Journal of Endocrinology, Metabolism and Diabetes of South Africa 2023; 28(1):18–28
https://doi.org/10.1080/16089677.2022.2084901

Open Access article distributed under the terms of the
Creative Commons License [CC BY-NC 4.0]
http://creativecommons.org/licenses/by-nc/4.0

JEMDSA
ISSN 1608-9677 EISSN 2220-1009
© 2022 The Author(s)
ARTICLE

Association between anthropometry and cardiovascular risk in patients attending a diabetic clinic

Kylie Divashnee Konar[a*] and **Somasundram Pillay**[b]

[a]*Phoenix Community Health Centre, Phoenix, South Africa*
[b]*Department of Internal Medicine at King Edward Hospital (KEH), Nelson R Mandela School of Clinical Medicine, University of KwaZulu-Natal, Durban, South Africa*
Correspondence: divashnee@hotmail.com

Check for updates

Background Obesity is a well-documented risk factor for cardiovascular disease (CVD) in type 2 diabetes mellitus (T2DM), with increasing evidence to suggest visceral adiposity as a greater risk factor for CVD than body mass index (BMI).
Objectives To determine a relationship between hypertension (HPT) and anthropometry in people living with diabetes (PLWD) in an HIV endemic area.
Methods This was a retrospective study analysing data captured from standardised clinic sheets from the DM clinic at the Harry Gwala Regional Hospital, Pietermaritzburg, South Africa, from January 1, 2019 to December 31, 2019.
Results Data from 957 PLWD were used for the study, the majority of whom had T2DM (811; 86.2%). Approximately one-sixth of the cohort had HIV infection (146; 15.3%). There was no significant difference in HPT prevalence between the HIV-uninfected (77.9%) and PLWD who had HIV (PLWDHIV) (78.1%). Multivariate analysis revealed females with increased waist circumference (WC) and waist-to-height ratio (WTHR) were 57.8 (95% CI 3.04–1096.33) ($p = 0.007$) and 87.2 (95% CI 4.88–1558.28) ($p = 0.002$) times more likely to be hypertensive respectively. By contrast, only BMI in males was associated with HPT with a AOR 5.294 (95% CI 1.54 - 18.22) ($p = 0.008$). HIV status was non-contributory to anthropometry in predicting HPT in PLWD.
Conclusion Our study found that anthropometric indices are not all equal predictors of HPT. The authors advocate for local guidance on gender-specific cut-offs on anthropometry in PLWD.

Keywords: Diabetes mellitus, waist circumference, waist-to-height ratio, body mass index, hypertension, HIV, cardiovascular disease

Background

Diabetes mellitus (DM), a chronic metabolic disorder, has been recognised as a major global public health burden.[1] By 2030, the number of adults with type 2 diabetes (T2DM) is expected to increase faster in low- to middle-income countries (LMICs) than in high-income countries (20%).[2]

Cardiovascular disease (CVD) is the leading cause of death worldwide.[3] T2DM is associated with increased cardiovascular morbidity and mortality.[4] Patients with T2DM have a two- to fourfold increase in risk of incident coronary heart disease ischaemic stroke and mortality. Hypertension (HPT), DM, dyslipidaemia and obesity are all well-documented modifiable risk factors for CVD.[4] Importantly, obesity is an independent risk factor for CVD, most notably HPT.[5–7] Patients with T2DM are generally more obese and have a greater number of cardiovascular risk factors when compared with patients without T2DM.[4,5] Weight loss is considered a key measure to the management of T2DM.[6,7] Increasing evidence suggests that visceral adiposity is a greater risk for CVD than body mass index.[4,5] Current evidence on anthropometric indices as a predictor for HPT remain unclear in PLWD. Most studies have described these indices in non-diabetic patients, which is not amenable to the PLWD profile.

Fat accumulation is generally described by these indices: waist circumference (WC), body mass index (BMI), and waist-to-height ratio (WTHR). [8] BMI is a simple index of weight-for-height that is commonly used to classify obesity in adults.[13] Globally, studies have found that patients with excess visceral adiposity, irrespective of their BMI, had increased cardiovascular risk.[8,9] In LMICs, the relationship between BMI and CVD displays vast heterogenicity as a result of both genetic and biological factors.[10] Despite its limitations, BMI has been adopted as a simple tool to categorise patients into risk groups.[11]

Both DM and human immunodeficiency virus (HIV) infection are independently associated with an increased risk of atherosclerosis.[12,13] Whether HIV infection is associated with increased DM risk, relative to uninfected controls, has been debated. Among large cohort studies, some have found an association of HIV with a higher risk of DM[14,15] while others have reported a similar[16] or even lower risk[17] compared with those who are uninfected. It is proposed that the combined effects of the chronic inflammation milieu caused by the HIV and the associated metabolic effects of antiretroviral therapy (ART) can increase cardiovascular risk.[18]

When compared with BMI, studies have shown that WC is a more effective measure of body fat distribution.[19,20] However, WC remain uncertain in certain populations. In 2020, Xing et al. stated that WC was not a sufficient predictor of major cardiovascular adverse events in female T2DM patients as compared with their male counterparts.[21] Patients with T2DM tend to be more obese, hence research from non-diabetic populations may not correlate well in patients with T2DM.[22]

WTHR has been described to be cheaper, and lacks the need for a scale and calibration with easier boundaries that may be used from consumer-friendly charts as compared with BMI.[19] WTHR values of 0.5 or above may indicate increased cardiovascular disease risk across sub-populations.[23]

Obesity is a well-documented risk factor for CVD, with increasing evidence to suggest visceral adiposity as a greater risk for CVD than BMI. Current evidence on anthropometric indices as a predictor for cardiovascular risk remain unclear in PLWD. Most studies describe anthropometric indices in general cohorts rather than in PLWD.

The purpose of this retrospective study was to determine whether all anthropometric indices, namely WC, WTHR or BMI, were equal predictors of HPT in PLWD in an HIV endemic area. As highlighted, a cheaper cost-effective tool to measure obesity can help prevent HPT and resultant complications in PLWD. This is particularly attractive in LMICs where community interventions need to be cost-effective to be sustainable.

Methods

Study design

A retrospective, analytical cohort study was performed using data collected from patients who attend a specialised diabetes clinic at the Harry Gwala Regional Hospital, Pietermaritzburg, KwaZulu-Natal. Clinicians used a standardised, comprehensive clinic sheet for all patients consulted in this clinic, which has been approved by the University of KwaZulu-Natal Biomedical Research and Ethics Committee (BREC)—BCA 194/15. The data for this study included all patients 18 years or older who attended the diabetes clinic between January 1, 2019, and December 31, 2019.

Patient demographics, family history of DM, mean glycated haemoglobin (HbA1c %), random blood glucose (mmol/l), HIV status, type of DM (Type 1 or 2), total cholesterol (TC) (mmol/l), triglyceride level (mmol/l), high-density lipoprotein (HDL) (mmol/l), low-density lipoprotein (LDL) (mmol/l), blood pressure (mmHg), GFR (glomerular filtration rate), social factors (diet adherence, exercise) were recorded. The presence of microvascular complications of DM (nephropathy, peripheral neuropathy, proliferative and non-proliferative retinopathy) and macrovascular complications of DM (stroke or cerebrovascular accident [CVA]), ischaemic heart disease (IHD) and myocardial infarction (MI) was also recorded.

