

Cardiovascular risk factors in a treatment-naïve, human immunodeficiency virus-infected rural population in Dikgale, South Africa

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Objective: The objective was to determine lipid levels and cardiovascular risk factors in treatment-naïve, human immunodeficiency virus (HIV)-infected rural African people in Limpopo province.

Design: This was a case control study.

Setting and subjects: The setting was Dikgale Health and Demographic Surveillance System Centre, Limpopo province. Treatment naïve, HIV-infected and HIV-negative people participated in the study.

Outcome measures: Demographic, lifestyle and chronic disease data were collected using the World Health Organization stepwise approach to surveillance (STEPS) questionnaire. Biochemical parameters were tested using standard biochemical methods. HIV testing and CD4 counts were performed using the Alere Determine™ HIV 1/2 Ag/Ab kit and The Alere Pima™ Analyser. Insulin resistance, low-density lipoprotein cholesterol (LDL cholesterol), and non-high-density lipoprotein cholesterol (non-HDL cholesterol) levels were calculated.

Results: The mean age of participants (years) was 49.7 ± 16.6 . More HIV-infected than HIV-uninfected women consumed alcohol (25.4% vs. 11.9%, p -value < 0.05), and the prevalence of abdominal obesity was higher in HIV-uninfected than in HIV-infected women (74.6% vs. 54.8%, p -value < 0.05). Levels of total cholesterol (TC), HDL cholesterol, non-HDL cholesterol, LDL cholesterol and apolipoprotein A1 (ApoA1) were significantly lower in the HIV-infected than in the HIV-uninfected group. The prevalence of low HDL cholesterol was higher in HIV-infected than in HIV-uninfected people (62.4% vs. 41.6%, p -value < 0.01). HIV infection increased the likelihood of low HDL cholesterol by 2.7 times (p -value 0.001). Male gender and alcohol use decreased the likelihood of low HDL cholesterol by 61% (p -value 0.002) and 48% (p -value 0.048), respectively. HIV infection was associated with low HDL cholesterol, ApoA1, LDL cholesterol and TC. Low CD4 count was associated with low body mass index, LDL cholesterol and high diastolic blood pressure.

Conclusion: The prevalence of cardiovascular risk factors was equally high in HIV-infected and in HIV-uninfected rural people, except for low HDL and alcohol consumption, which were significantly higher in HIV-infected people, while abdominal obesity was significantly higher in HIV-uninfected people. There is a need to raise awareness of cardiovascular risk factors in rural people in Limpopo province.

Keywords: abdominal obesity, alcohol, diabetes, hypertension, lipids

Introduction

Human immunodeficiency virus (HIV) is one of the greatest worldwide public health challenges. An estimated 22.5 million people lived with HIV in sub-Saharan Africa in 2007, which comprised approximately 68% of global infection.¹ Cardiovascular disease (CVD) is becoming a significant cause of morbidity and mortality in HIV-infected patients.²

HIV is a risk factor for CVD.³ It induces chronic inflammation that leads to several CVD-associated risk factors, such as type 2 diabetes, insulin resistance (IR), hypertension and dyslipidaemia,^{4–6} mediated by decreased adiponectin levels.⁶ Dyslipidaemia in HIV infection is characterised by decreased levels of total cholesterol (TC), low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol.^{7–10} Higher triglycerides (TG) levels were reported in advanced disease (acquired immune deficiency syndrome).^{11,12} Additionally, high levels of tobacco use, alcohol consumption and lower body mass index (BMI) are associated with CVD in HIV-infected people.^{13–15}

Studies on the lipid profiles of treatment-naïve, HIV-infected people in South Africa show contradictory results on TC and TG levels, while HDL cholesterol and LDL cholesterol levels

were reported by only one study.^{9,16} Little is known of CVD risk factors in HIV-infected people in the rural province of Limpopo. The purpose of our study was to determine whether or not CVD risk factors were more elevated in HIV-infected than in HIV-uninfected rural people. We also determined associations between CVD risk factors and HIV and CD4 count. We searched for major predictors of low HDL cholesterol, an independent risk factor for CVD.

