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Alcohol and the Heart: A Study of Echocardiographic Changes Among a Population of Male Consumers

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ABSTRACT **Original Research Article**

Background: Alcohol intake has been implicated in various cardiovascular complications. However, limited echocardiographic data exist on habitual alcohol consumers within the Nigerian population. Echocardiography offers a non-invasive and reliable method for evaluating cardiac structure and function. This investigation explored the long-term effects of regular alcohol consumption on cardiac morphology and function, focusing on residents of the Rumuekini communities in Rivers State.

Methods: Forty-eight adult males voluntarily participated in the study, each completing a structured questionnaire prior to echocardiographic assessment. Information on demographic profile, drinking habits, symptoms, and clinical examination findings was collected by a trained interviewer. Cardiac imaging was performed using an ATL high-definition ultrasound system, adhering to a standardized protocol. Measurements were compared with established Nigerian reference values for normal echocardiographic parameters. Approval for the study was granted by community authorities.

Results: The participants had a mean age of 33.9 ± 16.1 years, average BMI of 23.83 ± 3.43 kg/m², mean systolic blood pressure of 129.8 ± 21.1 mmHg, and diastolic pressure of 77.48 ± 13.9 mmHg. The reported average daily alcohol consumption was 74.88 ± 45.5 units. Echocardiography revealed a mean left atrial diameter of 3.48 ± 0.45 cm, left ventricular mass index (LVM/BSA) of 181.2 ± 284 g/m², ejection fraction of 61.66 ± 15.1%, and E/A ratio of 1.48 ± 0.49.

Conclusion: Regular alcohol consumption in this population was linked to notable alterations in cardiac structure and function affecting both the left and right sides of the heart.

Keywords: Alcohol consumption, Echocardiography, Ethanol, Cardiac structure, Left ventricular hypertrophy, Diastolic dysfunction, Cardiovascular health, Rumuekini, Nigeria.

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Introduction

In 2022, the *World Heart Federation* issued a policy brief titled *"Myths and Measures"*, addressing the relationship between alcohol intake and cardiovascular health.[1] This document directly refuted the long-standing belief that moderate alcohol use lowers the risk of heart disease, categorizing it as a misconception. The brief advocated for tighter regulatory control on alcohol consumption and linked part of the almost 100% global increase in cardiovascular disease prevalence over the past two decades to alcohol intake. Despite this stance, several studies[2–5] report divergent opinions among researchers regarding the influence of moderate alcohol use; however, there is consensus that excessive consumption produces harmful cardiovascular effects.[6–8] In light of the Federation's recent position, it becomes important to re-examine these contrasting findings.

Ethnicity and genetic background significantly influence disease patterns, presentation, and pharmacological or

physiological responses to various agents.[9,11] The present series of investigations was designed to explore and expand the body of knowledge on this critical subject.

The cardiovascular impact of alcohol ingestion is dose- and duration-dependent.[10] Short-term effects include transient tachycardia and a brief rise in blood pressure, typically resolving within minutes after drinking.[11] In contrast, long-term heavy consumption is associated with sustained adverse effects—ranging from benign premature beats to severe arrhythmias such as atrial fibrillation. Chronic alcohol use has been repeatedly linked to arrhythmias, including premature atrial complexes, premature ventricular complexes, and sinus tachycardia. One study noted that heart rate increases may peak four hours after alcohol ingestion.[13]

Hypertension is also widely regarded as one of the most frequent complications of sustained alcohol use. Although in over 95% of cases hypertension has no clearly identifiable cause, it is influenced by multiple risk factors—both modifiable and non-modifiable.[14] Alcohol intake is considered a modifiable risk factor in the pathogenesis of elevated blood pressure, partly through its influence on vascular intima-media thickness (IMT) and arterial diameter. Findings from an earlier study in this research series revealed increased vessel wall thickness and stiffness, alongside reduced lumen size, in heavy drinkers compared to non-drinkers.[15] Such vascular narrowing is likely a result of alcohol-induced changes in vascular smooth muscle tone, leading to vasoconstriction and higher blood pressure.

Another pathway involves alcohol's effect on vascular endothelial function. It has been associated with modulation of nitric oxide release, influencing vascular relaxation and tone.[16,17] Genetic predisposition may further modify this relationship; for example, research in Japanese hypertensive patients found that those carrying the inactive ALDH2*2 variant (aldehyde dehydrogenase gene) exhibited greater blood pressure reductions after alcohol intake than individuals homozygous for the active ALDH2*1 allele.[18,19] This effect is attributed to acetaldehyde-mediated vasodilation following alcohol ingestion.

