EFFECTS OF ALCOHOL ON OXIDATIVE PARAMETERS OF ALLOXAN INDUCED DIABETIC ALBINO RAT

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ABSTRACT

The effects of alcohol consumption on lipid peroxidation and antioxidant status were investigated in the alloxan induced diabetic rats. Plasma from the diabetic rats not treated with alcohol (DNT); diabetic rats treated with alcohol (DT) and non diabetic rats (ND) were analysed for their malondialdelyde (MDA) and vitamin C levels. Both the glucose level and the body weight were also studied. The mean weights of the rats in the different groups were the same until the onset of diabetes and alcohol ingestion when the weight decreased. After nine (9) days of alcohol supplementation, the DT rats weighed 114.00 ± 0.41 g, and the DNT rats weighed 121.00 ± 1.22 g while the rats in the controlled group weighed 146.33 0.14 g. The glucose levels for DT, DNT and ND were 29.56, ± 0.56, 28.81 ± 0.87 and 5.42 ± 0.19 nmol/l respectively. Analysis of the lipid peroxidation product (MDA) obtained showed a significant (P < 0.05) increase in MDA values from – DT rate (38.63 ± 3.88) nmol/ml to DNT rats (28.63 ± 1.38 nmol/lml), while MDA value for ND rats was 7.88 ± 1.38 nmol/l. Plasma vitamin C values of 0.62 ± 0.05 mg/100ml, 1.107 ± 0.13 mg/100ml and 1.79 ± 0.15 mg/100ml for DT, DNT and ND respectively were obtained.

Keywords: Alcohol, Antioxidant, Lipid peroxidation, Diabetes, Rat

INTRODUCTION

Diabetes mellitus has been considered an important health hazard because of the morbidity and mortality associated with it. The fact that it cannot be cured but only managed calls for a serious concern by patients and health workers.

Diabetes mellitus may be caused and or exacerbated by certain chemicals or compounds which elicit oxidative stress (Traverso *et al.,* 1999, Ogugua, 2000) in the exposed individual. On the other hand, antioxidants have been involved in the amelioration of oxidative stress – mediated pathologies (Halliwell *et al.,* 1992; Stern, 1993). Hence oxidative stress and antioxidants have been weighed side by side in diseases states including diabetes mellitus.

The natural quest for alcohol consumption has made it "a free for all drink" despite the obvious consequences of its acute and chronic intoxication (Nwodo, 1999). The morbidity and mortality of the diseases associated with alcohol intake is both a social and health problem and the complication of diabetes mellitus may be a double tragedy for alcoholic diabetics. Fatty liver, cirrhosis and hepatitis have been associated with high intake of alcohol (Ewa and Arthur, 1996; Nwodo, 1999). This suggests that liver damage may be as a consequence of alcohol ingestion. Presence of iron in beer has been implicated in the generation of reactive oxygen species and amplification of disease conditions associated with consumption of alcoholic beverages.

Cardiac arrhythmiasis has been associated with alcohol ingestion (Finch and Huebers, 1982; Ruskin, 1989). Thus alcohol ingestion can suppress the hearts pace and thus endanger lives. Alcohol consumption can increase pulse rate and blood pressure and hence decreases the strength of the pumping action of the heart (January and Fozzard, 1988). Some unexplained heart diseases could be due to chronic heavy drinking of alcohol. According to Belotsky *et al.* (1990) alcohol irritates the interior lining – mucosa – of the oesphagus, and induces stomach erosion causing inflammation and bleeding. Also alcohol causes diarrhoea through changes in intestinal motility and rate of propulsion of materials through the small intestine (Okeagu, 1999).

The inference from the above stipulates suggests that while normal persons may suffer complications of acute and chronic alcohol, ingestion diabetics could suffer more. The thirst for alcohol has made it such that even diabetics could not resist the taste and urge and in the rural settings diabetics and people prove to it consume alcohol without reservations. Hence the thrust of the research is to follow up diabetic animals ingesting alcohol by monitoring indices of oxidative stress – glucose level, lipid peroxidation product and antioxidant vitamin C. The outcome of the result may help in the management of diabetes mellitus.

MATERIALS AND METHODS

Eighteen albino rats obtained from animal house of the Department of Pharmacology and Toxicology, University of Nigeria, Nsukka were used for the experiment. These rats with average weight of 150 g were divided into three groups of six rats each and housed in stainless steel cages. They were fed with normal commercial chow and were allowed free access to water.

