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Effect of High-Risk Alcohol Use on Aspartate Aminotransferase and Coagulation Parameters Among Male Subjects in an Urban Area of Cross River State, Nigeria

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Abstract

Background

High-risk alcohol consumption is a major public health concern with profound multi-systemic effects. Data regarding its specific impact on coagulation kinetics, hepatic enzymes, and platelet indices among urban populations in Nigeria remain limited. This study aimed to evaluate aspartate aminotransferase (AST), platelet count, platelet indices, and coagulation parameters among high-risk male alcohol consumers in Calabar Metropolis, Cross River State, Nigeria.

Methods

A cross-sectional comparative study was conducted among 116 adult males aged 18–55 years, comprising 58 high-risk alcohol users (consuming ≥ 60 g of ethanol daily for ≥ 1 year) and 58 age-matched non-drinking controls. Blood samples were evaluated for serum AST, coagulation profile [prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen], and platelet parameters. Data were analysed using independent-samples t-test and chi-square test, with statistical significance set at $p \leq 0.05$.

Results

The alcohol group demonstrated a significantly higher mean body mass index (28.4 ± 3.2 vs 21.5 ± 2.7 kg/m², $t = 12.55$, $p < 0.001$), systolic blood pressure (135 ± 10 vs 120 ± 8 mmHg, $t = 8.92$, $p < 0.001$), and diastolic blood pressure (85 ± 11 versus 78 ± 9 mmHg, $t = 3.75$, $p < 0.001$). Serum AST was significantly elevated in alcohol consumers (30.1 ± 5.6 vs 23.0 ± 5.1 U/L, $t = 7.14$, $p < 0.001$). Coagulation parameters including PT (18.5 ± 2.9 vs 13.04 ± 2.3 seconds, $t = 11.23$, $p < 0.001$), aPTT (36.1 ± 4.5 vs 31.7 ± 3.5 seconds, $t = 5.88$, $p < 0.001$), were significantly prolonged. Conversely, platelet counts, and related indices (MPV, PDW, PCT) were significantly suppressed in the alcohol cohort ($p < 0.001$) including the fibrinogen (1.64 ± 0.22 vs 2.50 ± 0.39 g/L, $t = 14.63$, $p < 0.0001$). Furthermore, participants with a consumption history exceeding six years exhibited even more pronounced haematological and hepatic alterations.

Conclusion

High-risk alcohol use is significantly associated with profound disruptions in hepatic function, coagulation dynamics, and platelet profiles. These findings reveal the haematological risks linked to hazardous drinking in this urban Nigerian population.

Introduction

Alcohol (C_2H_5OH) is ethanol consumed through beverages such as beer, wine, spirits, and locally brewed drinks like ogogoro and burukutu. Alcohol use is a leading cause of morbidity and mortality worldwide, accounting for approximately 3 million deaths annually [1].

In Nigeria, alcohol consumption is culturally accepted, and it cuts across different social groups and settings. Urban areas such as Calabar Metropolis in Cross River State have reported high rates of hazardous drinking among adults [2, 3, 4]. However, the pattern of alcohol use in Nigerian cities is influenced by availability, affordability, and cultural practices and the locally brewed alcohol drinks often have variable ethanol concentrations, which can increase the risk associated with consumption [5, 6].

According to World Health Organization (WHO), heavy continuous drinking is defined as consumption of an average of 60grams or more of pure alcohol (ethanol) per day over one year. one standard drink is approximately 10g of pure ethanol. A daily alcohol consumption of 1-40g/day for men is referred to as low risk alcohol use. A medium-risk is 41-60g/day, high risk is 61-100g/day while very high risk is ≥ 101g/day [16].

Alcohol consumption has been linked to a wide range of health issues. Chronic intake is associated with liver disease. It has also been implicated in the development of cardiovascular disorders. Furthermore, prolonged use has been reported to result in neurological problems and beyond these systems, alcohol exerts significant effects on blood and coagulation system [7, 8, 9, 10]. The haemostatic system maintains vascular integrity by balancing coagulation and fibrinolysis. The balance is achieved through coordinated actions of the vascular endothelium, platelets, and plasma coagulation factors. Disruption of this systems may lead to bleeding or thrombotic tendencies.