Weight, height, WC, WTHR and BMI were measured by trained healthcare workers following standardised protocols, as set by the World Health Organization (WHO).[24] To perform height, weight and BMI readings, the Adam® Equipment MDW-300L scale (Adam Equipment Co, Milton Keynes, UK) was used. The patient's WC was taken at the end of normal expiration with a measuring tape at a point midway between the lower ribcage and the superior iliac crest in the midaxillary line. BMI was calculated as weight in kilograms divided by the square of height in metres. WTHR was calculated as WC (cm) divided by height (cm). Three measurements were taken for all indicators, and the averages were used for further analyses.

The values were categorised according to standardised guidelines. BMI (kg/m^2) was calculated and recorded in four categories defined with overweight as > 25 kg/m^2, 30–34.9 (Class 1 obesity), 35–39.9 (Class 2obesity), ≥ 40 (Class 3 obesity).[24] Abnormal WC was classified as > 94 cm for males and > 90 cm for females.[25] According to a systemic review, the mean boundary values for WTHR covering all cardiometabolic outcomes from studies in 14 different countries and including Caucasian, Asian and Central American subjects were 0.5 for both men and women, hence this was used in this study as a cut-off point for both men and women.[26]

Missing or incomplete or incorrectly completed data sheets were not considered. Good glycaemic control was defined as a HbA1c value < 7%.[25] The Bio-Rad D-10 machine (Bio-Rad Laboratories, Hercules, CA, USA) was used for analysing the HbA1c values at the laboratory. Both the laboratory and the machines are NGSP (National Glycohemoglobin Standardisation Programme) accredited to maintain standardisation of HbA1c results, while the random glucose measurement (mmol/l) was determined using an Accu-Chek® glucometer (Roche, Basel, Switzerland). Blood pressure (BP) and pulse were recorded using a Mindray® VS-800 machine. BP was recorded, as described in the 2014 South African hypertension guidelines.[27] Raised TC was defined as ≥ 4.5 mmol/l, triglycerides ≥ 1.7 mmol/l, LDL ≥ 1.8 mmol/l.[25] Reduced HDL was recorded as < 1 mmol/l for males and < 1.2 mmol/l for females. [25] Abnormal GFR < 60 was considered as chronic kidney disease (CKD). [28]

Clinicians used a standardised, comprehensive clinic sheet for all patients seen for consultation in this clinic, which has been approved by the University of KwaZulu-Natal Biomedical Research and Ethics Committee (BREC)—BCA 194/15. The data cohort for this study included all patients 18 years or older who attended the diabetes clinic between January 1, 2019, and December 31, 2019.

Statistical analysis

Statistical analysis was conducted with numerical data using ANOVA, while categorical data relationships were determined using either chi-square or Fisher's exact tests. A p-value < 0.05 was used as indicator of significance. Data were analysed by the Statistical Package for the Social Sciences (SPSS) version 25 for Windows (IBM Corp, Armonk, NY, USA). We used standardised reporting of non-parametric data as median throughout the article as ± interquartile range (IQR).

Results

Epidemiology

Data of 957 PLWD were used for the study: T2DM (811; 86.2%). T2DM was the most common type of DM in both PLWD and those PLWD with HIV infection (PLWDHIV) ($p < 0.001$). Although the proportion of the type of DM was similar between PLWD and PLWDHIV, there was a greater number of PLWD with T2DM ($p < 0.001$). Over two-thirds (69.4%) of PLWD were female ($p < 0.001$). Approximately one-sixth of the cohort had HIV infection (146; 15.3%). Of this cohort with DM, 129 (88.4%) were on a fixed-dose combination of anti-retroviral therapy (ART) of which 65.1% were on the fixed dose combination of tenofovir, emcitribine and efavirenz.

PLWDHIV were significantly younger than their uninfected counterparts (48.0 vs. 58.0 years, respectively, $p < 0.001$). PLWDHIV had a shorter median duration of DM of 7.0 (2.0–11.0) vs. 9.0 (3.0–17.0) years ($p < 0.001$) years as compared with their uninfected counterparts.

Patients living with diabetes (PLWD): overall study population

Anthropometric indices

The median BMI for PLWD was 32.0 (26.0–37.0). Three-quarters (75.8%) of PLWD had a BMI ≥ 25 kg/m^2. The median WTHR for PLWD was 0.66 (0.57–0.73) with 88.7% of PLWD having an

Table 1: Characteristics of the study population

Item	Number of participants ($n = 957$)	%	*p*-value
Age (years), median (IQR)	56.0 (44.0–64.0)		
Gender:			
• Females	664	69.4%	< 0.001
• Males	293	30.6%	
Duration of DM (years), median (IQR)	8.0 (3.0–15.0)		
Diabetes type:			
• Type 1	132[a]	13.8%	< 0.001
• Type 2	825*	86.2%	
Number (*n*) of patients:			
• With HIV infection	146	15.3%	
• On ART	129	88.4%	
• On Tenofovir/emcitribine/efavirenz (Tribus®/Atrozia®)	84	65.1%	
Following diet (*n*):			
• Overall	807*	84.9%	< 0.001
• Female	558	69.1%	
• Male	249	30.9%	
Following exercise (*n*):			
• Overall	722*	75.9%	< 0.001
• Female	495	68.6%	
• Male	227	31.4%	
With hypertension (*n*):			
• Overall	746*	78%	< 0.001
• Female	545	73.1%	
• Male	201	26.9%	
HbA1c %, median (IQR)	9.5 (7.7–11.2)		
Number of patients with poor glycaemic control (HbA1C ≥ 7%):			
• Overall	761*	79.5%	< 0.001
• Female	539	70.8%	
• Male	222	29.2%	
BMI (kg/m^2), median (IQR)			
• Overall	32.0 (26.0-37.0)		< 0.001
• Female	34.0 (29.0-39.0)		
• Male	27.0 (23.0-32.0)		
Number of patients with BMI > 25:			
• 25–29.9 (overweight)	192	21.9%	0.002
• 30–34.9 (Class 1 Obesity)	218	24.9%	
• 35–39.9 (Class 2 Obesity)	162	18.4%	
• ≥ 40 (Class 3 Obesity)	153	17.5%	
WTHR, median (IQR):			
• Overall	0.66 (0.57–0.73)		< 0.001
• Female	0.69 (0.63–0.75)		
• Male	0.57(0.49–0.63)		
Number of patients with abnormal WTHR > 0.5:			
• Overall	761*	88.7%	< 0.001
• Female	572	75.2%	
• Male	189	24.8%	
Waist circumference (cm), median (IQR):			
• Overall	105.0 (93.0–115.0)		< 0.001
• Female	109.0 (99.0–118.0)		
• Male	95.0 (81.0–106.5)		
Number of patients with abnormal WC (M > 94 cm, F > 90 cm):			
• Overall	716*	83.1%	< 0.001
• Female	574	80.2%	

(*Continued*)

Table 1: Continued.

