Analysis of Creatine Kinase-MB, Renal Biochemical Markers and Hematological Parameters among Alcohol Consumers in Oye-Ekiti, Ekiti State

Adegbola Abdullahi Adesina[1], Babajide Adewoyin Adeleke[2], Boluwatife Samuel Fatokun[3], Odunayo Iyanuoluwa Oluwasegun[4], Folorunsho Davies Olumide[5], Olubodun Olaotan Esther[6], Aladesuru Sharon Feyisayo[7], Shittu Roqeebat Mosunmola[8]

[1,2,4,5,6,7,8] Department of Medical Laboratory Science, Faculty of Basic Medical Sciences, Federal University Oye-Ekiti, Ekiti State, Nigeria

[3]Department of Chemical Pathology and Immunology, Federal Teaching Hospital Ido, Ekiti-State, Nigeria

ABSTRACT

Background: Alcohol is widely consumed in Nigeria, but its combined cardiac, renal and haematological effects at community level are poorly documented. This study compared serum creatine kinase-MB (CK-MB), renal markers, electrolytes and haematological parameters between alcohol consumers and non-consumers in Oye-Ekiti. Methods: In this cross-sectional study (January to May 2026), 100 adults (50 alcohol consumers and 50 non-consumers) were recruited. Questionnaire, anthropometric and blood pressure data were collected. Venous blood was analysed for full blood count and serum biochemistry. CK-MB was measured by fluorescence immunoassay, urea, creatinine and uric acid on an automated analyser, and electrolytes by ion-selective electrode; eGFR was calculated. Data were analysed with chi-square, t-test and ANOVA ($p < 0.05$). Results: Alcohol use was associated with male sex, transport occupations and secondary education ($p < 0.01$). Consumers had significantly higher CK-MB (5.32 ± 0.96 vs 1.68 ± 0.98 ng/mL), urea, creatinine, sodium, potassium, chloride, packed cell volume, haemoglobin and red cell count, and lower eGFR, uric acid, white cell and platelet counts ($p < 0.05$). Neutrophil and monocyte percentages were lower, lymphocyte and eosinophil percentages were higher, and red cell indices did not differ. Nine parameters, including CK-MB and creatinine, differed across drinking categories ($p < 0.05$). Conclusion: Alcohol consumers had higher CK-MB, urea and creatinine, lower eGFR and uric acid, and altered haematological indices than non-consumers, suggesting early cardiac, renal and haematological changes. Community-based screening of high-risk drinkers, particularly commercial drivers and motorcycle riders, merits consideration.

KEYWORDS

Alcohol consumption, creatine kinase-MB, renal function, electrolytes, haematological parameters

I. INTRODUCTION

Alcohol (ethanol) is a psychoactive and toxic compound that can produce dependence and acts on almost every organ system.1 The World Health Organization attributed about 2.6 million deaths worldwide to alcohol in 2019, or 4.7% of all deaths that year.1 The beverages involved range from commercial beer, wine and spirits to traditional products such as palm wine, burukutu, pito and locally distilled ogogoro, whose ethanol content is seldom tested or standardised.2 Herbal mixtures and bitters add to the exposure. Ethanol concentrations of 1.20% to 20.84% have been reported in herbal mixtures sold in Nigeria, 3 and 30% to 52% in selected herbal bitters sold in south-western Nigeria.4

A meta-analysis of 35 Nigerian studies covering 37,576 people estimated that 34.3% of those aged 15 years and above engage in harmful alcohol use (95% CI 28.6–40.1%). The prevalence was higher in men (43.9%) than in women (23.9%), and higher in rural (40.1%) than in urban areas (31.2%).5 Oye-Ekiti is a growing university town in which traditional drinking habits coexist with a large, mobile population of students, commercial drivers and motorcycle riders.6 In a semi-rural Nigerian community screened with the WHO ASSIST tool, current alcohol use was 23.7%, and 69.1% of current drinkers were at moderate or high health risk.7

Chronic heavy drinking is strongly associated with disease of the cardiovascular, renal and haematopoietic systems.8, 9 The older idea of a protective, J-shaped relationship between light drinking and heart health has largely been set aside, since large genetic and population studies indicate that no level of intake is completely safe.10 Alcohol is metabolised mainly in the liver, where acetaldehyde and reactive oxygen species are produced and cause cellular damage and inflammation.11

Prolonged heavy drinking causes alcoholic cardiomyopathy, in which the myocardium weakens and the ventricles dilate, eventually leading to heart failure.9 Creatine kinase-MB (CK-MB) is an is enzyme found mainly in cardiac muscle and has long been used as an indicator of myocardial injury. It usually rises in myocardial infarction, but non-ischaemic causes such as alcohol toxicity can also raise it.12 Raised CK-MB activity has been described in alcoholic patients without myocardial infarction.13 Cardiac troponins have largely replaced CK-MB in acute diagnosis because they are more specific, but CK-MB remains a practical option where troponin testing is unavailable or too costly, as is the case in many primary care clinics in this part of Nigeria.14

The kidneys keep fluid and electrolytes in balance and remove metabolic waste. Alcohol interferes with all three tasks at once by injuring tubular cells, altering renal blood flow and disturbing electrolyte handling.15 Urea, creatinine and uric acid are the standard biochemical indicators of kidney function.16 Creatinine alone can mislead in chronic drinkers, because loss of muscle mass may lower creatinine even when the kidneys are unaffected, so eGFR derived from creatinine gives a better picture of overall function.15 Serum sodium, potassium and chloride were also measured, because heavy alcohol intake can cause hypernatremia, hypokalaemia, hypomagnesaemia and hypophosphataemia.17

The blood is affected too, although the pattern is less predictable than in the heart and

kidneys. Chronic alcohol use has been linked with anaemia (often macrocytic), thrombocytopenia and leukopenia, which can weaken immunity and raise the risk of infection.18 A systematic review of 22 observational and cohort studies, including studies from Nigeria, Iraq and India, found that long-term drinkers often had macrocytic anaemia with higher mean corpuscular volume (MCV) and mean corpuscular haemoglobin (MCH), lower haemoglobin and red cell counts, and frequently lower platelet and white cell counts.19 The direction of change is not constant, however. Because alcohol is a diuretic, haemo concentration can raise packed cell volume (PCV), haemoglobin and red cell counts, whereas malnourished drinkers show the depressed pattern.

