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Research article

Alcohol consumption and HIV disease prognosis among virally unsuppressed in Rural KwaZulu Natal, South Africa

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Abstract: Background: The effect of alcohol consumption and human immunodeficiency virus (HIV) disease prognosis has been examined in several studies with inconsistent findings. We sought to determine the effect of alcohol consumption on HIV disease prognosis by examining $CD4^+$ T cell count/μL ($CD4^+$ count) and HIV RNA concentration [HIV viral load (VL)] independent of anti-retroviral therapy (ART). **Methods:** A secondary analysis was performed on a cross-sectional survey data of 1120 participants between 2018 and 2020. Questionnaires were used to obtain the participants' history of alcohol consumption. Blood samples were assayed for $CD4^+$ T cell count/μL ($CD4^+$ count) and HIV RNA concentration (HIV viral load). The history of alcohol consumption was categorized into non-alcohol consumers, non-heavy alcohol consumers, and heavy-alcohol consumers. Age, cigarette smoking, gender, and ART use were considered potential confounders. Participants were categorized into two cohorts for the analysis and a multivariate logistic regression was used to establish relationships among virally unsuppressed participants who were ART-experienced and ART-naïve. **Results:** A total of 1120 participants were considered for analysis. The majority were females (65.9%) between 15–39 years (72.4%). The majority were non-smokers

and non-alcohol consumers (88% and 79%, respectively). ART-experienced females had an increased risk of having a higher VL (VL > 1000). This finding was statistically significant [RR, 0.425, 95% CI, (0.192–0.944), p-value, 0.036]. However, ART-experienced participants aged above 64 years had an increased risk of having a lower VL (VL < 1000 copies/mL) and a lower risk of having a higher VL (VL > 1000). However, ART-naïve participants aged between 40–64 years had a significantly lower risk of having higher CD4 count ($CD4^+$ > 500 cells) and an increased risk of having a lower CD4 count [OR, 0.566 95% CI, (0.386–0.829), p-value, 0.004]. History of alcohol consumption did not have a significant effect on $CD4^+$ cell count and VL in neither the ART-experienced nor the naïve cohort. **Conclusions:** Female middle-aged people living with HIV (PLWH) are more likely to have a poorer HIV disease state, independent of alcohol consumption. Alcohol consumption may not have a direct effect on $CD4^+$ cell count and VL in either ART-naïve or experienced patients.

Keywords: HIV disease; alcohol consumption; CD4 count; anti-retroviral therapy

1. Introduction

Human immunodeficiency virus (HIV) disease continues to have disturbing health effects globally, with about 510000–860000 HIV/AIDS-related deaths and more than 38.4 million people who are currently living with HIV disease (PLWH) [1]. Globally, sub-Saharan Africa (SSA) drives incident cases and deaths in HIV [1–3]. Anti-retroviral therapy (ART), which is used in the management of PLWH, has a good prognosis by suppressing viral load when individuals living with HIV adhere to their treatment regimen. However, the success of ART can be marred by unhealthy lifestyles such as heavy alcohol consumption and cigarette smoking, which appear to be common phenomena among PLWH [4,5].

Alcohol consumption has merited studies because it can jeopardize the treatment outcome of PLWH. Alcohol consumption itself is considered a risk factor in HIV infection transmission [6] and it increases the burden of the disease state [6]. Moreover, it reduces adherence to treatment regimens, thereby increasing the morbidity and mortality of HIV/AIDS [7]. Though the net effect of the above-stated effects of alcohol consumption is linked to the progression of the HIV disease [8], reports on its association with $CD4^+$ cell count and viral load (VL) still remains controversial. For instance, one prospective longitudinal study showed an increased suppression of $CD4^+$ T cell counts in PLWH with frequent alcohol use [9], while another reported that heavy alcohol consumption negatively impacted $CD4^+$ cell counts solely in ART-naïve subjects [10].

Other investigations failed to establish an association between heavy alcohol consumption and $CD4^+$ T cell decline [10–13]. Heavy alcohol consumption has been identified as a significant contributor to poor ART adherence [14]. In pre-clinical studies utilizing well-controlled behavioral and environmental conditions, an animal model of simian immunodeficiency virus (SIV) infected macaques have provided significant insight on the interaction of HIV and heavy drinking [15]. Additional studies [15,16] have shown a significant temporal acceleration to end-stage disease in the absence of ART, with consistently higher plasma, cerebrospinal fluid and tissue VLs among chronic high alcohol administered animals compared to controls.