Beyond vascular and rhythm disturbances, alcohol can impair myocardial structure and function, leading to alcoholic cardiomyopathy—a mixed form of primary cardiomyopathy with both acquired and genetic components.[20] While its development is linked to chronic alcohol exposure, genetic factors, such as mutations in the TTNtv gene, may increase susceptibility.[20,21] Chronic exposure results in mitochondrial damage and release of cytolytic enzymes, contributing to myocardial injury. There is also growing interest in understanding the acute consequences of binge drinking.[22,23]

Alcohol misuse has been tentatively associated with infective endocarditis, although this remains underexplored. The organisms most often implicated include *Diplococcus pneumoniae* and *Streptococcus agalactiae*.[24,25] Alcohol-related endocarditis is often severe and potentially fatal. Experimental evidence has linked alcohol exposure to endomyocardial fibrosis (EMF) in animal models, although human studies are lacking.[26,27]

Alcohol's absorption and distribution characteristics also raise questions about its effects on the pericardium. Alcohol-related pericarditis, thought to be mediated by pro-inflammatory cytokine release, has been documented, prompting investigations into its pathophysiology.[28] Notably, most structural and functional effects of alcohol seem to be more pronounced in the left heart than the right, possibly reflecting distribution patterns and direct vascular exposure to circulating ethanol.[29]

Taken together, alcohol-induced myocardial disease may be better understood as a spectrum involving the endocardium, myocardium, and pericardium. This comprehensive view has implications for prevention, treatment strategies, and disease progression monitoring.

While some studies suggest potential cardiovascular benefits from alcohol consumption, debate persists regarding whether these effects stem from ethanol itself or from other beverage constituents such as polyphenols, flavanols, anthocyanins, phenolic acids, and resveratrol.[30] Resveratrol, for instance, has been shown to inhibit LDL oxidation, reduce platelet aggregation, and counteract cholesterol's prothrombotic effects.

Alcohol may also increase circulating levels of tissue plasminogen activator (t-PA), a fibrinolytic enzyme, potentially conferring protection against thrombosis.[31] Furthermore, ischemic preconditioning effects have been reported in association with alcohol intake.[32] Lipid profile changes—such as increased HDL through reduced cholesteryl ester transfer protein (CETP) activity and lowered LDL—add to the proposed protective mechanisms. Nevertheless, alcohol has also been found to reduce net protein synthesis in the myocardium, particularly under stress conditions, an effect observed in multiple studies though not yet fully explained.[33]

The present study applies these insights to echocardiographic evaluation of habitual alcohol consumers in the Rumuekini community, with the goal of further clarifying alcohol's structural and functional cardiac effects.

Methods

Study Design

This investigation employed a prospective, cross-sectional, community-based approach.

Study Location

The research was conducted in a temporary echocardiography facility set up within the Rumuekini town hall, a peri-urban settlement in Rivers State.

Ethical Approval

Authorization for the study was granted by the Rumuekini Community Development Committee and the traditional ruler of the community. Written informed consent was obtained from each participant after a thorough explanation of the study's objectives and procedures. Participant privacy was maintained throughout, and no form of judgement or discrimination was expressed toward subjects.

Study Population

A total of 48 male residents voluntarily participated in the echocardiographic arm of a larger investigation into cardiovascular effects among alcohol consumers. No women were recruited. All volunteers were counselled about the protocol and provided informed consent prior to enrolment.

Exclusion Criteria

Capillary blood glucose levels were measured in all participants, and those diagnosed with diabetes mellitus were excluded from the study.

Data Collection

A structured questionnaire captured demographic data, occupation, presenting symptoms, preferred type of alcoholic beverage, estimated daily alcohol consumption, and smoking status. This questionnaire was integrated with a proforma for documenting clinical examination results, drug history, and findings from electrocardiography, echocardiography, and carotid Doppler studies. The present paper reports on the echocardiographic outcomes and the prevalence of structural and functional abnormalities among heavy alcohol users in Rumuekini.

