

Full Length Research Paper

Drinking patterns: biochemical and haematological findings in alcohol consumers in Ile-Ife, Nigeria

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Effect of drinking patterns on biochemical and haematological parameters was conducted on 200 Nigerian men, categorized into non-drinkers (control), occasional, moderate and heavy drinkers, using standard techniques. Their ages ranged between 20 and 57 years. The values obtained for occasional and moderate drinkers showed no significant difference ($p > 0.05$) from those of non-drinkers based on their biochemical and haematological parameters. However, there was significant difference ($p < 0.05$) in the values obtained for heavy drinkers and those of other categories. This study showed that occasional and moderate drinking had no effect on biochemical and haematological parameters while heavy drinking had some effect. Some of the results in conjunction with the clinical history would also be useful in diagnosing and management of alcoholics.

Key word: Drinking patterns, alcohol, biochemical, haematological, parameters.

INTRODUCTION

Alcoholism represents one of the most serious worldwide socioeconomic and health problems. An alcoholic is a person who consumes an amount of alcohol capable of producing pathological changes (Criteria committee, 1972). The amount of alcohol capable of producing disease depends on a variety of factors, including genetic predisposition (Bailey et al., 1976), malnutrition (Mendenhall, 1984), and concomitant viral infections of the liver (Hall, 1985).

Marmot et al. (1981), Klatsky et al (1992), Fuchs et al. (1995) and Renaud et al. (1999) reported association of heavy alcohol intake with a significant increase of all-cause and non-cardiovascular mortality rates especially by cirrhosis, cancer and violent deaths. They also reported that all-cause mortality rates are lower for moderate drinkers than for non-drinkers, because of a

lower heart disease. This beneficial effect of moderate alcohol consumption might be explained by a rise of high density lipoprotein cholesterol (HDL-c) induced by alcohol consumption (Rimm et al., 1999), but also by other mechanisms such as alcohol anti-aggregation properties (Meade et al., 1985). Heavy alcohol intake was reported to be associated with an increase in blood pressure by Milon et al. (1982), Saunders (1987) and Marmot et al. (1994).

Acute or chronic alcohol consumption causes degeneration in different internal organs and systems of adults (Watabiki et al., 2000; Mezey, 1985; Persson et al., 1990; Benicky et al., 2000; Bralowsky and Garcia, 1999; Fortunato and Gates, 2000). In the same way, Denker and Ericksson (1998), Oyama et al. (2000) and Butters et al. (2000) reported the effect of maternal alcohol consumption on different organs and systems of the developing fetus. Important functional disorders of these organs and systems occur frequently because of these negative effects. Mezey (1985) stated that high alcohol intake is a known cause of diarrhoea, other gastro-intestinal symptoms and in advanced states,

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decreases in body weight. Generally, it is accepted that, in individuals with high alcohol consumption, malnutrition develops depending on the possible changes in intestinal absorption mechanisms and dysfunction of some organs such as the liver and the pancreas (Hornby, 1991; Metcalf, 1993; Mezey, 1985; Persson et al., 1990). Furthermore, maternal alcohol during gestation is known to cause fetal growth retardation in humans and laboratory animals (Lin, 1981; Leichter, 1989), an effect persisting for a long period after parturition (Sanchis-segura et al, 2000; Juarez et al., 2000).

It has been reported that medically diagnosed alcoholics can be differentiated from non-alcoholics using clinical laboratory tests. Moreover, distinguishing alcoholic from non-alcoholic liver disease has important implications for treatment and management (Ryback, 1982). Heavy drinking induces changes in some biological parameters such as γ -glutamyl transferase (GGT) (Whitehead et al, 1978; Yersin et al, 1995; Hoffmeister et al., 1999) or mean corpuscular volume (MCV) (Whitehead et al., 1978; Yersin et al., 1995), which are the most widely used among markers of excessive alcohol drinking, though their diagnostic accuracy remains controversial (Hoeksema and de Bock, 1993).

A large number of scientific papers have been published concerning biological markers of alcohol consumption in different parts of the world, but none have been reported at Ile-Ife. Hence we undertook this study to examine to what fashion biochemical and haematological parameters were affected in abstainers, occasional drinkers, moderate drinkers and heavy drinkers in Ile-Ife.

