Effect of Alcohol Consumption On Platelets Count, Intrinsic and Extrinsic Pathways of Blood Coagulation

By

Oke Olusegun Taiwo
Adeyanju Oluwaseyi Adeteju
Oyedeji Samuel Oyewole
Adedire Olusoji Aderemi
Adewuyi Isaac Kayode
Grace Oludayo
Research Article

Effect of Alcohol Consumption On Platelets Count, Intrinsic and Extrinsic Pathways of Blood Coagulation

Oke Olusegun Taiwo¹*, Adeyanju Oluwaseyi Adeteju², Oyedeji Samuel Oyewole¹, Adedire Olusoji Aderemi¹, Adewuyi Isaac Kayode¹, Grace Oludayo³

¹School of Medical Laboratory Science, Obafemi Awolowo University Teaching Hospital Complex, P.M.B.5538. Ile-Ife.
²Haematology Department, University College Hospital Ibadan.
³Anesthetic Department, Obafemi Awolowo University Teaching Hospital Complex, P.M.B.5538. Ile-Ife.

*Corresponding Author’s Email: oketaiwo@yahoo.com, Phone: +2348033772582

ABSTRACT

Alcohol is like a cankerworm that has eaten deep into the fabrics of our society. Contrary to popular belief it is not a habit, rather it is a disease. It can also be viewed as a form of drug addiction. Alcoholism is a degenerative disease that often causes long term physical, emotional and behavioral complication for the victim. Effect of alcohol on platelets count, intrinsic and extrinsic pathways of blood coagulation were determined on one hundred (100) willingly volunteered subjects. Twenty five (25) of them served as control and the remaining seventy five (75) were grouped into three, twenty five (25) in each group as heavy, moderate and occasional drinkers. The grouping was done based on the number of beer bottles they took and how regular they took it. The occasional drinkers were those that took three to four bottles of beer a week and the drinking pattern was not regular. Moderate drinkers took 5 to 7 bottles a week while the heavy drinkers took more than seven bottles per week. Estimated parameters were platelets counts, prothrombin time test and activated partial thromboplastin time test. It was observed that there were statistically significant differences (p<0.05) in all the parameters estimated in moderate and heavy drinkers, while the experimental differences seen in the parameters of occasional drinkers were not statistically significant (p>0.05).

Keywords: Platelets, Prothrombin time, Activated Partial thromboplastin time test, Alcohol, Intrinsic, Extrinsic.

INTRODUCTION

Alcohol consumption from many epidemiological studies has shown that it has some beneficial effect to the body system. Moderate alcohol consumption (1 – 2 standard drinks per day or 10 – 20g/d) can reduce the risk of heart disease and ischemic stroke by 20% to 60% and death of all causes by 10% to 20% (Lazarus et al., 1991). The underlying factors contributing to the protective or pathophysiologic effects related to alcohol consumption are not well understood. Nevertheless, the adverse effect of alcohol if consumed in excess is dangerous. Alcohol is a drug that depresses the central nervous system like sedative and anesthetics. It is not a stimulant as widely believed but speech becomes free and social inhibition may be forgotten since it affects the portion of the brain that control judgment (Leiber, 2000). It infiltrates the brain, liver heart, pancreas, lung and kidney within minutes and passes into the blood stream (Epstein, 1992). Alcohol has been reported to induce anaemia, prolonged prothrombin time (PT) and Partial thromboplastin time test with decreased albumin production. Also vitamin K dependent factors could be deficient due to malabsorption of vitamin K in the upper gastro intestinal tract as a result of excessive alcohol intake which damages the gastric mucosal (Ratnoff, 1997).