Item	Number of participants ($n = 957$)	%	p-value
• Male	142	19.8%	
Total cholesterol(mmol/l), median (IQR):			
• Overall	4.4 (3.6–5.3)		< 0.001
• Female	4.6 (3.7–5.4)		
• Male	4.1 (3.4–5.1)		
Number of patients with raised TC ≥ 4.5 mmol/l:			
• Overall	436*	49.5%	< 0.001
• Female	328	75.2%	
• Male	108	24.8%	
Triglyceride (mmol/l), median (IQR):			
• Overall	1.5 (1.0–2.3)		0.409
• Female	1.6 (1.1–2.3)		
• Male	1.5 (0.9–2.3)		
Number of patients with raised triglyceride ≥ 1.7 mmol/l:			
• Overall	378*	43.2%	< 0.001
• Female	271	71.7%	
• Male	107	28.3%	
LDL (mmol/l), median (IQR):			
• Overall	2.4(1.8–3.1)		0.030
• Female	2.5(1.8–3.2)		
• Male	2.2(1.7–3.0)		
Number of patients with raised LDL ≥ 1.8 mmol/l:			
• Overall	420*	73.2%	< 0.001
• Female	296	70.5%	
• Male	124	29.5%	
HDL (mmol/l), median (IQR):			
• Overall	1.2 (1.0–1.4)		< 0.001
• Female	1.2 (1.0–1.5)		
• Male	1.1 (0.9–1.4)		
Number of patients with reduced HDL < 1 mmol/l for male; < 1.2 mmol/l for female:[∂]			
• Overall	315*	75.9%	< 0.001
• Female	239	24.1%	
• Male	76		
Number of patients with cardiovascular complications (n):			
• Previous CVA	32	3.3%	
• Previous MI	6	0.6%	
• IHD/angina	11	1.1%	
• CKD Stage 4 (GFR < 60)	329	39.4%	
Retinopathy (n):			
• Proliferative	23	2.4%	
• Non-proliferative	84	8.8%	
Peripheral neuropathy (n)	374	39.1%	

*Missing variables.
[∂]Percentage of patients with reduced HDL in each subgroup.
Keywords: BMI = body mass index; CVA = cerebrovascular accident (stroke); CKD = chronic kidney disease; DM = diabetes mellitus; HBA1C = glycated haemoglobin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; IHD = ischaemic heart disease (angina); IQR = interquartile range; LDL = low-density lipoprotein; MI = myocardial infarction; TC = total cholesterol; WC = waist circumference; WTHR = waist-to-height ratio; ART = antiretroviral therapy.

abnormal WTHR > 0.5. The median WC in PLWD was 105.0 cm (93.0–115.0) with 83.1% of PLWD having an abnormal WC. Females were found to have greater median BMI, WC and WTHR than their male counterparts ($p < 0.001$) (Table 1).

Hypertension

Approximately four-fifths (78%) of the study population had HPT (Table 1) with a greater number of females having HPT as compared with males ($p < 0.001$) (Supplementary Table 1). Increasing BMI ≥ 25, abnormal WC and WTHR ≥ 0.5 were all significantly associated with the presence of HPT in both male ($p < 0.001$) and female PLWD ($p < 0.001$) (Supplementary Table 1). In male PLWD a WC ≥ 94cm ($p < 0.001$) and a WTHR ≥ 0.5 ($p < 0.001$) were significantly associated with HPT as compared with male PLWD with WC < 94cm ($p = 0.373$) and WTHR < 0.5 ($p = 0.204$) (Supplementary Table 1).

Table 2: Overall bivariate and multivariate analysis including gender

	Bivariate analysis				Multivariate analysis		
Variable	**Unadjusted OR**	**CI**	**Pearson Chi-Square Value**	***p*-value**	**Adjusted OR**	**CI**	***p*-value**
BMI	3.607	2.49–5.23	49.506	< 0.001	2.494	1.30–4.80	0.006
WC	4.638	3.18–6.76	79.686	< 0.001	2.131	1.08–4.19	0.028
WTHR	7.582	4.83–11.91	95.823	< 0.001	3.009	1.38–6.55	0.005
Gender	0.477	0.35–0.66	21.486	< 0.001	0.852	0.48–1.50	0.578
Age	1.162	1.139-1.186	489.341	<0.001	1.120	1.090-1.151	<0.001

A chi-square test of independence was done for the bivariate analysis. Each of the independent conditions was significantly related to HPT ($p < 0.001$).

Patients living with diabetes mellitus and HIV infection (PLWDHIV)

The median BMI, WTHR and WC in PLWDHIV were 30.0 (26.0–35.0), 0.63 (0.55–0.69) and 102.0cm (91.0–111.0) respectively (Table 3). PLWDHIV were found to have a similar proportion of participants who were overweight and had abnormal WTHR as compared with their HIV-uninfected counterparts (Table 3). There was a greater proportion of HIV-uninfected PLWD with an abnormal WC as compared with PLWDHIV ($p < 0.001$) (Table 3).

Hypertension

There was a similar proportion of HPT in HIV-uninfected PLWD (77.9%) as compared with PLWDHIV (78.1%) (Table 3). In both HIV-uninfected PLWD and in PLWDHIV, an increasing BMI and WTHR ≥0.5 were associated with HPT ($p < 0.001$) (Supplementary Table 2). Within the PLWDHIV cohort, significantly more females with HPT had an abnormal WC > 80 cm ($p < 0.001$) (see Supplementary Table 2). By contrast, for HIV-infected males with HPT, WC was found to be non-significant ($p = 0.330$) as compared with HIV-uninfected males with HPT ($p < 0.001$) (see Supplementary Table 2).

Binary logistic regression (BLR)

BLR was performed on the data with HPT as the dependent variable and each of BMI, WC and WTHR as independent variables (Table 2). The table below also includes gender as a dependent variable.

Overall analysis

After adjusting for the following variables: CVA (Stroke) (1 = Yes), Peripheral Neuropathy (PN) (1 = Yes), Non-Proliferative Retinopathy (1 = Yes), Proliferative Retinopathy (1 = Yes), HIV Status (1 = Positive), Total Cholesterol (1 ≥ 4.5), Triglycerides (1 ≥ 1.7), HDL (1 ≥ 1.2), LDL (1), Systolic BP (1 ≥ 140), Diastolic BP (1 ≥ 90), GFR (1 = Abnormal), Exercise (1 = Yes) and Mean HbA1c (1 ≥ 7) we showed that BMI, WC and WTHR remained significant, but gender became non-significant ($p = 0.578$). Overall, WTHR had the highest odds for predicting HPT in PLWD. Despite a decrease in odds ratio after adjustment, the odds for BMI, WC and WTHR remained significant. Furthermore, it was found that the two main contributors to the adjustment variables were raised systolic BP > 140 and CKD (GFR < 60) (Supplementary Table 3).

Analysis by gender

Females only

Females with abnormal WC and WTHR were 14.6 and 22.6 times more likely to be hypertensive respectively ($p < 0.001$) (Supplementary Table 4). These odds were significantly increased after adjustment. After adjustment, females with abnormal WC and WTHR were 57.8 ($p = 0.007$) and 87.2 ($p = 0.002$) times more likely to be hypertensive, respectively (Supplementary Table 4). WTHR was found to have the greatest odds in predicting HPT in female PLWD. After adjustment, increasing BMI in females became non-significant for HPT ($p = 0.087$) (Supplementary Table 4).

Males only

In the bivariate analysis, all anthropometric indices were significantly associated with HPT in males ($p < 0.001$) (Supplementary Table 5). After adjusting for dependent variables, only increasing BMI was significantly associated with HPT in males ($p = 0.008$). Males with increasing BMI were 5.3 times more likely to be HPT than those with normal BMI (Supplementary Table 5).

Effect of HIV on the multivariate analysis

Overall, HIV had no significant change in odds for HPT as compared with HIV-uninfected PLWD for all three anthropometric indices (Table 4).

Discussion

Globally, more than 90% of PLWD are categorised as T2DM.[29] Our study results were in keeping with this finding, with 86.2% of PLWD having been diagnosed with T2DM ($p < 0.001$). In 2020, a report on the global burden of DM found males to have a higher prevalence of DM than females.[30] Additionally, the age of onset of new diagnosis was shown to be earlier among males with expected patterns of rising DM prevalence with increasing age, and with an incidence peak at 55–59 years, in keeping with the median age of our study population of 56.0 (44.0–64.0) years. Some 51% of patients were found to be between 50 and 70 years old. This implies that PLWD have a longer period of their lives potentially exposed to the adverse cardiometabolic complications associated with DM. An opportunity to be able to predict the risk of developing CVD should be of high clinical priority in the management of DM.