SUBJECTS AND METHODS

Subjects

This study was conducted on 200 adult Nigerian males categorized into four groups: none drinkers, occasional drinkers, moderate drinkers and heavy drinkers. Fifty subjects were in each group, their ages ranged between 20 and 57 (average 33.4) years. Questionnaires about socio-economic status, smoking habit, frequency, quantity and type of alcohol were filled for each subject. Frequency, quantity and type of alcohol intake were used to categorize the drinkers. Non drinkers were those that have not taken alcohol before in their life, occasional drinkers were those that took 1 or 2 bottles of beer during ceremonies (one or twice in a month, two or three months) moderate drinkers took 1 bottle of beer per day and heavy drinkers took 3 bottles or more of beer on daily basis. The volume of a bottle of beer is 60 cl, which is approximately 1 pint of beer. Smokers were excluded from the study.

Materials

20 ml of blood was obtained by clean venepuncture from each subjects. 2.5 ml was dispensed into fluoride oxalate bottle and 10 ml into lithium heparin bottle. 4.5 ml was dispensed into a bottle containing 0.5 ml sodium citrate (3.8%) and the remaining 3 ml dispensed into dipotassium ethylenediamine tetraacetic acid

(K₂EDTA).

Biochemical analysis

Blood glucose was determined from fluoride oxalate bottles, sodium (Na⁺), Potassium (K⁺), bicarbonate (HCO₃⁻), Chloride (Cl⁻), urea, uric acid, total protein, albumin, globulin, cholesterol, γ -glutamyl transferase (GGT), aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (Alk.phos) were determined from lithium heparin bottles using standard techniques

Haematological analysis

Haemoglobin (Hb), packed cell volume (PCV), white cell count (WCC) (total and differential) and platelets were determined from K₂EDTA bottles using standard techniques. Mean cell volume (MCV), mean cell haemoglobin (MCH) and mean cell haemoglobin concentration (MCHC) were calculated. Prothrombin time (PT) and activated partial thromboplastin time were performed on citrated samples using standard techniques.

Statistics

The mean and standard deviation and the level of significance for the difference between means were computed by SPSS 6.

Table 1. Distribution of drinking pattern in the study population.

Category	No	%
None	50	25
Occasional	50	25
Moderate	50	25
Heavy	50	25

RESULTS

A total of 200 subjects were recruited for this study and were categorized into four with fifty in each group (Table 1). The effect of drinking patterns on biochemical parameters is shown in Table 2. The values obtained for glucose, cholesterol, GGT, ALT, AST and Alk.phos were significantly higher ($p < 0.05$) in heavy drinkers than values obtained for these parameters in other categories. The values obtained for sodium and potassium were significantly lower ($p < 0.05$) in heavy drinkers than in other categories. The values for the remaining parameters in all the groups were comparable.

The effect of drinking patterns on haematological parameters is shown in Table 3. As shown in the table, the values obtained for all the parameters with the exception of MCV showed no statistical significant difference ($p > 0.05$) in all the categories of alcohol consumers. The values obtained for MCV in heavy drinkers was significantly higher ($p < 0.05$) than the values obtained for other categories.

Table 2. Biochemical parameters in the four different categories of alcohol consumers.

Parameters	Alcohol intake			
	None	Occasional	Moderate	Heavy
Glucose (mmol/L)	4.1±0.23	4.1 ±0.71	4.1 ± 0.45	5.0 ± 0.21
Sodium (mmol/L)	134 ± 2.70	134 ± 3.01	135 ± 3.13	124 ± 3.25
Potassium (mmol/L)	3.7±0.24	3.7±0.33	3.7 ± 0.66	3.3 ± 0.93
Bicarbonate (mmol/L)	25 ± 3.24	24 ± 3.69	25 ± 3.78	25 ± 3.91
Chloride (mmol/L)	99 ± 5.32	98 ± 6.11	98 ± 6.48	100 ± 6.39
Urea (mmol/L)	3.4 ± 0.15	3.4 ± 0.62	3.4 ± 0.85	3.4 ± 1.10
Uric acid (mmol/L)	0.21 ± 0.01	0.21 ± 0.01	0.21 ± 0.01	0.2 ± 0.02
Total protein (g/L)	75 ± 3.5	76 ± 3.9	75 ± 4.1	76 ± 3.7
Albumin (g/L)	45 ± 2.10	46 ± 2.20	45 ± 2.31	46 ± 2.16
Globulin (g/L)	30 ± 2.19	30 ± 2.15	30 ± 2.31	30 ± 1.39
Cholesterol (mmol/L)	4.0 ± 0.21	4.0 ± 0.27	4.2 ± 0.31	6.0 ± 0.89
GGT (IU/L)	9.5 ± 0.75	9.4 ± 0.63	9.5 ± 0.70	18.1 ± 0.87
ALT (IU/L)	7.3 ± 0.67	7.2 ± 0.51	7.3 ± 0.42	10.7 ± 0.81
AST (IU/L)	7.9 ± 0.62	7.8 ± 0.39	7.9 ± 0.43	12.0 ± 0.9
Alk.phos (IU/L)	95 ± 3.90	94 ± 3.75	95 ± 4.10	124 ± 3.69