Method

Study design and setting

The study was a cross-sectional, nested, case control substudy of the main project, *The Prevention, Control and Management of Chronic Disease project in a rural population, South Africa*. The study was conducted in Dikgale Health and Demographic Surveillance System (HDSS) Centre, situated 50 km north east of Polokwane, the capital city of Limpopo province. There are approximately 35 000 inhabitants in the Dikgale HDSS Centre, most of whom speak Northern Sotho.

Study participants

Eight hundred and fifteen randomly selected people participated in the main project. Participants who received pre-counselling were tested for HIV. Participants who tested

HIV-positive and who were not on antiretroviral treatment according to the questionnaire formed the cases (89). The controls (178), matched for gender and age (± 2 years), were randomly selected from those who tested negative for HIV.

Pregnant women were excluded from the study. Participants received information on the study prior to participation through door-to-door visits by trained fieldworkers and signed consent forms. Despite providing consent, some participants indicated that they did not want to know their results. Consenting participants were advised of participation dates, the local venue and times a week in advance. Participants received pre-counselling from trained counsellors on the scheduled date. The HIV results were revealed in post-counselling to participants who wanted to know their status. HIV-positive participants, and those with other abnormal biochemical abnormalities parameters, were referred to the local clinic or hospital for further analysis and management. Trained fieldworkers revisited participants and administered the questionnaire.

Ethical considerations

Permission for the study was sought from tribal chiefs. Ethical approval was obtained from the Ethics Committee of the University of Limpopo, Medunsa Campus. Written informed consent was obtained from participants and guardians of minors (< 18 years) prior to the study.

Data collection

The World Health Organization (WHO) stepwise approach to surveillance (STEPS) questionnaire¹⁷ was used to obtain information on the medical condition of the subjects. Tuberculosis and HIV information was obtained using an additional questionnaire. The data collection was conducted from August 2011 to April 2012.

Anthropometric measurements

Weight was measured using Omron® BF 400 (Omron Healthcare, Kyoto, Japan). Subjects were asked to take off their shoes and heavy coats. Weight was measured to the nearest 0.1 kg. Height was measured with a stadiometer. Barefoot subjects were asked to stand in an upright position. Height was measured to the nearest 0.1 cm. BMI was calculated by dividing weight (kg) by height (m^2). A BMI of 18.50–24.99 kg/m^2 was considered to be normal, 25–29.99 kg/m^2 overweight and ≥ 30 kg/m^2 obese.¹⁸

Waist circumference (WC) and hip circumference (HC) were measured using a measuring tape. WC was measured around the area between the last rib and the hip bone, while HC was measured around the widest part in the gluteal area. Both parameters were measured to the nearest 0.1 cm, and were used to calculate the waist to hip ratio.

Blood pressure measurements

Blood pressure (BP) was measured using the Omron® M5-1 (Omron Healthcare, Kyoto, Japan). The subjects were asked to sit and relax for five minutes before the first measurement was taken, and were restricted from talking during the measurement procedure. Three measurements were taken, with a few minutes' break inbetween. The mean of the last two values was calculated for systolic blood pressure (SBP) and diastolic blood pressure (DBP). High BP was defined as SBP above 140 mmHg and/or DBP above 90 mmHg,¹⁹ and/or a self-reported history of antihypertensive drug use.

Blood collection

Fasting venous blood samples were drawn by registered nurses. Whole blood was used to measure CD4 count on the day of collection. Serum from clotted blood and plasma from whole blood were separated through centrifugation at 2 000 rpm for 15 minutes. Glucose and HIV status were analysed soon after centrifugation using plasma. The remaining samples were stored at $-80^\circ C$ until analysis.