The effect also depends on the amount and duration of exposure.18 A narrow set of tests may miss subtle changes in the white cell differential, such as relative lymphocytosis with neutropenia, which a falling total count does not reveal.16

To our knowledge, no published community-based study has examined CK-MB, renal biochemical markers and a full haematological panel together in adult alcohol consumers in Oye - Ekiti. This study compared serum CK-MB, renal biochemical markers (urea, creatinine, uric acid, electrolytes and eGFR) and haematological parameters between alcohol consumers and non-consumers in Oye-Ekiti, Ekiti State, Nigeria.

II. METHODOLOGY

A. Study Design

This was a comparative cross-sectional study of alcohol consumers and non-consumers in Oye-Ekiti, Ekiti State, carried out over five months, from January to May 2026. Participants were recruited purposively from motor parks, beer parlours, palm wine tapping points and community gatherings. Oye-Ekiti is the administrative headquarters of Oye Local Government Area (LGA) in south-western Nigeria. The town was chosen because few community-based studies have examined alcohol-related organ damage in the rural and semi-urban parts of Ekiti State, and because it gave access to students, commercial drivers and motorcycle riders who drink locally produced spirits such as ‘pelebe’ and *ogogoro*.

B. Ethical Approval and Informed Consent

Ethical approval was obtained from the Research Ethics Committee of the Ekiti State Ministry of Health, Ado-Ekiti (EKHREC/EA/U/206) and the College of Medicine Research Ethics Committee, Federal University Oye-Ekiti (FUO/COMREC/MLS/2026/076). Before any data or samples were collected, the purpose, procedures, risks and benefits of the study were explained to each participant in English or Yoruba, and written informed consent was obtained from all participants. Confidentiality and anonymity were maintained throughout, participants were free to withdraw at any time without penalty, and the study followed the principles of the Declaration of Helsinki.

C. Sample Size

A minimum sample size of 70 participants was required, based on a prevalence of 23.9% for alcohol consumption in Ekiti State reported by Usman *et al.* (2024).[20] To compensate for possible non response and to improve the precision of the study, the sample size was

increased to 100 participants. Hence, a total of 100 subjects were recruited, comprising 50 alcohol consumers who served as the test group and 50 apparently healthy non-consumers, matched for age and sex, who served as control subjects.

D. Study Participants

A total of one hundred (100) adults aged 18 to 60 years who lived in Oye-Ekiti were recruited, comprising fifty (50) alcohol consumers (test group) and fifty (50) non-consumers (control group). They included commercial drivers, motorcycle riders, university students and labourers, and both men and women were enrolled. Alcohol consumers were individuals who reported regular, long-term intake of alcoholic beverages, including commercial beers, spirits and local brews such as ogogoro or palm wine, for at least two years.

Non-consumers reported no history of alcohol intake, or only very occasional and negligible intake, and were selected to match the consumers as closely as possible by age and sex. Individuals with known chronic conditions such as diabetes mellitus, hypertension, or established chronic kidney or heart disease were excluded. Others excluded were those taking medicines known to affect cardiac, renal or haematological parameters (for example diuretics, nephrotoxic drugs or chemotherapy), pregnant or lactating women, and anyone who declined to give consent.

E. Data Collection and Laboratory Analysis

A semi-structured, interviewer-administered questionnaire was used to collect socio-demographic data (age, sex, occupation, educational level and marital status), the pattern of alcohol consumption (duration, frequency, type of beverage and quantity per sitting), medical history (renal disease, diabetes mellitus and malignancy), cigarette smoking, previous blood transfusion and family history of relevant conditions. Weight was measured using a weighing balance to the nearest 0.1 kg with participants in light clothing, and height to the nearest 0.1 cm with a studio meter.

Waist and hip circumference were measured using a tape rule. After at least five minutes of seated rest, systolic and diastolic blood pressure were measured on the left arm with a validated electronic monitor (Omron M X2 Basic, Omron Health Care Co. Ltd, Kyoto, Japan). Venous blood (10 mL) was drawn from the cubical fossa by a qualified Medical Laboratory Scientist under aseptic conditions, with a tourniquet on the upper arm. Five (5) mL was dispensed into a plain bottle and 5 mL into an EDTA bottle. The plain sample was allowed to clot and was centrifuged at 3,000 rpm for 5 minutes, and the serum was separated and stored at −20 °C until analysis. The EDTA samples were mixed gently by inversion and analysed within 4 hours of collection.

Serum urea (urease–Berthelot method), [21] creatinine (modified Jaffe method) [22] and uric acid (uricase–peroxidase method) [23] were measured on a Chemray Rayto 240 automated chemistry analyser with Agappe reagents (Agappe Diagnostics Ltd, Kerala, India), following the manufacturer's instructions.

The eGFR was calculated manually from serum creatinine, expressed in mL/min/1.73 m². Serum CK-MB was measured by fluorescence immunoassay on the Finecare™ FIA Meter (Guangzhou Wondfo Biotech Co., Ltd).[24] the meter reported CK-MB in ng/mL from the lot-specific calibration. Serum sodium, potassium and chloride were determined on a Q-LYTE 60 ion-selective electrode analyser after two-point calibration with standards A and B.[25] A full blood count was run on a Sysmex XN-1000 analyser (Sysmex Corporation, Japan), [26]

which gave haemoglobin (Hb), PCV, red blood cell (RBC) count, white blood cell (WBC) count with differential, platelet count, MCV, MCH and mean corpuscular haemoglobin concentration (MCHC).

Control samples were run with every batch and checked against the manufacturers' ranges before results were accepted. For CK-MB, the internal control line of each test cartridge was verified, and external control material of known concentration was run with each new reagent lot and at regular intervals. Daily internal quality control on the Sysmex XN-1000 used XN-CHECK material.

F. Statistical Analysis

Data were analysed with the Statistical Package for the Social Sciences (SPSS) version 27.0. Categorical variables were compared with the chi-square test, or with Fisher's exact test when expected cell counts were below 5. The independent-samples t-test was used to compare means between the two groups, and one-way analysis of variance (ANOVA) was used to compare variables across categories of alcohol quantity per sitting. Results were expressed as mean ± standard deviation (SD), and $p < 0.05$ was considered statistically significant. The findings were presented in tables.

III. RESULTS

A. Socio demographic characteristics of the study participants

Table 1 shows the socio demographic characteristics of the 100 participants by study group. Alcohol consumption was significantly associated with sex, occupation and educational level, but not with age, marital status or ethnicity. Consumers made up a higher proportion of males (46/81, 56.8%) than females (4/19, 21.1%; Fisher's exact test, $p = 0.009$). The proportion of consumers did not differ significantly across age groups (range 40.9% to 58.8% among those aged 20 years and above; $\chi^2 = 3.441$, $p = 0.487$), and was higher, though not significantly, among married (34/58, 58.6%) than single participants (16/42, 38.1%; $p = 0.068$).

