P<0.05$) levels in chronic burukutu consumers when compared with the control subjects. Chronic beer consumers also showed significant increase in ALP levels when compared with the control subjects ($P<0.05$). Other liver biomarkers such as albumin, total and direct bilirubin as well as ALT showed slight increase in both chronic burukutu and beer consumers when compared with the control subjects. The increased levels were however not significant ($P>0.05$). The mean value (2.0 ± 0.21) of AST/ ALT ratio of the chronic burukutu consumers was indicative of alcohol abuse. [11,12]

Table 4: Comparison of liver biomarkers in chronic burukutu consumers according to average volume consumed per day.

Volume consumed (Litres)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ ALT
1-2	64.00 ± 3.32	33.50 ± 1.94	9.83 ± 1.16	3.65 ± 1.00	164.00 ± 54.85	15.53 ± 1.70	30.63 ± 5.45	2.00 ± 0.43
3-4	64.81 ± 1.48	35.75 ± 0.64	14.40 ± 0.84	4.24 ± 031	156.20 ± 16.08	26.88 ± 2.32	54.96 ± 6.80	2.04 ± 021
5+	62.40 ± 2.10	35.80 ± 1.79	12.97 ± 1.31	3.41 ± 0.46	191.30 ± 28.14	39.23 ± 9.18	58.89 ± 10.87	2.10 ± 0.53

Values are given as Mean ± SEM. Statistical comparison was done using the Student's t-test

Table 5: Comparison of liver biomarkers according age group in subjects who are chronic beer consumers.

Age Group (Years)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ ALT
20-29	63.10 ± 1.45	37.10 ± 0.96	15.00 ± 1.19	4.29 ± 0.35	128.70 ± 8.98	26.14 ± 2.90	38.84 ± 4.86	1.77 ± 0.23
30-39	62.00 ± 2.30	35.20 ± 1.39	12.90 ± 1.82	2.98 ± 0.47	138.00 ± 25.52	22.84 ± 4.76	41.30 ± 5.69	2.02 ± 0.38
50-59	57.40 ± 3.28	39.40 ± 0.51	12.20 ± 1.41	4.62 ± 0.50	152.00 ± 31.37	40.78 ± 11.16	50.18 ± 8.90	1.42 ± 022

Values are given as Mean ± SEM. Statistical comparison was done using the Student's t-test.

Table 6: Comparison of liver biomarkers in chronic beer consumers according to average period of consumption.

Period of consumption (Years)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ ALT
1-5	64.29 ± 1.68	38.07 ± 1.20	15.11 ± 2.58	4.12 ± 0.36	129.90 ± 10.26	26.52 ± 3.75	38.87 ± 6.43	1.81 ± 0.31
6-10	61.10 ± 1.60	36.00 ± 1.21	13.29 ± 1.12	3.87 ± 0.51	146.00 ± 18.42	27.84 ± 6.48	42.10 ± 5.28	1.81 ± 0.24
□10	59.57 ± 3.13	38.00 ± 1.13	14.73 ± 2.12	4.54 ± 0.68	124.00 ± 17.74	35.44 ± 6.07	45.86 ± 6.95	1.34 ± 0.13

Values are given as Mean ± SEM. Statistical comparison was done using the Student's t-test.

Table 7: Comparison of liver biochemical parameters in chronic beer consumers according to average number of bottles consumed per day.

Volume consumed (bottles)	Total Protein (g/L)	Albumin (g/L)	Total bilirubin (µmol/L)	Direct bilirubin (µmol/L)	ALP (U/L)	ALT (U/L)	AST (U/L)	AST/ ALT
1-2	62.26 ± 1.78	37.31 ± 1.24	14.92 ± 1.47	4.36 ± 0.38	144.30 ± 14.01	24.77 ± 3.43	43.53 ± 6.69	1.81 ± 0.31
3-4	61.50 ± 1.97	36.75 ± 1.03	13.77 ± 1.41	3.96 ± 0.48	124.70 ± 12.26	31.58 ± 5.81	39.37 ± 4.66	1.81 ± 024
5+	58.50 ± 1.19	38.25 ± 1.65	16.53 ± 3.34	4.90 ± 0.50	149.00 ± 24.91	40.15 ± 8.79	52.68 ± 8.13	1.34 ± 0.13

Values are given as Mean ± SEM. Statistical comparison was done using the Student's t-test.

The significant increase levels of ALP activity observed in this study may be due to the impact of alcohol content of burukutu and beer on the liver hepatocytes since serum ALP activity is mainly from the liver with 50% contributed by the bone. [13] This is because alcohol is known to inflict injury on the liver through oxidative stress resulting from the breakdown of its metabolic products especially acetaldehyde. [14] Despite the limitation of this study of not estimating gamma glutamyl transferase (GGT) along with the ALP which helps in differentiating whether the high levels of serum ALP is from the liver or not, the fact still remains that chronic alcohol consumption is a factor for elevated values of ALP as seen in alcoholic hepatitis with cholestasis. [15] Hence, this significant increase in ALP in chronic burukutu consumers ($P<0.001$) and chronic beer consumers ($P<0.05$), may be suggestive of cholestatic reaction.