Biochemical analysis

Triglycerides (TGs), TC, HDL cholesterol, glucose and creatinine levels were determined on ILab® 300 Plus Chemistry System (Instrumentation Laboratory Company, Milan, Italy). Insulin levels were measured on Beckman® Access Immunoassay System (Beckman Coulter, USA). Apolipoprotein B (ApoB), Apolipoprotein A1 (ApoA1) and high-sensitivity C-reactive protein (hsCRP) levels were measured on the IMMAGE® Immunochemistry System (Beckman Coulter, USA). Determine™ HIV-1/2 Ag/Ab Combo was initially used for HIV testing. Positive samples and 10% of negative samples were re-run using DoubleCheckGold™ Ultra HIV 1/2 kit. Both kits are Elisa®-based and supplied by Inverness Medical, Tokyo, Japan. CD4 count was measured using the Pima® Analyser (Inverness Medical, Tokyo, Japan). LDL cholesterol and IR were calculated using Friedewald²⁰ and homeostatic model assessment-insulin resistance²¹ formulas, respectively. Non-HDL cholesterol was determined by subtracting the concentration of cholesterol in the HDL from that in the total plasma.

Statistical analysis

Statistical analysis was performed with Statistical Package for Social Science® version 20. Variables were tested for normality using frequency histograms and line graphs. Data that were not normally distributed were logarithmically transformed for analysis. To make a comparison of group differences in respect of socio-demographic characteristics and lipid profiles, the independent Student's *t*-test was used for continuous variables and the chi-square test for categorical variables. Normally and not normally distributed data were presented as mean $\pm$ standard deviation and median interquartile range, respectively. Bivariate correlation was used to establish associations between HIV, CD4 count and CVD-associated risk factors. Bivariate logistic regression was used to ascertain the individual influence of CVD-associated risk factors on HDL cholesterol levels. Multivariate stepwise forward and backward regression modelling was used to determine significant predictors of low HDL cholesterol levels.

Results

The mean age (years) of the participants was 49.7 ± 16.6 . HIV-infected participants and HIV-uninfected participants had similar measurements in weight, height, BMI, WC, HC, hsCRP, insulin, DBP and SBP. Equally high prevalence rates of tobacco use (18% vs. 15.2%), IR (23.9% vs. 21.9%), hypertension (42.7% vs. 45.5%) and diabetes mellitus (13.5% vs. 13.5%) were observed between the HIV-positive and HIV-negative participants, respectively. The prevalence rate of low HDL cholesterol was significantly higher in HIV-infected than in HIV-uninfected individuals (62.4% vs. 41.6%, *p*-value < 0.01). A significantly higher percentage of HIV-positive women than HIV-negative women consumed alcohol (25.4% vs. 11.9%, *p*-value < 0.05), and abdominal obesity was present in a significantly higher percentage of HIV-negative women (74.6% vs. 54.8%, *p*-value < 0.05), while their BMI remained below the threshold of 30 (Table 1).

Table 1: Characteristics of human immunodeficiency virus-infected and human immunodeficiency virus-uninfected rural African people