Occupation showed the strongest association ($\chi^2 = 31.791$, $p < 0.001$): 85.7% of transport workers (30/35) were consumers, compared with 28.6% of students (6/21) and 25.8% of skilled or technical workers (8/31). Consumers were also proportionally more common among participants with secondary education (28/40, 70.0%) than those with tertiary education (19/53, 35.8%; $\chi^2 = 12.312$, $p = 0.006$). Ethnicity was not significantly associated with alcohol use ($\chi^2 = 3.342$, $p = 0.188$).

Table 1: Socio demographic characteristics of the study participants by group

Variable	Category	Alcohol consumer's n (%) (n = 50)	Non-consumers n (%) (n = 50)	Test statistic	p-value
Sex	Male	46 (56.8)	35 (43.2)	Fisher's exact	0.009*
	Female	4 (21.1)	15 (78.9)		

Age (years)	<20	2 (100.0)	0 (0.0)	$\chi^2 = 3.441$	0.487
	20–29	9 (40.9)	13 (59.1)		
	30–39	17 (51.5)	16 (48.5)		
	40–49	12 (46.2)	14 (53.8)		
	≥50	10 (58.8)	7 (41.2)		
Marital status	Single	16 (38.1)	26 (61.9)	Fisher's exact	0.068
	Married	34 (58.6)	24 (41.4)		
Occupation	Student	6 (28.6)	15 (71.4)	$\chi^2 = 31.791$	<0.001*
	Transport workers	30 (85.7)	5 (14.3)		
	Skilled/technical workers	8 (25.8)	23 (74.2)		
	Business/trading	4 (36.4)	7 (63.6)		
	Farming	2 (100.0)	0 (0.0)		
Educational level	No formal education	1 (100.0)	0 (0.0)	$\chi^2 = 12.312$	0.006*
	Primary	2 (33.3)	4 (66.7)		
	Secondary	28 (70.0)	12 (30.0)		
	Tertiary	19 (35.8)	34 (64.2)		
Ethnicity	Yoruba	40 (46.5)	46 (53.5)	$\chi^2 = 3.342$	0.188
	Igbo	9 (69.2)	4 (30.8)		
	Hausa	1 (100.0)	0 (0.0)		

*Statistically significant at $p < 0.05$. Values are frequencies, with percentages within each category in parentheses. Fisher's exact test was used when ≥20% of cells had expected frequencies below 5 or when any expected cell count was below 5; χ^2: Pearson chi-square. Transport workers include bike riders and drivers; skilled/technical workers include bricklayers, welders, engineers, painters, mechanics, quality surveyors and public servants.

B. Anthropometric measurements and blood pressure of alcohol consumers and non-consumers

Table 2 compares the anthropometric and blood pressure measurements of alcohol consumers and non-consumers using the independent-samples t-test. Waist circumference was the only measure that differed significantly between the groups, being greater in consumers than in non-consumers (86.88 ± 16.17 vs 76.96 ± 20.92 cm; mean difference 9.92 cm, $t = 2.653$, $p = 0.009$). Height (166.76 ± 11.03 vs 164.70 ± 8.73 cm; $t = 1.036$, $p = 0.303$), weight (75.14 ± 13.60 vs 79.40 ± 10.08 kg; $t = -1.780$, $p = 0.078$), hip circumference (94.26 ± 11.25 vs 87.94 ± 37.15 cm; $t = 1.151$, $p = 0.252$), systolic blood

pressure (119.68 ± 15.15 vs 117.88 ± 17.95 mmHg; t = 0.542, p = 0.589) and diastolic blood pressure (80.26 ± 11.16 vs 80.68 ± 11.99 mmHg; t = −0.181, p = 0.856) did not differ significantly.

Table 2: Independent-samples t-test comparison of anthropometric and blood pressure measurements between alcohol consumers and non-consumers

Variable	Alcohol consumers Mean ± SD (n = 50)	Non-consumers Mean ± SD (n = 50)	Mean difference	t-value	p-value
Height (cm)	166.76 ± 11.03	164.70 ± 8.73	2.06	1.036	0.303
Weight (kg)	75.14 ± 13.60	79.40 ± 10.08	−4.26	−1.780	0.078
Waist circumference (cm)	86.88 ± 16.17	76.96 ± 20.92	9.92	2.653	0.009*
Hip circumference (cm)	94.26 ± 11.25	87.94 ± 37.15	6.32	1.151	0.252
Systolic BP (mmHg)	119.68 ± 15.15	117.88 ± 17.95	1.80	0.542	0.589
Diastolic BP (mmHg)	80.26 ± 11.16	80.68 ± 11.99	−0.42	−0.181	0.856

C. Alcohol consumption pattern and lifestyle characteristics of the study participants

Table 3 describes the pattern of alcohol use and the lifestyle characteristics of the two groups. All 50 consumers reported alcohol use and all 50 non-consumers reported none (Fisher's exact test, p < 0.001). Most consumers (33/50, 66.0%) had drunk alcohol for more than 10 years, 9 (18.0%) for 1 to 5 years, 4 (8.0%) for 6 to 10 years and 3 (6.0%) for less than 1 year, and the duration was other or unclear for 1 (2.0%). Sixteen consumers (32.0%) drank daily and 19 (38.0%) drank occasionally. Most consumers (35/50, 70.0%) took 1 to 2 drinks per sitting, while 6 (12.0%) took 3 to 4 drinks and 9 (18.0%) took more than 4. Spirits (32/50, 64.0%), beer (28/50, 56.0%), palm wine (26/50, 52.0%) and local alcoholic beverages (14/50, 28.0%) were each reported significantly more often by consumers (all p < 0.001).

Regular physical exercise was less common among consumers than non-consumers (19/50, 38.0% vs 34/50, 68.0%; p = 0.005), whereas smoking status, history of cardiovascular disease, diagnosis of diabetes and current medication use did not differ significantly between the groups (all p > 0.05).

