SOME TRACE ELEMENTS AND LIVER ANALYTES IN CONSUMERS OF A LOCAL ALCOHOLIC BEVERAGE (BURUKUTU)

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Abstract

Aim: Trace elements, such as zinc and copper, have been reported to affect liver function. This study was therefore designed to determine the effect of "burukutu" (a local alcoholic beverage) on the serum levels of zinc and copper and to compare this with that of controls.

Method: A total of 96 age-matched adult males made up 56 "burukutu" consumers, 20 refined alcohol consumers and 20 controls were recruited for the study. Serum zinc, copper, transaminases and total bilirubin were analysed.

Results: These indicated that there was no significant difference ($p>0.05$) in serum zinc and copper between consumers of "burukutu" and the controls. However, there was a significant difference ($p<0.05$) in serum copper between refined alcohol consumers and the controls as well as in the serum zinc and copper between "burukutu" and refined alcohol consumers. Zinc deficiency was observed in moderate to severe liver disease in refined alcohol consumers but not in "burukutu" consumers.

Conclusion: "Burukutu does not significantly affect serum levels of zinc and copper and by extension, the liver function of consumers.

Key words: Trace elements, Burukutu, Refined alcohol, liver disease.

Introduction.

It has long been known that excessive intake of alcohol leads to end organ damage in the liver. Alcohol consumption is second only to hepatitis, as the most common cause of liver disease in the developing countries[1]. In the Savanna region of Nigeria, different types of alcoholic drinks, including "Burukutu" are consumed.

"Burukutu" is an alcoholic beverage prepared locally from grains of corn and sorghum, the final product is reddish - brown in colour and the alcoholic content is about 3%. [2]

Zinc deficiency in liver diseases, including alcohol induced liver disease has been documented.[3, 4] Besides nutritional factors, many diseases and medical treatments may produce conditional zinc deficiency. Zinc deficiency in hepatic cirrhosis patients (e.g. alcohol and viral hepatitis) is characterized by low serum and hepatic zinc levels along with increased excretion of zinc. Multiple mechanisms for zinc deficiency or altered zinc metabolism in alcoholic liver disease among others include poor intake, increased urinary loss and depressed absorption[3]. Zinc deficiency in liver disease may affect levels of potentially hepatotoxic metals such as copper and iron. The role of intestinal metallothionein in zinc and copper bioavailability helped to clarify potential mechanisms for these mineral-mineral interactions.[4, 5] High zinc consumption induced intestinal metallothionein, which inhibits both zinc and copper absorption. However, metallothionein has a greater affinity for copper than zinc and high-dose zinc could induce copper toxicity.[6]

The metabolic functions of zinc and copper as an essential component of many metaloenzymes involved in virtually all aspects of metabolism cannot be over emphasized.[6] Zinc has been shown to be an important element in wound healing and many studies have implicated zinc as a necessary factor in the biosynthesis and integrity of connective tissue. Consequently adequate zinc nutrition is especially important for the post- surgical patient. [9] Furthermore, there is increasing evidence that zinc deficiency compromises immune response. Deficiency causes deficits in lymphocyte and thymocyte functions that are reversed by zinc supplementation. In renal patients undergoing regular haemodialysis, cellular immunity was found to be impaired in untreated patients compared with those receiving zinc supplementation.[9] Therefore, since there is evidence of zinc deficiency in alcoholic liver disease and zinc deficiency may lead to copper toxicity, this study is carried out to estimate the serum levels of these trace elements in consumers of this local alcoholic beverage. The integrity of the liver was assessed by estimating serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST) and total bilirubin.

Materials and Methods.

A total of ninety-six (96) low to middle - level income subjects were recruited for the study after an informed consent was obtained from each subject. Fifty-six (56) of them were consumers of not less than one calabash (approximately one liter) of "burukutu" per day for not less than five years. Twenty (20) were consumers of refined alcohol (not less than three bottles of beer per day) and 24 others who neither took refined alcohol nor "burukutu", also for not less than five years, served as controls. All

subjects were adult males and were age matched with age range of 21 to 56 years and living in Jos or environs.

Subjects who had a history of yellowish colouration of the eyes and urine were excluded. Also excluded were those who had right hypochondrial pains, those who either take burukutu or refined alcohol in less than 5 years prior to this study and those who take both burukutu and refined alcohol. Females were also excluded because they were too few in number.

All blood samples were collected at the time convenient to subjects. 5.0 ml of blood was taken from each subject under aseptic conditions into dry, plain bottles, allowed to clot and then centrifuged at 3000rpm for 5 minutes and sera separated into clean dry bottles and stored at $-20^{o}C$ until analyzed within 72hrs of sample collection.

Both serum copper and zinc were measured by atomic absorption spectrophotometry.[8]

Results.

Table 1 shows the mean and standard deviation (SD) of the trace elements and metabolites analyzed in all the three categories of subjects, and shows the inverse relation of zinc and copper.

Table 1:Mean (SD) of assayed parameters in consumers of "burukutu" and refined alcohol and the control subjects.

Analytes	"Burukutu" consumers	Refined alcohol consumes	Controls
Zinc (mmol/L)	0.049 (0.020)	0.060 (0.016)	0.050 (0.020)
Copper (mmol/L)	0.023 (0.010)	0.017 (0.005)	0.023 (0.006)
[a]ST (I.U)	13.82 (6.08)	16.45 (4.90)	11.03 (4.15)
ALT (I.U)	13.12 (8.70)	13.46 (4.08)	12.79 (4.79)
Bilirubin (umol/L)	10.50 (9.06)	12.17 (9.00)	8.40 (4.49)

[a]AST - Aspartate aminotransferase, [b]ALT - Alanine aminotransferase.