Characteristics	All participants		Males		Females	
	HIV-negative n = 178	HIV-positive n = 89	HIV-negative n = 52	HIV-positive n = 26	HIV-negative n = 126	HIV-positive n = 63
Age	49.7 ± 16.6	49.7 ± 16.8	54.4 ± 18.3	55 ± 18.8	47.7 ± 15.5	47.6 ± 15.6
Weight (kg)	68.3 ± 15.7	69.2 ± 17.9	66.4 ± 15.3	65.9 ± 11	69.1 ± 15.8	70.6 ± 19.9
Height (m)	1.60 ± 0.09	1.62 ± 0.09	1.66 ± 0.09	1.68 ± 0.09	1.58 ± 0.08	1.60 ± 0.08
Body mass index	26.2 ± 6.5	25.9 ± 7.2	23.7 ± 5.4	23.1 ± 5.2	27.2 ± 6.7	27 ± 7.7
WC (cm)	88.1 ± 14.4	85.5 ± 16.5	83.5 ± 13.5	80.9 ± 10.7	90.0 ± 14.3	87.3 ± 18.1
HC (cm)	102.3 ± 14.4	100.8 ± 13.5	93.8 ± 11.4	97.3 ± 7.4	105.8 ± 14.1	102.1 ± 15.2
WC to HC	0.87 ± 0.12	0.84 ± 0.09*	0.90 ± 0.15	0.83 ± 0.08*	0.85 ± 0.11	0.84 ± 0.09
DBP (mmHg)	81.8 ± 12.3	81.1 ± 15.8	83.1 ± 11.7	80.0 ± 13.2	81.3 ± 12.5	81.6 ± 16.8
SBP (mmHg)	129.5 ± 21.6	128.2 ± 24.8	136.0 ± 22.1	129.9 ± 26.4	126.8 ± 20.9	127.5 ± 24.3
Alcohol, n (%)	35 (19.7)	26 (29.2)	20 (38.5)	10 (38.5)	15 (11.9)	16 (25.4)*
Tobacco use, n (%)	27 (15.2)	16 (18)	17 (32.7)	8 (30.8)	10 (7.9)	8 (12.7)
IR,* n (%)	39 (21.9)	21 (23.9)	13 (25)	8(30.8)	26 (20.6)	13 (21)
Hypertension,** n (%)	81 (45.5)	38 (42.7)	29 (55.8)	10 (38.5)	52 (41.3)	28 (44.4)
Diabetes,*** n (%)	24 (13.5)	12 (13.5)	6 (11.5)	5 (19.2)	18 (14.3)	7 (11.1)
Abdominal obesity,**** n (%)	105 (59)	36 (41.4)*	11 (21.2)	2 (8)	94 (74.6)	34 (54.8)*
Low HDL cholesterol,***** n (%)	74 (41.6)	53 (62.4)**	10 (19.2)	14 (53.8)**	64 (50.8)	39 (66.1)

DBP: diastolic blood pressure, HC: hip circumference, HDL: high-density lipoprotein, IR: insulin resistance, SBP: systolic blood pressure, WC: waist circumference

Values are mean ± standard deviation, median interquartile range

* p -value < 0.05, ** p -value < 0.01

* Insulin resistance ≥ 2.5

** Hypertension or systolic blood pressure > 140 mmHg, diastolic blood pressure > 90 mmHg, and a history of high blood pressure

*** Diabetes glucose > 7 mmol/l and a history of diabetes

**** Abdominal obesity [waist circumference > 94 cm (males) and waist circumference > 80 cm (females)]

***** Low high-density lipoprotein cholesterol [high-density lipoprotein cholesterol ≤ 1.3 mmol/l (females), and high-density lipoprotein cholesterol ≤ 1.1 mmol/l (males)]

Total cholesterol, HDL cholesterol, non-HDL cholesterol and LDL cholesterol levels were significantly lower (p -value < 0.05) in the HIV-infected than in the control group, while no statistical difference was observed between TG levels (Table 2).

The pattern persisted in males, while LDL cholesterol and non-HDL cholesterol levels were not significantly different between HIV-positive and HIV-negative females. ApoB levels were not significantly different in HIV-positive than in HIV-positive people. ApoA1 levels were significantly lower in the HIV-positive group than in the control group. Glucose levels were significantly lower in HIV-infected than in the control group

(p -value 0.04). Glucose levels remained significantly lower (p -value 0.01) in females, while no significant difference was observed in males.

HIV infection significantly correlated negatively with HDL cholesterol ($r = -0.23$, p -value < 0.00), ApoA1 ($r = -0.24$, p -value 0.00), LDL cholesterol ($r = -0.18$, p -value 0.00) and TC ($r = -0.23$, p -value 0.00), while CD4 count [only available for 52 participants (mean value 397 cells/μl)] significantly correlated positively with BMI ($r = 0.37$, p -value 0.00) and LDL cholesterol ($r = 0.30$, p -value 0.03), but negatively with DBP ($r = -0.43$, p -value 0.00) (Table 3).