Our study showed that despite the majority of PLWD having reported to have followed a diet (84.9%) and exercise regimen (75.9%), respectively, more than three-quarters (75.8%) of these PLWD were overweight ($p = 0.002$). The significant majority (75.9%) of our patients were also found to have poor glycaemic control.

The indices of truncal obesity in our study (WC and WTHR) identified more PLWD with obesity (83.1% and 88.7% respectively) when compared with BMI (82.6%). Body mass index is widely used for the diagnosis of obesity.[31] Research has demonstrated that people with higher BMI are more likely to have T2DM.[32] Although BMI is the most used anthropometric measurement in epidemiological studies to indicate obesity, available evidence is pointing towards the better performance of other measures of adiposity (this especially in sub-Saharan

Table 3: Characteristics of the HIV positive vs. negative PLWD

Item	HIV-negative (*n* = 811)	HIV-positive (*n* = 146)	*p*-values
Age (years), median (IQR)	58.0 (44.0–66.0)	48.0 (42.0–54.0)	< 0.001
Gender:			
• Overall	811 (84.7)	146 (15.3%)	< 0.001
• Females	568 (70.0)	96 (65.8%)	< 0.001
• Males	243 (30.0)	50 (34.2%)	< 0.001
Duration of DM (years), median (IQR)	9.0 (3.0–17.0)	7.0 (2.0–11.0)	0.004
Diabetes type:			
• Type 1	115*(14.2%)	17 (11.6%)	< 0.001
• Type 2	693 *(85.8%)	129 (88.4%)	< 0.001
Following diet:			
• Overall	675* (83.9%)	132* (90.4%)	< 0.001
• Female	473 (70.1%)	85 (64.4%)	< 0.001
• Male	202(29.9%)	47 (35.6%)	< 0.001
Following exercise:			
• Overall	603* (74.9%)	119* (81.5%)	< 0.001
• Female	415 (68.8%)	80 (67.2%)	< 0.001
• Male	188 (31.2%)	39 (32.8%)	< 0.001
With hypertension:			
• Overall	632* (77.9%)	114* (78.1%)	< 0.001
• Female	469 (74.2%)	76 (66.7%)	< 0.001
• Male	163 (25.8%)	38 (33.3%)	< 0.001
HbA1c %, median (IQR):			
• Overall	9.5 (7.8–11.2)	8.9 (7.2–11.1)	0.052
• Female	9.6 (7.8–11.4)	8.7 (7.1–11.2)	0.031
• Male	9.5(7.6–10.9)	9.4 (7.7–11.0)	0.905
Number of patients with poor glycaemic control (HbA1C ≥ 7%):			
• Overall	655* (86.1%)	106* (13.9%)	< 0.001
• Female	469 (71.6%)	70 (66.0%)	< 0.001
• Male	186 (28.4%)	36 (34.0%)	< 0.001
BMI (kg/m^2), median (IQR):			
• Overall	32.0 (26.0–38.0)	30.0 (26.0–35.0)	0.049
• Female	34.0 (29.0–39.0)	32.5 (28.0–37.0)	0.149
• Male	27.0 (23.0–32.0)	27.0 (23.5–30.0)	0.862
Number of patients with BMI > 25:			
• Overall	612* (82.6%)	112* (83.6)	< 0.001
• 25–29.9 (overweight)	151 (20.4%)	41 (30.6%)	< 0.001
• 30–34.9 (Class 1 Obesity)	181 (24.4%)	37 (27.6%)	< 0.001
• 35-39.9 (Class 2 Obesity)	138 (18.6%)	23 (17.2%)	< 0.001
• ≥ 40 (Class 3 Obesity)	142 (19.2%)	11 (8.2%)	< 0.001
WTHR, median (IQR):			
• Overall	0.66 (0.58–0.74)	0.63 (0.55–0.69)	0.008
• Female	0.70 (0.64–0.76)	0.68 (0.62–0.72)	0.007
• Male	0.57 (0.49–0.64)	0.55 (0.51–0.61)	0.527
Number of patients with abnormal WTHR > 0.5:			
• Overall	643* (88.7%)	118* (88.7%)	< 0.001
• Female	492 (96.5%)	80 (95.2%)	< 0.001
• Male	151 (70.2%)	38 (77.6%)	< 0.001
Waist circumference (cm), median (IQR):			
• Overall	106.0 (94.0–116.0)	102.0 (91.0–111.0)	0.042
• Female	109.0 (99.0–119.0)	105.5 (97.0–114.0)	0.095
• Male	96.0 (80.0–107.0)	92.0 (86.0–103.0)	0.228
Number of patients with abnormal WC (M > 94 cm. F > 80 cm):			
• Overall	616* (84.5%)	100* (75.2%)	< 0.001

(*Continued*)

Table 3: Continued.

Item	HIV-negative ($n = 811$)	HIV-positive ($n = 146$)	p-values
• Female	494 (79.9%)	80 (80.0%)	< 0.001
• Male	122 (19.8%)	20 (20.0%)	< 0.001
TC (mmol/l), median (IQR):			
• Overall	4.3 (3.6–5.2)	4.9 (4.1–5.6)	< 0.001
• Female	4.5 (3.7–5.3)	4.9 (4.2–5.5)	0.004
• Male	4.0 (3.4–5.0)	4.8 (3.7–5.8)	0.023
Number of patients with raised TC ≥ 4.5 mmol/l:			
• Overall	348* (46.8%)	88* (64.2%)	< 0.001
• Female	268 (77.0%)	60 (68.2%)	< 0.001
• Male	80 (23.0%)	28 (31.8%)	< 0.001
Triglyceride (mmol/l), median (IQR):			
• Overall	1.5 (1.0–2.3)	1.7 (1.2–2.6)	0.036
• Female	1.6 (1.0–2.3)	1.6 (1.1–2.4)	0.974
• Male	1.4 (0.9–2.1)	1.9 (1.4–3.0)	< 0.001
Number of patients with raised triglyceride ≥ 1.7 mmol/l:			
• Overall	308* (41.6%)	70* (52.2%)	< 0.001
• Female	233 (75.6%)	38 (54.3%)	< 0.001
• Male	75 (24.4%)	32 (45.7%)	< 0.001
LDL (mmol/l), median (IQR):			
• Overall	2.3 (1.7–3.1)	2.6 (1.8–3.2)	0.079
• Female	2.4 (1.8–3.1)	2.7 (1.9–3.3)	0.167
• Male	2.1 (1.7–2.9)	2.4 (1.6–3.1)	0.811
Number of patients with raised LDL ≥ 1.8 mmol/l:			
• Overall	355* (72.7%)	65* (75.6%)	< 0.001
• Female	249 (70.1%)	47 (72.3%)	< 0.001
• Male	106 (29.9%)	18 (27.7%)	< 0.001
HDL (mmol/l), median (IQR):			
• Overall	1.2 (1.0–1.4)	1.3 (1.1–1.6)	0.002
• Female	1.2 (1.0–1.4)	1.4 (1.2–1.6)	< 0.001
• Male	1.1 (0.9–1.3)	1.1 (0.9–1.5)	0.501
Number of patients with reduced HDL < 1 mmol/l for male; < 1.2 mmol/l for female:[∂]			
• Overall	282*	33*	< 0.001
• Female	218 (63.7%)	21 (47.7%)	< 0.001
• Male	64 (37.4%)	12 (60.0%)	
Number of patients with cardiovascular complications:			
• Previous CVA	28 (3.5%)	4 (2.7%)	< 0.001
• IHD/angina	11 (1.4%)	0 (0.0%)	< 0.001
• Previous MI	5 (0.6%)	1 (0.7%)	0.102
• CKD (GFR < 60)	281 (40.0%)	48 (36.1%)	< 0.001
Retinopathy:			
• Proliferative	22 (2.7%)	1 (0.7%)	< 0.001
• Non-proliferative	79 (9.7%)	5 (3.4%)	< 0.001
Peripheral neuropathy	313 (38.6%)	61 (41.8%)	< 0.001