Table 2 shows comparisons of the variables between "burukutu" consumers and the controls. There was no significant difference ($p>0.05$) in either serum zinc or copper between the two test groups, The means of AST, ALT, and total bilirubin were generally higher in burukutu consumers than in the controls, but the differences were not significant ($p>0.05$).

Table 2:Comparing analytes between consumers of "burukutu" and control subjects

Analytes	"Burukutu" consumers	Controls	p values.	Inferences
Zinc (mmol/L)	0.049	0.050	>0.05	NS
Copper (mmol/L)	0.023	0.023	>0.05	NS
[a]AST (I.U)	13.83	11.03	>0.05	NS
[b]ALT (I.U)	13.12	12.79	>0.05	NS
Bilirubin (umol/)	10.50	8.40	>0.05	NS

NS = Not significant.

Table 3 shows the comparisons of the analytes between refined alcohol consumers and the controls. The result shows that there are significant differences ($p<0.05$) in the serum copper and AST between the two groups. The differences between the other analytes were not significant ($p>0.05$).

Table 3:Comparing analytes between consumers of refined alcohol and controls

Analytes	Refined Alcohol consumers	Controls	p values	Inferences
Zinc (mmol/L)	0.060	0.050	>0.05	NS
Copper (mmol/L)	0.017	0.023	<0.05	S
[a]AST (I.U)	16.45	11.03	<0.05	S
[b]ALT (I.U)	13.46	12.79	>0.05	NS
Bilirubin (umol/L)	12.17	8.40	>0.05	NS

NS = Not significant, S = Significant

Table 4 shows comparisons of the analytes between burukutu and refined alcohol consumers. The differences in serum zinc and copper between the two groups were significantly different ($p<0.05$), while the differences between the rest of the analytes were not significant (p.0.05).

Table 4:Comparing analytes between consumers of "burukutu" and refined alcohol

Analytes	Burukutu consumers	Refined Alcohol consumers	p values	Inferences
Zinc (mmol/L)	0.049	0.060	<0.05	S
Copper (mmol/L)	0.023	0.017	<0.05	S
[a]AST (I.U)	13.82	16.45	>0.05	NS
[b]ALT (I.U)	13.12	13.46	>0.05	NS
Bilirubin (umol/L)	10.50	12.17	>0.05	NS

Discussion.

Physiologically, zinc and copper absorption occurs mostly in the duodenum and proximal jejunum. The absorption process is active, energy-dependant and apparently mediated by specific zinc transport (binding) ligands. The comparative effects of refined alcohol and "burukutu" are highlighted and discussed.

Table 1 shows the similarity in zinc and copper serum levels of "burukutu" consumers and that of the controls. This may substantiate the assertion that "burukutu" is only dietary as claimed by its consumers and documented by Ogbonna[2]. The higher zinc values in those who take refined alcohol than in both "burukutu" consumers and the controls is contrary to the reported deficiency of zinc in liver diseases, including alcoholic liver disease in consumers. However when the zinc level of 0.06mmol. /L was compared to that of the controls (0.05mml. /L), there was no significant difference. Zinc deficiency is usually associated with hepatic cirrhosis patients (e.g. alcoholism and viral hepatitis) and is characterized by low serum and hepatic zinc levels along with increased excretion of zinc. Concurrent with the zinc levels in both refined alcohol and "burukutu" consumers, were the inverse levels of copper. It was however noted that the ratio of Copper in "burukutu" consumers and controls (1:2)

were similar, while that between consumers of refined alcohol and controls (1:3) was higher. This suggests that the concentration of alcohol being consumed affects the zinc-copper ratio. It has been documented that serum copper increases in liver cirrhosis due to accumulation since excess copper, which would have been excreted via bile is retained. Alongside this, serum zinc decreases in the same condition caused either by alcoholism or viral hepatitis.[10]

The decreased level of copper in refined alcohol consumers when compared to the controls was significant and agrees with the findings of Ross[8]. This is consistent with the inverse relationship between zinc and copper[6]. Human copper deficiency has been associated with several conditions. It has been observed in pre-maturity, malnutrition, malabsorbtion and chronic diarrhoea. Hypocupremia has been associated with abnormal copper metabolism in liver diseases such as portal cirrhosis, billiary tract disease and hepatitis The increased Aspartate aminotransferase (AST) level in refined alcohol consumers in relation to the controls was also significant and has been similarly reported.[9] Alanine aminotransferase (ALT) and bilirubin levels in both refined alcohol and Burkutu" consumers, were not significantly different although they were higher than the control values. It was however expected that both AST and ALT are to be raised in alcoholic liver disease.[9] A further index of alcoholic liver disease is the AST/ALT ratio, but when this was calculated in all the groups, none of them had a ratio of up to 2:1, which is the minimum in alcoholic hepatitis.[9] Virtually all of them had a ratio of approximately 1:1. This implies that neither the liver of refined alcohol consumers nor that of "burukutu" consumers had been adversely affected enough to offset the serum levels of AST and ALT. Aminotransferase levels in alcoholic liver disease are reduced in proportion to the degree of liver damage since alcohol depletes vitamin- B_6 – dependent, pyridoxal-5-phosphate, an essential precursor of aminotransferase synthesis. The elevation in bilirubin was also not significant and like the AST and ALT, its measure of increase does not indicate a diagnosis of alcoholic liver disease.

It was therefore concluded, that the consumption of "burukutu", although unrefined, is less harmful to the liver than refined alcohol and this may be due to its lower alcoholic content. The duration of consumption and its effect on the serum levels of these trace elements and enzymes, which are metabolized by the liver, could not be ascertained in the course of this work.

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