Echocardiographic Parameters and Definitions of Abnormality

1. **Left Atrial Diameter (LAD):** A measurement >4.0 cm was considered abnormal, based on reference values reported by Oyati et al.^34
2. **Left Ventricular Internal Diameter (LVID):** An abnormal threshold was defined as >5.6 cm.^34
3. **Relative Wall Thickness (RWT):** RWT was calculated as 2 × posterior wall thickness ÷ LV internal diameter. Values >0.43 were classified as concentric LV hypertrophy, while <0.26 indicated eccentric LV hypertrophy, in accordance with the American Society of Echocardiography (ASE) guidelines.^35
4. **Left Ventricular Mass Indexed to Body Surface Area (LVM/BSA):** Normal male reference values generally range between 70–120 g/m².^35
5. **Left Ventricular Ejection Fraction (LVEF):** LVEF <50% was considered reduced.^35
6. **Diastolic Function:** Evaluated using the E/A ratio and deceleration time (DecT).^35

Data Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 23. Continuous variables were presented as mean ± standard deviation (SD), while categorical variables were expressed as percentages. Comparisons between means were made using paired t-tests, and categorical variables were compared using chi-square tests. A *p*-value <0.05 was considered statistically significant.

Results

Alcohol Consumption Patterns

Alcoholic drinks consumed included beers (*Star*, *Golder*, *Guinness*, *Heineken*, *Harp*, *Legend*), locally brewed gin (*kai-kai*), palm wine, whisky, and *Squadron*. Beer was the most frequently consumed beverage. Alcohol content estimates were based on 50 g/cl for beer,[36] 20–40 g/l for whisky and similar liquors,[36 40] g/l for local gin,[36] while palm wine alcohol content varied depending on fermentation time after tapping.

Classification of Drinkers

Although all participants were heavy consumers, they were further stratified into:

- **Mild drinkers:** ≤50 g/day
- **Moderate drinkers:** 51–100 g/day
- **Excessive drinkers:**>100 g/day

The total daily intake ranged from 15 g to 190 g.

Demographics and Clinical Profile

The mean age was 33.9 ± 16.1 years (range: 19–91 years). Mean BMI was 23.83 ± 3.43 kg/m². Mean systolic BP was 129.8 ± 21.1 mmHg and mean diastolic BP 77.48 ± 13.9 mmHg. The mean estimated daily alcohol intake was 74.88 ± 45.5 g. Students accounted for the largest occupational group (18 participants, 40.42%), while 16 (33%) reported smoking.

Symptoms and Clinical Findings

Chest pain was the most frequently reported symptom, followed by palpitations and dyspnoea. Three individuals met Framingham's criteria for congestive cardiac failure, presenting with displaced apex beats, elevated jugular venous pressure, and cardiac murmurs.

Regression Analysis

- **E/A Ratio:** BMI emerged as the sole significant predictor ($p = 0.040$), with a negative correlation. Other variables (age, alcohol quantity, SBP, DBP) were non-significant. Model $R^2 = 14.3\%$.
- **Ejection Fraction (EF):** None of the predictors reached significance; $R^2 = 4.9\%$, suggesting unmeasured factors contribute more strongly to EF variability.
- **LVM/BSA:** SBP was the only significant predictor ($p = 0.041$) with a positive correlation. Age, BMI, alcohol quantity, and DBP showed no significant association. Collinearity diagnostics indicated no multicollinearity among predictors in all models.

Table 1. Cardiovascular and echocardiographic parameters

Cardiovascular Parameter	Mean ± SD
Age (years)	33.9 ± 16.1
BMI	23.83 ± 3.43
SBP	129.8 ± 21.1
DBP	77.48 ± 13.9
PR	74.39 ± 10.9
QUANTITY	74.88 ± 45.5
LAD	3.478 ± 0.45
ACS	2.277 ± 0.48
IVSD	1.117 ± 0.53
LVPWD	1.645 ± 1.68
LVIDD	4.684 ± 0.79
RWT	0.722 ± 0.67
LVM	177.73 ± 36.9
LVM/BSA	92.17 ± 16.0
EF	61.66 ± 15.1
FS	33.31 ± 12.6
E/A	1.481 ± 0.49

Table 2. Participant characteristics and echocardiographic findings

Parameter	N	Percentage (%)
Students	23	36.8%
Smokers	16	33.33%
Hypertensives	10	20.83%
Ejection Fraction – Normal	36	75.00%
Ejection Fraction – Depressed	8	16.67%
Ejection Fraction – Exaggerated	4	8.33%
Diastolic Function – Normal	24	50.00%
Diastolic Function - Grade 1	10	14.58%
Diastolic Function - Grade 2	7	10.42%
Diastolic Function - Grade 3	7	12.5%
LVH – Normal	23	47.92%
LVH – Concentric	23	47.92%
LVH – Eccentric	2	4.17%