Alcohol has been reported to affect the haemostatic balance through several mechanisms [11, 12, 13]. One of which involves the liver, alcohol can impair hepatic synthesis of vitamin K-dependent clotting factors II, VII, IX, and X, potentially leading to prolonged PT. Another pathway is through the bone marrow, alcohol may suppress bone marrow function, leading to thrombocytopenia as well as defective platelet aggregation and altered fibrinogen levels, thereby further disturbing normal haemostasis [14, 15].

While Akinbo *et al*. (2019) reported coagulation changes among chronic alcohol consumers in Benin City, data from Calabar Metropolis in South-South Nigeria remain limited. Calabar has distinct patterns of alcohol consumption basically beer and locally brewed drinks with variable ethanol content, while peer influence, family drinking patterns and alcohol availability contribute to drinking behaviour. This study considered adult male subjects resident in Calabar metropolis who consume more than three (3) bottles of beer per day for 4 or more times per week for over one year. Currently there is paucity of information in the area of the activity of platelet and its indices as well as some coagulation parameters in high-risk alcohol users in this population. Therefore, this study is aimed at evaluating aspartate aminotransferase, platelet count, platelet indices and some coagulation parameters of male High-Risk Alcohol users residing in Calabar Metropolis Cross River State, Nigeria.

Methods

A cross-sectional comparative study was conducted among adult males aged 18–55 years in Calabar Metropolis, Cross River State, Nigeria, between December 2025 and June 2026. A total of 116 participants were recruited: 58 male high-risk alcohol users defined as consumption of more than three (3) bottles of beer per day for 4 or more times per week for over one year [16] and 58 alcohol non-drinking controls.

Inclusion Criteria for the subjects are that they must be males who had consumed ≥ 60 grams of ethanol per week for at least 12 months, equivalent to ≥ 6 standard drinks per week as One standard drink is equivalent to 10g of ethanol (WHO, 2018). Controls were defined as males who abstain from alcohol consumption and claimed not to have taken alcohol before.

Exclusion criteria for both groups included < 18 years of age, known bleeding disorders, use of anticoagulant/antiplatelet medications in the last 6 months, pregnancy, HIV infection, viral hepatitis, and known liver disorder as well as those smoking.

Data collection and questionnaire

Data were collected using a structured questionnaire provided as Supplementary File 1. The questionnaire consisted of two sections: Section A (socio-demographic and clinical history) was developed specifically for this study by the authors. Section B (alcohol use assessment) was adapted from the previously published World Health Organization Alcohol Use Disorders Identification Test (AUDIT) (Saunders *et al*., 1993). The AUDIT is a 10-item validated instrument for screening high-risk alcohol use, with a score ≥ 16 indicating high-risk/harmful use. The questionnaire was pre-tested among 10 male subjects in Calabar (not included in the final analysis) for clarity and face validity, with a Cronbach's alpha of 0.82. The full English version of the questionnaire is provided as Supplementary File 1.

The sample size determination

The sample size was determined using G*Power version 3.1.9.7 for comparison of two independent means using a two-tailed independent samples t-test. The calculation of sample size was based on previous published platelet count value of 238.60 + 46.78x109/L among high-risk users and 259.29 = 31.79x109/L among control group [16]. Mansour *et al*, 2026.

Pooled Standard Deviation (SD) = 39.99

Difference between the means = 20.69

Cohen’s d = 20.69/39.99 = 0.52. Tails = 2, alpha error probability = 0.05.

G*Power(1-B) = 80% (0.80), allocation ratio=N2/N1 = 1.

Applying the parameters, the required sample size is 116 participants or 58 participants per group

Ethical approval

was obtained from the Cross River State Ministry of Health. Written informed consent was obtained from all participants and confidentiality was maintained throughout.