Table 3: Patterns of alcohol consumption and lifestyle characteristics by study group

Variable	Category	Alcohol consumer's n (%) (n = 50)	Non-consumers n (%) (n = 50)	Test statistic	p-value
Alcohol consumption	Yes	50 (100.0)	0 (0.0)	Fisher's exact	<0.001 *
	No	0 (0.0)	50 (100.0)		
Duration of alcohol use	<1 year	3 (6.0)	0 (0.0)		
	1–5 years	9 (18.0)	0 (0.0)		
	6–10 years	4 (8.0)	0 (0.0)		
	>10 years	33 (66.0)	0 (0.0)		
	Other/unclear	1 (2.0)	0 (0.0)		
Frequency of alcohol use	Daily	16 (32.0)	0 (0.0)		
	Occasionally	19 (38.0)	0 (0.0)		
Beer consumption	Yes	28 (56.0)	0 (0.0)	Fisher's exact	<0.001 *
	No	22 (44.0)	50 (100.0)		
Palm wine consumption	Yes	26 (52.0)	0 (0.0)	Fisher's exact	<0.001 *
	No	24 (48.0)	50 (100.0)		
Spirits (gin/vodka)	Yes	32 (64.0)	0 (0.0)	Fisher's exact	<0.001 *
	No	18 (36.0)	50 (100.0)		
Local alcoholic beverages	Yes	14 (28.0)	0 (0.0)	Fisher's exact	<0.001 *
	No	36 (72.0)	50 (100.0)		
Average drinks per sitting	1–2 drinks	35 (70.0)	0 (0.0)	χ^2 = 100.000	<0.001 *
	3–4 drinks	6 (12.0)	0 (0.0)		
	>4 drinks	9 (18.0)	0 (0.0)		
	None	0 (0.0)	50 (100.0)		
Smoking status	Yes	7 (14.0)	4 (8.0)	Fisher's exact	0.525

	No	43 (86.0)	46 (92.0)		
Regular physical exercise	Yes	19 (38.0)	34 (68.0)	Fisher's exact	0.005*
	No	31 (62.0)	16 (32.0)		
Cardiovascular disease history	Yes	3 (6.0)	5 (10.0)	Fisher's exact	0.715
	No	47 (94.0)	45 (90.0)		
Diabetes diagnosis	Yes	0 (0.0)	1 (2.0)	Fisher's exact	1.000
	No	50 (100.0)	49 (98.0)		
Current medication use	Yes	16 (32.0)	12 (24.0)	Fisher's exact	0.504
	No	34 (68.0)	38 (76.0)		

D. Serum CK-MB and renal biochemical markers of alcohol consumers and non-consumers

Figure 1 shows a clear difference in serum CK-MB between the two groups. The mean was approximately 5.32 ng/mL (95% CI 5.05 to 5.58 ng/mL) in alcohol consumers and approximately 1.68 ng/mL (95% CI 1.40 to 1.96 ng/mL) in non-consumers. As shown in Table 4, consumers had a markedly higher mean CK-MB (5.317 ± 0.957 vs 1.680 ± 0.981 ng/mL), and the difference was statistically significant ($t = 18.767$, $p < 0.001$). As shown in Table 5, alcohol consumption was significantly associated with changes in the renal biochemical parameters. Consumers had significantly higher serum urea (3.254 ± 0.537 vs 2.982 ± 0.474 mmol/L; $t = 2.686$, $p = 0.008$) and creatinine (65.100 ± 11.543 vs 53.560 ± 7.804 µmol/L; $t = 5.856$, $p < 0.001$). Their mean eGFR was significantly lower (118.731 ± 12.285 vs 124.475 ± 10.534 mL/min/1.73 m²; $t = -2.510$, $p = 0.014$), and serum uric acid was also significantly lower (0.346 ± 0.137 vs 0.428 ± 0.143 mmol/L; $t = -2.925$, $p = 0.004$).

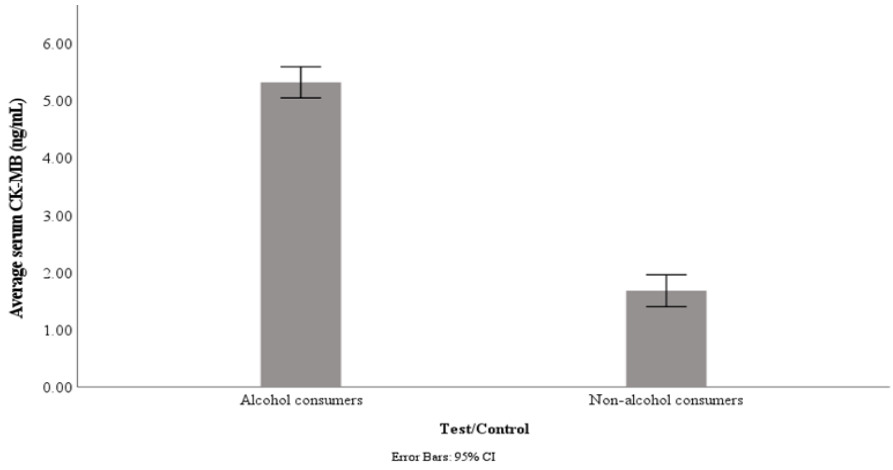


Figure 1: Serum level of creatine kinase-MB (CK-MB) among study participants

Table 4: Comparison of serum creatine kinase-MB (CK-MB) levels between alcohol consumers and non-consumers

Variable	Alcohol consumers Mean ± SD (n = 50)	Non-consumers Mean ± SD (n = 50)	t-value	p-value
CK-MB (ng/mL)	5.317 ± 0.957	1.680 ± 0.981	18.767	<0.001 *

Table 5: Independent-samples t-test comparison of urea, creatinine, eGFR and uric acid between alcohol consumers and non-consumers

Variable	Alcohol consumers Mean ± SD (n = 50)	Non-consumers Mean ± SD (n = 50)	t-value	p-value
Urea (mmol/L)	3.254 ± 0.537	2.982 ± 0.474	2.686	0.008*
Creatinine (μmol/L)	65.100 ± 11.543	53.560 ± 7.804	5.856	<0.001*
eGFR (mL/min/1.73 m²)	118.731 ± 12.285	124.475 ± 10.534	−2.510	0.014*
Uric acid (mmol/L)	0.346 ± 0.137	0.428 ± 0.143	−2.925	0.004*

E. Hematological parameters of alcohol consumers and non-consumers

Table 6 compares the hematological parameters of the two groups. Consumers had significantly higher PCV (42.72 ± 3.83% vs 39.80 ± 3.90%; t = 3.777, p < 0.001), hemoglobin (14.27 ± 1.27 vs 13.31 ± 1.33 g/dL; t = 3.684, p < 0.001) and RBC count (4.75 ± 0.42 vs 4.41 ± 0.45 $\times 10^{12}$/L; t = 3.818, p < 0.001). Their WBC count was significantly lower (4.14 ± 2.11 vs 5.01 ± 1.32 $\times 10^{9}$/L; t = −2.465, p = 0.015).