Table 1: Weight, Glucose, malondialdehyde and Vitamin C levels of diabetic and non-diabetic rats exposed to alcohol treatments

Group*	Weight (g) mean ± SD	Glucose level (mmol/l)	MDA nmol/ml Plasma	Vitamin C mg/100 ml
Non diabetic rats (ND)	146.33 ± 0.14	5.42 ± 0.19	7.88 ± 1.38	2.79 ± 0.15
Diabetic rats not treated with alcohol (DNT)	121.0 ± 1.22g	28.81 ± 0.87	28.63 ± 1.38	1.11 ± 0.52
Diabetic rats treated with alcohol (DT)	114.00 ± 0.41g	29.56 ± 0.56	38.63 ± 3.88	0.62 ± 0.52

**Results are mean ± SD; n = 6.*

The group A were the control rats while groups B and C rats were induced with diabetes mellitus. Group C rats were further treated with ethanol.

Diabetes was induced by intraperitonial injection of alloxan (200 mg/kg). The body weights of all the animals were determined before the induction of diabetes and during the experiment. Five millilitres volume of alcoholic beverage was given to each rat in group C daily for nine (9) days.

Blood glucose levels were determined daily using One Touch Blood Glucose Kit (Glucometer). At the end of the nine days the animals were sacrificed and other parameters determined as follows: Serum Malonyldialdelyde level was determined using the method of Albro *et al.* (1986) and Das *et al.* (1990) and while ascorbic acid (Vitamin C) level was determined using Tietz (1970).

RESULTS AND DISCUSSION

Table 1 showed that the normal rats had the highest weight while the diabetic rats had lower weight. Treatment with alcohol resulted to further loss in weight compared with the diabetic non-alcohol treated.. This suggests that diabetic condition could cause a reduction in weight and ethanol (beer) ingestion by diabetics compounded the problem. Decrease in weight of diabetic subjects had earlier been reported (Traverso *et al.,* 1999, Ogugua 2000). Generally, oxidative stress could lead to loss in weight which may be severed in the ethanol treated rats. Alcohol in this study increased oxidative stress which might have led to loss in body weight of the animals stressed.

Table 1 showed high levels of glucose and malondialdehyde in diabetic not treated rats (DNT) which further increased in alcohol treated diabetic rats (DT). There was above 2.6 % increased in blood glucose level of diabetic alcohol treated rats when compared with other treatments. Earlier reports proposed an overall reduction of blood glucose by alcohol (Nwodo, 1999). Prolonged ingestion of alcohol could trigger off excess production of reactive oxygen species leading to increased blood glucose level. Increased malondialdelyde level has been associated with increased glucose level (Reaven, 1995). The high MDA level in this work (Fig. 1) lay credence to this speculation. However, ethanol in low quantity may be antioxidative (hence may lower glucose level and oxidative stress index). Glucose autoxidation and increased oxidative stress has been reported (Hunt & Stocker 1990; Tukuncu *et al* 1998). The magnitude of reactive oxygen species production in the presence of ethanol may therefore modulate the level of glucose in such system. Generally, copious generation of reactive oxygen species could trigger off normal mechanism, in this case a reduction mechanism may be effected and hence elevation of blood glucose (Ogugua, 2000).

The vitamin C level was low in DNT rats compared with ND rats while DT rats had the lowest vitamin C levels (Table 1). In this system, vitamin C acted as an antioxidant and so was depleted in the process. Frei (1991) reported that vitamin C was the first antioxidant to be encountered during lipid peroxidation and so diminished in organic system. These findings were also corroborated by the report of Ogugua (2000) that Vitamin C diminished in diabetic rabbits monitored over time. For more information on the role of antioxidants in oxidative stress, both Ogugua (1994) and Fakoya *et al.* (1998) laid credence to the present findings.

Our results showed that oxidative stress became amplified in ethanol treated diabetics. Ethanol has been reported to induce oxidative stress and mediated lipid peroxidation; (Diluzo and Stefe, 1977; Bosch *et al.,* 1998; Ren *et al.,* 2000). The very low level of vitamin C in the alcoholic diabetics rats suggests aggravated depletion of vitamin C as it encounters free radicals. Thus the antioxidant status of the system is compromised in diabetics ingesting alcohol.

One may therefore re-iterate this point like a town crier that diabetics should avoid ingesting alcoholic drinks and at the same time suggest that supplementation with vitamin C or any antioxidant vitamin may help in the management of subjects with diabetes mellitus consuming alcohol.

People who are prone to diabetes (latent diabetes) may have it triggered off with alcohol ingestion while those who have developed diabetes would tend to severe complications. Hence medically challenged individual – diabetics should completely avoid alcoholic beverages.

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