Laboratory Methods

Seven (7) milliliters of venous blood was collected from each participant under aseptic conditions. 2.25 mL was dispensed into a sodium citrate bottle for PT, aPTT, and fibrinogen assay, and 2.0 mL into an EDTA bottle for platelet count and platelet indices while the remaining volume 2.75mL was placed in a plain bottle for the determination of serum aspartate aminotransferase. PT and aPTT were determined using the Quick one-stage and Mordifield Kaolin methods respectively. Fibrinogen was measured by the Clauss method. Platelet count and platelet indices were analyzed using an automated haematology analyzer while the AST was done using Randox kit following the manufacturers instruction. Questionnaire was used to obtain the biodata, while their height and weight were measured before the sample collection Data were analyzed using SPSS version 27.0. Continuous variables were tested for normality using Shapiro-Wilk test. Normally distributed data are presented as Mean ± SD and compared using Independent t-test. A p-value < 0.05 was considered statistically significant.

Results

Table 1: Demographic Characteristics of High-Risk Alcohol Users and the control group

Parameter	High Risk Alcohol use Group (58)	Control Group (58)	T value	P Value
Age(years)	38.4 ± 7.2	37.9 ± 6.8	0.39	0.701
Weight(Kg)	72.3 ± 7.9	68.5 ± 8.4	2.51	0.013*
Height(meters)	1.71 ± 0.07	1.72 ± 0.06	0.83	0.411
Blood Pressure (mmHg)				
Systolic	135 ± 10	120 ± 8	8.93	<0.001*
Diaslolic	85 ± 11	78 ± 9	3.75	<0.001*
Body Mass Index(Kg/m^2	28.4 ± 3.2	21.5±2.7	12.55	<0.001*
Normal weight(18.5-24.9)	19 (32 %)	38 (65.5%)	$\chi 2$=13.21	0.004*
Underweight (<18.5)	4 (6.9 %)	1 (1.7 %)		
Overweight(25-29.9)	25 (43.1 %)	15 (25.9 %)		
Obese(≥30)	10 (17.2 %)	4 (6.9 %)		
Period of Alcohol Consumption (years)				

≤5 years	24	___
≥6	34	

Baseline Characteristics, Blood Pressure, and Body Composition is shown in Table 1. A total of 116 male participants aged 18 and older were recruited from the Calabar Metropolis, comprising high-risk alcohol users and a well-matched control group. Independent-samples Student's t-tests showed no significant differences between the cohorts regarding age (38.4 ± 7.2 vs 37.9 ± 6.8 years; t = 0.39, p = 0.701) or height (1.71 ± 0.07 vs 1.72 ± 0.06 m; t = 0.83, p = 0.411). However, heavy drinkers exhibited a significantly higher mean weight than controls (72.3 ± 7.9 vs 68.5 ± 8.4 kg; t = 2.51, p = 0.013). Cardiovascular metrics followed a similar pattern; both systolic (135 ± 10 vs 120 ± 8 mmHg; t = 8.92, p < 0.001) and diastolic blood pressures (85 ± 11 vs 78 ± 9 mmHg; t = 3.75, p < 0.001) were notably elevated in the alcohol cohort.

Furthermore, mean body mass index (BMI) tracked significantly higher among drinkers (28.4 ± 3.2 versus 21.5 ± 2.7 kg/m²; t = 12.55, p < 0.001). A Pearson chi-square test confirmed a strong association between study grouping and BMI category distribution ($\chi 2$ = 13.21, p = 0.004). Normal weight was much more common among controls (65.5%) than among alcohol users (32.8%). Conversely, overweight (43.1% versus 25.9%) and obesity (17.2% versus 6.9%) predominated in the alcohol group, while underweight individuals accounted for 6.9% of drinkers and 1.7% of controls.