In the differential count, neutrophils (30.10 ± 10.11% vs 52.04 ± 11.84%; t = −9.965, p < 0.001) and monocytes (0.62 ± 1.76% vs 3.78 ± 2.21%; t = −7.917, p < 0.001) were significantly lower in consumers, while lymphocytes (62.82 ± 11.92% vs 40.62 ± 12.64%; t = 9.035, p < 0.001) and eosinophils (6.10 ± 9.46% vs 3.18 ± 3.29%; t = 2.062, p = 0.042) were significantly higher. Platelet count was significantly lower in consumers (234.34 ± 26.67 vs 248.86 ± 28.70 $\times 10^{9}$/L; t = −2.621, p = 0.010). MCV, MCH, MCHC and basophils did not differ between the groups (p > 0.05).

Table 6: Independent-samples t-test comparison of hematological parameters between alcohol consumers and non-consumers

Variable	Alcohol consumers Mean ± SD (n = 50)	Non-consumers Mean ± SD (n = 50)	Mean difference	t-value	p-value
PCV (%)	42.72 ± 3.83	39.80 ± 3.90	2.92	3.777	<0.001*
Hb (g/dL)	14.27 ± 1.27	13.31 ± 1.33	0.96	3.684	<0.001*
RBC ($\times 10^{12}$/L)	4.75 ± 0.42	4.41 ± 0.45	0.33	3.818	<0.001*
WBC ($\times 10^{9}$/L)	4.14 ± 2.11	5.01 ± 1.32	−0.87	−2.465	0.015*
MCH (pg)	30.06 ± 0.21	30.17 ± 0.66	−0.12	−1.196	0.235

Neutrophils (%)	30.10 ± 10.11	52.04 ± 11.84	−21.94	−9.965	<0.001*
Lymphocytes (%)	62.82 ± 11.92	40.62 ± 12.64	22.20	9.035	<0.001*
Monocytes (%)	0.62 ± 1.76	3.78 ± 2.21	−3.16	−7.917	<0.001*
Eosinophils (%)	6.10 ± 9.46	3.18 ± 3.29	2.92	2.062	0.042*
Basophils (%)	0.36 ± 0.53	0.38 ± 0.49	−0.02	−0.197	0.844
Platelets (×10⁹/L)	234.34 ± 26.67	248.86 ± 28.70	−14.52	−2.621	0.010*
MCV (fL)	89.99 ± 1.58	90.34 ± 5.01	−0.35	−0.474	0.637
MCHC (g/dL)	33.41 ± 0.59	33.49 ± 1.87	−0.08	−0.296	0.768

F. Serum electrolytes of alcohol consumers and non-consumers

Table 7 compares serum electrolyte levels between the two groups. All three electrolytes were significantly higher in consumers: sodium 138.880 ± 2.256 vs 134.260 ± 2.423 mmol/L ($t = 9.869$, $p < 0.001$), potassium 3.932 ± 0.467 vs 3.320 ± 0.375 mmol/L ($t = 7.223$, $p < 0.001$) and chloride 98.680 ± 2.832 vs 97.380 ± 2.490 mmol/L ($t = 2.438$, $p = 0.017$). The difference was smaller for chloride than for sodium and potassium.

Table 7: Serum electrolyte levels (sodium, potassium and chloride) in alcohol consumers and non-consumers

Variable	**Alcohol consumers Mean ± SD (n = 50)**	**Non-consumers Mean ± SD (n = 50)**	**t-value**	**p-value**
Sodium (mmol/L)	138.880 ± 2.256	134.260 ± 2.423	9.869	<0.001*
Potassium (mmol/L)	3.932 ± 0.467	3.320 ± 0.375	7.223	<0.001*
Chloride (mmol/L)	98.680 ± 2.832	97.380 ± 2.490	2.438	0.017*

G. Analysis of variance of CK-MB, renal biochemical markers and hematological parameters by quantity of alcohol consumed per sitting

Table 8 shows the one-way ANOVA relating CK-MB, renal biochemical markers and hematological parameters to the number of drinks taken per sitting. CK-MB differed significantly across the groups ($F = 115.395$, $p < 0.001$): it was lowest in non-drinkers (1.68 ± 0.98 ng/mL) and between 5.13 and 5.37 ng/mL in the three drinking categories (5.34 ± 0.98, 5.13 ± 1.04 and 5.37 ± 0.92 ng/mL for 1–2, 3–4 and more than 4 drinks, respectively). Creatinine also differed ($F = 12.126$, $p < 0.001$), from 53.56 ± 7.80 µmol/L in non-drinkers to 69.33 ± 12.45 µmol/L in those taking more than 4 drinks, as did uric acid ($F = 3.846$, $p = 0.012$), which was highest in non-drinkers (0.43 ± 0.14 mmol/L). Urea ($F = 2.534$, $p = 0.061$) and eGFR ($F = 2.289$, $p = 0.083$) did not differ significantly, although consumers tended to have slightly higher urea and lower eGFR.

Among the hematological parameters, PCV ($F = 4.747$, $p = 0.004$), hemoglobin ($F = 4.456$, $p = 0.006$) and RBC count ($F = 4.798$, $p = 0.004$) were significantly different across the

groups, all being higher in drinkers than in non-drinkers. WBC count differed significantly ($F = 2.917$, $p = 0.038$) and fell from 5.01 ± 1.32 $\times 10^9$/L in non-drinkers to 3.48 ± 1.15 $\times 10^9$/L in those taking more than 4 drinks. Neutrophil percentage fell from 52.04 ± 11.84% in non-drinkers to about 30% in drinkers ($F = 32.508$, $p < 0.001$), whereas lymphocyte percentage rose from 40.62 ± 12.64% to 67.22 ± 8.91% in those taking more than 4 drinks ($F = 30.032$, $p < 0.001$). Monocyte percentage was much lower in drinkers ($F = 20.990$, $p < 0.001$), and eosinophil percentage differed significantly ($F = 5.216$, $p = 0.002$), with the highest mean (13.83 ± 14.91%) in the 3–4 drinks group. MCH ($F = 0.542$, $p = 0.654$), basophils ($F = 2.445$, $p = 0.069$), platelets ($F = 2.462$, $p = 0.067$), MCV ($F = 0.216$, $p = 0.885$) and MCHC ($F = 0.297$, $p = 0.828$) did not differ significantly.

