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Evaluation of the effects of Maternal alcohol consumption on some selected biochemical parameters

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ABSTRACT

Alcohol ingestion can result in fetal disturbances in the offsprings. In this research, the effects of maternal alcohol consumption on the biochemical parameters were carried out. Three groups A, B and C were used in this study. Group A was the control group while groups B and C served as test groups. Group B was exposed to 16 to32g of alcohol daily for 3months while group C was administered 60 to 80g of alcohol within the same period. A total of 90 people from the three groups were used in the investigations. 3mls of blood was collected from the vein. Vitros 360 chemistry autoanalyzer was used for biochemical assay. Mean values of bilirubin were significantly elevated ($p<0.05$) in test groups (B & C) compared with control while the mean value of glucose were significantly increased. Furthermore, while the mean value of glucose concentrations were significantly increased ($p<0.05$), mean values of cholesterol and creatinine levels were only significantly increased in week 5 in the test groups compared with control ($P<0.05$). However, there was no significant elevation ($p>0.05$) in mean value of urea in the test groups in all the weeks understudy. These investigations have therefore demonstrated that chronic alcohol consumption has some deleterious and toxic effect on some biochemical parameters and has revealed potential risks in the consumers. Therefore, there is the need for controlled alcohol consumption.

Keywords: Alcohol, Consumption, human, Biochemical and Parameters.

INTRODUCTION

Alcoholism has become a serious socio economic and health problems [1,2,3]. Acute and chronic alcohol misuses have been shown to cause reproductive function derangements in human and experimental animals [4,5,6]. Alcohol ingestion during pregnancy can result in fetal disturbances in their off-springs [7,8]. In experimental animal models the syndrome is characterized by retardation of fetal life. Fetal alcohol syndrome is a pattern of mental and physical defects that can develop in fetus in association with high levels of alcohol consumption during pregnancy [9,10]. Damage to the central nervous system (CNS) has emerged as one of the most serious consequences of fetal alcohol syndrome [11,12] Alcohol crosses the placental barriers and can stunt fetal growth or weight, create distinctive facial stigmata, damage neurons and brain structures which can result in physiological or behavioral problems and causes other physical damage [13,14]. Studies in human and primates on brain structure and function now strongly suggest that maternal alcohol consumption can affect fetal brain structure and functions [15,16]. The main effect of fetal alcohol syndrome is permanent central neurons system damage especially to the brain. Developing brain cells and structures can be malformed. Alcohol exposure can create cognitive and functional disabilities including poor memory, attention deficits and impulsive behaviour [17,18]. This could cause the disruption of hypothalmic pituitary gonadol axis which plays a regulatory role in reproduction. Gonadotropin stimulation starts from release of leutenising hormone releasing hormone (LHRH) from hypothalamus and is released to the pituitary. In response the pituitary produces luteinizing hormone (LH) and follicle stimulating hormone FSH. LH stimulates testosterone production

