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Relationship between duration of harmful alcohol consumption and serum lipids in some hospitalized Nigerian alcoholics undergoing detoxification treatment

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ABSTRACT

Heavy consumption of alcohol has been established to be the most common causative factor of liver disease, pancreatitis, upper gastrointestinal and neurological disorders. However, the influence of harmful duration of alcohol consumption on some "Syndrome X" risk factors and associated secondary cardiovascular dysfunction, is largely unknown. The study investigated the relationship of serum lipids in alcoholics and their duration of harmful alcohol consumption. One hundred and seven chronic alcoholics who were referred consecutively for detoxification were selected, and their drinking pattern was sufficient for them to be included in the study. History of alcohol abuse as well as drinking behaviour in the last 6 months were assessed by a semi-structured interview, and serum lipids were determined by standard procedures. Statistical analysis revealed that the duration of harmful alcohol consumption had the highest influence on the degree of brain injuries and withdrawal seizures. Serum cholesterol, triacylglycerol (TAG), HDL-,LDL- and VLDL-cholesterol, and blood pressure (BP) measures were elevated in alcoholics compared with control subjects. In recent times, increase in serum TAG and TAG/HDL-cholesterol ratio have been reported to be among the biochemical indicators of "Syndrome X", an emerging disease. Thus, the prevalence of this emerging metabolic disorder among alcoholics deserve further investigation, because it might be yet another alcohol-related disorder study suggests.

Keywords: Alcoholics, Pancreatitis, "Syndrome X", Cholesterol, Triacylglycerol.

INTRODUCTION

The production of alcoholic beverages appears to have been one of the earliest discoveries and it was recognized that among those who consumed it, were some who developed an uncontrollable necessity for continued use regardless of the consequences. Alcoholics therefore, are those excessive drinkers whose dependence on alcohol has attained such a degree that is shows a noticeable, mental disturbance or an interference with their bodily and mental health, their inter personal relationships, and their smooth socioeconomic functioning. Alcoholism, one of the most common chronic disorders in our society, promotes a plethora of diseases and the social health care costs of harmful alcohol consumption are enormous[1].

The metabolism of alcohol occurs principally in the liver where the zinc-containing metalloenzyme, alcohol dehydrogenase, located in the cytosol of the hepatocyte oxidizes the alcohol (ethanol) to acetaldehyde. About 75% of ethanol taken up by the liver is released as acetate into the circulation[2]. Acetate is then oxidized to carbon (iv)

oxide via the citric acid cycle. Intermediates in the metabolism of ethanol can initiate a wide range of biochemical alterations in man. So sufficient intake of alcohol over a fairly long period of time could disturb serum lipid levels. Population studies have revealed significant correlation between alcohol consumption and serum triacylglycerol (TAG) levels especially in hypertriglyceridaemic alcoholics[3]. Time-dependent changes in TAG levels have been noted during abstinence[4]. Acute doses, when given to habitual drinkers caused hypertriglyceridaemia[5]. An investigation into the HDL sub-classes in male chronic alcoholics showed the HDL concentration in alcoholics to be significantly higher than control, but returned to normal within one week of withdrawal[6]. Alcohol consumption caused an increase in HDL levels and thus, could be protective against cardiovascular disease[7]. However, only in the absence of other aetiologic factors. Acute doses of ethanol caused a mean increase of about two fold in the TAG content and a decrease in the cholesterol/TAG ratio in LDL[8].

Reports have also shown that excessive consumption of alcohol correlates with the prevalence of high blood pressure[9]. It has been, suggested that such prevalence could be due to the ethanol-induced hypertriglyceridaemia[10]. Although, alcohol consumption has been reported to increase plasma TAG and blood pressure[3], there are little information on the changes in cardiovascular parameters in serum of some alcoholics currently undergoing detoxification treatment. Therefore, this study was aimed at evaluating the relationship between harmful drinking duration and changes in serum lipids (the biochemical markers of cardiovascular complications) in some Nigerian alcoholics during and after detoxification.

MATERIAL AND METHODS

Choice of subject

One hundred and seven male chronic alcoholics referred consecutively for detoxification to Uselu Psychiatric Hospital, Uselu, Benin City, were selected for the study. History of alcohol misuse including drinking duration, drinking pattern and physical illnesses, were assessed by a semi-structured interview according to the documentation standards of the German Society for Addiction Research and Therapy[11]. Sixty-six alcoholics (mean age±SD;44.2±8.3 years; mean duration of harmful drinking: 4.2±2.3years), and 41 alcoholics (mean age±SD: 42.8±7.9years; mean duration of harmful drinking: 8.6±2.1years) constitute group A and B patients, respectively.

