Effect of chronic alcohol consumption on haematological and cardiohepatic function markers among commercial motor cyclists in Owerri, Nigeria

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ABSTRACT

It is a common practice among commercial motor cyclists to take alcohol particularly in the early hours of the day with the notion to wade off cold. When in fact previous studies have indicted alcohol to potentiate hepatic damage and cause various damages to different organs of the body. Based on this, we investigated the effect of chronic alcohol consumption in motor cyclists using some haematological indices together with some cardiohepatic parameters to determine any possible effect of alcohol in motorcyclists. The study involved 50 males age 26-35 years who take between 100-200mls of alcohol [74 to 148 grams (< 40%) alcohol] each day for more than two years. Ten non-alcoholic motorcyclists served as control. Parameters monitored were haemoglobin (Hb), packed cell volume (PCV) and white blood cells counts (WBC). Also, assayed were serum enzymes-aspartate aminotransferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), and creatine kinase (CK). Cholesterol level was also determined. Our data show that the alcoholic motorcyclists had significant decrease in Hb, PCV and WBC (P<0.05). On serum enzymes monitored, the result showed a significant increase (P< 0.05) in the activities of AST, GGT and ALP in the alcoholic motorcyclists. There was also a significant increase in the activity of CK (P <0.05) in these alcoholic individuals. Although, the activity of LDH and total cholesterol level were slightly increased in alcoholic motor cyclist studied.

Key words: Cytochrome P450, Alcohol, Cardiac dysfunctions, Cholesterol, Motor cyclist

INTRODUCTION

Motor cycle is no doubt one of the quickest transport systems in Nigeria. Among its advantages is easy accessibility to remote areas of the country where cars cannot reach.

In the face of high rate of unemployment in the country some young male graduates even use this means to sustain themselves while waiting to secure their dream jobs.

Unfortunately, it is becoming a common practice that some of these motor cyclists take alcohol especially during the early hours of the day with the notion to wade off cold. The alcohol may be taken plain or together with root extracts often alleged to cure different ailments such as malaria and some stomach discomforts.

When in fact, alcohol causes body to loose heat by widening the small blood vessels making the body to release heat from within to the surface. The person may feel temporarily warm when actually the body loses a greater amount of heat. Continuous lost of the body’s heat can lead to hypothermia, which is a very dangerous condition (Igboh et al., 2006).

The brain is usually markedly affected by alcohol than any other organ of the body. Alcohol depresses the brain and decreases its activity.
resulting to sedation and impaired judgment and this could be responsible for a number of motorcycle accidents on our roads.

Incidentally, various animal and human studies have demonstrated the deleterious effects of alcohol on the various organs of this body (Igboh, 2003, Niemela, 2004, Lavala, et al., 2004). Several other studies have also revealed that alcohol potentiates hepatocyte damage through its microsomal metabolism via cytochrome P<sub>450</sub> especially (CYP<sub>450</sub> 2E1) which results in a significant release of free radicals particularly reactive Oxygen species (ROS). These in turn deplete reduced glutathione (GSH) and other defense systems necessary to combat oxidative stress (Igboh et al., 2006; Guerri and Grisolia, 1980; Lieber, 1997; 2000a and b).

Other studies have implicated the ROS generated from alcohol metabolism to cause many clinical conditions apart from alcoholic liver disease; others include autoimmune disease e.g. rheumatoid arthritis, haemachromatosis, atherosclerosis and cardiovascular disease (Griendling, 2003). It was based on these findings that the investigators became inquisitive in studying the effects of alcohol on motor cyclists actually focusing precisely on haematological and biochemical changes that could occur.

**MATERIAL AND METHODS**

**Subjects**

Fifty (50) consenting males, age 26-35 years, who take between 100-200mls of alcohol (74 to 148 grams < 40% alcohol) each day for more than two years, within Owerri were investigated. Ten non-alcoholics motorcyclists served as control. Blood samples were taken for analysis of haemoglobin (Hb), packed cell volume (PCV), white blood cell (WBC) counts and lymphocytes Counts. Monitored were serum enzymes-aspartate aminotransferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and creatine kinase (CK). Serum cholesterol level was also determined.

**Sample collection**

Blood samples were collected with bottles containing EDTA anticoagulant for Hb, PCV, WBC and lymphocytes determinations and counts. Other bottles without anticoagulant were used to collect blood for the assay of serum enzymes – AST, CK, LDH, GGT and ALP. Cholesterol was also determined.

**Analysis**

For Hb, the cyanomethaemoglobin method of Fairbanks (1982) was used. While the methods of Dacie and Lewis (1991), were used for PCV, Lymphocytes and WBC counts.

AST was determined using Reitmen and Frankel, (1956) method. ALP activity was determined employing Klein et al. (1960); method. CK, LDH and GGT were determined using the methods of Rosalki, (1967), Amador, et al. (2002) and Szasz, (1969) respectively. Cholesterol was determined using Abell et al. (1952) method.

**Statistics**

The statistical analysis used was the one-way analysis of variance (ANOVA). This was used for haematological parameters, while the Student’s T-test was used in evaluating the activity of the enzymes. P < 0.05 was regarded as significance (Obi, 1986).