Table 8: Analysis of variance of CK-MB, renal biochemical markers and hematological parameters by number of drinks per sitting

Variables	**Non-drinkers (n = 50)**	**1–2 drinks (n = 35)**	**3–4 drinks (n = 6)**	**>4 drinks (n = 9)**	**F-value**	**p-value**
Cardiac and renal parameters (mean ± SD)						
CK-MB (ng/mL)	1.68 ± 0.98	5.34 ± 0.98	5.13 ± 1.04	5.37 ± 0.92	115.395	<0.001*
Urea (mmol/L)	2.98 ± 0.47	3.27 ± 0.47	3.12 ± 0.44	3.28 ± 0.83	2.534	0.061
Creatinine (µmol/L)	53.56 ± 7.80	64.29 ± 11.69	63.50 ± 9.42	69.33 ± 12.45	12.126	<0.001*
eGFR (mL/min/1.73 m²)	124.47 ± 10.53	117.88 ± 13.19	121.14 ± 9.57	120.45 ± 10.82	2.289	0.083
Uric acid (mmol/L)	0.43 ± 0.14	0.37 ± 0.15	0.27 ± 0.08	0.32 ± 0.10	3.846	0.012*
Haematological parameters (mean ± SD)						
PCV (%)	39.80 ± 3.90	42.77 ± 3.77	43.17 ± 4.12	42.22 ± 4.32	4.747	0.004*
Hb (g/dL)	13.31 ± 1.33	14.25 ± 1.25	14.40 ± 1.35	14.26 ± 1.44	4.456	0.006*
RBC ($\times 10^{12}$/L)	4.41 ± 0.45	4.74 ± 0.42	4.80 ± 0.45	4.73 ± 0.48	4.798	0.004*
WBC ($\times 10^9$/L)	5.01 ± 1.32	4.40 ± 2.39	3.60 ± 1.04	3.48 ± 1.15	2.917	0.038*
MCH (pg)	30.17 ± 0.66	30.05 ± 0.24	30.00 ± 0.00	30.12 ± 0.11	0.542	0.654

Neutrophils (%)	52.04 ± 11.84	29.80 ± 9.15	31.50 ± 16.96	30.33 ± 9.45	32.508	<0.001*
Lymphocytes (%)	40.62 ± 12.64	63.43 ± 10.96	52.67 ± 16.95	67.22 ± 8.91	30.032	<0.001*
Monocytes (%)	3.78 ± 2.21	0.57 ± 1.90	1.33 ± 2.16	0.33 ± 0.50	20.990	<0.001*
Eosinophils (%)	3.18 ± 3.29	5.80 ± 8.83	13.83 ± 14.91	2.11 ± 3.76	5.216	0.002*
Basophils (%)	0.38 ± 0.49	0.40 ± 0.55	0.67 ± 0.52	0.00 ± 0.00	2.445	0.069
Platelets ($\times 10^9$/L)	248.86 ± 28.70	232.86 ± 28.34	242.50 ± 22.30	234.67 ± 23.90	2.462	0.067
MCV (fL)	90.34 ± 5.01	90.18 ± 0.74	89.92 ± 0.21	89.27 ± 3.50	0.216	0.885
MCHC (g/dL)	33.49 ± 1.87	33.32 ± 0.06	33.36 ± 0.08	33.79 ± 1.39	0.297	0.828

Values are significantly different at $p < 0.05$. CK-MB: creatine kinase-MB; eGFR: estimated glomerular filtration rate; PCV: packed cell volume; Hb: hemoglobin concentration; RBC: red blood cell count; WBC: white blood cell count; MCH: mean corpuscular hemoglobin; MCV: mean corpuscular volume; MCHC: mean corpuscular hemoglobin concentration; SD: standard deviation.

IV. DISCUSSION

A. Discussion of the Study

This study compared serum CK-MB, renal biochemical markers, electrolytes and haematological parameters between alcohol consumers and non-consumers in Oye-Ekiti. Serum CK-MB was markedly higher in consumers than in non-consumers. This agrees with the cardiovascular effects of alcohol described in the American Heart Association statement, particularly the association between heavy alcohol consumption and myocardial dysfunction.8 However, CK-MB is not completely cardiac specific and may also rise with skeletal muscle injury.12 The finding may therefore reflect increased release of enzyme from cardiac or skeletal muscle associated with alcohol exposure, but CK-MB elevation alone cannot establish myocardial injury or myocardial infarction. The renal findings also differed significantly. Consumers had higher mean urea and creatinine and a lower eGFR, consistent with evidence linking alcohol exposure to alterations in renal haemodynamic and function.15 They are also broadly consistent with Joo et al., who reported an association between certain patterns of alcohol consumption and progression of chronic kidney disease.27 Although eGFR was lower in consumers, the values remained within a generally preserved range,28 which suggests a difference in renal filtration rather than established chronic kidney disease.

Serum uric acid showed the opposite pattern, being significantly lower in consumers (0.346 ± 0.137 vs 0.428 ± 0.143 mmol/L; p = 0.004). This differs from the commonly reported association between alcohol consumption and hyperuricaemia.29 Kawamoto et al., however, showed that the relationship between alcohol, uric acid and renal function is more complex and may vary with drinking pattern and renal handling.30 The lower uric acid here may reflect drinking pattern, diet, renal urate handling, hydration status or other characteristics of the study population. None of these was directly assessed, so they cannot be established as explanations.

Sodium, potassium and chloride were significantly higher in consumers, which differs from the electrolyte disturbances commonly associated with alcohol-induced diuresis.15 the simultaneous rise in PCV, haemoglobin and red cell count raises the possibility that relative haemo concentration contributed to the higher electrolyte concentrations. This remains only a possible explanation, because hydration status was not directly examined.

The haematological findings were mixed. Consumers had higher PCV, haemoglobin and red cell count, but lower white cell, neutrophil, monocyte and platelet counts, with higher lymphocyte and eosinophil percentages. The lower white cell, neutrophil and platelet counts are broadly consistent with the haematological alterations reported by Maduka and Onuoha among alcohol consumers in Bayelsa State, 31 and are compatible with those reported by Oke et al. among chronic consumers in Akure, Southwest Nigeria.32 In contrast, the higher PCV and haemoglobin here differ from the lower PCV reported by Oke et al., 32 which suggests that factors such as hydration status may have influenced the red cell findings.