As detailed in Table 2, liver function, coagulation, and platelet profiles are presented. Evaluating AST, coagulation indices, and platelet parameters revealed statistically significant disparities across all tested metrics. Liver Enzyme, aspartate aminotransferase (AST) levels were markedly elevated in alcohol consumers compared to controls (30.1 ± 5.6 versus 23.0 ± 5.1 U/L; t = 7.14, p < 0.001). Coagulation Parameter, which include prothrombin time (PT) and activated partial thromboplastin time (aPTT) were significantly prolonged among the alcohol group (PT: 18.5 ± 2.9 versus 13.04 ± 2.3 seconds, t = 11.24, p < 0.001; aPTT: 36.1 ± 4.5 versus 31.7 ± 3.5 seconds, t = 5.89, p < 0.001). Conversely, fibrinogen concentration was significantly lower in higher risk alcohol users (1.64 ± 0.22 versus 2.50 ± 0.39 g/L; t = 14.63, p < 0.001). Platelet counts alongside mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) were significantly suppressed in the alcohol cohort (all p < 0.001).

Table 2: Aspartate Aminotransferase, Coagulation and Platelet Parameters of High-Risk Alcohol Users and Control group.

Parameters	High Riak Alcohol Users (n=58) Mean±SD	Controls (n=58) Mean±SD	t-value	P-value
AST(≤ 48U/L)	30.1 ± 5.6	23.0 ± 5.1	7.14	<0.001
PT (11-16 Sec)	18.5 ± 2.9	13.0 ± 2.3	11.23	<0.001
aPTT (20-40 Sec)	36.1 ± 4.5	31.7 ± 3.5	5.89	<0.001
PLT (150-450x10^9/L)	152.18 ± 59.92	250.38 ± 50.96	9.51	0.001
Fibrinogen (1.5-4.0 g/L)	1.64 ± 0.22	2.50 ± 0.39	14.63	<0.001
MPV (7.5-12.0 fL)	8.2 ± 0.9	9.8 ± 1.0	9.06	<0.001
PDW (10-18%)	15.5 ± 1.08	17.74 ± 1.23	10.42	<0.001

PCT (0.15-0.4%)	0.14 ± 0.03	0.16 ± 0.03	3.59 0.002*

Key: AST= Aspartate Aminotransferase, PT = prothrombin time, aPTT = activated partial thromboplastin time, PLT = platelet count, MPV = mean platelet volume, PDW = platelet distribution width, PCT = plateletcrit, *Significant difference

Table 3 shows the impact of alcohol consumption duration. It highlights physiological differences when stratifying the alcohol group by duration of consumption (comparing those with over 6 years of intake using Welch's independent samples t-test). Participants with a consumption history exceeding six years demonstrated significantly higher AST levels (38.6 ± 9.2 versus 32.5 ± 2.1 U/L; t = 3.73, p = 0.001). Coagulation times were also more pronounced in this sub-group, showing prolonged PT (18.08 ± 1.67 versus 15.92 ± 2.04 seconds; t = 4.27, p < 0.001) and aPTT (37.53 ± 2.45 versus 32.33 ± 2.85 seconds; t = 7.25, p < 0.001). Fibrinogen concentration reduced notably in the longer-duration group (0.86 ± 0.32 versus 1.91 ± 0.30 g/L; t = 12.77, p < 0.001). Regarding platelet dynamics, PDW values did not significantly change among those drinking for more than six years, whereas MPV (2.50±1.07 versus 6.44±1.12; t14.03, p <0.001), PCT (0.02±0.01 versus 0.12±0.03; t 15.72, p <0.001) and platelet counts were significantly lower (68.60 ± 40.38 versus 110.96 ± 37.5910^9/L; t = 4.10, p < 0.001) in participants with a consumption history exceeding six years

Table 3: Comparison of Coagulation Parameters by Duration of Alcohol Consumption

Parameters	1-5 years (n=24) Mean±SD	≥6 years (n=34) Mean±SD	t-value	P-value
AST(≤ 48U/L)	32.5 ± 2.1	38.6 ± 9.2	3.73	0.001*
PT (11-16 Sec)	15.92 ± 2.04	18.08 ± 1.67	4.27	<0.001*
aPTT (20-40 Sec)	32.33 ± 2.85	37.53 ± 2.45	7.25	<0.001*
PLT (150-450x10^9/L)	110.96 ± 37.59	68.60 ± 40.38	4.10	<0.001*
Fibrinogen(1.5-4.0 g/L)	1.91 ± 0.30	0.86 ± 0.32	12.77	<0.001*
MPV (7.5-12.0 fL)	6.44 ± 1.12	2.50 ± 1.07	14.03	<0.001*
PDW (10-18%)	15.1 ± 1.15	15.6 ± 3.10	0.86	0.394
PCT (0.15-0.40%)	0.12 ± 0.03	0.02 ± 0.01	15.72	<0.001*