Table 2: Biochemical characteristics of human immunodeficiency virus-infected and human immunodeficiency virus-uninfected rural African people

Characteristics	All participants		Males		Females	
	HIV-negative n = 178	HIV-positive n = 89	HIV-negative n = 52	HIV-positive n = 26	HIV-negative n = 126	HIV-positive n = 63
hsCRP (mg/l)	2.32 (0.88-6.09)	1.87 (0.68-5.64)	1.86 (0.87-3.17)	3.79 (0.93-10.03)	2.81 (0.88-6.99)	1.58 (0.61-5.23)
Glucose (mmol/l)	5.79 ± 2.78	5.22 ± 1.63*	5.29 ± 0.93	5.51 ± 2.13	6.00 ± 3.23	5.11 ± 1.38*
Insulin (uIU/ml)	6.28 (3.27-9.42)	4.91 (2.56-9.33)	5.80 (2.74-9.63)	4.85 (2.37-15.20)	6.37 (3.43-9.43)	4.91 (3.12-9.10)
TG (mmol/l)	1.18 (0.83-1.70)	1.12 (0.78-1.67)	1.29 (0.87-1.74)	1.03 (0.74-2.15)	1.14 (0.79-1.70)	1.15 (0.78-1.52)
TC (mmol/l)	4.74 ± 1.15	4.16 ± 1.27*	4.74 ± 1.00	3.92 ± 1.02*	4.75 ± 1.21	4.27 ± 1.35*
LDL cholesterol (mmol/l)	2.76 ± 0.95	2.37 ± 1.10*	2.69 ± 0.85	2.15 ± 1.03*	2.79 ± 0.99	2.47 ± 1.12
HDL cholesterol (mmol/l)	1.37 ± 0.35	1.17 ± 0.44*	1.39 ± 0.33	1.07 ± 0.36*	1.36 ± 0.36	1.21 ± 0.46*
Non-HDL cholesterol (mmol/l)	3.38 ± 1.05	2.99 ± 1.10*	3.36 ± 0.94	2.85 ± 0.98*	3.39 ± 1.10	3.05 ± 1.15
ApoB (mg/dl)	89.0 ± 27.9	83.9 ± 24.3	85.8 ± 26.3	78.8 ± 26.7	90.3 ± 28.5	86.3 ± 23.0
ApoA1 (mg/dl)	150.5 ± 32	133.4 ± 33.3*	153.4 ± 33.3	121.8 ± 25.8*	149.3 ± 31.4	138.5 ± 35.3*

ApoA1: apolipoprotein A1, ApoB: apolipoprotein B, HDL: high-density lipoprotein, hsCRP: high-sensitivity C-reactive protein, LDL: low-density lipoprotein, TC: total cholesterol, TG: triglycerides

Values are presented as mean ± standard deviation, median interquartile range

* p -value < 0.05

Cut-offs: Low-density lipoprotein cholesterol < 3 mmol/l, total cholesterol < 5 mmol/l, and triglycerides < 1.7mmol/l

Table 3: Bivariate correlations of cardiovascular disease-associated factors with human immunodeficiency virus and CD4 count

	HDL cholesterol	ApoA1	LDL cholesterol	TC	BMI	DBP
HIV	–0.239*	–0.240*	–0.179*	–0.225*	–0.02	–0.02
CD4 count	–0.04	–0.21	0.303**	0.25	0.370 *	–0.43*

ApoA1: Apolipoprotein A1, BMI: body mass index, DBP: diastolic blood pressure, HDL: high-density lipoprotein, HIV: human immunodeficiency virus, LDL: low-density lipoprotein, TC: total cholesterol

Values are correlation coefficients

* p -value < 0.01, ** p -value < 0.05

The current study focused on HDL cholesterol levels as the latter is regarded as a significant independent risk factor for CVD.^{2,22–24} Bivariate analysis showed that an HIV-positive person is 2.3 times more likely to have low HDL cholesterol than an HIV-negative person (Table 4). HIV infection increased the likelihood of low HDL cholesterol by 2.7 times in a multivariate analysis. Overall, HIV-positive status, gender and alcohol consumption were significant predictors of low HDL cholesterol in this study.