*Missing variables.
[∂]Percentage of patients with reduced HDL in each subgroup.
Keywords: BMI = body mass index; CVA = cerebrovascular accident (stroke); CKD = chronic kidney disease; DM = diabetes mellitus; HBA1C = glycated haemoglobin; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; IHD = ischaemic heart disease (angina); IQR = interquartile range; LDL = low-density lipoprotein; MI = myocardial infarction; TC = total cholesterol; WC = waist circumference; WTHR = waist-to-height ratio; ART = antiretroviral therapy.

Africa), such as WC and WTHR, in predicting CVD.[24] A growing body of evidence suggests that central obesity, or visceral adiposity, is a better predictor of cardiovascular risk in T2DM than BMI.[33] Central obesity could be associated with the higher risk of T2DM prevalence in men, given that men are more prone to android adiposity with greater abdominal adiposity, as compared with women who are more likely to exhibit gynoid adiposity.[34] However, there remain multiple disparities in the levels of underweight and obesity across African regions, for example in Southern African countries comprising Botswana[35], Namibia[36] and South Africa,[37,38] countries with the highest obesity index. In 2008, South Africa was rated as the country with the highest BMI globally, with a median population level score approximated at 26.9 kg/m^2 among males (versus world average of 23.8 kg/m^2), and 29.5 kg/m^2 among females (versus world average of

Table 4: Effect of HIV on multivariate analysis

		Multivariate analysis		
Variable		**Adjusted OR**	**CI**	***p*-value**
BMI	Overall	0.958	0.443–2.072	0.958
	Female	0.857	0.324–2.265	0.756
	Male	1.045	0.245–4.467	0.952
	Overall	1.045	0.481–2.268	0.912
WC	Female	0.888	0.325–2.428	0.888
	Male	1.660	0.385–7.156	0.496
	Overall	0.984	0.452–2.140	0.968
WTHR	Female	0.852	0.311–2.339	0.756
	Male	1.324	0.313–5.605	0.703

24.1 kg/m^2), respectively. In South Africa, Goetjes et al. found that men were 11.32% less likely to be obese than women,[39] this more especially among the black African race. Our study concurred with these findings and found that female PLWD had higher median BMI, WC and WTHR as compared with male PLWD ($p < 0.001$). Previous studies in America and Europe have also documented a higher BMI among females.[40,41] In South Africa, rapid urbanisation characterised by the adoption of unhealthy energy-dense diets and physical inactivity have contributed to the steadily increasing obesity epidemic, with 69% of women and 39% of men being classified as overweight or obese.[42] In African women, the prevalence of T2DM doubled from 4.1% in 1980 to 8.9% in 2014.[43] African women are disproportionately affected by diabetes compared with their male counterparts, often taking the position of unpaid caregiver roles for affected family members in addition to taking care of their own diabetes.[44] Children born to diabetic mothers are at an increased risk of developing T2DM in adulthood,[45] thereby accelerating the intergenerational risk of T2DM. Mapping the prevalence of T2DM in this population is important as it has implications for future trends, as well as allowing for monitoring of the burden of T2DM in Africa.

HPT remains the most important CVD risk factor globally, this more especially in PLWD. The coexistence of HPT in DM has been shown to increase the risk of death and overall cardiovascular events by 44% and 41%, respectively, as compared with 7% and 9% in PLWD without HPT.[46] Globally, rates of CVD in patients without DM at all ages are higher in men than in women.[47] However, these CVD event rates are shown to be similar among those women and men living with diabetes.[48] We found that over three-quarters (78%) of PLWD had HPT with 70.5% of female PLWD having HPT. In our study, abnormal WC ($p = 0.007$) and WTHR ($p = 0.002$) had the highest odds in predicting HPT in females, while BMI was shown to be the best predictor of HPT in males ($p = 0.008$). Results of studies from Asia support our findings with reports of a WTHR > 0.5 being a better predictor of HPT in PLWD as compared with other indices.[49–51] Similarly, in Brazil, Castanheira et al.[52] found that a WTHR ≥ 0.5 was a better predictor of HPT in both sexes as a compared with other indices, while in Chile, a study also found similar WTHR cut-off points of ≥ 0.59 in females and ≥ 0.55 in males. [53] In contrast, a 2021 study from Nigeria found a higher proportion of males with cardiovascular risk factors as compared with females (38.2% vs 24.2%). They found that BMI and WTHR had the highest area under curve (AUC) of 0.613 and 0.577 respectively.[54] However, this could be region specific. Some earlier studies had suggested that using a cut-off point of 0.5 for WTHR may not be suitable for African black patients and the one-size fits all approach needs to be reviewed.[55,56] Our study found WTHR > 0.5 significant in females in predicting HPT ($p = 0.002$), while non-significant in males (0.053). This shows there can be disparities in gender and ethnic populations using WTHR, especially amongst African people.

WC is an easy-to-use, inexpensive measure of obesity. In our study, WC was significantly associated with HPT in females ($p = 0.007$) while non-significant in males ($p = 0.156$). Given that women have a higher proportion of total subcutaneous fat distribution as compared with men,[57] the potential risk of misclassification of women as having excessive visceral adiposity by using values of WC to predict other components of the metabolic syndrome (METs)[58] cannot be ignored. There is a need to validate the WC ≥ 94 cm for men and ≥ 90 cm for women observed in this present study for other sub-Saharan African populations. Country, ethnic and gender-specific WC cut-off points are needed, because adopting other WC criteria to diagnose African black populations may either under- or overestimate the presence of METs. A cross-sectional study in Ghana found the prevalence of abdominal obesity was 77.0% and was significantly higher in women than in men. They showed a positive correlation between WC ($r = 0.56$, $p < 0.001$), female gender ($r = 0.73$, $p < 0.001$) and age ($r = 0.20$, $p < 0.001$). Most studies in South Africa have demonstrated the need for different WC thresholds in local populations.[59–62]

Isolated systolic hypertension is a major cardiovascular risk factor at all ages and in both sexes.[63] In our study the presence of a GFR < 60 and elevated systolic blood pressure was found to have significantly impacted our findings. Diabetic kidney disease is a common complication of diabetes, is mediated by hypertension and is associated with poor quality of life, multiple hospital admissions and increased burden of disease.[64]

A 2010 report found that the prevalence of DM among HIV-infected adults was 10.3% (95% CI 9.2–11.5%).[65] The prevalence of DM was also shown to be 3.8% (CI 1.8–5.8%) higher in HIV-infected adults as compared with the adults in the general population.[43] Factors associated with this increased DM prevalence among HIV-infected adults included increasing age, duration of HIV infection, lower cluster of differentiation-4 (CD4) cell count and obesity. There are reported lower BMI and obesity rates in PLWDHIV in Africa ranging from 3.2% in Ethiopia,[66] to 3.8% in Malawi[67] and –8.3% in Nigeria[68] Despite our PLWDHIV having a lower median BMI ($p < 0.049$), WC (0.042) and WTHR (0.002) as compared with their HIV-uninfected counterparts, our study found substantially higher obesity rates amongst PLWDHIV (83.6%) as compared with global trends, which we attributed to the presence of DM. Similarly, a recent South African study showed that obesity was significantly more prevalent than in other African countries, especially in women (40.8% vs. 28.3%) and among older women (> 30 years old) ($p = 0.039$).[69] One theory proposed was that African women with HIV preferred to be overweight as this was associated with improved ability to breast-feed.[70]