n = 50 for each category.

Table 3. Haematological parameters in the four different categories of alcohol consumers.

Parameters	Alcohol intake			
	None	Occasional	Moderate	Heavy
Haemoglobin (g/dl)	14.4 ± 1.3	14.3 ± 1.3	14.5 ± 1.2	14.8 ± 1.2
PCV (%)	44.6 ± 4.2	43.9 ± 4.7	44.2 ± 3.7	45.3 ± 3.8
RCC (x 10 ⁶ / mm ³)	4.991 ± 5672.3	4.983 ± 5231.4	4.899 ± 4911.2	4.997 ± 5031.1
WCC (mm ³)	5543.3 ± 994.2	4666.7 ± 2065.4	4516.7 ± 2825.6	4733.3 ± 1400.6
Neutrophils (%)	62.1 ± 4.5	60.2 ± 7.3	60.7 ± 5.8	62.6 ± 5.3
Lymphocytes (%)	36.0 ± 4.6	37.4 ± 7.3	36.8 ± 5.3	35.7 ± 4.8
Eosinophils (%)	1.7 ± 0.8	2.4 ± 1.2	1.5 ± 0.8	2.4 ± 1.7
Monocytes (%)	3.0 ± 1.8	1.9 ± 0.9	3.0 ± 1.8	2.8 ± 1.7
Basophils (%)	1.0 ± 0.00	1.0 ± 0.00	1.0 ± 0.00	1.0 ± 0.00
Platelets (mm ³)	227933.33 ± 56898.5	243033.3 ± 76519.3	211733.3 ± 49906.8	217966.8 ± 41736.0
MCV (µm ³)	85.3 ± 9.7	84.9 ± 8.8	84.9 ± 9.1	89.7 ± 9.7
MCH (pg/L)	28.5 ± 2.9	27.4 ± 3.5	28.4 ± 4.1	28.9 ± 4.3
MCHC (%)	32.3 ± 1.8	32.7 ± 0.9	1.9 ± 1.9	32.4 ± 1.8
PT (seconds)	13.5 ± 0.7	13.1 ± 1.4	13.7 ± 1.3	13.2 ± 1.3
APTT (seconds)	34.1 ± 3.3	34.7 ± 3.7	34.7 ± 2.8	34.7 ± 3.5

N = 50 for each parameter category.

DISCUSSION

The findings of this study have shown the effect of drinking patterns on biochemical and haematological parameters in alcohol consumers in Ile-Ife. Conigrave et al. (1995) stated that a variety of blood tests have been used to aid the assessment of drinking history and that more recently, laboratory tests based on urine, breath

and sweat analyses have been investigated. However, there has been a great deal of controversy over the usefulness of these markers. Many conventional tests have only limited sensitivity and specificity, and there have been doubts whether there is sufficient benefit to warrant their use.