It has been well documented that the environment (e.g. economic, psychosocial, physical, food insecurity or environmental difficulties) has the ability to accelerate to end-stage disease much faster [17]. Therefore, we analyzed cross-sectional data obtained from a 2018–2021 Vukuzazi study to assess the effect of alcohol consumption on $CD4^+$ T cell count/μL (CD4 count) and HIV RNA concentration (HIV VL). We hypothesized that alcohol consumption would not be associated with a lower $CD4^+$ count and a higher VL.

2. Material and methods

2.1. Study setting, study design, and recruitment

The uMkhanyakude district is one of the 11 districts in the Province of KwaZulu Natal, which is considered deprived according to the District Health Barometer [18]. This study was a secondary analysis of an 18-month (between 2018 and 2020) observational study performed by the Vukuzazi team. About 39000 individuals were eligible a year before data collection. During the period of data collection, about 3000 of these individuals had either died or moved out of the study area. About 18024 participants completed the study questionnaire, and 17871 had their anthropometry checked and recorded. Out of theses participants, 1120 individuals were virally unsuppressed and were considered for this analysis. Study participants were individuals aged 15 years and older who were residents of the uMkhanyakude district of KwaZulu-Natal [19]. However, this secondary analysis included participants aged 18 years and above.

2.2. Ethical consideration

The original work performed by the Vukuzazi team received approval from the Ethics Committees of the University of KwaZulu-Natal, the London School of Hygiene and Tropical Medicine, the Partners Institutional Review Board, and the University of Alabama at Birmingham.

The current study received ethical approval from both the Africa Health Research Institute Institutional Review Board and the University of Limpopo, with a project number TREC/112/2021: IR. Additionally, permission was obtained from the Vukuzazi team to access the database for the secondary analysis.

2.3. Invitation process at the participant's homestead and informed consent

The current study was solely based on the analysis of secondary data from the Vukuzazi program; therefore, informed consent was waived on behalf of the informed consent obtained during data acquisition from the Vukuzazi team. Before choosing the final sites, permission was requested from the local traditional authority and leaders. Each participant brought a special invitation card to the health camp [19]. Those who agreed to participate were given a barcoded wristband that served as a special identification during their encounter with the Vukuzazi camp, verifying their identification at each station. At the Vukuzazi health camp, a formal informed consent and enrollment process was followed by a household visit, during which, all eligible participants were invited to participate [19].

2.4. Field and laboratory procedures

At the Vukuzazi camp, research nurses administered questionnaires to assess the individual's history of HIV, hypertension, and diabetes [19]. Anthropometric and blood pressure measurements were performed using the WHO STEPS protocol. A blood sample was taken from each participant.

2.5. Study variables, measurements, and their definitions

In this study, $CD4^+$ cell count/μl and plasma HIV RNA/ml (VL) were the outcome variables. In the hospital laboratories, flow cytometry was used to calculate the $CD4^+$ count. VL was assessed using either a polymerase chain reaction or a branched-chain assay. A poor HIV disease state was defined as a $CD4^+$ count less than 500 count/μl [20].

Alcohol consumption history was categorized into non-alcohol consumers, non-heavy alcohol consumers, and heavy-alcohol consumers. Non-alcohol consumers were defined as respondents who did not take alcohol. Non-heavy alcohol consumers were defined as respondents who consumed alcohol occasionally in the past 30 days and who, on average, consumed less than 5 drinks per occasion for men or less than 4 drinks per occasion for women. On the other hand, heavy alcohol consumers were operationally defined as someone who consumed alcohol frequently in the past 30 days and who, on average, consumed at least 5 drinks per occasion for men or at least 4 drinks per occasion for women.

Participants were considered virally unsuppressed if their viral load was more than 200 copies/mL [20]. Participants were categorized as either ART-experienced or ART-naïve. ART-experienced individuals were PLWH who were receiving ART or had ART in the past [21]; alternatively, ART-naïve individuals were PLWH who have not started ART [13]. The body mass index (BMI) was also calculated as the weight of patients in kilograms divided by the square of the height in metres^2 and was defined as normal (18.5–24.9 kg/m^2), underweight (< 18.5 kg/m^2), overweight (25.0–29.9 kg/m^2) and obese (≥ 30 kg/m^2). Waist-to-hip ratio (WHR) was calculated and defined as normal and abdominal obese. WHR > 9.0 in males and >8.5 in women were classified as abdominal obesity.