We found a greater proportion of PLWDHIV with hypertension as compared with their HIV-uninfected counterparts ($p < 0.001$), and 65.8% of those were female ($p < 0.001$). This could be explained by the fact that the majority of PLWDHIV included in our study were on ART (88.4%). It is known that widespread use of ART has significantly increased the life expectancy of PLWDHIV, which supports an ageing HIV population who are

at risk of developing comorbidities such as DM that occur at earlier ages than in the general population.[71,72]

Our study is the first to describe HPT and anthropometry specifically in PLWDHIV. Some studies in PLWHIV in Brazil have found BMI to be a poor tool to identify each metabolic change separately but it was suggested that it is best to identify metabolic syndrome as a whole.[73] In Africa, BMI was significantly associated with HPT in Tanzania[74] and Cameroon.[75] Increased BMI increases the risk of HIV patients developing DM.[76] Increasing the duration of patients on ART also increases the risk of gaining weight and the risk factor for non-communicable diseases (NCDs). Despite this, there was no significant difference from anthropometric indices in predicting HPT in PLWDHIV in our study. This could be due to smaller sample size in relation to the study population.

Based on existing population projections, a study in Tanzania and Uganda estimated that the economic health service burden in people living with comorbidity (HIV and/or DM and/or HPT) will go up by 21.5% (range, 19.3%, 23.4%) over the next 5 years.[77] An integrated one-stop management of chronic conditions – specifically HIV, DM and HPT – which could reduce costs per patient between 34% and almost 50%, as compared with managing each condition separately, is required, especially in LMICs in Africa.

Conclusion

Diabetes mellitus and hypertension remain major causes of premature cardiovascular morbidity and mortality globally, while obesity is a well-documented risk factor for CVD, HPT and DM. Our study found that anthropometric indices are not all equal predictors of HPT. WC and WTHR were found to be better predictors of HPT in female PLWD while BMI was found to be a better predictor of HPT in male PLWD. This is of clinical significance in LMIC as simple anthropometric measures are cost-effective, non-invasive and universally acceptable measures of body habitus. This is especially useful in a resource-constraint setting like South Africa. Disparities we found in this study by gender in anthropometry are also important. We advocate that local guidance on gender- and ethnic-specific cut-offs needs to be devised to prevent over- or underestimation of cardiovascular risk in PLWD. HIV status was non-significant with regard to anthropometric indices in predicting hypertension in PLWDHIV.

Study limitations — As this was a retrospective study no causal relationships could be determined; rather, associations were defined. The effect of ART on obesity and cardiovascular risk in PLWDHIV could be further explored.

Acknowledgements — Mr D Singh is thanked for his help in generating statistical analyses for the study. The authors would also like to thank Sister Lungi Ndaba and her nursing team at the Harry Gwala Regional Hospital diabetes clinic.

Compliance with ethical standards — Ethics approval was obtained from the Biomedical Research Ethics Committee, University of KwaZulu-Natal (number 3287/2021).

Data availability statement — Data sharing is not applicable to this article, as no new data were created or analysed in this study.

Disclosure statement — No potential conflict of interest was reported by the authors.

Disclaimer — The views and opinions expressed in this article are those of authors and do not necessarily reflect the position of any affiliated agency of the authors.

Supplementary material — available online at https://doi.org/10.1080/16089677.2022.2084901

ORCID

Kylie Divashnee Konar http://orcid.org/0000-0002-0492-218X
Somasundram Pillay http://orcid.org/0000-0002-5604-645X

References

1. World Health Organization. Diabetes. 2021. World Health Organization. Available at: https://www.who.int/health-topics/diabetes (Accessed on 21 May 2021).
2. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. *Lancet*. 2016;387(10027):1513–1530. https://doi.org/10.1016/S0140-6736(16)00618-8.
3. Roth GA, Abate D, Abate KH, et al. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet*. 2018;392(10159):1736–1788. https://doi.org/10.1016/S0140-6736(18)32203-7.
4. Sarwar N, Gao P, Seshasai SR, et al. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. *Lancet*. 2010;375(9733):2215–2222. https://doi.org/10.1016/S0140-6736(10)60484-9.
5. Eckel RH, Kahn SE, Ferrannini E, et al. Obesity and type 2 diabetes: what can be unified and what needs to be individualized? *J Clin Endocrinol Metab*. 2011;96(6):1654–1663. https://doi.org/10.1210/jc.2011-0585.
6. Hardy OT, Czech MP, Corvera S. What causes the insulin resistance underlying obesity? *Curr Opin Endocrinol Diabetes Obes*. 2012;19(2):81–87. https://doi.org/10.1097/MED.0b013e3283514e13.
7. World Health Organization. Cardiovascular diseases. WHO Factsheet, 2021. (Accessed on 10 June 2021).
8. Després JP. Body fat distribution and risk of cardiovascular disease: an update. *Circulation*. 2012;126:1301–1313. https://doi.org/10.1161/CIRCULATIONAHA.111.067264.
9. Neeland IJ, Ross R, Després JP, et al. International atherosclerosis society; International chair on Cardiometabolic Risk working group on visceral obesity. visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol*. 2019;7:715–725. https://doi.org/10.1016/S2213-8587(19)30084-1.
10. Bowry A, Lewey J, Dugani SB, et al. The burden of cardiovascular disease in Low- and middle-income countries: Epidemiology and management. *Can J Cardiol*. 2015;31:1151–1159. https://doi.org/10.1016/j.cjca.2015.06.028
11. Piché M, Tchernof A, Després JP. Obesity phenotypes, diabetes, and cardiovascular diseases. Obesity, metabolic syndrome, and cvd compendium. *AHA J*. 2020;126:1477–1500.
12. Grunfeld C, Delaney JA, Wanke C, et al. Preclinical atherosclerosis due to HIV infection: carotid intima-medial thickness measurements from the FRAM study. *AIDS (London, England)*. 2009;23(14):1841–1849.
13. Triant VA, Lee H, Hadigan C, et al. Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. *J Clin Endocrinol Metab*. 2007;92(7):2506–2512.
14. Tien PC, Schneider MF, Cole SR, et al. Antiretroviral therapy exposure and incidence of diabetes mellitus in the women's interagency HIV study. *AIDS (London, England)*. 2007;21(13):1739–1745.
15. Rasmussen LD, Mathiesen ER, Kronborg G, et al. Risk of diabetes mellitus in persons with and without HIV: a danish nationwide population based cohort study. *PloS ONE*. 2012;7(9):e44575. https://doi.org/10.1371/journal.pone.0044575
16. Ledergerber B, Furrer H, Rickenbach M, et al. Factors associated with the incidence of type 2 diabetes mellitus in HIV-infected participants in the Swiss HIV cohort study. *Clin Infect Dis*. 2007;45(1):111–119.