Seventy-two age- and sex- matched, non-alcohol drinkers in apparent good health were included as control subjects. All the subjects (107 alcoholics and 72 control individuals) under-went comprehensive clinical examination, and were tested on admission and three weeks later.

Sample collection

Five millilitres (5ml) of fasting intravenous blood sample was collected from each subject using sterile needle and syringe into plain sterile bottle, centrifuged and the supernatant (serum) was collected and labeled, ready for analysis.

Method used for serum sample analysis

Determination of the serum total cholesterol was done using the Allain *et al*.[12] enzymatic-colorimetric method. Trinder[13] GPO-PAP enzymatic colorimetric method was used to analyze the serum TAG levels in the samples. The HDL-cholesterol was estimated using the Burstein and Mortin[14] enzymatic-colorimetric method. The Friedwald *et al*.[15] formulae was used to estimate the serum levels of VLDL-and LDL-cholesterol. The commercial kits containing the reagents used for these laboratory analyses were supplied by Reactivos Cromatest Laboratories, Knickerbocker, SAF, Spain. Blood pressure was measured by sphygnomanometer (ACCOSSON MERCURY, CE 0120) in a well seated position after some few minutes of rest and prior to sample collection.

Statistics

All statistical calculations were performed using the SPSS-PC programme package (version 7.5).

RESULTS AND DISCUSSION

The proportion of alcohol-related medical disorders was similar in both groups (A and B) of alcoholic patients (Table 1). However, group B

Table 1: Information on alcohol-related disorders and changes in serum lipid measures obtained from alcoholics and control (non-alcohol drinkers) subjects

Alcohol-Related Disorders	Group A Alcoholics (n=66)	Group B Alcoholics (n=41)	Control Subjects (n=72)
Alcohol-Related Disorders	33.3	31.7	0.00
Chronic gastritis (%)	07.6	12.2	0.00
Gastroinstinal bleeding(%)	31.8	36.6	0.00
Polyneuropathy (%)	39.4	41.5	0.00
Withdrawal seizures (%)	47.0	61.0	0.00
Pancreatitis (%)	34.9	39.0	0.00
Delirium (%)	40.9	43.9	0.00
Severe brain injuries (19.7%)	46.3	0.00	
Liver cirrhosis (%)	21.2	24.4	0.00
Alcoholic hepatitis	25.8	36.0	0.00
Oesophageal varices	09.1	12.2	0.00
Fatty liver	53.0	48.8	0.00
Contributory Factors			
Alcohol dose (g/kg)	2.02±0.5	2.08±0.4	-
Age (years)	44.2±8.3	42.8±7.9	43.6±8.6
Mean duration of harmful alcohol consumption (yr)	4.2±2.3	8.5±2.1	-
Laboratory Investigations in serum			
Total cholesterol (mmol/L)	5.53±1.42 (10.6)	5.80±1.80 (16.0)	5.00±2.20
Triacylglycerol,TAG (mmol/L)	1.89±0.05 (48.9)	2.28±0.03* (79.5)	1.27±0.13
HDL-cholestrol (mmol/L)	1.24±0.07 (33.3)	1.29±0.04 (38.7)	0.93±0.06
TAG:HDL-cholesterol ratio	1.52±0.24 (11.0)	1.77±0.28 (29.2)	1.37±0.19
LDL-cholesterol (mmol/L)	1.52±0.24 (11.0)	4.05±0.29 (6.0)	3.82±0.26
VLDL-cholesterol (mmol/L)	0.38±0.02 (52.0)	0.46±0.06* (84.0)	0.25±0.03
Systotic blood pressure (mmHg)	142.6±3.4* (29.5)	150.8±5.3* (37.0	110.1±3.2
Diastolic blood pressure (mmHg)	105.0±2.2* (32.9)	108.0±4.2* (36.7)	79.0±2.1

The data on serum laboratory investigations are expressed as Mean±SD of 'n' subjects.
Values in parenthesis are the percentage increase in alcoholics values compared with control subjects
*P<0.05 compared with control values
Data were generated during admission for detoxification, and those obtained after 3 weeks of admission were not significantly different (P<0.05)

alcoholics (patients with longer duration of harmful drinking: 8.5±2.1 years) showed a history of brain injuries, withdrawal seizures and erectile dysfunction more often than the group A alcoholics with mean duration of harmful drinking of 4.2±2.3 years.