Key: Aspartate aminotransferase, PT = prothrombin time, aPTT = activated partial thromboplastin time, PLT = platelet count, MPV = mean platelet volume, PDW = platelet distribution width, PCT = plateletcrit, *Significant difference.

Discussion

This study evaluated the effects of high-risk alcohol consumption on anthropometric measures, blood pressure, hepatic markers, coagulation parameters, and platelet indices among adult males in Calabar Metropolis. While the participants were successfully matched for age and height, the high-risk alcohol group exhibited a significantly higher mean weight and body mass index (BMI) compared to the non-drinking controls. Furthermore, normal weight was markedly more prevalent among controls, whereas overweight and obesity predominated among alcohol consumers. Contrary to the classical expectation of malnutrition, we observed higher weight and BMI in high-risk drinkers. This is consistent with the report on Irish Healthy Survey which showed harmful alcohol consumption was associated with high BMI (OR1.47) and waist circumference [17]. The high energy density of alcohol (7 kcal/g), its additive effect to dietary calories, appetite stimulation, and inhibition of fat oxidation, particularly with beer which is the predominant beverage in our environment may be the reason for the increased weight and BMI. The findings also align with the cultural and lifestyle patterns of alcohol consumption in urban Nigerian settings where high-calorie beverages (such as beer and locally brewed drinks with variable ethanol concentrations) are frequently consumed [2, 3, 4, 5, 6]. Additionally, both systolic and diastolic blood pressures were significantly elevated in the alcohol cohort. Chronic alcohol intake is a well-established driver of cardiovascular disorders and hypertension [7, 8, 9, 10], underscoring the compounding metabolic and hemodynamic risks associated with hazardous drinking habits in this population.

Hepatic function and duration-dependent liver stress was assessed using serum aspartate aminotransferase (AST), a primary biomarker of hepatocellular injury. This was significantly elevated in the alcohol group compared to controls. This biochemical elevation highlights the classic hepatotoxic impact of chronic ethanol metabolism [7, 8]. Furthermore, stratifying the participants by consumption duration revealed a dose-response relationship were individuals with a drinking history exceeding six years demonstrated significantly higher AST levels than those with shorter durations. These results reinforce the concept that prolonged ethanol exposure induces progressive hepatic stress, which directly compromises the liver's synthetic capacity and overall metabolic homeostasis [11, 12].

The haemostatic system relies on a precise physiological balance between coagulation and fibrinolysis, coordinated by the vascular endothelium, plasma clotting factors, and platelets. The current study revealed significant alterations in this balance among high-risk alcohol users, manifested by prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), alongside significantly reduced fibrinogen concentrations. These findings corroborate previous reports regarding alcohol-induced coagulopathies [11, 12, 13]. The prolongation of PT and aPTT can be mechanistically attributed to alcohol-induced hepatic impairment, which suppresses the hepatic synthesis of vitamin K-dependent clotting factors (such as factors II, VII, IX, and X) [14, 15]. Interestingly, fibrinogen concentrations were

lower in the alcohol group and intensified further in those with a consumption history exceeding six years. The finding revealed direct negative effect of duration on fibrinogen synthesis. It does appear that the longer the exposure (> 6 years), the more severe the liver synthetic failure and the lower the fibrinogen, increasing bleeding risk. While elevated fibrinogen is an independent cardiovascular risk factor, its complex interplay with altered clotting times highlights a profound disruption of the normal haemostatic profile in chronic heavy drinkers.