17. Butt AA, McGinnis K, Rodriguez-Barradas MC, et al. HIV infection and the risk of diabetes mellitus. *AIDS*. 2009;23(10):1227–1234.
18. Fahme SA, Bloomfield GS, Peck R. Hypertension in HIV-infected adults: novel pathophysiologic mechanisms. *Hypertension*. 2018;72 (1):44–55.
19. Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous and how do we measure them? *Int J Epidemiol*. 2006;35(1):83–92.
20. Janssen I, Heymsfield SB, Allison DB, et al. Body mass index and waist circumference independently contribute to the prediction of nonabdominal, abdominal subcutaneous, and visceral fat. *Am J Clin Nutr*. 2002;75(4):683–688.
21. Xing Z, Peng Z, Wang X, et al. Waist circumference is associated with major adverse cardiovascular events in male but not female patients with type-2 diabetes mellitus. *Cardiovasc Diabetology*. 2020;19:39. https://doi.org/10.1186/s12933-020-01007-6.
22. Xing Z, Pei J, Huang J, et al. Relationship of obesity to adverse events among patients with mean 10-year history of type 2 diabetes mellitus: results of the ACCORD study. *J Am Heart Assoc*. 2018;7(22): e010512. https://doi.org/10.1161/JAHA.118.010512.
23. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. *Int J Food Sci Nutr*. 2005;56(5):303–307. https://doi.org/10.1080/09637480500195066.
24. The WHO STEPwise approach to noncommunicable disease risk factor surveillance. (2017). Available at: https://www.who.int/ncds/surveillance/steps/STEPS_Manual.pdf (Last accessed on 23/02/2022).
25. SEMDSA Type 2 Diabetes Guidelines Expert Committee. JEMDSA 2017; 22(1)(Supplement 1): S1-S196. (Last accessed on 23/02/2022).
26. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0·5 could be a suitable global boundary value. *Nutr Res Rev*. 2010;23:247–269.
27. Hypertension guideline working group, et al. South African hypertension practice guideline 2014. *Cardiovasc J Afr*. 2014;25:288–294. https://doi.org/10.5830/CVJA-2014-062.
28. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of Chronic Kidney disease. *Kidney Int Suppl*. 2013;3:1–150.
29. Amod A, Berg G. The 2012 SEMDSA guidelines for the management of type 2 diabetes. *J Endocrinol Metab Diabetes S Afr*. 2012;17(2):S4), http://www.cdediabetes.co.za/uploads/images/files/SEMDSA%20 Guideline%20for%20the%20Management%20of%20Type%202%20Diabetes_2012.pdf (accessed 18 October 2019).
30. Khan MAB, Hashim MJ, King JK. Epidemiology of type 2 diabetes - Global Burden of Disease and forecasted trends. *J Epidemiol Glob Health*. 2020;10(1):107–111. https://doi.org/10.2991/jegh.k.191028.001.
31. Tran NTT, Blizzard CL, Luong KN, et al. The importance of waist circumference and body mass index in cross-sectional relationships with risk of cardiovascular disease in Vietnam. *PLoS ONE*. 2018;13:e0198202. https://doi.org/10.1371/journal.pone.0198202.
32. Mutyambizi C, Pavlova M, Chola L, et al. Cost of diabetes mellitus in Africa: A systematic review of existing literature. *Global Health*. 2018;14(1):3. https://doi.org/10.1186/s12992-017-0318-5.
33. Nordström A, Hadrévi J, Olsson T, et al. Higher prevalence of type 2 diabetes in Men than in women Is associated With differences in visceral Fat mass. *J Clin Endocrinol Metab*. 2016;101(10):3740–3746. https://doi.org/10.1210/jc.2016-1915.
34. Kautzky-Willer A, Harreiter J, Pacini G. Sex and gender differences in risk, pathophysiology and complications of type 2 diabetes mellitus. *Endocr Rev*. 2016;37(3):278–316. https://doi.org/10.1210/er.2015-1137.
35. Keetile M, Navaneetham K, Letamo G, et al. Socioeconomic and behavioural determinants of overweight/obesity among adults in Botswana: cross-sectional survey. *BMJ Open*. 2019;9:12. http://doi.org/10.1136/bmjopen-2019-029570.
36. Craig LS, Gage AJ, Thomas AM. Prevalence and predictors of hypertension in Namibia: a national-level cross-sectional study. *PLoS ONE*. 2018;13:e0204344. https://doi.org/10.1371/journal.pone.0204344.
37. Kruger HS. Obesity among women: a complex setting. *South Afr J Clin Nutr*. 2018;31:4–5.
38. Cheteni P, Khamfula Y, Mah G. Exploring food security and household dietary diversity in the Eastern Cape province, South Africa. *Sustainability*. 2020;12:1851. https://doi.org/10.3390/su12051851.
39. Goetjes E, Pavlova M, Hongoro C, et al. Socioeconomic inequalities and obesity in South Africa—A decomposition analysis. *Int J Environ Res Public Health*. 2021;18:9181. https://doi.org/10.3390/ijerph18179181.
40. Tauqeer Z, Gomez G, Stanford FC. Obesity in women: insights for the clinician. *J Womens Health (Larchmt)*. 2018;27:444–457. https://bit.ly/3FfDNpT.
41. Garawi F, Devries K, Thorogood N, et al. Global differences between women and men in the prevalence of obesity: is there an association with gender inequality? *Eur J Clin Nutr*. 2014;68:1101–1106. https://bit.ly/30h2bsn.
42. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in Children and adults during 1980–2013: A systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2014;384:766–781.
43. Kengne AP, Bentham J, Zhou B, et al. Trends in obesity and diabetes across Africa from 1980 to 2014: an analysis of pooled population-based studies. *Int J Epidemiol*. 2017;46:1421–1432. https://doi.org/10.1093/ije/dyx078.
44. WHO. Montevideo roadmap 2018-2030 on NCDs as a sustainable development priority, 2017.
45. Clausen TD, Mathiesen ER, Hansen T, et al. High prevalence of type 2 diabetes and pre-diabetes in adult offspring of women with gestational diabetes mellitus or type 1 diabetes: the role of intrauterine hyperglycemia. *Diabetes Care*. 2008;31:340–346. https://doi.org/10.2337/dc07-1596.
46. Emdin CA, Rahimi K, Neal B, et al. Blood pressure lowering in type 2 diabetes: a systematic review and meta-analysis. *JAMA*. 2015;313 (6):603–615.
47. Prospective Studies Collaboration, Asia Pacific Cohort Studies Collaboration. Sex-specific relevance of diabetes to occlusive vascular and other mortality: a collaborative meta-analysis of individual data from 980 793 adults from 68 prospective studies. *Lancet Diabetes Endocrinol*. 2018;6(7):538–546. https://doi.org/10.1016/S2213-8587(18)30079-2.
48. Kalyani RR, Lazo M, Ouyang P, et al. Sex differences in diabetes and risk of incident coronary artery disease in healthy young and middle-aged adults. *Diabetes Care*. 2014;37(3):830–838. https://doi.org/10.2337/dc13-1755.
49. Zeng Q, He Y, Dong S, et al. Optimal cut-off values of BMI, waist circumference and waist:height ratio for defining obesity in Chinese adults. *Br J Nutr*. 2014;112(10):1735–1744. https://doi.org/10.1017/S0007114514002657.
50. Cai L, Liu A, Zhang Y, et al. Waist-to-height ratio and cardiovascular risk factors among Chinese adults in Beijing. *PLoS One*. 2013;8(7): e69298. https://doi.org/10.1371/journal.pone.0069298.
51. Zhang Y, Gu Y, Wang N, et al. Association between anthropometric indicators of obesity and cardiovascular risk factors among adults in Shanghai, China. *BMC Public Health*. 2019;19:1035. https://doi.org/10.1186/s12889-019-7366-0.
52. Castanheira M, Chor D, Braga JU, et al. Predicting cardiometabolic disturbances from waist-to-height ratio: findings from the Brazilian longitudinal study of adult Health (ELSA-brasil) baseline. *Public Health Nutr*. 2018;21(6):1028–1035. https://doi.org/10.1017/S136898001700338X.
53. Petermann-Rocha F, Ulloa N, Martínez-Sanguinetti MA, et al. ELHOC-Chile Research consortium. Is waist-to-height ratio a better predictor of hypertension and type 2 diabetes than body mass index and waist circumference in the Chilean population? *Nutrition*. 2020;79-80:110932. https://doi.org/10.1016/j.nut.2020.110932.
54. Azeez TA, Esan A, Yemitan AI, et al. Anthropometric indices as predictors of 10-year cardiovascular risk among Sub-Sahara Africans with type 2 diabetes. *J Cardiovasc Med Cardiol*. 2021;8(4):072–078. https://doi.org/10.17352/2455-2976.000174.