From our work, the values obtained for biochemical and haematological parameters in occasional and moderate

drinkers showed no significant difference ($p > 0.05$) to those obtained for non-drinkers which served as control group. This may mean that occasional and moderate drinking has no effect on blood biochemistry and haematology. However, in heavy drinkers, there were significant differences ($p < 0.05$) in some of the biochemical and haematological results when compared to those of abstainers, occasional and moderate drinkers. The values obtained for glucose, cholesterol, GGT, ALT, AST and Alk.phos were significantly higher and sodium and potassium were significantly lower ($p < 0.05$) in heavy drinkers than in other categories. Some of the findings are comparable to previous reports on alcohol intake and biochemistry, especially GGT. Sharper et al. (1985) stated that the marked influence of alcohol consumption on GGT is well recognized and that no study has identified a more sensitive single biochemical marker of alcohol consumption. The increases observed in other liver enzymes could be due to the effect of alcohol on liver. This was recognized by Nelpas and Berthold (1991), Seitz et al. (1992) and Reinke and Mccay (1997) who observed that alcohol affects primarily the liver in humans. Whitehead et al. (1978) and Chalmers et al. (1981) in recognizing the effect of alcohol on liver enzymes also suggested that other liver enzyme tests should be considered as additional markers of heavy alcohol consumption. Significant increase in AST activity in acute alcohol intake was also reported by Marway et al. (1993). Significant reduction in sodium levels observed in heavy drinkers is consistent with the findings of Shaper et al. (1985) and Marway et al. (1993). Shaper et al. (1985) reported that alcohol intake has highly significant positive associations with globulin, potassium, haemoglobin, PCV, WCC and calcium; highly significant negative associations with urea, RCC, sodium and creatinine; and no association with total cholesterol. Our findings with respect to potassium, haemoglobin and PCV were comparable to their results. In our study, the MCV was significantly higher ($p < 0.05$) in heavy drinkers than in other categories. Elevated MCV in heavy drinkers have been reported by previous workers (Vincent Betaille, 2003). All other haematological parameters in this work showed no statistical significant difference in all categories of alcohol consumers when compared with non-drinkers (control).

In conclusion, heavy drinking have been shown to affect some biochemical haematological parameters while occasional and moderate drinking had no effects, hence some of the parameters that had association with heavy drinking could be used in conjunction with clinical history for the diagnosis and management of alcoholism.

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REFERENCES

- Bailey RJ, Krasner N, Feddleston ALW (1976). Histo compatibility antigens, autoantibodies, and immunoglobulins in alcoholic liver disease. *Br. Med. J.* 2: 727-729.
- Benicky J, Nikodemova M, Scsukova S, Zorad S, Strbak V (2000). Four-week ethanol drinking increases both thyrotropin-releasing hormone (TRH) release and content in rat pancreatic islets. *Life Sci*, 66: 629 – 39.
- Butters NS, Gibson MA, Reynolds JN, Brien JF (2000). Effects of chronic prenatal ethanol exposure on hippocampal glutamate release in the postnatal guinea pig. *Alcohol* 21: 1 – 9.
- Conigrave KM, Degenhardt LJ, Whitfield JB Saunders JB, Helander A, Tabakoff B (2002). CDT, GGT and AST as markers of alcohol use: the WHO / ISBRA collaborative project. *Alcohol Clin. Exp. Res.* 26: 332-339.
- Criteria Committee, National Council on Alcoholism (1972), Criteria for the diagnosis of alcoholism. *Ann. Intern. Med.* 77: 249 – 258.
- Fortunato F, Gates LK (2000). Alcohol feeding and lipopolysaccharide injection modulate apoptotic effectors in the rat pancreas *in vivo*. *Pancreas* 21: 174- 180.
- Fuchs CS, Stampfer MJ, Colditz GA, Giovannuchi EL, Manson JE, Kawachi I, Hunter DJ, Hankinson SE, Hennekens CH, Rosner B, Speizer FE, Willett WC (1995). Alcohol Consumption and mortality among women. *New Engl. J. Med.* 332: 1245-1250.
- Hall P editor (1985). *Alcoholic liver disease: pathobiology epidemiology, and clinical aspects*, New York, Wiley & Sons.
- Hoeksema HL, de Bock GH (1993). The value of laboratory tests for the screening and recognition of alcohol abuse in primary care patients. *J. Family Practise* 37: 268 – 276.
- Hoffmeister H, Schelp FP, Mensink GBM, Dietz E, Bohning D (1999). The relationship between alcohol consumption, health indicators and mortality in the German population. *Int. J. Epidemiol.* 28: 1066 – 1072.
- Juarez J, Barrios De Tomasi E, Vazquez C (2000). Alcohol treatment during lactation produces an advance in the onset of puberty in female rats. *Alcohol* 21: 181-5.
- Klatsky AL, Armstrong MA, Friedman GD (1992). Alcohol and mortality. *Annals of Internal Medicine*, 117: 646 – 654.
- **Lin GW (1981). Fetal Malnutrition: a possible cause of the fetal alcohol syndrome. *Prog. Biochem Pharmacol*, 18: 115-121.
- Marmot MG, Elliot P, Shipley MJ, Dyer AR, Ueshima H, Beevers DG, Stamler R, Kesteloot H, Rose G, Stamler J (1994). Alcohol and blood pressure: The INTERSALT Study. *Br. Med. J.* 308: 1263 –1267.
- Marmot MG, Shipley MJ, Rose G, Thomas BJ (1981). Alcohol and mortality: a U-shaped curve. *Lancet* 1: 580 – 583.
- Marway JS, Keating JW, Reeves J, Salisbury JR, Preedy VR (1993). Seromuscular and mucosal protein synthesis in various anatomical regions of the rat gastrointestinal tract and their response to acute ethanol toxicity. *Eur. J. Gastroenterol. Hepatol.* 5: 27 – 34.
- Meade TW, Vickers MV, Thompson SG, Stirling Y, Haines AP, Miller GJ (1985). Epidemiological characteristics of platelet aggregability. *Br. Med. J.* 290: 428 – 432.
- Mezey E (1985). Effect of ethanol on intestinal morphology In: HK Seitz.B. Kommerell (Eds). *Alcohol related diseases in gastroenterology*. Springer Verlag,Berlin.
- Milon H, Froment A, Gaspard P, Guidollet J, Ripoll JP (1982). Alcohol consumption and blood pressure in a French epidemiological Study. *Eur. Heart J.* 3: 59 – 64.
- Nelpas B, Berthold P (1991). Alcoholic liver disease. Current opinion in *Gastroenterol.* 7: 383 – 387.
- Oyama LM, Couto Rc, Couto GE, Damaso AR, Oller do Nascimento CM (2000). Ethanol intake during lactation II. Effects on pups' liver and brain metabolism. *Alcohol* 21: 201 – 206.
- Persson J, Berg NO, Sjolund K, Stenling R, Magnusson PH (1990). Morphological changes in the small intestine after chronic alcohol consumption. *Scand. J. Gastroenterol.* 25: 173 – 184.
- Reinke LA, Mccay PB (1997). Spin trapping studies of alcohol-initiated radicals in rats liver: influence of dietary fat. *J. Nutr.* 127: 899s – 902s.
- Renaud SC, Gueguen R, Siest G, Salamon R (1999). Wine, beer and