**RESULTS**

The results obtained are shown in Table 1. Table 1, records the changes in some haematological parameters and cardiohepatic function markers among commercial motor cycles riders who drink or do not drink alcohol. Tables 1 showed the effect of alcohol on some haematological and biochemical indices in alcohol; non-alcoholic motorcyclists.

Form the result of the study, the alcoholics motorcyclist exhibited significant decrease in Hb, PCV, WBC, and Lymphocyte counts (P < 0.05). On serum enzymes monitored, the result showed a significant increase in the activities of AST, GGT and ALP in alcoholics motorcyclist (P < 0.05). There was a slight but significant increase in the activity of CK (P < 0.05) in these individuals. The activity of LDH and total cholesterol level were slightly high in alcoholics motorcyclist studied.
DISCUSSION

The decline in Hb, PCV, WBC and Lymphocytes, is probably due to generation of free radicals (particularly reactive oxygen species, ROS through the microsomal metabolism of alcohol via cytochrome P<sub>450</sub>. ROS is known to deplete antioxidants and depletion of antioxidants render blood cells very fragile, thus leading to accelerated destruction of the blood cells (Plit et al., 1998; Van Antwerpen et al., 1994; Van Antwerpen et al., 1995; Van Antwerpen et al., 1995; Davis,1995).

The low Hb, PCV, WBC and Lymphocytes are indicative of massive destruction of blood cells, which can lead to anaemia and weaken immune system. Again, low Hb, PCV, WBC and L could be as a result of malnutrition. Alcohol suppresses appetite and causes ulceration of the intestine. These factors could interfere with nutrient availability and utilization by the body, which can lead to malnutrition.

The elevated activity of AST is not surprising considering that AST is richly present in blood cells, and the destruction of these cells will liberate the enzyme into the plasma. Elevation of GGT and ALP are indicative of hepatic derangement, the hepatocytes metabolise alcohol. The higher ROS generated from such metabolism, attack hepatocytes causing cell death. Consequently, the death of the hepatocytes will cause hepatic enzymes to be liberated to the plasma resulting in high activity of these enzymes. The CK and LDH are slightly elevated in alcoholics motorcyclists compared with their non-alcoholic counterparts. Again, this is an indication of destruction of the cardiac cells. Equally, pointing to the possibility of cardiac dysfunction. Increase in cholesterol level is indicative of the possibility of atherosclerotic disorder. Alcohol encourages lipolysis and lipogeneosis depending on the energy state of the body. When there is energy deficit, (since alcohol suppresses appetite) the body falls back on energy reserves. This eventually leads to lipolysis. However, when there is excess energy, lipogeneosis takes place. Both, lipolysis and lipogeneosis result to elevated plasma cholesterol level. From the result of the study, the work support the suggestion that alcohol may predispose alcoholic motorcyclists to haematological abnormalities, hepatic derangement and possible cardiac dysfunction. To prevent these dysfunctions among other effects of alcohol, like the increased motor cycle accidents, the use of alcohol should be strongly discouraged among commercial motorcyclists.

Table 1: Changes in haematological, cardiac and hepatic function markers among commercial motor cycle riders who either consume or do not consume alcohol

<table>
<thead>
<tr>
<th>Diagnostic markers</th>
<th>Alcohol drinkers (n=50)</th>
<th>Non-alcohol drinkers (n=50)</th>
</tr>
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<tbody>
<tr>
<td>Haematological Parameters</td>
<td></td>
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<tr>
<td>- Haemoglobin (g/dL)</td>
<td>8.8±1.5*</td>
<td>12.7±2.9</td>
</tr>
<tr>
<td>- Packed Cell Volume (%)</td>
<td>26.4±1.3*</td>
<td>34.1±2.9*</td>
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<tr>
<td>- White Blood Cell Count (x10&lt;sup&gt;9&lt;/sup&gt;/L)</td>
<td>3.8±0.2</td>
<td>4.0±0.5</td>
</tr>
<tr>
<td>- Lymphocyte Count (mm&lt;sup&gt;3&lt;/sup&gt;)</td>
<td>55.0±1.4*</td>
<td>70.0±3.6*</td>
</tr>
<tr>
<td>Cardiac marker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Creative Kinase (U/L)</td>
<td>193.5±4.0*</td>
<td>136.0±3.2</td>
</tr>
<tr>
<td>- Lactate dehydrogenase (U/L)</td>
<td>126.3±3.6*</td>
<td>103.7±1.9</td>
</tr>
<tr>
<td>- Serum Cholesterol (mg/dL)</td>
<td>225.8±3.0*</td>
<td>135.0±2.3</td>
</tr>
<tr>
<td>Hepatic marker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Aspartate aminotransferase (U/L)</td>
<td>25.4±3.2*</td>
<td>19.0±1.5</td>
</tr>
<tr>
<td>- Gamma glutamyltransferase (U/L)</td>
<td>32.3±2.5*</td>
<td>20.1±1.1</td>
</tr>
<tr>
<td>- Alkaline phosphatase (U/L)</td>
<td>109.0±2.6*</td>
<td>69.3±1.5</td>
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</table>

Values are expressed as Mean ± SEM for 'n' subjects
*Significant difference (P<0.5) from non-drinkers value
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REFERENCES