2.6. Statistical analysis

Data was imported using STATA/SE, version 14.2, and was cleaned for statistical analysis. Descriptive analyses such as frequencies, percentages, and figures were used to describe the study population. The primary exposure of interest in this analysis was alcohol consumption. Covariates included history of smoking, obesity, gender, age, and ART use; these were treated as potential confounders. The null hypothesis was that alcohol consumption is not related to a poor HIV disease state, while the alternate hypothesis was that alcohol consumption was associated with a poor HIV disease state. ART duration was considered as one of the covariates and was factored in the model used.

Chi-square was used to analyze relationships between background characteristics and was stratified by their ART status. $CD4^+$ less than 500 and VL more than 200 copies/mL were used as the base outcome of the dependent variables. A multivariate logistic regression was used to analyze

relationships between $CD4^+$ cell count, VL, and predictor variables controlling the confounding variables. p-values < 0.05 were considered statistically significant.

3. Results

3.1. Background characteristics of study participants

A total of 1120 participants were considered for the analysis. Table 1 shows the background characteristics of the participants. The majority of the PLWH were females (65.89%) between 15–39 years (72.4%) with a normal BMI (48.7%) and hip-to-waist ratio (56.25), non-smokers and non-alcohol consumers (88% and 79%, respectively).

Table 1. Distribution of background characteristics.

Background variable	Frequency	Percentage
Sex		
Male	382	34.11
Female	738	65.89
Total	1120	100
Age (years)		
15–39	811	72.41
40–64	284	25.36
Above 64	25	2.23
Total	1120	100.00
BMI (Kg/m^2)		
Underweight	42	3.75
Normal	545	48.66
Overweight	250	22.32
Obese	283	25.27
Total	1120	100.00
Waist-Hip-Ratio		
Normal	630	56.25
Abdominal obesity	490	43.75
Total	1120	100.00
Cigarette smoking history		
Abstainers	983	87.77
Current cigarette smokers	131	11.70
Ex-smoker	6	0.54
Total	1120	100.00

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Background variable	Frequency	Percentage
Alcohol consumption		
Non-alcohol consumers	887	79.20
Non-heavy alcohol consumers	217	19.38
Heavy-alcohol consumers	16	1.43
Total	1120	100.00
ART duration		
Less than 5 years	98	35.38
5–10 years	95	34.30
Above 10 years	84	30.32

Table 2 shows the distribution of the median $CD4^+$ count and VL. VL greater than 1000 copies/mL and $CD4^+$ less than 500 count/µl were seen among participants who do not consume alcohol. Although the highest number of participants with a low $CD4^+$ count was seen among participants who do not consume alcohol, the highest median was observed among participants who consume alcohol heavily. Although the highest number of participants with viral load greater than 1000 copies/mL was observed among those who do not consume alcohol, the highest of the median VL was seen among those who consume alcohol.

Table 2. Distribution of $CD4^+$ count and Viral with the median values.

Covariate	Non-alcohol consumers (N)	Median (IQR)	Moderate consumers	Median (IQR)	Heavy consumers	Median (IQR)
$CD4^+$						
$CD4^+ > 500$	348	706(240)	64	658.5(274)	7	675(258)
$CD4^+ < 500$	539	277(191)	153	265(249)	9	331(165)
VL						
VL > 1000	741	16472(50669)	195	28052(71833)	13	3110(29816)
VL < 1000	146	442(377)	22	729(290)	3	598(496)

Note: VL: Viral load; IQR: Interquartile range.

Table 3 shows the distribution of $CD4^+$ count and VL of the PLWH. Out of the 1120 PLWH, 387 were on ART. Almost 64 % of ART-experienced participants were females (63.6%) with $CD4^+ < 500$ count/µl compared to their male counterparts; additionally, they had a higher VL of more than 1000000 copies/mL (66.7%) compared to their male counterparts.

Table 3. Distribution of CD4+ count and viral load of the ART-experienced participants.