- mortality in middle-aged men from eastern France. *Arch. Internal Med.* 159: 1865 – 1870.
- Rimm EB, William P, Fosher K, Criqui M, Stampfer MJ (1999). Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *Br. Med. J.* 319: 1523 – 1528.
- Sanchis-Segura C, Correa M, Aragon CM (2000). Lesions on the hypothalamic arcuate nucleus by estradiol valerate results in a blockade of ethanol-induced locomotion. *Behav. Brain Res.* 114: 57 – 63.
- Saunders JB (1987). Alcohol: an important cause of hypertension. *Br Med. J.* 294: 1045 – 1046.
- Seitz HK, Xu Y, Simmanowski UA, Osswald B (1992). Effect of age and gender on in vivo ethanol elimination, hepatic alcohol dehydrogenase activity, and NAD⁺ availability in Fisher F344 rats. *Res Exp Med*, 1992: 205-212.
- Shaper AG, Pocock SJ, Ashby D, Walker M, Whitehead TP (1985). Biochemical and haematological response to alcohol intake. *Annals of Clinical Biochemistry* 22: 50 – 61
- Watabiki T, Okii Y, Tokiyasu T, Yoshimura S, Yoshida M, Akane A, Shikata N, Tsubura A (2000). Long-term ethanol consumption in ICR mice causes mammary tumour in females and liver fibrosis in males. *Alcohol Clin Exp Res*, 24 (4 suppl): 117s – 122s.
- Whitehead TP, Clarke CA, Whitfield AGW (1978). Biochemical and haematological markers of alcohol intake. *Lancet*, I; 978-981.
- Yersin B, Nicolet JF, Decrey H, Burnier M, Van Melle G, Pecoud A (1995). Screening for excessive alcohol drinking. *Arch. Internal Med.* 155:1907–1911.