Table 3. Paired samples test and correlations between smoking and non smoking population

Variable	Non-Smokers Mean± SD	Smokers Mean (S)± SD	SEM	t	P-value
AGEyrs	56.47±17.62	52.93±17.62	4.55	0.78	0.45
BMI (kg/m$^{2)}$	23.49±4.15	23.54±4.15	1.20	-0.05	0.97
SBP (mmHg)	128.93±18.31	125.53±18.31	4.73	0.72	0.48
DBP (mmHg)	82.13±13.32	75.93±13.32	3.44	1.80	0.09
Alcohol Quantityg/dl	75.93±81.76	74.33±81.76	21.11	0.08	0.94
LAD (cm)	3.53±0.55	3.33±0.55	0.15	1.33	0.21
LVM/BSA (Kg/cm	180.00±533.69	196.03±533.69	137.80	-0.12	0.91
EF (%)	69.13±19.73	61.03±19.73	5.09	1.59	0.13
FS (%)	37.47±14.92	32.11±14.92	3.85	1.39	0.19
E/A	1.58±0.56	1.44±0.56	0.18	0.80	0.44
RWT	1.06±0.75	0.85±0.78	0.31	0.69	0.50

Table 4. Regression analysis for EF

Predictor	Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig.
(Constant)	57.78	20.91		2.76	0.01
AGE	0.08	0.16	0.08	0.47	0.64
BMI	-0.13	0.77	-0.03	-0.17	0.87
QUANTITY	0.04	0.05	0.14	0.85	0.4
SBP	-0.11	0.14	-0.16	-0.77	0.45
DBP	0.204	0.215	0.196	0.95	0.35

Table 5. Regression analysis for LVM/BSA

Predictor	Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig.
(Constant)	274.7	382.937		0.72	0.48
AGE	-3.463	2.95	-0.186	-1.17	0.25
BMI	-11.43	14.17	-0.13	-0.81	0.43
QUANTITY	-0.48	0.95	-0.08	-0.51	0.62
SBP	5.52	2.61	0.42	2.12	0.04
DBP	-4.93	3.94	-0.25	-1.25	0.22

Table 6. Regression analysis for LAD

Predictor	Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig.
(Constant)	1.93	0.51		3.77	0.0
BMI	0.02	0.02	0.14	0.98	0.33
AGE	-0.0	0.0	-0.04	-0.28	0.7
SBP	0.01	0.0	0.49	2.65	0.01
DBP	-0.0	0.01	-0.09	-0.5	0.62
QUANTITY	0.0	0.0	0.17	1.23	0.22

Discussion

This cross-sectional study evaluated the echocardiographic profile of male residents in Rumuekini Community, Rivers State, Nigeria, with the primary aim of assessing the cardiovascular impact of chronic alcohol consumption. A total of 48 male participants were enrolled, with a mean age of 33.9 ± 16.1 years, capturing a broad age spectrum from late adolescence to elderly adulthood. This wide age distribution allows for a broader interpretive lens on how alcohol may differentially affect cardiac function across the lifespan. Although the sample size is relatively modest, it surpasses the minimum calculated sample size of 33 required for statistical significance at a 95% confidence level, thus providing sufficient power to detect meaningful associations and group differences. [36]

Participants exhibited a mean Body Mass Index (BMI) of 23.83 ± 3.43 kg/m², placing the average subject within the normal weight range, although values ranged from underweight to borderline obese categories. Blood pressure measurements revealed a mean systolic pressure of 129.8 ± 21.1 mmHg and a diastolic pressure of 77.48 ± 13.9 mmHg, reflecting a population at the threshold of pre-hypertension. This may hint at early cardiovascular stress in this community even among ostensibly healthy individuals.

The majority of respondents were either self-employed or students, with 36.8% identifying as students, pointing to a youthful and active demographic. Importantly, 33.33% of participants were smokers, while 66.67% were non-smokers. This is a substantial proportion, suggesting that tobacco use remains a significant modifiable risk factor in this population and warrants public health attention.