Alcohol has anti-aggregatory effect on platelet suggesting the beneficial effect of alcohol in preventing coronary heart disease but when consumed moderately. It was also found that alcohol decreases both platelet aggregation and the circulating fibrinogen level (Renaud and De Lorgerri, 1992). Alcohol consumption affects plasma lipid profiles and has been shown to raise high-density lipoprotein levels (Fraser et al., 1983). In the United states, the Beverage Guidance Panel has been formed to “provide guidance on the relative health and nutritional benefits and risks of various beverage categories” and to “help consumers select a variety of beverages” (Popkin et al., 2006). Recognizing the health benefits of the moderate consumption of alcohol, the Beverage Guidance
Panel released the following recommendations in 2006 in the new proposal guidance system for beverages’ alcoholic beverages 0 to 1 drinks/day for women and 0 to 2 drinks/day for men. A number of studies have indicated that ethanol also directly affects haemostasis via a number of mechanisms, including modulation of plasma coagulation factors, fibrinolysis and platelet function (Dimmit et al., 1998; Hillbom and Neiman, 1998; Mikhailidis et al., 1986). Chronic alcohol consumption can predispose to bleeding. A study of patients in United States and Sweden showed that the baseline incidence of acute upper gastrointestinal bleeding increased by 3-fold as alcohol consumption increased from 1 drink of fewer per week to more than 20 drinks per week (Kaufman et al., 1999). There is also a linear association between the consumption of ethanol and the risk of haemorrhagic stroke (Donahue et al., 1986).

The aim of this research work is to assess the effect of quantity of alcohol consumed per day on the coagulation parameters.

MATERIALS AND METHODS

Subject Selection

A total of 100 subjects comprising of 75 alcoholics and 25 non alcoholics. The alcoholics were further grouped into heavy drinkers (25), moderate (25) and occasional drinkers (25). Blood samples were collected from all of them having given consent to participate in the study. The blood samples collected were used for platelet count (PLT), prothrombin time (PT) and activated Partial thromboplastin time test (APTT). The analysis was done at the Department of Haematology, Obafemi Awolowo University Teaching Hospital Complex, Ile – Ife.

Specimen collection and Laboratory analysis

The blood sample was collected under aseptic conditions. Nine milliliters (9ml) of blood was collected from cubital vein by venepuncture into 0.5ml of 3.8% sodium citrate in a plastic tube and commercially prepared Ethylene Diamine Tetra acetic acid (EDTA) plastic tube. The blood collected into sodium citrate plastic tube was centrifuged immediately at 2500g for 15minutes and the plasma separated and stored into stopper tubes and used within 4 hours of collection for prothrombin time (PT) and activated Partial thromboplastin time test (APTT). The EDTA blood sample was used for platelet count (PLT). Stasis was avoided during blood collection to prevent activation of clotting factors. The manual methods by Dacie and Lewis was adopted for the estimation of platelets count (Dacie and Lewis, 2001), while the screening kits of thromboplastin and haemoscann were used for APTT and PT.

PROTHROMBIN TIME PROCEDURE USING PLASMACANN KIT

**Principle:** Prothrombin time is the time in seconds necessary for plasma to clot at 37°C. Once an external coagulation factors (Thromboplastin) and Calcium chloride has been added. Manufacturer procedure was adopted. Normal range 10-15 seconds.

**Activated Partial Thromboplatin Time;**

Same principle was added and the manufacturer procedure equally adopted. Normal range 33-45 seconds

Results

This study was carried out among the inhabitant of Ile-Ife, who gave the consent of participation. One hundred subjects that volunteered were recruited. The recruited subject were grouped into four (4). The first group was the control subjects (25), the second were the occasional drinkers (25), the third groups were moderate drinkers (25) and the fourth groups were the heavy drinkers (25). Table 1 shows the overall result of some coagulation parameters in alcoholics regardless of their classes of classification. Statistically significant differences were seen in all the parameters of coagulation estimated (p<0.05).

The coagulation parameters shown in table 2 belong to the first group called occasional drinkers. This group drinks occasionally, they took between 3 and 4 bottle of beer, the drinking was not regular. Statistically significant differences were not found in all the parameters estimated, (PLT, PT and PTTK) (p<0.05). The results of table 3 belong to the moderate drinkers who drank between 5–7 bottles of alcoholic drinks per week. Statistically significant differences were observed in all the parameters estimated which are PLT, PT and PTTK (p<0.05). Table 4 shows the
Table 1: Showing the overall result of some coagulation parameters in alcoholics regardless of their classes.

