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Comorbidities of Obesity in a Rural African Population Residing in Limpopo Province, South Africa: A Comparison between General and Central Obesity

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Abstract: Obesity is a growing epidemic that threatens to deplete healthcare resources by increasing the prevalence of diabetes, heart disease, high blood pressure, cancer, and chronic kidney disease. The prevalence of general and central obesity among the rural Black population in Limpopo Province is high; however, few studies have evaluated the association between obesity and its comorbidities among the rural Black population, and, hence, this study aims to determine the relationship between obesity and associated comorbidities. This study was cross-sectional and retrospective in design, using secondary data from the Africa Wits-INDEPTH Partnership for Genomic (AWI-Gen) research phase 1 study. A sample size of 791 participants was collected conveniently. Data were analysed using the Statistical Package for Social Sciences version 27 (SPSS). A chi-square, unpaired Student's *t*-test, bivariate and partial correlation, and multivariate regression analysis were used for analysis. General obesity correlated positively and significantly with LDL-C/HDL-C ratio and hypertension, while central obesity correlated positively and significantly with diabetes, hypertension, chronic kidney disease, LDL-C/HDL-C ratio, and TC/HDL-C ratio. In the present study, obese participants were more likely to be hypertensive, and have an increased LDL-C/HDL-C ratio. Centrally obese participants were more likely to be diabetic, hypertensive, have dyslipidaemia, and exhibit reduced kidney function.

Keywords: LDL-C/HDL-C; TC/HDL-C ratio; hypertension; kidney dysfunction; diabetes

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1. Introduction

Obesity is defined as a physical condition that results from excessive storage of fat in the body [1,2]. It is diagnosed by measuring body mass index (BMI), which is weight in kilograms divided by the square of the height in meters. Individuals with a body mass index (BMI) 30 kg/m^2 and above are considered obese. Obesity can also be determined by a waist circumference (WC) of ≥94 cm and ≥80 cm in men and women, respectively. The global prevalence of obesity has increased by more than double between 1980 and 2014 [3]. The estimated global total prevalence of central obesity was 31.3% in the 1990s (1985–1999) and increased to 48.3% between 2010–2014 [4]. Overweight and obesity are responsible for the deaths of at least 2.8 million individuals and 2.3% of disability-adjusted life years worldwide [5]. It is estimated that more than half of adults in Europe aged 35–65 years are either overweight or obese [6].

Countries in the process of transitioning from underdeveloped to developed status, such as China, Brazil, and South Africa, are particularly affected by an increase in obesity rates across all economic levels and age groups. The prevalence of obesity in South Africa was

reported to be 67.0% [7]. Obesity is more prevalent in women compared to men and higher in rural, as compared to urban, areas [8]. Obesity is a risk factor for high blood pressure, diabetes mellitus, and other cardiovascular disease (CVD)-related morbidities [2,9,10].

The increased cost of treating associated comorbid diseases related to obesity presents a massive challenge to both private and public healthcare systems [11,12]. It has been estimated that the economic impact of obesity in South Africa amounts to approximately ZAR 33 billion each year [13]. This includes obesity-related costs such as loss of productivity, medical spending, and absenteeism [13]. In South Africa, the National Development Plan (NDP) aims to reduce obesity through providing an enabling environment for easy access to healthy food and physical activity opportunities and enabling people to make healthy food choices from a position of knowledge and understanding [14,15]. This strategy is in line with the national strategic plan for the prevention and control of noncommunicable diseases, 2022–2027, which aims to reduce noncommunicable diseases (NCDs) through strengthening the healthcare system [16]. Although strategies to reduce obesity have been put in place, there is limited published evidence about the effectiveness of measures adopted to reduce obesity, especially in rural areas, thus making it difficult to monitor obesity trends.