Background variable	ART experienced (n = 387)			
	$CD4^+ \geq 500$	$CD4^+ < 500$	VL > 1000(%)	VL < 1000(%)
Sex				
Male	29(23.02)	95(36.40)	93(31.53)	31(33.70
Female	97(76.98)	166(63.60)	202(68.47)	61(66.30)
Total	126(100)	261(100)	295(100.00)	92(100.00)
Age (years)				
15–39	98(77.78)	159(60.92)	207(70.17)	50(54.35)
40–64	26(20.63)	98(37.55)	84(28.47)	40(43.48)
Above 64	2(1.59)	4(1.53)	4(1.36)	2(2.17)
Total	126(100)	261(100)	295	92(100)
BMI (Kg/m^2)				
Underweight	9(7.14)	16(6.13)	22(7.46)	3(3.26)
Normal	43(34.13)	143(54.79)	146(49.49)	40(43.48)
Overweight	37(29.37)	54(20.69)	67(22.71)	24(26.09)
Obese	37(29.37)	48(18.39)	60(20.34)	25(27.17)
Total	126(100)	261(100)	295(100)	92(100)
Waist-Hip-Ratio				
Normal	75(59.52)	165(63.22)	169(57.29)	46(50)
Abdominal obesity	51(40.48)	96(36.78)	126(42.71)	46(50)
Total	126(100)	261(100)	295(100)	92(100)
Smoking History				
Abstainers	114(90.48)	228(87.36)	258(87.46)	84(91.30)
Current	12(9.52)	31(11.88)	35(11.86)	8(8.70)
Ex-smoker		2(0.77)	2(0.68)	0
Total	126(100)	261(100)	295(100)	92(100)
Alcohol consumption				
Non-alcohol consumers	109(86.51)	199(76.25)	233(78.98)	75(81.52)
Non-heavy alcohol consumers	17(13.49)	57(21.84)	59(20.00)	15(16.30)
Heavy-alcohol consumers		5(1.92)	3(1.02)	2(2.17)
Total	126(100)	261(100)	295(100)	
ART duration				
Less than 5 years	40(42.55)	58(31.69)	82(38.86	16(24.24)
5–10 years	31(32.98)	64(34.97)	70(33.18)	25(37.88)
Above 10 year	23(24.47)	61(33.33)	59(27.96)	25(37.88)
Total	94(100)	183(100)	211(100)	66(100)

Note: Unless otherwise stated, the table above shows the distribution of participants and their $CD4^+$, VL categorization of those who were on ART. ART: Antiretroviral therapy; VL: Viral load.

3.2. Predictors of poorer HIV disease state

ART-experienced females had a lower risk of having a lower VL (VL < 1000 copies/mL) and an increased risk of having a higher VL (VL > 1000). This finding was statistically significant [RR, 0.425, 95% CI, (0.192–0.944), p-value, 0.036]. However, ART-experienced participants aged above 64 years had an increased tendency of having a lower VL (VL < 1000 copies/mL) and a lower risk of having a higher VL (VL > 1000). This finding was also statistically significant [RR, 11.020, 95% CI, (1.191–101.982), p-value, 0.035]. Alcohol consumption did not have a significant effect on an increased VL greater than 1000 copies/mL [non-alcohol consumers, RR, 0.796 95% CI, (0.298–2.123), p-value, 0.648]. Table 4 shows the VL of the virally unsuppressed ART-experienced participants.

Table 4. Multivariate logistic regression of predictors of reduced viral load among ART-experienced participants.

Viral load	Model 1-unadjusted			Model 2-adjusted			
	RR	P > t	95% Conf. Interval	RR	P > t	95% Conf. Interval	
VL > 1000	(base outcome)						
Alcohol consumption History							
Non-alcohol consumers	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Moderate consumers	0.796	0.625	0.319	1.988	0.796	0.648	0.298	2.123
Heavy consumers	2.357	0.524	0.169	32.888	2.357	0.439	0.268	20.707
_cons	0.105	0.006	0.0213	0.5166	0.015	0.000	0.010	0.021

Note: Unadjusted model Number of obs = 277, LR $chi^2(13)$ = 23.70, Prob > Chi^2 = 0.0340, Pseudo R^2 = 0.0779. Adjusted model: Number of obs = 277, Wald $chi^2(13)$ = 376.99, Prob > Chi^2 = 0.0000, Pseudo R^2 = 0.077. Analysis was conducted by multinomial logistics regression model with two models. Model 1 is unadjusted. Model two adjusted for gender, age, BMI, WHR, Smoking history, and ART medications.

* p-value < 0.05 was considered statistically significant. Multivariate logistic regression was used to obtain values. RR: Relative risk; CI: Confidence interval. ART: Antiretroviral therapy; _cons: Constant.

Table 5 shows the multivariate logistic regression of predictors of reduced VL among the ART-naïve participants. The relative risk of female ART-naïve participants having a lower VL (VL < 1000) was 2.256 times greater than the male ART-naïve participants. Female ART-naïve participants had a greater risk of having a lower VL than their male counterparts [RR, 2.256, 95% CI, (1.165–4.366), p-value, 0.016]. Alcohol consumption did not have a significant effect on an increased VL greater than 1000 copies/mL [moderate alcohol consumers, RR, 0.796 95% CI, (0.298–2.123), p-value, 0.648].