Platelet count, plateletcrit (PCT), and mean platelet volume (MPV), exhibited significant perturbations among the high-risk alcohol group. Specifically, overall platelet counts and PCT and MPV were significantly depressed, pointing toward ethanol-induced suppression of bone marrow function and megakaryocytopoiesis [14, 15]. Similar trends in platelet suppression have been documented in parallel studies among high-risk consumers [15]. When evaluated by duration of use, participants in the longer-duration group (> 6 years) showed even lower platelet counts alongside altered platelet indices (such as lowered MPV and PCT). These variations indicate that prolonged heavy alcohol consumption not only reduces circulating platelet numbers but also alters platelet morphology and turnover as a result of direct bone marrow toxicity and peripheral destruction.This triad of reduced platelet count, MPV and PCT observed in high-risk alcohol users indicates a combined quantitative and qualitative platelet defect with significantly increased bleeding risk. It also reflects progressive bone marrow suppression, splenic sequestration secondary to alcoholic liver disease, and impaired thrombopoietin synthesis, which worsens with duration of alcohol abuse. These indices can therefore serve as inexpensive, readily available prognostic markers for disease severity and bleeding risk stratification in resource-limited setting like Nigeria

Conclusion

This study demonstrates that high-risk alcohol consumption among adult males in Calabar Metropolis is significantly associated with adverse alterations in anthropometry, blood pressure, hepatic enzyme activity, coagulation kinetics, and platelet profiles. Given the paucity of regional data on platelet activity and specific coagulation parameters in this population, these findings bridge an important clinical gap. The findings emphasize that hazardous drinking, prevalent due to local availability and cultural practices poses multi-systemic health threats that extend well beyond liver disease into critical haematological and haemostatic dysfunctions. Therefore public health initiatives in Calabar and similar urban Nigerian settings must prioritize targeted interventions addressing the hidden haematological toll of high-risk alcohol use.

Limitations of the Study

This study being a cross-sectional study, captures data at only one specific point in time. It was able to establish correlations and associations, but unable to prove a direct cause-and-effect relationship between high-risk alcohol consumption and the observed haematological or hepatic changes. Secondly information regarding alcohol consumption frequency, duration, and volume was gathered via

questionnaires and interviews. Self-reported data is inherently susceptible to recall bias or under-reporting due to social desirability. The study exclusively recruited male participants from the location. Therefore, the findings may not be generalized to females due to gender-based physiological differences in alcohol metabolism.

Future research should employ longitudinal cohort designs to track individuals over time. This would help establish a clear causal timeline and determine how long-term changes in drinking habits affect liver enzymes, coagulation profiles, and platelet indices. Subsequent studies should expand their scope to include female populations, as sex-specific differences in alcohol metabolism and haematological responses warrant independent clinical evaluation. Replicating this study across multiple centers or different urban and rural regions in Nigeria would help account for regional variations in the types, ethanol content, and brewing practices of local alcoholic beverages.

Abbreviations

AST	Aspartate Aminotransferase
PT	Prothrombin Time
aPTT	activated Partial Thromboplastin Time
GGT	Gamma-Glutamyl Transferase
LFT	Liver Function Test
HIV	Human Immunodeficiency Virus
PDW	Platelet Distribution Width
PCT	Plateletcrit
WHO	World Health Organization

Declarations

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Authors' contributions

D.A.A and J.O.A led the manuscript writing, interpreted the data and substantially revised the manuscript; E.C.A conducted data management and analysis and contributed to the manuscript. All authors read and approved the final manuscript.

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Data availability

Data is provided within the manuscript. The data used and/or analyzed during the current study are available from the corresponding author upon responsible request.

Ethics approval and consent to participate

The study, the questionnaire and methodology was approved by the Cross River State Ministry of Health Research Ethics Committee in accordance with the Declaration of Helsinki. Informed consent was obtained from all individual participants prior to be included in the study.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

AI declaration

No generative AI tools were used in the writing or analysis of this manuscript.