and FSH plays role in sperm maturation [19].
Fetal gonadotropins provide the stimulus for the maturation of Fetal Leydig cells and onset of testosterone secretion [20]. In adults, alcohol is known to disturb many of the rhythms of neuroendocrine functions, probably through its actions on the hypothalamus [21]. This could be as a result of disruption of the hypothalamic pituitary gonadal axis. In chronic alcohol groups with male rats it was observed that testicular, prostate and seminal vesicle atrophy occurs in addition to lowered plasma testosterone. Some biochemical studies revealed among others that chronic alcohol groups produce increase in β endorphin, prolactin and produces decrease in LH hormone in male [22]. Heavy alcohol intake had been associated with a significant increase of all-cause and non-cardiovascular mortality rates especially by cirrhosis, cancer and violent deaths [23]. Alcohol consumption has been found to have considerable effect in the liver [24]. This has aroused serious medical interest. Alcohol has been found to injure the nervous system by inhibiting growth processes. It can attack the brain function and may have metabolic effects on the liver function enzymes-the liver being one of the most important organs in drug metabolism [25]. Alcohol consumption may bring about changes that may alter the release of aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP). Heavy drinking is associated with major liver diseases such as fatty liver, alcoholic hepatitis and cirrhosis [8]. Moderate consumption of alcohol can help improve cardiovascular health. This beneficial effect of moderate alcohol consumption might be explained by arise of high density lipoprotein cholesterol (HDL-c) induced by alcohol consumption [11]. Heavy drinking can increase the amount of triglycerides in the blood. This can increase the risk of heart diseases, high blood pressure, obesity and type 2 diabetes mellitus [22]. Studies have shown that subjects with alcohol consumption has high level of serum triglycerides, high density lipoprotein cholesterol (HDL),uric acid, estimated creatinine clearance rate (CCr) and glomerular filterate rate (GFR) values than none drinkers [12]. Also alcohol reduces functional hepatic nitrogen clearance and acutely down regulate urea synthesis in normal men [13].
One of the pharmacological actions of alcohol is to lower blood glucose. While moderate amounts of alcohol can cause blood glucose to rise, excess alcohol can actually decrease blood sugars [14]. Alcohol has direct effect on the glucose levels of diabetics. Alcohols like beer and sweet wine contain carbohydrate and may raise the glucose level of a diabetic. Acute or chronic alcohol consumption causes degeneration in different internal organs and systems of adults [15]. Also the effect of maternal alcohol consumption on different organs and systems of the developing fetus have been reported by [20,21]. A Large number of research had exposed the deleterious effect of alcohol consumption during pregnancy on both the mother and fetus [20]. Following reported increase in alcohol consumption in Nnewi and environs, this research is designed to investigate the effects on some biochemical parameters on both moderate and heavy drinkers.

MATERIALS AND METHODS

RESEARCH DESIGN

The volunteers were grouped into three (A, B, & C) with 30 people in each group. Group A served as control while groups B and C were used as test groups. During investigation, the control group A was fed with water and normal diets while groups B and C where fed with 16 to 32grams of ethanol and60 to 80 grams respectively for 5 weeks.

Sample Collection and Analysis

About 3mls of blood samples were collected by venous puncture and placed into plain tubes. The samples were allowed to clot and centrifuged at 3000 g for 10 minutes. The serum was separated into plain test tubes for biochemical analyses. The samples were analyzed for total bilirubin, cholesterol, glucose, urea and creatinine levels using Vitros auto analyzer.

Estimation of Biochemical Parameters

All biochemical analyses in this work was carried out with Vitros 360 auto analyzer according to manufacturers instructions for use. This system makes use of thin film analyzer that uses dry reagent spread in extremely thin layer on a plastic slide to which the sample is added. When light is incident on the slide, light passes through different layers of the slide, namely the spreading layer, reagent layer, indicator layer and support layer. The amount of light that enters the slide is different from the amount that leaves the slide due to absorption of light at the reagent layer. The difference in light intensity is directly proportional to quantity of analyte present in the sample and is used to compute the value of the analyte. Each biochemical parameter uses a different slide with the apparatus.

Statistical Analysis

Data collected were subjected to statistical analysis using the analysis of variance (ANOVA). Values were deemed significant if $p<0.05$.

RESULTS

Table 1: Comparism of mean total bilirubin levels of the control, moderate and heavy alcohol consumers (mg/dl).

Age in weeks	**Control groups**	**Moderate alcohol exposed**	**heavy alcohol exposed**	**groups**
	Mean ± SD	mean± SD	Mean ± SD	**P value**
2	0.3000±.12	0.5638±.17	0.5387±.17	**0.0004**
3	0.2863±.09	0.4313±.16	0.6088±.15	**0.000**
4	0.3100±.07	0.3975±.12	0.7813±.21	**0.000**
5	0.2725±.09	0.3587±.08	0.8638±.14	**0.000**
6	0.2563±.08	0.2875±.07	0.9500±.24	**0.000**

Table 2

Comparism of cholesterol assay levels on the of the control, moderate and heavy alcohol exposed using mean ± SD (mg/dl).

Age in weeks	**Control groups**	**Moderate alcohol exposed groups**	**Heavy alcohol exposed**	**groups**
	Mean ± SD	Mean ± SD	Mean ± SD	**P value**
1	48.250±13.41	53.000±13.82	56.250±13.16	**0.501**
2	59.500±14.69	61.250±13.18	61.000±15.96	**0.967**
3	48.250±12.21	58.000±15.31	62.000±15.78	**0.175**
4	48.500±11.84	57.500±14.99	66.500±12.45	**0.041**
5	**64.500±5.21**	**73.250±7.09**	**70.500±8.40**	**0.059**

Table 3

Comparism of glucose levels on the control, moderate and heavy alcohol exposed in Albino rats using mean ± SD (mg/dl).