Table 5. Multivariate logistic regression of predictors of reduced viral load among ART-naïve respondents.

Viral load (VL)	Model 1-unadjusted				Model 2-adjusted			
	RR	P > t	5% Conf. interval		RR	P > t	95% Conf. Interval	
VL > 1000	(base outcome)							
Alcohol consumption History								
Non-alcohol consumers	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Moderate consumers	0.528	0.162	0.215	1.292	0.528	0.142	0.225	1.239
Heavy consumers	0.844	0.875	0.103	6.927	0.844	0.876	0.100	7.105
_cons	0.194	0.020	0.049	0.769	0.194	0.023	0.047	0.802

Note: Unadjusted model: Number of obs = 733, LR $chi^2(11)$ = 22.40, Prob > chi^2 = 0.0215, Pseudo R^2 = 0.0447. Adjusted: Number of obs = 733, Wald $chi^2(11)$ = 20.04, Prob > chi^2 = 0.0448, Pseudo R^2 = 0.0447. Analysis was conducted by multinomial logistics regression model with two models. Model 1 is unadjusted. Model two adjusted for gender, age, BMI, WHR, Smoking history, and ART medications. RR: Relative risk; CI: Confidence interval. ART: Antiretroviral therapy; _cons: Constant.

Table 6 shows the $CD4^+$ count of virally unsuppressed ART-experienced participants. ART-experienced participants aged between 40–64 had an increased risk of having a higher CD4 count ($CD4^+$ > 500 cells/mm^3) and a lower risk of having a lower CD4 count ($CD4^+$ < 500 cells/mm^3). This finding was statistically significant [RR, 0.360 95% CI, (0.182–0.714), p-value, 0.003]. Alcohol consumption did not have a significant effect on reduced $CD4^+$ cell counts less than 500 cells/mm^3 [moderate alcohol consumers, RR, 0.462 95% CI, (0.194–1.099), p-value, 0.081].

Table 6. Multivariate logistic regression of predictors of reduced $CD4^+$ ART-experienced respondents.

CD4 count	Model 1-unadjusted				Model 2-adjusted			
	RR	P > t	5% Conf. interval		RR	P > t	95% Conf. Interval	
$CD4^+$ < 500	(base outcome)							
Alcohol consumption History								
Non-alcohol consumers	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Moderate consumers	2.148	0.080	0.9130	5.052	2.148	0.080	0.912	5.059
Heavy consumers	0.000	0.983	0.000	0.000	0.000	0.000	0.000	0.000
_cons	1.193	0.751	0.400	3.553	1.193	0.738	0.424	3.355

Note: Unadjusted model: Number of obs = 277, LR $chi^2(11)$ = 34.52, Prob > chi^2 = 0.0010, Pseudo R^2 = 0.0973. Adjusted: Number of obs = 277, Wald $chi^2(13)$ = 570.36, Prob > Chi^2 = 0.0000, Pseudo R^2 = 0.0973. Analysis was conducted by multinomial logistics regression model with two models. Model 1 is unadjusted. Model two adjusted for gender, age, BMI, WHR, Smoking history, and ART medications.

* p-value < 0.05 was considered statistically significant. Multivariate logistic regression was used to obtain values. RR: Relative risk; CI: Confidence interval. ART: Antiretroviral therapy; _cons: Constant.

Table 7 shows the $CD4^+$ count of the virally unsuppressed ART-naïve participants. ART-naïve participants aged between 40–64 years had a significantly lower risk of having a higher CD4 count ($CD4^+ > 500$ cells) and an increased risk of having a lower CD4 count [OR, 0.566 95% CI, (0.386–0.829), p-value, 0.004]. Alcohol consumption did not have a significant effect on $CD4^+$ count [moderate consumers, RR, 0.890 95% CI, (0.568–1.394), p-value, 0.611, heavy consumers, RR, 2.640 95% CI, (0.726–9.605), p-value, 0.141]. Among the participants who were ART naïve, females had a significantly increased $CD4^+$ count compared to their male counterparts [RR, 2.021 95% CI, (1.298–3.147), p-value, 0.002].

Table 7. Multivariate logistic regression of predictors of reduced $CD4^+$ among ART-naïve respondents.