<table>
<thead>
<tr>
<th>Test</th>
<th>PLT</th>
<th>PT</th>
<th>PTTK</th>
</tr>
</thead>
<tbody>
<tr>
<td>75</td>
<td>183,620.33±14,641.64</td>
<td>33.96±18.85</td>
<td>50.23±10.24</td>
</tr>
<tr>
<td>25</td>
<td>233,953.67±24,183.14</td>
<td>15.60±3.75</td>
<td>39.26±6.68</td>
</tr>
<tr>
<td>P-Value</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Table 2: Showing the platelet count, PT, and PTTK results in Occasional drinkers

<table>
<thead>
<tr>
<th>Test</th>
<th>PLT</th>
<th>PT</th>
<th>PTTK</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>228,731.00±51,985.69</td>
<td>16.39±6.86</td>
<td>40.21±11.84</td>
</tr>
<tr>
<td>25</td>
<td>233,953.67±24,183.14</td>
<td>15.60±3.75</td>
<td>39.26±6.68</td>
</tr>
<tr>
<td>P-Value</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table 3: Showing the result of Platelet count, PT, and PTTK in moderate drinkers

<table>
<thead>
<tr>
<th>Test</th>
<th>PLT</th>
<th>PT</th>
<th>PTTK</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>185,830.00±40,005.95</td>
<td>26.50±10.31</td>
<td>47.82±9.29</td>
</tr>
<tr>
<td>25</td>
<td>233,953.67±24,183.14</td>
<td>15.60±3.75</td>
<td>39.26±6.68</td>
</tr>
<tr>
<td>P-Value</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Table 4: Showing the result of Platelet count, PT, and PTTK in heavy drinkers

<table>
<thead>
<tr>
<th>Test</th>
<th>PLT</th>
<th>PT</th>
<th>PTTK</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>170,300.00±60,500.41</td>
<td>54.00±5.43</td>
<td>58.16±12.28</td>
</tr>
<tr>
<td>25</td>
<td>233,953.67±24,183.14</td>
<td>15.60±3.75</td>
<td>39.26±6.68</td>
</tr>
<tr>
<td>P-Value</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

DISCUSSION

One of the most serious socioeconomic problems worldwide is alcoholism. It poses a major health hazard to human health because it is toxic to almost all the organs in the body and hence affects all the metabolic processes going on in the body. A person who consumes an amount of alcohol capable of producing pathological changes is defined as an alcoholic (Criteria Committee, 1972). The overall result of this research work showed thrombocytopenia of statistically significant in alcoholic compared to the control (non alcoholics). Thrombocytopenia mean reduced level of platelets and platelets major function is in blood clotting. Reduced platelets level may be due to impair production or destruction of platelets in the circulation. When either of this occur the normal function of platelets among which; maintaining the integrity of vascular endothelium, performing the primary haemostatic plug following vessel injury, activation of blood coagulation system, producing mediators involved in vessel wall repair and regulation of vascular toxicity, as well as inflammatory reaction and producing growth factors (Stiene-Martin et al., 1998) will be affected. According to Dailey (2002), impaired platelet production may occur during heavy alcohol ingestion and this usually disappears after 3-7 days. Weed and Reed (1983) also said that the inhibition of bone marrow by alcohol can result in a low number of circulating platelets. The thrombocytopenia seen in this work is due to the effect of alcohol and this is pronounced in moderate and heavy alcoholics.

Hard and Ballard (1997) said, apart from acquired immune deficiency syndrome (AIDS), alcoholism probably is the leading cause of thrombocytopenia. Moreover, alcohol-related thrombocytopenia generally is transient and platelet counts usually return to normal within one week of abstinence (Harold and Ballard, 1997). Alcohol affects not only platelet production but also platelet function. Thus, patients who consume excessive amounts of alcohol can exhibit a wide spectrum of platelet abnormalities. These abnormalities include impaired platelet aggregation, decreased secretion or activity of platelet-derived proteins involved in blood clotting and prolongation of bleeding in the absence of thrombocytopenia. The overall result of this work is in line with the previous work done, where
thrombocytopenia were recorded. Though it was recorded that alcohol has adverse effects on the blood building or hematopoietic system, both are direct and indirect. The direct consequences of excessive alcohol consumption include toxic effects on the bone marrow; the blood cell precursors; and the mature red blood cells (RBC’s), white blood cells (WBC’s) and platelets. Alcohol’s indirect effects include nutritional deficiencies that impair the production and function of various blood cells. These direct and indirect effects of alcohol can result in serious medical problems for the drinker. (Harold and Ballard, 1997).