Discussion

The mean age (years) of the participants was 49.7 ± 16.6 . There was no significant difference in mean anthropometric values, BP measurements and insulin levels between HIV-positive and HIV-negative people. These similarities may indicate that our study population was in the early to mid stage of HIV infection, which was confirmed by the mean CD4 count of nearly 400 cells/ μ l. Similarities in BMI, WC and HC between treatment-naïve HIV-infected and HIV-uninfected people have also been observed elsewhere.²⁵

The prevalence rates of diabetes mellitus, hypertension and IR were not different between HIV-positive and HIV-negative males and females. However, overall, the prevalence rates were high, putting both HIV-positive and HIV-negative people at risk of CVD occurrence.

Similar prevalence rates of diabetes mellitus between treatment-naïve HIV-infected and HIV-uninfected women were reported in a menopause study,²⁵ and are consistent with those found in the current study. Higher prevalence rates of diabetes mellitus were reported in HIV-uninfected than in HIV-infected men (12% vs. 7%, p -value < 0.0001) by Womack et al.²⁶ Several hypothesis have been made about the relationship between HIV and diabetes, including the chronic inflammation state, leading to decreased adiponectin levels and IR, genetic susceptibility, obesity prevalence, sedentariness

and the use of opiates.^{4,25,27} A significant difference in the hypertension prevalence rate between HIV-infected and HIV-uninfected people was not found in this study. Similar results were reported by Baekken et al.²⁸ Other investigators found a significantly higher prevalence rate of hypertension in HIV-uninfected women relative to that in HIV-infected women (31.4% vs. 20.1%, p -value < 0.001), while no significant difference was observed in men.²⁹ These inconsistencies could relate to differences in HIV duration and different populations.⁵

Abdominal obesity was more predominant in HIV-negative than in HIV-positive females (59% vs. 41.1%, p -value < 0.05). Although this study did not include a dietary analysis, earlier reports found that the Dikgale HDSS community consumed high carbohydrates and low-fat, low-dietary fibre which may favour the development of abdominal obesity.³⁰ Similarly, a study conducted in a South African rural area showed obesity to be significantly more common in HIV-uninfected than in HIV-infected adults (24.5% vs. 20%, p -value < 0.001), and this difference was largely contributed by women (34% HIV-uninfected vs. 23.8% HIV-infected), while obesity rates were not significantly different in HIV-infected and HIV-uninfected men.²⁹

A significantly higher prevalence rate of HIV-infected than HIV-uninfected females consumed alcohol (25.4% vs. 11.9%, p -value < 0.05), while similar rates were observed in HIV-infected and HIV-uninfected males. Dikgale HDSS is a poor rural community, and poverty is a driver of commercial sex work for most women, exposing them to the sale of alcohol and the risk of HIV infection. Significantly higher alcohol use by HIV-positive than HIV-negative women has been reported elsewhere.³¹ The current study observed similar tobacco usage in HIV-positive and HIV-negative males and females. Higher tobacco use in HIV-positive than in HIV-negative people was reported elsewhere.³²

Table 4: Predictors of low high-density lipoprotein cholesterol levels in participants in the Dikgale Health and Demographic Surveillance System Centre ($n = 267$)

Predictors	Bivariate logistic regression		Multivariate logistic regression	
	OR (95% CI)	p -value	Adjusted OR (95% CI)	p -value
HIV-positive	2.33 (1.37–3.96)	0.002	2.69 (1.52–4.75)	0.001
Male	0.35 (0.20–0.62)	< 0.001	0.39 (0.22–0.71)	0.002
Alcohol	0.49 (0.27–0.90)	0.02	0.52 (0.27–0.99)	0.047
WC obesity	1.63 (1–2.66)	0.05		
IR	1.21 (0.67–2.16)	0.53		
hsCRP	2.07 (0.97–4.40)	0.06		
Hypertension	0.91 (0.56–1.48)	0.71		
Smoking	0.48 (0.24–0.96)	0.04		
Diabetes	0.78 (0.38–1.59)	0.49		