The burden of obesity-related noncommunicable diseases in South Africa remains persistent, with poor and Black South African women being particularly at risk [17]. Research has highlighted significant links between body weight, sleep duration, and the consumption of sugar-sweetened beverages among rural Black South Africans, particularly in women [17]. In addition, the prevalence of double malnutrition is rising in Sub-Saharan Africa due to rapid epidemiological and nutritional transitions [18]. In this region, most studies on double malnutrition have been conducted at the country and household levels, with individual-level studies mainly focusing on children and women of reproductive age [18]. Interventions aimed at improving nutrition in similar contexts should target individuals across the life course, with a particular focus on women [18].

As the prevalence of obesity increases, so too does the burden of its associated comorbidities. The prevalence of general and central obesity has also been increasing among rural Black populations in Limpopo Province, South Africa, a region characterized by high poverty rates and limited access to healthcare, which may exacerbate the impact of these conditions [19]. However, much of the existing literature focused on urban areas or high-income countries, leaving rural communities underrepresented. Hence, this study aims to quantify the strength of the association between obesity and its comorbidities in this population and to identify potential socioeconomic mediators of this relationship [19]. The results may contribute to public health strategies and policies tailored to these vulnerable communities [19].

2. Materials and Methods

2.1. Study Design

The study used Africa Wits INDEPTH-partnership for Genomic Research (AWI-Gen) phase 1. This population-based cross-sectional study was conducted with 791 participants aged $\geq$ 40 (242 men and 549 women). The sample size was calculated using sample size estimation with correlation coefficient. The minimum sample size for the study was 402 participants. The research took place in the Dikgale, Mamabolo, and Mothiba Health Demographic Surveillance Site (DIMAMO HDSS) area, originally known as Dikgale Health Demographic Surveillance Site (Dikgale HDSS), located in the Capricorn District of Limpopo Province, South Africa. Ethics to conduct the study were granted by the Turfloop Research Ethics Committee (TREC)(TREC/264/2021:PG). The Dikgale Tribal Authority authorized the execution of the study, and each participant provided written informed consent.

2.2. Measurements

Participants' information on age, gender, socioeconomic factors, and obesity risk factors were collected using the AWI-Gen H3Africa Questionnaire. A qualified research nurse with the assistants of trained research assistants collected anthropometric and ultrasound measurements and blood samples. Body weight was measured to the nearest 0.1 kg with the participant wearing light clothes and without shoes using the Omron Measuring Scale manufactured by Omron Healthcare (INC, CHINA). Height was collected using a stadiometer while participants were standing vertically barefoot, and the measurements were rounded to the nearest 0.1 m. Body mass index (kg/m^2) was computed by dividing the measured weight by height squared. The waist circumference was measured using a measuring tape (manufactured by SECA, Hamburg, Germany). Logic ultrasound (GE Healthcare, Chicago, IL, USA) was used to measure subcutaneous and visceral fat. Visceral adipose tissue was measured as the distance, in centimetres (cm), between the peritoneum and spine when there was a clear space between the vertebra and the aorta. Subcutaneous adipose tissue thickness was measured as the depth (cm) from the skin to the linea alba. Visceral fat was measured using a 4C abdominal convex transducer placed longitudinally and subcutaneous fat with a 9 L small parts linear transducer placed transversely; both measurements were made where the xiphoid line crosses the waistline. The Omron blood pressure monitor (manufactured by Omron Healthcare Inc., Shanghai China) was used to measure blood pressure; three readings were taken, and the mean of the last two readings was recorded. A Randox Plus clinical chemistry analyser (UK) was used to analyse biochemical parameters (total cholesterol (TC), triglycerides (TG), high-density-lipoprotein-cholesterol (HDL-C), glucose, insulin, albumin-to-creatinine ratio (ACR), and estimated glomerular filtration (EGF). The measurements of TC, HDL-C, and TG were used for the calculation of low-density lipoprotein-cholesterol (LDL-C) by the Friedewald formula [20], in mmol/L: LDL-C = (TC) − (HDL-C) − (TG/5). The formula was not applicable at TG concentrations greater than 4.5 mmol/L. Details about the methods are reported elsewhere [21].