The effect of alcohol consumption is more pronounced in the heavy drinkers more than the moderate drinkers. The effect seen might be due to the toxic effect on the bone marrow, where the platelets and other blood cells are been produced as pointed out by Harold and Ballard. This is dangerous for the alcoholics.

The coagulation mechanism occur in the intrinsic and extrinsic pathways follows 3 steps which are the formation of thromboplastin, conversion of prothrombin to thrombin and thirdly formation and stabilization of fibrin (Ruf and Edigton 1994 and Nemerson 1998). The prothrombin time and activated partial thromboplastin time tests are the screening tests for the extrinsic and intrinsic pathways of blood coagulation. Abnormalities in the result of any of these tests indicate a deficient of one or more of the coagulation factors in the pathway of blood coagulation. Almost all the coagulation factors are proteinous in nature except factor four which is calcium and majority of them are produced from the liver. Liver is regarded as the body factory, where some substances that are useful to the body are built and other substances that are injurious to the body are detoxified and rendered harmless.

Liver is the organ that breaks down alcohol into harmless byproducts and clearing it from the body. Excessive consumption of alcohol can cause damage and prolonged dysfunction of the liver, such as liver cirrhosis. When this happen, the function of the liver such as synthesis of some of the coagulation factors is hindered and this leads to non availability or reduced level of these factors resulting into prolonged result of the PT and APT. Alcohol also impair the absorption of some vitamins and folic acid in the body, among the vitamins is vitamin K which is essential for the production of some of the coagulation factors in these pathways. Reinke and Mccay in 1997 said that alcohol disturbs liver functions and this will affect the synthesis of coagulation factors, hence prolongation of PT and APTT. (Reinik and Mccay, 1997). Vitamin K is an essential factor in the synthesis of factors II, VII, IX and X, this vitamin is confirmed deficient in the alcoholics and this will lead to prolong PT and APT (Ochei and Kolhatkar, 2004). This was buttressed by the work of Ratnoff, where he recorded that vitamin K dependent factors could be deficient due to malabsorption of vitamin K in the upper gastro intestinal tract as a result of excessive alcohol intake which damages the gastric mucosal (Ratnoff, 1997).

Keneth and his colleagues reported that light-to-moderate alcohol use was associated with lower levels of fibrinogen, plasma viscosity, factor VII, and vWF. On the other hand, fibrinolytic potential was lower with increasing alcohol consumption, most dramatically at ≥7 drinks weekly. They also found similar findings for beer, wine and liquor drinkers, but wine drinkers generally had the lowest PAI-1 levels at moderate levels of consumption (Keneth et al., 2001). The specific mechanisms by which alcohol may alter haemostatic parameters remain uncertain (Research Monograph no 13 1996).

A number of studies have indicated that ethanol also directly affects haemostasis via a number of mechanisms, including modulation of plasma coagulation factors, fibrinolysis and platelet function. Chronic alcohol consumption can predispose to bleeding. A study of patients in the United States and Sweden showed that the baseline incidence of acute upper gastrointestinal bleeding increased by 3-fold as alcohol consumption increased from 1 drink or fewer per week to more than 20 drinks per week (Kaufman et al., 1999). It was reported that prolonged liver dysfunction, such as liver cirrhosis resulting from excessive alcohol consumption, can harm the brain, leading to a serious and potentially fatal brain disorder known as hepatic encephalopathy (Butterworth 2003). Hepatic encephalopathy can cause changes in sleep patterns, mood and personality; psychiatric conditions such as anxiety and depression; severe cognitive effects such as shortened attention span; and problems with coordination such as a flapping or shaking of the hands (called asterixis). In the most serious cases, patients may slip into a coma (i.e., hepatic coma), which can be fatal (Alcohol Research and Health 2003).

In conclusion, alcohol consumption has some beneficial effect if it is consumed occasionally, but the evil effect to body is more than its benefit. The result of this work predict bleeding tendency for the alcoholics, all the parameters associated with blood clotting were affected in alcoholics compared to non alcoholics. Though it is more difficult to forsake alcohol once started, it is more advisable for people not to indulge in it and stay away from it because of the danger associated to it.

REFERENCES

Alcoholic Brain Damage (2003). The journal Alcohol Research & Health, (Vol. 27, No. 2,)