Across categories of drinks per sitting, CK-MB, creatinine, uric acid, PCV, haemoglobin, red cell count, white cell count, neutrophils and lymphocytes differed significantly, whereas urea, eGFR, platelet count and the red cell indices did not. The relationship between alcohol consumption and these laboratory parameters was therefore not uniform across markers. Overall, the findings show significant differences in selected cardiac, renal, electrolyte and haematological parameters between consumers and non-consumers. Some are consistent with previous evidence and others differ from commonly reported patterns.

B. Strength and Weaknesses of the Study

The study recruited participants from motor parks, beer parlours, and palm wine tapping points and community gatherings. This community-based approach reached habitual and occupational drinkers who may be under-represented in hospital-based or campus-based studies. Cardiac, renal, electrolyte and haematological markers were measured in the same individuals, and testing was done on automated analysers with internal quality control on each batch and daily XN-CHECK controls for the full blood count. Alcohol exposure was self-reported and was not checked against objective markers such as gamma-glutamyl transferees (GGT), carbohydrate-deficient transferrin (CDT) or phosphatide ethanol (PEth). Hydration status was not assessed, troponins were not measured alongside CK-MB.

C. Implications of the Findings of the Study

Regular alcohol consumers may benefit from routine health assessment that includes cardiovascular, renal and haematological parameters where clinically indicated. Periodic

measurement of serum creatinine, urea and eGFR should be considered in people who drink heavily or frequently, so that biochemical changes are picked up early. Alcohol education and risk-reduction programmes should be strengthened among groups with high exposure, notably students, commercial drivers and motorcycle riders. This study adds local data from Oye-Ekiti on an integrated cardiac, renal, electrolyte and haematological profile in drinkers drawn from occupational and social groups that laboratory-based alcohol research often misses. Future studies should confirm self-reported alcohol exposure with objective markers such as GGT, CDT or PEth.

V. CONCLUSION

This study found significant differences in selected cardiac, renal, electrolyte and haematological parameters between alcohol consumers and non-consumers in Oye-Ekiti. Consumers had higher serum CK-MB, urea and creatinine and a lower eGFR, together with significant differences in sodium, potassium and chloride and in several haematological parameters, particularly WBC, neutrophils, lymphocytes, monocytes and platelets. The lower uric acid and the higher PCV, haemoglobin, RBC count and electrolytes in consumers differed from some commonly reported patterns and may reflect hydration status and other features of this population. The findings indicate an association between alcohol consumption and altered laboratory parameters across several systems, while the cross-sectional design limits causal interpretation.

RECOMMENDATIONS

Regular alcohol consumers should undergo periodic health screening incorporating renal parameters and cardiovascular enzymes to detect subclinical organ dysfunction early. Alcohol risk-reduction and educational interventions should be prioritized among high-exposure occupational groups, particularly commercial drivers and motorcycle riders in community settings. Future studies should confirm self-reported alcohol intake using objective biomarkers such as GGT, CDT, or PEth while evaluating cardiac troponins alongside CK-MB to clarify the nature of muscle involvement. Finally, longitudinal designs with direct assessment of hydration status are recommended to establish causal relationships and validate these multi-system alterations.

AUTHORS CONTRIBUTIONS AND DECLARATION

Adegbola Abdullahi Adesina: conceptualization, research execution, data and sample collection, laboratory analysis, data analysis, project coordination, writing of the original draft, writing and review of the final draft. Babajide Adewoyin Adeleke: project coordination, journal communication, sample collection and laboratory analysis, data analysis, writing and review of the final draft. Boluwatife Samuel Fatokun: sample collection, data analysis, writing of the original draft, writing and review of the final draft. Odunayo Iyanuoluwa Oluwasegun: data and sample collection, writing of the original draft. Folorunsho Davies Olumide: research execution, writing of the original draft. Olubodun Olaotan Esther: data and sample collection, writing and review of the final draft. Aladesuru Sharon Feyisayo:

research execution, writing and review of the final draft. Shittu Roqeebat Mosunmola: research execution, data collection, writing and review of the final draft.

A. Ethics Approval and Consent to Participate:

Ethical approval was obtained from the Research Ethics Committee of the Ekiti State Ministry of Health, Ado-Ekiti (EKHREC/EA/U/206) and the College of Medicine Research Ethics Committee, Federal University Oye-Ekiti (FUO/COMREC/MLS/2026/076).

B. Consent for Publication:

Informed consent was obtained from all participants in this research.

C. Availability of Data and Materials:

The datasets generated and analysed during this study are available from the corresponding author on reasonable request.

D. Competing Interests:

No competing interests were declared.

E. Funding:

We have not received any financial support for this research or manuscript.

F. Acknowledgment:

None.

All co-authors approved the final manuscript.

All co-authors have read and approved the submission.

ABBREVIATIONS

ANOVA: analysis of variance
ASSIST: Alcohol, Smoking and Substance Involvement Screening Test
BP: blood pressure
CDT: carbohydrate-deficient transferrin
CI: confidence interval
CK-MB: creatine kinase-MB
eGFR: estimated glomerular filtration rate
EDTA: ethylenediaminetetraacetic acid
FIA: fluorescence immunoassay
GGT: gamma-glutamyl transferase
Hb: haemoglobin
LGA: Local Government Area
MCH: mean corpuscular haemoglobin
MCHC: mean corpuscular haemoglobin concentration
MCV: mean corpuscular volume
PCV: packed cell volume

PEth: phosphatidylethanol
POD: peroxidase
RBC: red blood cell
SD: standard deviation
SPSS: Statistical Package for the Social Sciences
WBC: white blood cell
WHO: World Health Organization

REFERENCES

[1] World Health Organization. Global status report on alcohol and health and treatment of substance use disorders. Published 2024. Accessed October 25, 2026. https://www.who.int/publications/i/item/9789240096745

[2] Ibraheem W. Traditional alcoholic beverages in Nigeria: A review. J Ethn Foods. 2018; 5(3):177-183.