2.3. Diagnosis

Normal cut-off values for serum lipid were total TG level < 1.7 mmol/L, LDL-C level < 3.0 mmol/L, TC level < 5.00 mmol/L, and HDL-C > 1.0 mmol/L for men and > 1.3 mmol/L for women [22,23]. The optimal cut-off values for lipid ratios were TC/HDL-C < 5.3, TG/HDL-C < 4.70 for men, and < 3.7 for women [24].

Individuals with a history of hypertension or blood pressure of either or blood pressure of ≥140 systolic mmHg or ≥90 diastolic mmHg or both were considered to be hypertensive [25]. Participants with a history of diabetes, fasting blood glucose of ≥7 mmol/L, or random blood glucose of ≥11.1 mmol/L were considered diabetic in the present study [26].

2.4. Statistical Analysis

Data were analysed using Statistical Package for Social Sciences (SPSS) version 27.0. Categorical data were reported as frequencies and percentages, and continuous variables that were normally disturbed were presented as mean ± standard deviation while those that were not normally distributed were presented as median (interquartile range). A comparison of proportions was performed using chi-square, whilst a comparison of means was performed using an unpaired Student's *t*-test. Mann–Whitney test was used to compare variables that were not normally distributed. The association between comorbidities with general obesity and central obesity was analysed using bivariate correlation and partial correlation. In bivariate and partial correlation, the association was between binary variables which were dichotomized in terms of yes/no. To determine the relationship between general obesity and central obesity with their comorbidities, we performed multivariate logistic regression analysis. In this analysis, the comorbidities (diabetes, hypertension, dyslipidaemia, kidney diseases) were the dependent variables, while general or central obesity served as the independent variables. In the model, we adjusted for age, gender (male/female), educational status marital status (married, single divorced, and married),

current smoker (yes/no), and current alcohol consumers (yes/no). A *p*-value of less than 0.05 was considered statistically significant.

3. Results

Table 1 shows the characteristics of the participants. The study consisted of 791 respondents; 549 participants were females and 242 were males. The mean age was 52.47 ± 8.24 with no significant difference in mean age between the two genders. The mean BMI of the total population was 28.01 ± 8.24. BMI was significantly higher in women (30.81 ± 8.05) as compared to men (21.67 ± 4.08, $p < 0.001$). The prevalence of general obesity in the total population was 35.4%, and significantly more women were obese (49.5% vs. 3.3%, $p < 0.001$) as compared to men. In the total population, the mean subcutaneous fat (SAT) was 1.84 ± 1.08. Significantly more women had higher mean SAT as compared to men (2.24 ± 1.038 vs. 0.94 ± 0.50) ($p < 0.001$). The prevalence of central obesity in the total population was 59.9%, and more women were significantly and centrally obese (79.6% vs. 15.3%, $p < 0.001$) as compared to men. The mean waist circumference in the total population was 90.12 ± 16.07, and waist circumference was significantly higher in women (94.36 ± 15.89, $p < 0.001$) as compared to men (80.60 ± 11.83, $p < 0.001$). In the total population, the mean visceral fat (VAT) was 6.56 ± 2.17. Significantly more women had higher mean VAT as compared to men (6.78 ± 2.23 vs. 6.04 ± 1.96) ($p < 0.001$).

Table 1. Characteristics of participants by gender.