Age in weeks	**Control groups**	**moderate Alcohol exposed groups**	**Heavy Alcohol exposed groups**	
	Mean ± SD	Mean ± SD	Mean ± SD	**P value**
1	129.125±10.76	121.125±6.47	126.625±10.20	**0.238**
2	128.125±10.37	119.000±6.82	120.625±13.84	**0.217**
3	131.875±10.02	119.375±7.63	120.750±7.69	**0.015**
4	130.375±8.75	120.625±6.41	112.250±8.70	**0.001**
5	**131.750±12.23**	**123.750±8.97**	**107.625±14.28**	**0.002**

Table 4

Comparism of mean serum urea levels of the control, moderate and heavy alcohol exposed (mg/dl).

Age in weeks	**Control groups**	**Moderate alcohol exposed group B**	**Heavy alcohol exposed groups C**	
	Mean ± SD	Mean ± SD	Mean ± SD	**P value**
2	17.0375±1.35	17.6000±1.25	17.3500±1.50	**0.716**
3	17.2375±1.54	18.9750±2.80	19.0625±4.20	**0.416**
4	17.3250±1.69	18.1250±1.97	17.4375±1.13	**0.580**
5	17.8625±.83	17.6625±1.41	17.6875±1.04	**0.927**
6	17.475±1.03	17.2875±.76	16.6750±1.93	**0.470**

Table 5

Comparism of creatinine values of control, moderate and heavy alcohol exposed i using mean ± SD (mg/dl).

Age in weeks	**Control groups**	**Moderate alcohol exposed groups B**	**Moderate Alcohol exposed group C**	
	Mean ± SD	Mean ± SD	Mean ± SD	**P value**
2	0.7939±.27	0.9088±.19	1.0750±.39	**0.189**
3	0.7812±.21	0.8938±.34	1.0200±.34	**0.315**
4	0.7687±.15	1.0250±.40	1.2563±.50	**0.057**
5	0.8038±.20	0.8250±.35	1.2063±.42	**0.045**
6	**0.8138±.20**	**0.8713±.35**	**1.2225±.52**	**0.089**

DISCUSSION

These investigations have apparently demonstrated that alcohol consumption can cause a lot of changes in some biochemical parameters. Bilirubin estimation which is an important parameter in hepatitis and other liver diseases was investigated. In this work it was observed that there was an elevation of bilirubin ($P<0.05$) in all the weeks under study for both moderate and heavy alcohol exposed groups B and C. It was observed that bilirubin levels differ in both group B and C in week 2 compared with control. It has been demonstrated that many diseases of the liver are accompanied by jaundice [8].The diseases of the liver include fatty liver, alcoholic hepatitis and cirrhosis [11]. Volunteers used in this investigation were classified as heavy drinkers. They are groups that took alcohol consistently on daily basis [12]. From the pattern of results the bilirubin levels of those exposed to alcohol moderately appeared significantly lower when compared with rats exposed to alcohol heavily. It has been demonstrated that not all heavy drinkers develop alcohol hepatitis or cirrhosis [15]. These findings suggest that other factors ranging from hereditary to environmental may affect bilirubin level.

Cholesterol mean values were elevated in group C. Liver mitochondria can convert acetate to acetylCoA in a reaction requiring ATP and catalyzed by the enzyme thiokinase. However further processing of the acetyCoA by the citric acid cycle is blocked, because NADH inhibits two important regulatory enzymes isocitrate dehydrogenase and α-Ketoglutarate dehydrogenase. This results in accumulation of acetylcoA, a precursor in the cholesterol synthesis. Chronic alcohol ingestion will result in accumulation of acetylCoA and increases cholesterol synthesis [11]. Findings in week 5 agree with earlier studies that moderate consumption of alcohol increases high density lipoprotein cholesterol (HDL) by as much as 4mg/dl within 24 hours. According to Adam, (2011), HDL protects against arteriosclerosis and heart attack. It has been established that excessive alcohol consumption increases the amount of triglycerides which can increase low density lipoprotein.LDL increases the risk of cardiovascular disease, high blood pressure and obesity [7,9,17].