Only 20.83% of participants were hypertensive. While this may initially appear to be a modest proportion, the clinical significance is heightened by the relatively young mean age of 33.9 years. This early onset of hypertension raises important red flags, especially in light of concurrent lifestyle risk factors such as tobacco use (33.33%) and frequent alcohol consumption. In populations where cardiovascular risk factors traditionally emerge later in life, such findings suggest a shifting epidemiological trend—one in which younger individuals are developing subclinical cardiovascular remodeling earlier, likely driven by modifiable behaviors.

The observation aligns with emerging data from African populations where urbanization, stress, poor dietary habits, and substance use are increasingly contributing to early vascular aging and premature hypertensive heart disease.[37]

The study exclusively recruited male participants, which limits its applicability to women, who may exhibit different

patterns of alcohol metabolism, cardiovascular response, and disease trajectory. Nevertheless, focusing on an all-male cohort allows for gender-specific insights that are often overlooked in cardiovascular research, especially within Sub-Saharan African communities

The echocardiographic evaluation among the 48 male participants revealed several pertinent structural and functional cardiac insights. The mean left atrial diameter (LAD) was 3.48 ± 0.45 cm, while the mean left ventricular mass indexed to body surface area (LVM/BSA) stood at 181.2 ± 284 g/m². Left ventricular systolic function, assessed via ejection fraction (EF), had a mean of 61.66 ± 15.1%, while the mean E/A ratio—a surrogate for diastolic function—was 1.48 ± 0.49.

A majority (75%) of participants had normal ejection fractions. However, 16.67% demonstrated depressed systolic function, while 8.33% had exaggerated EF—often reflective of hyperdynamic circulation or volume overload states, potentially linked to chronic alcohol consumption.

Diastolic dysfunction (DD) was notably present in 50% of the cohort, indicating subclinical impairment in ventricular relaxation. Specifically, Grade 1 DD (Impaired relaxation) was present in 10 participants (14.58%),

Grade 2 (Pseudo-normal filling) in 7 participants (10.42%), and Grade 3 (Restrictive filling) in 7 participants (12.5%).

Left ventricular hypertrophy (LVH) was prevalent in 25 individuals (52.08%), with a striking dominance of concentric LVH (47.92%), and only 2 participants (4.17%) manifesting eccentric LVH. Interestingly, left atrial enlargement (LAE) was seen in 5 subjects (10.42%), and left ventricular internal dimension in diastole (LVIDd) > 5.6 cm, suggestive of chamber dilatation, was also present in only 5 participants (10.42%).

These findings converge on the insight that alcohol consumption in this cohort is more strongly associated with concentric LVH and diastolic dysfunction than with overt systolic dysfunction or marked chamber dilatation. Concentric remodeling, a hallmark of pressure overload, may reflect alcohol's hypertensive effect on the myocardium, while early diastolic dysfunction could denote subclinical myocardial stiffening, especially among those with elevated body mass indices. The predominance of concentric left ventricular hypertrophy (LVH) in our cohort aligns a Nigerian echocardiographic study by Adebayo et al. (2015)[37] among hypertensive males showed a similar pattern, with concentric LVH being the most prevalent geometric alteration, likely due to chronic pressure overload.

In contrast, alcoholic cardiomyopathy (ACM) often associated with longstanding excessive alcohol intake is typically characterized by eccentric LVH and dilated cardiac chambers, evolving into a phenotype similar to dilated cardiomyopathy (DCM). However, in our cohort, eccentric LVH and overt ventricular dilation were observed in only a minority (4.17% and 10.42%, respectively), possibly reflecting either: it remains an area of debate whether concentric LVH inevitably transitions into DCM. Some longitudinal studies suggest that sustained afterload (hypertension, alcohol toxicity) can lead to myocyte apoptosis, interstitial fibrosis, and ultimately chamber dilation. However, this progression is neither linear nor inevitable. Concentric hypertrophy may remain stable for years, particularly if hypertension is controlled or alcohol exposure reduced.[38-40]

As for eccentric LVH, while traditionally associated with volume overload states and systolic dysfunction, emerging imaging techniques like strain echocardiography and cardiac MRI have shown that viable myocardial tissue may persist, especially in early stages.[41- 44] This suggests potential reversibility or at least stabilization with abstinence and medical therapy a key message in patient counseling.

A South African study by Ntusi et al.[45] also noted that early alcoholic cardiac changes may mimic hypertensive heart disease, complicating the diagnostic distinction. Moreover, in resource-limited settings, the overlap of nutritional deficiency, alcohol use, and undiagnosed hypertension muddies the pure phenotype of "alcoholic cardiomyopathy."