55. Mabchour AE, Delisle H, Vilgrain C. Specific cut-off points for waist circumference and waist-to-height ratio as predictors of cardiometabolic risk in black subjects: a cross-sectional study in Benin and Haiti. *Diabetes Metab Syndr Obes*. 2015;8:513–523. https://doi.org/10.2147/DMSO.S88893.
56. Matsha TE, Kengne A-P, Yako YY, et al. Optimal Waist-to-Height ratio values for cardiometabolic risk screening in an ethnically diverse sample of South African urban and rural school boys and girls. *PLoS ONE*. 2013;8:e71133. https://doi.org/10.1371/journal.pone.0071133.
57. Owolabi EO, Goon DT, Adeniyi OV, et al. Prevalence and correlates of metabolic syndrome among adults attending healthcare facilities in Eastern Cape, South Africa. *Open Public Health J*. 2017;10:63–68. https://doi.org/10.2174/1874944501710010148
58. Hayashi T, Boyko EJ, McNeely MJ, et al. Minimum waist and visceral fat values for identifying Japanese Americans at risk for the metabolic syndrome. *Diabetes Care*. 2007;30(1):120–127. https://doi.org/10.2337/dc06-0739.
59. Motala AA, Esterhuizen T, Pirie FJ, et al. The prevalence of metabolic syndrome and determination of the optimal waist circumference cutoff points in a rural South African community. *Diabetes Care*. 2011;34:1032–1037. https://doi.org/10.2337/dc10-1921.
60. Crowther NJ, Norris SA. The current waist circumference cut point used for the diagnosis of metabolic syndrome in sub-Saharan African women is not appropriate. *PLoS ONE*. 2012;7:e48883. https://doi.org/10.1371/journal.pone.0048883.
61. Kalk WJ, Joffe BI, Sumner AE. The waist circumference of risk in black South African men is lower than in men of European ancestry. *Metab Syndr Relat Disord*. 2011;9:491–495. https://doi.org/10.1089/met.2011.0063.
62. Peer N, Steyn K, Levitt N. Differential obesity indices identify the metabolic syndrome in black men and women in Cape Town: the CRIBSA study. *J Public Health (Oxf)*. 2016;38:175–182. https://doi.org/10.1093/pubmed/fdu115.
63. James PA, Oparil S, Carter BL, et al. Evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the eighth joint national Committee (JNC 8). *JAMA*. 2014;311(5):507–520. https://doi.org/10.1001/jama.2013.284427.
64. Thurlow JS, Joshi M, Yan G, et al. Global epidemiology of end-stage kidney disease and disparities in kidney replacement therapy. *Am J Nephrol*. 2021;52(2): 98–107. https://doi.org/10.1159/000514550.
65. Hernandez-Romieu AC, Garg S, Rosenberg ES, et al. Is diabetes prevalence higher among HIV-infected individuals compared with the general population? evidence from MMP and NHANES 2009-2010. *BMJ Open Diabetes Res Care*. 2017;5(1):e000304. https://doi.org/10.1136/bmjdrc-2016-000304.
66. Fiseha T, Belete AG, Dereje H, et al. Hypertension in HIV-infected patients receiving Antiretroviral Therapy in northeast Ethiopia. *Int J Hypertens*. 2019;4103604; https://doi.org/10.1155/2019/4103604.
67. Divala OH, Amberbir A, Ismail Z, et al. The burden of hypertension, diabetes mellitus, and cardiovascular risk factors among adult Malawians in HIV care: consequences for integrated services. *BMC Public Health*. 2016;16(1):1243. https://doi.org/10.1186/s12889-016-3916-x.
68. Ekrikpo UE, Akpan EE, Ekott JU, et al. Prevalence and correlates of traditional risk factors for cardiovascular disease in a Nigerian ART-naive HIV population: a cross-sectional study. *BMJ Open*. 2018;8:e019664. https://doi.org/10.1136/bmjopen-2017-019664.
69. Hanley S, Moodley D, Naidoo M. Obesity in young South African women living with HIV: A cross-sectional analysis of risk factors for cardiovascular disease. *PLoS ONE*. 2021;16(11):e0255652. https://doi.org/10.1371/journal.pone.0255652.
70. Croffut SE, Hamela G, Mofolo I, et al. HIV-positive Malawian women with young children prefer overweight body sizes and link underweight body size with inability to exclusively breastfeed. *Matern Child Nutr*. 2018;14(1):e12446. https://doi.org/10.1111/mcn.12446.
71. Rasmussen LD, May MT, Kronborg G, et al. Time trends for risk of severe age-related diseases in individuals with and without HIV infection in Denmark: a nationwide population-based cohort study. *Lancet HIV*. 2015;2:e288–e298. https://doi.org/10.1016/S2352-3018(15)00077-6.
72. Schouten J, Wit FW, Stolte IG, et al. Cross-sectional comparison of the prevalence of age-associated comorbidities and their risk factors between HIV-infected and uninfected individuals: the AGEhIV cohort study. *Clin Infect Dis*. 2014;59:1787–1797. https://doi.org/10.1093/cid/ciu701.
73. Beraldo RA, Meliscki GC, Silva BR, et al. Comparing the ability of anthropometric indicators in identifying metabolic syndrome in HIV patients. *PLoS ONE*. 2016;11(2):e0149905. https://doi.org/10.1371/journal.pone.0149905.
74. Kagaruki GB, Mayige MT, Ngadaya ES, et al. Magnitude and risk factors of non-communicable diseases among people living with HIV in Tanzania: A cross sectional study from mbeya and Dar es Salaam regions. *BMC Public Health*. 2014;14(1):904. https://doi.org/10.1186/1471-2458-14-904.
75. Dimala CA, Atashili J, Mbuagbaw JC, et al. Prevalence of hypertension in HIV/AIDS patients on highly active Antiretroviral Therapy (HAART) compared with HAART-naive patients at the limbe Regional hospital, Cameroon. *PLoS ONE*. 2016;11(2):e0148100. https://doi.org/10.1371/journal.pone.0148100.
76. Galli L, Salpietro S, Pellicciotta G, et al. Risk of type 2 diabetes among HIV-infected and healthy subjects in Italy. *Eur J Epidemiol*. 2012;27(8):657–665. https://doi.org/10.1007/s10654-012-9707-5.
77. Shiri T, Birungi J, Garrib AV, et al. Patient and health provider costs of integrated HIV, diabetes and hypertension ambulatory health services in low-income settings — an empirical socio-economic cohort study in Tanzania and Uganda. *BMC Med*. 2021;19:230. https://doi.org/10.1186/s12916-021-02094-2.

Received: 28-02-2022 Accepted: 30-05-2022