Variables	Total Mean ± SD/%(*n*)	Women Mean ± SD/%(*n*)	Men Mean ± SD/%(*n*)	*p* Value
N	791	69.4 (549)	30.6 (242)	
Age(years)	52.47 ± 8.24	52.48 ± 8.06	54.45 ± 8.64	0.952
BMI (kg/m^2)	28.01 ± 8.24	30.81± 8.05	21.67 ± 4.08	<0.001
General obesity (%)	35.4 (280)	49.5 (272)	3.3 (8)	<0.001
Waist circumference (cm)	90.12 ± 16.07	94.36 ± 15.89	80.60 ± 11.83	<0.001
Central obesity (%)	59.9 (474)	79.6 (437)	15.3 (37)	<0.001
Visceral adipose tissue (cm)	6.56 ± 2.17	6.78 ± 2.23	6.04 ± 1.96	<0.001
High VAT(%)	61.3 (485)	82.5 (400)	17.5 (85)	<0.001
Subcutaneous adipose tissue (cm)	1.8 ± 1.07	2.21 ± 1.01	0.9 ± 0.52	<0.001
High SAT (%)	55.0 (482)	96.0 (411)	4.0 (17)	<0.001
Hypertension (%)	28.7 (227)	31.0 (170)	23.6 (57)	0.040
Diabetes (%)	6.3 (50)	6.9 (38)	5.0 (12)	0.344
Kidney disease (%)	12.4 (98)	13.3 (73)	10.3 (25)	0.292
LDL-C/HDL-C	2.26 (3.08–1.53)	2.37 (3.16–1.67)	2.04 (2.80–1.20)	0.362
TC/HDL-C	3.06 (4.40–2.89)	3.71 (4.51–2.89)	3.28 (4.12–2.57)	0.036
TG/HDL-C	0.88 (1.32–0.56)	0.877 (1.34–0.58)	0.88 (1.28–0.55)	0.322
Smoking status (%)	21.3 (148)	9.5 (14)	90.5 (134)	<0.001
Alcohol consumption (%)	33.7 (215)	35.3 (76)	64.7 (139)	<0.001

Table 2 shows the comparison of comorbidities of obesity between obese and nonobese participants. The proportion of hypertension was significantly higher in participants with obesity than in those without obesity (35.4% vs. 25.0%, $p = 0.002$). The proportion of high LDL-C/HDL-C ratio was significantly higher in obese participants than those without obesity (55.1% vs. 32.3%, $p < 0.001$). Moreover, the proportion of high TC/HDL-C ratio was also significantly higher in obesity as compared to nonobesity (14.5% vs. 8.9%, $p = 0.010$).

There was no significant difference in diabetes kidney disease and TG/HDLC ratio between nonobese and obese.

Table 2. Comparison of comorbidities between general obesity and nongeneral obesity.

Characteristics	Nongeneral Obesity %(*n*)	General Obesity %(*n*)	*p*-Value
Diabetes	5.7 (29)	7.5 (21)	0.359
Hypertension	25.0 (128)	35.4 (99)	0.002
High ACR	21.3 (43)	25.7 (28)	0.398
Low eGFR	4.9 (25)	3.2 (9)	0.359
Kidney disease	11.9 (61)	13.2 (37)	0.652
High LDL-C/HDL-C	32.3 (159)	55.1 (145)	<0.001
High TC/HDL-C	8.9 (45)	14.5 (40)	0.022
High TG/HDL-C	1.6 (8)	0.7 (2)	0.508

Table 3. In bivariate correlation, diabetes correlated positively (r = 0.078) and significantly with obesity (p = 0.028). Hypertension correlated positively (r = 0.139) and significantly with obesity (p < 0.001). High LDL-C/HDL-C ratio correlated positively (r = 0.258) and significantly with obesity (p < 0.001). High TC/HDL-C ratio correlated positively (r = 0.100) and significantly with obesity (p = 0.005). There was no association between TG/HDH-C, low eGFR, ACR, and kidney disease with obesity.

Table 3. Bivariate correlation between general obesity and comorbidities.

Variables	Correlation	*p* Value
Diabetes	0.078	0.028
Hypertension	0.139	<0.001
High ACR	0.090	0.113
Low eGFR	−0.001	0.976
Kidney disease	0.068	0.056
High LDL-C/HDL-C	0.258	<0.001
High TC/HDL-C	0.100	0.005
High TG/HDL-C	−0.018	0.617

Table 4. After controlling for age and gender in partial correlation, only the correlation of hypertension, diabetes, and LDL-C/HDL-C with obesity remained significant, whilst that of kidney disease and related functional tests (ACR and low eGFR) was lost. There was no significant correlation between obesity with kidney disease, high TC/HDL-C, and TG/HDL-C.

Table 5 presents multivariate regression between comorbidities and general obesity after adjusting for age, gender, marital status, highest level of education, current smoker, and current alcohol consumption. Obesity was associated with hypertension (aOR = 1.884, 95% CI: 1.479–2.400), LDL/HDL-C (aOR = 2.275, 95% CI: 1.783–2.902), and TC/HDL-C (aOR = 1.976, 95% CI: 1.363–2.864). There was no association between general obesity with diabetes and kidney disease.