When intra- group comparison of liver biomarkers in burukutu and beer subjects were made according to age brackets, a significant ($P<0.05$) increase in level of ALP was found in the age bracket of (20–29) years as against (40–49) years in burukutu consumers only. This observation may be due to the effect of alcohol content of burukutu on the liver and even the bones of this age group. Studies have shown that chronic heavy drinking of alcohol is detrimental to bone health; and this effect is said to be striking in younger people. [16] In addition, this age bracket may be more prone to risky behaviour like binge drinking, which is an emerging type of drinking habit, than 40-49 yrs age group.

ALP was also observed to have decreased significantly in chronic burukutu consumers with respect to average period of exposure to chronic burukutu consumption in group (6-10) years ($P<0.05$) and>10 years ($P<0.001$) when compared with the group of (1-5) yrs period. This data indicates that there is a relationship between the period of exposure to chronic drinking and the plasma level of ALP. This may not be attributed to the impact of burukutu on the liver so to say; rather, it may be due to the impact of alcohol content of burukutu on the bones. The effect of chronic consumption of alcohol has been reported to have direct impact on bone cells by decreasing the number of osteoblasts, osteoid formation and osteoblasts proliferation as well as indirect effects through its action on mineral regulatory hormones. [17] This development affects osteoblastic activity and bone remodeling resulting in low generation of bone isoform of ALP which is a biomarker of bone formation. [16,18] However, calcium and phosphate were not estimated in this study to show correlation with the ALP decrease. Studies done on animal model (albino rats) on the effect of chronic alcohol consumption on bone health showed that serum activity of ALP was decreased; [19] and this agrees with our finding. Another plausible explanation for the decreased ALP, may be due to poor nutrition associated with those who consume burukutu as food [4], which is not a rich source of protein and other minerals especially zinc and magnesium which are needed for the synthesis of ALP. ALP is a metalloenzyme with zinc as its main prostatic group.

Albumin is the most abundant plasma protein and is produced by hepatocytes. [20] In this study, albumin was observed to have decreased significantly when data was compared between the group >10 yrs and 1-5 yrs according to average period of exposure to chronic consumption of burukutu. The data therefore showed that there is concomitance between average period of exposure to chronic drinking and the level of albumin in the serum. The significant decreased in albumin levels so observed in this study could be due to the impact of alcohol content of burukutu on the protein synthesis ability of the liver. This finding is in agreement with Osaretin et al. [21] who reported that heavy drinking affects protein synthesis. Similar work done by Stanley P. C, [22] found a progressive decrease in total protein and albumin in chronic drinkers of alcohol. The decrease in albumin levels could also be attributed to low supply of amino acids necessary for the synthesis of albumin due to the use of burukutu as a source of protein by many poor rural-urban populations. In addition, this study was conducted in typical peasant agrarian communities where the consumption of burukutu as breakfast in the morning, as a source of energy for daily farming activities especially among the male folk, is a norm.

Comparison of liver biomarkers of chronic burukutu consumers and beer according to the average volume of consumption per day did not show any significant differences. This implied that there was no direct relationship between volume of consumption of burukutu and the serum levels of the liver biomarkers. However, the study done by Magu et al. [23] in Dakachi, Kaduna state, Nigeria showed that there is a positive relationship between the volume of consumption of burukutu with the serum activity of liver enzymes (AST and ALT) in moderate and heavy drinkers. Our study was carried out in Akwanga, Nasarawa State, Nigeria. The plausible explanation for this difference in observation could be due to difference in study designs with particular reference to questionnaire and ethnicity or location. Pävikki et al. [24] in a study done on the biomarkers of liver status in heavy and moderate drinkers demonstrated that biomarkers of alcohol abuse and liver function may respond to even rather low levels of ethanol intake in a gender- dependent manner.

AST was significantly elevated in chronic burukutu consumers as against the control subjects. The significant AST increase observed in burukutu subjects in this study, points to the capacity of chronic alcohol consumption to induce enzyme leakage from the hepatocytes especially in alcoholic hepatitis as reported by Uchida et al. [15] Alcohol increases mitochondrial AST activity in plasma, whereas other causes of hepatitis typically do not. [25] It does so by inducing the release of mitochondrial AST from cells without visible cell damage. [26]

Conclusion

This study showed that chronic burukutu consumption had statistically significant impact on ALP, AST and Albumin liver biomarkers; while chronic consumption of commercial beer had statistically significant impact on AST only. This implies that chronic consumption of burukutu alcoholic beverage and beer has impact on the liver biomarkers but burukutu seems to have more effect. This may be due to its relatively high alcoholic content because its fermentation process during production is local and unstandardized. In addition, burukutu may have detrimental effect on the health of the bones. Hence, chronic consumption of these alcoholic drinks should be discouraged.

Conflict of Interest

All authors disclose that there was no conflict of interest.

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