Considering the fact the volunteers were exposed to heavy alcohol, one expected that the cholesterol level should be high in all the test groups as carried out by other researchers but the results in this investigation was on the contrary except in weeks 4 and 5 of group C where high cholesterol values were recorded. The reason for this picture is not understood. [12] reported that alcohol consumption at a level that does not affect calorie intake increases cholesterol concentration. The glucose levels of the alcohol exposed group B in weeks 2 and 3 did not reveal any significant change when compared with the control group. But alcohol affected the glucose levels in weeks 4, 5 and 6. Ethanol is metabolized primarily in the Liver. This metabolism occurs by two pathways. The first pathway comprises catalysis of ethanol to acetaldehyde by alcohol dehydrogenase. Acetaldehyde is further metabolized by aldehyde dehydrogenase to acetate. The two steps lead to accumulation of NADH as a result of continuous alcohol consumption. This high concentration of NADH inhibits gluconeogenesis by preventing the oxidation of lactate to pyruvate. The high concentration of NADH will lead to accumulation lactate and the consequences may be hypoglyceamia and lactic acidosis [8]. Previous works revealed that alcohol consumption has effects on glucose levels of those exposed directly to alcohol and this depends on the type of alcohol consumed and also affects the sugar levels in diabetics [20]. The liver performs the function of glycogenesis and glucogenolysis. This result is similar to those of [25]. Alcohol therefore reduces glucose levels on both heavy and moderate drinkers. This finding suggests that alcohol could be diabetogenic. Studies carried out by [11]

revealed that consumption of alcohol in diabetic rats decreases body weights. Though this work is not specifically on diabetic rats but has demonstrated that alcohol consumption both moderately and heavily can be deleterious on the health of consumers due to possible inhibition of gluconeogenesis.

In this study urea levels of the groups B and C showed no differences when compared with control group. According to [8] alcohol consumption acutely down regulates urea synthesis in healthy volunteers, favouring nitrogen preservation. These findings did not differ from our results because all our findings from moderate and heavy alcohol posed groups showed no differences ($P>0.05$) with the control groups. High urea levels are implicated in renal failure, dehydration, diabetes and diet. This suggests that alcohol may affect urea level only when it may cause some physiological changes like renal failure, dehydration and stress related situations [11]. This investigation cannot be reconciled with reported nitrogen wasting of chronic alcoholics. The research carried out observed that alcohol exposure to volunteers did not statistically affect the creatinine levels in weeks 2, 3, 4 and 6. There was an elevation of creatinine levels in week 5 of heavy alcohol exposed (Group C).

These findings did not suggest reason for the change in week 5. However there is strong suggestion that factors other than alcohol can influence such changes. Such factors are likely to be dehydration, renal impairment of some volunteers in that particular group and probably genetic. The association between alcohol consumption and renal function is poorly understood [6]. There was an evidence that chronic alcohol consumption may cause direct damage to the kidneys [11]. It may also indirectly alter renal function by elevating blood pressure.

Alcohol consumption was independently and significantly associated with a higher level of estimated Ccr and GFR as well as serum urea [16]. This assertion is in line with the pattern presented in week 5 of table 5 where we noticed significant changes in heavy alcohol exposed against control group. This implies that constant exposure to alcohol may affect creatinine levels as a result of blood pressure which may cause renal dysfunction [20]. However in a study carried out by [13] it was concluded that alcohol intake has no effect in glomerular filteration rate and serum creatinine levels. [17] in a prospective study showed that alcohol intake has no long term adverse effect on renal function as assessed by calculating creatinine clearance rate Ccr and glomerular filtrate rate (GFR) and may in fact have a renoprotective effect in women with hypertension. This study is in line with our findings in weeks 2, 3, 4 and 6 where alcohol intake in both B and C groups show no significant increase on the creatinine levels when compared with control group.

CONCLUSION

Alcohol consumption have been shown to affect some biochemical parameters on users. This could expose them to serious health challenges. It has been demonstrated that acute or chronic alcohol consumption can affect the liver, bilirubin levels and most biochemical activities. Therefore, people are advised to consume alcohol very minimally to avoid health risks.

RECOMMENDATIONS

This work is limited to biochemical parameters, more research is recommended to determine the effect o alcohol consumption on female and male reproductive organs.

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