In order to evaluate the influence of contributory factors, such as age, duration of harmful alcohol consumption, and estimated alcohol intake, on the occurrence of alcohol-related disorders, a stepwise logistic regression was performed. The statistical analysis revealed that higher doses of alcohol intake had the highest influence on the rate of withdrawal delirium, gastrointestinal bleeding and pancreatitis, the duration of harmful alcohol consumption on brain injuries and withdrawal seizures, and age on gastrointestinal bleeding, chronic gastritis, alcoholic hepatitis and liver cirrhosis. There was no significant contributory factor for oesophageal varices and fatty liver.

The measurement of laboratory parameters-serum lipids, yielded elevated mean levels of total cholesterol, triacylglycerol (TAG), HDL-, LDL- and VLDL-cholesterol, blood pressure (BP), and TAG/HDL-cholesterol ratio in all groups, but proportionately higher among the group B alcoholics. Increase in serum TAG and TAG/HDL- cholesterol ratio have been recently reported to be among the biochemical indicators of cardiovascular dysfunction – a secondary complication of "Syndrome X", an emerging disease [(16)]. The data obtained after three weeks of admission were not significantly different (Scheffe-test, $P<0.05$) from those obtained during admission.

From the results, it can be observed that in the alcoholics especially those with longer duration of harmful alcohol consumption, the mean TAG value was found to be significantly, higher when compared with the mean value of the control subjects. Elevated serum TAG level has been demonstrated in chronic alcoholics, in subjects with acute signs of intoxication, and in alcoholics with delirium tremens[17]. The association between hypertriglyceridaemia and alcohol intake has also been documented [8].

The mean serum total cholesterol levels for the alcoholics in both groups were moderately elevated compared with the control mean value. It has been demonstrated that neither an acute dose[5] nor a moderate intake[8] of alcohol has any appreciable influence on plasma total cholesterol. In two other studies of two separate groups of male alcoholics, only 6 out of 77 alcoholics[18] and 1 out of 38 alcoholics[6] had elevated cholesterol levels. Our findings are in agreement with these earlier observations, and they all suggest that alcohol does not seem to elevate plasma total cholesterol levels. Any observable slight increase may be due to alcohol-induced increase in the cholesterol fraction found in HDL, which further suggests the implication of other artherogenic risk factors.

The systolic and diastolic B.P. in both groups of the alcoholics investigated were found to be significantly higher, compared with the control mean values at the 5% probability level. There was a good correlation between TAG and B.P ($r=0.905$; $P<0.05$). This is in agreement with previous reports that have associated excessive alcohol intake with high B.P.[9]. There were no significant differences ($P<0.05$) in HDL,-LDL- and VLDL- cholesterol mean values for the alcoholic and control subjects. The statistical relationship between these fractions and B.P. was weak and negative for the HDL-component in the alcoholics. Excess alcohol consumption causes an increase in HDL levels, and so, could be protective against cardiovascular disease[19] since, high levels of HDL prevent the accumulation of cholesterol in the plasma by transporting excess cholesterol from the peripheral tissues to the liver for appropriate metabolism. However, such therapy could only be effective if increase in plasma cholesterol is the sole and only aetiologic risk factor involved. It has been shown that high levels of LDL-cholesterol will cause an increase in plasma cholesterol[10] and high VLDL-cholesterol levels caused an elevation of plasma TAG levels[4]. However, when the level of HDL-cholesterol increases, serum cholesterol decreases.

The metabolic relationship between "Syndrome X" risk factors and alcohol consumption

is being refined [20]. Increase in blood TAG concentrations have been shown to reduce the number of insulin receptors[21]. High TAG alone increases the risk of heart attack nearly three-fold, and people with the highest TAG to HDL-cholesterol ratio had 16 times the risk of heart attack as those with the lowest ratio[16]. Some researchers now think that plasma TAG levels may actually be more important than cholesterol level in establishing heart disease risk-a complication of "Syndrome X". Increase in VLDL-cholesterol has been linked to free radical generation. This study suggests that "Syndrome X" may be yet another alcohol-related medical disorder. Therefore, the prevalence of the emerging disorder- "Syndrome X", deserve further investigation among alcoholics.

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