Echocardiographic Differences between Smokers and Non-Smokers

When comparing echocardiographic parameters between the cohort of smokers and non-smokers, no statistically significant differences were observed. However, subtle trends emerged that warrant further investigation. The mean ejection fraction (EF) was lower among smokers (61.03 ± 19.73%) compared to non-smokers (69.13 ± 19.73%), suggesting early subclinical systolic dysfunction. Similarly, fractional shortening (FS) was reduced in smokers (32.11 ± 14.92%) versus non-smokers (37.47 ± 14.92%), although these differences did not reach statistical significance ($p = 0.13$ and $p = 0.19$, respectively).

Interestingly, smokers had slightly smaller left atrial diameters (3.33 ± 0.55 cm) than non-smokers (3.53 ± 0.55 cm), and lower E/A ratios, pointing toward possible diastolic dysfunction, though again non-significant ($p = 0.21$ and $p = 0.44$). Left ventricular mass indexed to BSA (LVM/BSA) was paradoxically higher in smokers (196.03 g/m²) than in non-smokers (180.00 g/m²), but the large standard deviation suggests underlying heterogeneity in cardiac remodeling. Relative wall thickness (RWT) was also lower among smokers (0.85 ± 0.78) compared to non-smokers (1.06 ± 0.75), a pattern that may reflect a shift toward eccentric geometry in some smokers.

Though these findings were not statistically significant, possibly due to small subgroup sizes, they align with existing literature[46-48] linking tobacco use to myocardial oxidative stress, endothelial dysfunction, and altered autonomic tone.

Larger, adequately powered studies may be needed to validate these early trends and elucidate the additive or synergistic cardiotoxicity of alcohol and tobacco combined.

Multivariate Predictors of Echocardiographic Parameters

The regression analysis revealed important insights into the predictors of echocardiographic changes in this cohort. Notably, systolic blood pressure (SBP) emerging as a significant predictor of both left ventricular mass index (LVM/BSA) ($p = 0.04$) and left atrial diameter (LAD) ($p = 0.01$). These findings reinforce the well-established role of pressure overload in driving structural cardiac remodeling, particularly concentric hypertrophy and left atrial enlargement, both of which were frequently observed in this study. Interestingly, no significant predictors were identified for ejection fraction (EF), suggesting that systolic function may be preserved in early stages of alcohol-related myocardial remodeling. This aligns with the known pathophysiological trajectory in which structural abnormalities—particularly diastolic dysfunction and hypertrophy—precede overt systolic impairment.

Although alcohol quantity and BMI did not emerge as statistically significant predictors, they demonstrated directional associations with key variables, such as E/A ratio and LVM/BSA, suggesting that with a larger sample, more robust associations might be observed.

In sum, these findings underscore the central role of elevated systolic blood pressure as a modifiable determinant of alcohol-related cardiac changes, while highlighting the complex, multifactorial nature of myocardial adaptation in chronic drinkers. They also support the need for early intervention, particularly through blood pressure control, to prevent the transition from subclinical to overt cardiac dysfunction.

Limitations

The study's relatively small sample size, (48 males) and all males, limits the generalizability of the findings to the broader population, including females. Self-Reported Data: Alcohol consumption data was self-reported, introducing potential recall bias. Also, the cross-sectional nature of the study precludes establishing causal relationships between alcohol consumption and echocardiographic abnormalities.

Confounding Factors: While some confounders were controlled for, other potential factors such as diet, physical activity, and genetic predisposition were not accounted for.

Significance of the Findings

The study highlights the significant impact of alcohol consumption on cardiovascular health within the Rumuekini community. The identification of BMI and SBP as significant predictors of echocardiographic parameters underscores the importance of monitoring these factors in individuals who consume alcohol. The findings contribute to the growing body of evidence on the adverse effects of alcohol on heart health and emphasize the need for public health interventions to reduce alcohol consumption and promote cardiovascular health.

Conclusion

In conclusion, this study provides valuable insights into the relationship between alcohol consumption and echocardiographic parameters in a Nigerian community. The findings are consistent with local, African, and international studies, highlighting the global relevance of the issue. Future research should focus on larger, more diverse populations and longitudinal designs to further elucidate the causal relationships and underlying mechanisms.

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