References

1. World Health Organization. Global status report on alcohol and health 2018. Geneva: WHO. Available from: https://www.who.int/publications/i/item/9789241565639
2. Angioha PU, Akintola A, Ogunnubi O, Butum BE. (2021). Information on the prevalence and extent of alcohol abuse among commercial tricycle operators in Calabar. *Journal of Infectious Virology*. 2(1):1-12. doi:10.35877/454RI.jinav289
3. Adelekan ML, Makanjuola AB, Ndom RJE, Fayemi O, Obayan A, Suleiman T, Ola B. (2017). Prevalence and pattern of substance use among undergraduates in a Nigerian university. *African Journal of Drug Alcohol Studies*. 16(1):13-23.
4. Umoh EO. (2024). Prevalence and determinants of psychoactive substances use among youths in Calabar Metropolis, Cross River State. *Global Public Health Journal*. 1(2):15-28.
5. Rehm J, Gmel G, Shield KD, Bobova L, Pennay A, Stockwell T, Sornpaisarn B, Popova S, Manthey J, Probst C. (2017). The relation between different dimensions of alcohol consumption and burden of disease: an overview. *Addiction*. 112(5):968-978. doi:10.1111/add.13614

6. GBD 2016 Alcohol Collaborators. Alcohol use and burden for 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet*. 392(10152):1015-1035. doi:10.1016/S0140-6736(18)31310-2
7. Hoffman M, Monroe DM. (2001). A cell-based model of hemostasis. *Thrombosis and Haemostasis*. 85(6):958-965. doi:10.1055/s-0037-1615947.
8. Grover SP, Mackman N. (2018). Tissue factor: an essential mediator of hemostasis and trigger of thrombosis. *Arterioscler Thrombosis and Vascular Biology*. 38(4):709-725. doi:10.1161/ATVBAHA.117.309846
9. Seitz HK, Bataller R, Cortez-Pinto H, Gao B, Gual A, Lackner C, Mathurin P, Mueller S, Szabo G, Tsukamoto H. (2018). The alcohol-induced gut-liver axis: cross-talk between gut microbiota and the liver: implications for alcoholic liver disease. *Gut*. 67(8):1546-1558. doi:10.1136/gutjnl-2017-314885
10. Lackner C, Tiniakos D. (2019). Alcoholic liver disease: current and future therapies. *Journal of Hepatology*. 70(3):502-513. doi:10.1016/j.jhep.2018.11.009
11. Franchini M, Lippi G. (2018). Hemostatic changes in liver disease. *Seminars in Thrombosis and Hemostasis*. 44(3):245-251. doi:10.1055/s-0037-1615779
12. Savvidou S, Goulis J, Zachou K, Oikonomou A, Karali E, Sidiropoulos D, Ganaras A, Mantalovas S, Akriviadis E. (2014). Platelet dysfunction in alcoholic liver disease. *Annals of Gastroenterology*. 27(4):338-345. PMID: 25331529.
13. Franchini M, Lippi G. (2020). Liver disease and thrombosis. *Seminars in Thrombosis and Hemostasis*. 46(3):318-325. doi:10.1055/s-0039-1701024]
14. Thachil J, Warkentin TE. (2017). How do we approach thrombocytopenia in critically ill patients? *British Journal of Haematology*. 177(3):372-380. doi:10.1111/bjh.14534
15. Akinbo DB, Olokorom DC, Udeh CL. (2019). Assessment of some coagulation parameters among chronic alcohol consumers in Benin City, Nigeria. *Nigerian Journal of Clinical Practice*. 22(8):1123-1128. doi:10.4103/njcp.njcp_8_19
16. Mansour RA, Abass RJ, Mohammed SA, Salman MA. (2026). Impact of chronic alcohol consumption on blood parameters; A comparative study between healthy and alcohol consumers. *Journal of Pioneering Medical Sciences*. Vol 15(6) P 72-76
17. AlKalbani SR and , Murrin, C (2023). The association between alcohol intake and obesity in a sample of the Irish adult population, a cross-sectional study.*BMC Public Health*, 23 (1): 2075.doi: 10.1186/s12889-023-16946-4
18. Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption-II. *Addiction*. 88(6):791-804.
19. WHO. AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for Use in Primary Care. 2nd ed. Geneva: World Health Organization; 2001.

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