[3] Kehinde OS, Adegoke AE. Taking alcohol by deception: An analysis of ethanol concentration of "paraga," an alcoholic herbal mixture in Nigeria. BMC Res Notes. 2012; 5:127. doi:10.1186/1756-0500-5-127

[4] Dike EN, Okafor CJ, Nwachukwu A. Assessment of ethanol and heavy metal content of commercial herbal bitters sold in South-western Nigeria. J Toxicol Environ Health Sci. 2020; 12(3):45-52. doi:10.5897/JTEHS2020.0448

[5] Adeloye D, Olawole-Isaac A, Auta A, et al. Epidemiology of harmful use of alcohol in Nigeria: A systematic review and meta-analysis. Am J Drug Alcohol Abuse. 2019; 45(5):438-450. doi:10.1080/00952990.2019.1628244

[6] Kumolalo FB, Obadeji A, Adegoke BO. Substance use trends among undergraduate students and its association with socio demographics and self-esteem. Taiwanese J Psychiatry. 2023; 37(1):41-46. doi:10.4103/TPSY.TPSY_5_23

[7] Lasebikan VO, Ola BA. Prevalence and correlates of alcohol use among a sample of Nigerian semirural community dwellers in Nigeria. J Addict. 2016; 2016:2831594. doi:10.1155/2016/2831594

[8] Piano MR, Marcus GM, Aycock DM, et al. Alcohol use and cardiovascular disease: A scientific statement from the American Heart Association. Circulation. 2025; 152(1):e7-e21. doi:10.1161/CIR.0000000000001341

[9] Gupta S, Ahimsadasan N, Dalsania K, et al. Alcohol and cardiovascular disease. Am J Cardiol. 2026; 259:132-138. doi:10.1016/j.amjcard.2025.09.035

[10] Wood AM, Kaptoge S, Butterworth AS, et al. Risk thresholds for alcohol consumption: Combined analysis of individual-participant data for 599,912 current drinkers in 18 countries. Lancet. 2018; 391(10129):1513-1523. doi:10.1016/S0140-6736(18)30134-X

[11] Contreras-Zentella ML, Villalobos-García D, Hernández-Muñoz R. Ethanol metabolism in the liver, the induction of oxidant stress, and the antioxidant defence system. Antioxidants. 2022; 11(7):1258. doi:10.3390/antiox11071258

[12] Kurapati R, Zubair M. Creatine kinase MB: Diagnostic utility and limitations. In: Stat Pearls. Stat Pearls Publishing; 2026. https://www.ncbi.nlm.nih.gov/books/NBK557591/

[13] Siegel AJ, Rybak SL, McArdle JJ. Increased CK-MB activity in alcoholic patients. Ann Intern Med. 1985; 103(6_Part_1):857-859.
[14] Ojo AA, Ojo OO, Ojo OS. Cardiac troponins in the diagnosis of myocardial infarction. Niger J Cardiol. 2026; 23(1):1-10.
[15] Yokus E, Erturk M, Aksoy M. Impact of alcohol misuse on renal function parameters and electrolyte balance. Ren Fail. 2026; 48(2):215-224. doi:10.1080/0886022X.2026.2301245
[16] Burtis CA, Ashwood ER, Bruns DE. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 6th ed. Elsevier; 2018.
[17] Nowak K, Surazynski A, Zalewska A. Electrolyte disturbances in chronic alcohol abuse. Nutrients. 2020; 12(9):2845. doi:10.3390/nu12092845
[18] Cytryn E, Schmidt N, Winters AC, Kushner T. No hepatic manifestations of alcohol use disorder and alcohol-related liver disease. Clin Liver Dis. 2026; 30(1):127-146. doi:10.1016/j.cld.2025.09.004
[19] Shimna CS, Girgla KK, Regalla SR, Srivastava S, Tyagi A. Impact of alcohol consumption on blood indices: Systematic review. Eur J Cardiovascular Med. 2025; 15(10):42-47.
[20] Usman DM, Sunday OK, Adekunle AO, et al. Alcohol consumption patterns and associated factors in Ekiti State, Nigeria: a state-wide, cross-sectional study. Arch Psychiatry Res. 2024; 60(3):219. doi:10.20471/may.2024.60.03.04
[21] Ojo TA, Adebayo OS, Bakare AA. Comparative analysis of renal function parameters using automated enzymatic methods in a Nigerian population. Afr J Biomed Res. 2023; 26(2):145-152.
[22] Soleimani M, Zare H, Mousavi SM. Kinetic determination of creatinine based on the Jaffe reaction using an automated clinical chemistry analyser. Anal Methods Clin Chem. 2023; 2023:5567890. doi:10.1155/2023/5567890
[23] Palekar S, Kumar A, Singh R. Optimization of uricase-peroxidase coupled enzymatic assay for serum uric acid determination. Biochem Eng J. 2025; 190:108754. doi:10.1016/j.bej.2024.108754
[24] Guangzhou Wondfo Biotech Co., Ltd. Fine care™ FIA Meter plus Operation Manual. Guangzhou Wondfo Biotech Co., Ltd.; 2016.
[25] El Bachiri K, Mousty C, El Rhazi M. Electrochemical detection of electrolytes using a solid-state ion-selective electrode system. Sensors. 2021; 21(8):2684. doi:10.3390/s21082684
[26] Chen Z, Zhang Y, Liu X. Performance evaluation of the Sysmex XN-1000 automated haematology analyser in a clinical laboratory setting. J Clin Lab Anal. 2022; 36(5):e24389. doi:10.1002/jcla.24389
[27] Joo YS, Koh H, Park J, et al. Alcohol consumption and progression of chronic kidney disease: Results from the Korean cohort study for outcome in patients with chronic kidney disease. Mayo Clin Proc. 2020; 95(2):293-305.
[28] Inker LA, Schmid CH, Tighiouart H, et al. New creatinine- and cystatin C–based equations to estimate GFR without race. N Engl J Med. 2021; 385(19):1737-1749. doi:10.1056/NEJMoa2102953
[29] Miake J, Hisatome I, Tomita K, et al. Impact of hyper- and hypo-uricemia on kidney function. Biomedicines. 2023; 11(5):1258. doi:10.3390/biomedicines11051258
[30] Kawamoto R, Kikuchi A, Akase T, Ninomiya D, Tokumoto Y, Kumagi T. Alcohol consumption and serum uric acid are synergistically associated with renal

dysfunction among community-dwelling persons. J Clin Lab Anal. 2021; 35(7):e23812. doi:10.1002/jcla.23812

[31] Maduka VA, Onuoha CE. Evaluation of some haematological parameters in alcoholics' residents in Bayelsa State, Nigeria. Asian J Biol Sci. 2023; 16(4):566-571. doi:10.3923/ajbs.2023.566.571

[32] Oke OA, Adeyemi O, Babatunde A. Haematological and biochemical alterations associated with local alcoholic beverage consumption in Nigerian adults. Niger Med J. 2026; 67(1):34-41.