CD^+ count	Model 1-unadjusted			Model 2-adjusted			
	RR	P > t	5% Conf. interval	RR	P > t	95% Conf. Interval	
$CD4^+ < 500$	(base outcome)						
Alcohol consumption history							
Non-alcohol consumers	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Moderate consumers	1.132	0.592	0.719	2.455	1.132	0.587	0.7235	1.772
Heavy consumers	0.387	0.151	0.106	1.414	0.387	0.154	0.1052	1.427
_cons	3.127	0.057	0.966	10.126	3.127	0.057	0.9675	10.111

Note: Unadjusted model: Number of obs = 733, LR $chi^2(11) = 39.58$, Prob > $chi^2 = 0.0000$, Pseudo $R^2 = 0.0401$. Adjusted: Number of obs = 733, Wald $chi^2(11) = 38.65$, Prob > $chi^2 = 0.0001$, Pseudo $R^2 = 0.0401$. Analysis was conducted by multinomial logistics regression model with two models. Model 1 is unadjusted. Model two adjusted for gender, age, BMI, WHR, Smoking history, and ART medications.

* p-value < 0.05 was considered statistically significant. Multivariate logistic regression was used to obtain values. RR: Relative risk; CI: Confidence interval. ART: Antiretroviral therapy; _cons: Constant.

4. Discussion

The main objective of this Vukuzazi dataset secondary analysis was to determine the effect of alcohol consumption on the prognosis of HIV disease among the virally unsuppressed PLWH who were both ART-experienced and ART-naive. The results add to the existing knowledge that ART-experienced PLWH who consume alcohol either moderately or heavily do not have reduced $CD4^+$ cell counts as compared to their counterparts who were non-alcohol consumers. This observation was also the same among the ART-naïve cohort.

History of alcohol consumption among ART-experienced PLWH was found not to be significantly associated with a poor HIV disease state compared to their counterparts who were non-alcohol consumers. This finding is contrary to the findings of previous studies [16,20,22–24]. Baum et al. [9] found that alcohol consumption was associated with higher HIV RNA levels and lower $CD4^+$ cell counts among PLWH who were receiving ART. However, our findings are similar to the findings of other studies, which found no significant association between alcohol consumption and a poor HIV disease state among ART-experienced PLWH [24,25].

Alcohol consumption has been shown to cause immunosuppression through impaired macrophage function [26], increased natural killer cell activity, increased spontaneous monocyte activation [26], and impaired antibody response [27]. A poor HIV disease state could also be explained by the deleterious effects of heavy alcohol consumption [28], which compromises the body's immunity. These outcomes were not shown by the current study.

Rather, our analysis showed a strong association between age (40–60 years) and a poor HIV disease state. Several studies have posited the relationship between age and HIV disease progression. Age has become a prognostic host factor because older age is associated with lower $CD4^+$ cell counts [29]. Moreover, studies have shown that increased age at the time of an AIDS diagnosis parallels the progressive rise of mean age at the time of the first recognition of HIV infection [30,31]. When individuals are unaware of their status, late detection of HIV disease is another contributing cause to the increasing frequency of newly confirmed HIV infection, with a worse disease state among the aged [30,31].

Additionally, our findings have shown a significant association between the female sex and a poor HIV disease state. The analysis showed that ART-experienced female participants had a higher risk of having an increased VL. Several studies have posited the female sex as a high risk for HIV infection [32–35] and disease progression. The unique characteristic of the female genital tract enhances the risk of HIV infection. One of such characteristics is the local changes in their genital tract induced by infection by other microorganisms [32]. Other factors predisposing females to HIV infection in SSA is the low-income status.

5. Conclusions

Female middle-aged PLWH are more likely to have a poor HIV disease state, independent of alcohol consumption. Alcohol consumption may not have a direct effect on $CD4^+$ cell count and VL in both ART-naïve and experienced patients.

6. Limitations

Our study could not be short of limitations. We were unable to determine the actual units of alcohol consumed by the PLWH. Another limitation to this analysis is the fact that alcohol consumption among the PLWH was obtained by self-reporting. Self-reporting of alcohol use is wroth with challenges of underreporting. Again, the current analysis did not have data on ART adherence which may better explain some observations found in the current secondary analysis.

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Authors' contributions

All authors made a significant contribution to this study, whether that is in conception, data analysis and interpretation. All authors also took part in the drafting, revising, and gave approval for the publication of this manuscript.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

Authors involved in this study have no conflict of interest to declare.

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