CI: confidence interval, HIV: human immunodeficiency virus, hsCRP: C-reactive protein, IR: insulin resistance, OR: odds ratio, WC: waist circumference. Values are unadjusted odds ratio (95% confidence interval) from bivariate logistic regression, and adjusted odds ratio (95% confidence interval) from multivariate stepwise forward and backward logistic regression modelling

The lipid profile in HIV-infected people (89) was altered, compared to that in HIV-uninfected people (Table II). Our study showed significant decreases in TC, LDL cholesterol and HDL cholesterol in HIV-positive people compared to that in HIV-negative people. This lipoprotein pattern is associated with the potential risk of premature CVD developing.³³ The dyslipidaemia caused by the effects of the virus itself results from the inflammatory cytokine response to HIV infection.^{34,35}

Several studies have reported similar decreases in TC, HDL cholesterol and LDL cholesterol,^{36–39} while higher LDL cholesterol levels have been reported elsewhere.^{40–42} Interestingly, a study¹⁶ conducted on urban South African women reported no significant difference in TC between treatment-naïve HIV-positive and HIV-negative women, while HDL cholesterol and LDL cholesterol were not reported. The current study observed no significant difference in TG levels between HIV-positive and HIV-negative people for the whole group, and for males and females separately. Some studies have demonstrated disparate TG results.^{9,38,42} Variations in TG levels were attributed to the difference in degree of immunosuppression, with TG levels escalating with an increase in immunosuppression.^{11,43}

Consistent with the current study, Baker et al⁴⁴ reported no significant association between HDL cholesterol and CD4 count. By contrast, two earlier studies by El-Sadr et al⁴⁵ and Rose et al⁴⁶ reported significant associations between HDL cholesterol and CD4 count.

Even though the contribution of HIV to lipid abnormalities may be difficult to distinguish from that of classical cardiovascular risk factors, in the current study, after controlling for the effect of each risk factor, the multivariate regression model indicated HIV infection, male gender and alcohol use as significant predictors of low HDL cholesterol. Having 2.8 times more likelihood of low HDL cholesterol in HIV-infected people therefore increases the risk of CVD in this HIV-infected rural population. Similarly, Oka et al¹² reported an association between HIV disease and lipid metabolism in a Japanese male population.

The strength of this study was that standardised techniques were used, which included the WHO STEPS questionnaire and the repeated measurement of BP and HIV, as well as the use of controls in analysing the biochemical parameters.

Limitations of the study included the relatively small number of study subjects. Moreover, the sample largely comprised people living at home, thus it was skewed towards an older sedentary group. Migrant workers and people with employment were away from home when the study was conducted. This could have biased the results. A similar study is needed on the migrant population. Information on smoking and alcohol use was obtained using the WHO STEPS questionnaire, considered to be a reliable instrument.

Conclusion

The treatment-naïve, HIV-infected rural population in the Dikgale HDSS had significantly lower levels of TC, LDL cholesterol, HDL cholesterol and ApoA1, but not TG, than an HIV-uninfected rural population. The study also revealed a high prevalence of CVD risk factors, such as hypertension, diabetes, IR, tobacco use and alcohol consumption in treatment-naïve, HIV-infected people, which confirms an earlier report on the general population.⁴⁷ To our knowledge, this study is the first to have reported on CVD risk factors in treatment-naïve,

HIV-infected people in the Limpopo province. HIV outreach programmes should focus more on the management of noncommunicable diseases in rural areas where elderly people reside. Further studies on subclasses of lipoproteins may provide more in-depth knowledge of the risk of CVD in this rural population.

Conflict of interest — The authors declare no conflict of interest.

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