Table 6 shows the comparison of comorbidities between central obese and noncentral obese. The proportion of diabetes was significantly higher in participants with central obesity than without obese central obesity (8.6% vs. 2.8%, p < 0.001). The proportion of hypertension was also significantly higher in participants with central obesity than in those without central obesity (34.6% vs. 19.9%, p = 0.001). The proportion of low eGFR was significantly higher in central obesity as compared to noncentral obesity (5.5% vs. 2.5%,

$p = 0.049$), and also the proportion of kidney disease was significantly higher in participants with central obesity as compared to those without central obesity (15.2% vs. 8.2%, $p = 0.004$). The proportion of high LDL-C/HDL-C ratio was significantly higher in central obese participants than those in noncentral obesity (49.8% vs. 27.5%, $p < 0.001$). Moreover, the proportion of high TC/HDL-C ratio was significantly higher in central obesity as compared to noncentral obesity (13.2% vs. 7.3%, $p = 0.010$).

Table 4. Partial correlation between general obesity and comorbidities.

Variables	Correlation	*p* Value
Diabetes	0.092	0.005
Hypertension	0.165	<0.001
High ACR	0.097	0.103
Low eGFR	−0.081	0.172
Kidney disease	0.092	0.119
High LDL-C/HDL-C	0.211	<0.001
High TC/HDL-C	0.090	0.129
High TG/HDL-C	0.039	0.516

Table 5. Multivariate logistic regression between comorbidities and general obesity.

Variables	1st Model (OR 95% CI) (Unadjusted)	*p* Values	Last Model (aOR 95% CI) (Adjusted)	*p* Value
Hypertension	1.878 (1.425;2.475)	<0.001	1.884 (1.479;2.400)	<0.001
Diabetes	1.355 (0.82;2.238)	0.235		
Kidney disease	0.865 (0.597;1.253)	0.443		
High LDL/HDL-C	2.382 (1.825;3.108)	<0.001	2.275 (1.783;2.902)	0.001
High TC/HDL-C	2.241 (1.439;3.490)	<0.001	1.976 (1.363;2.864)	<0.001
High TG/HDL-C	0.323 (0.066;1.576)	0.162		

The comorbidities were the dependent variable, adjusted for age, gender, marital status, the highest level of education, current smoker, and current alcohol consumption.

Table 6. Comparison of comorbidities between central obese and noncentral obese.

Characteristics	Noncentral Obese %(N)	Central Obese %(N)	*p* Value
Diabetes	2.8 (9)	8.6 (41)	0.001
Hypertension	19.9 (63)	34.6 (164)	<0.001
High ACR	18.2 (22)	25.8 (49)	0.129
Low eGFR	2.5 (8)	5.5 (26)	0.049
Kidney disease	8.2 (26)	15.2 (72)	0.004
High LDL-C/HDL-C	27.5 (84)	49.8 (220)	<0.001
High TC/HDL-C	7.3 (23)	13.2 (62)	0.010
High TG/HDL-C	0.9 (3)	1.5 (7)	0.748

In bivariate correlation results (Table 7), diabetes, glucose, hypertension low eGFR, and kidney disease correlated positively and significantly with central obesity. High LDL-C/HDL-C ratio and TC/HDL-C ratio also correlated positively and significantly with central obesity.

Table 7. Bivariate correlation between central obesity and comorbidities.

Variables	Correlation	*p* Value
Diabetes	0.117	0.001
Hypertension	0.160	<0.001
High ACR	0.088	0.120
Low eGFR	0.073	0.042
Kidney disease	0.104	0.003
High LDL-C/HDL-C	0.223	<0.001
High TC/HDL-C	0.094	0.008
High TG/HDL-C	0.024	0.498

In Table 8, for partial correlation, after controlling for age and gender, the association between central obesity with diabetes, hypertension, high LDL-C/HDL-C ratio, and TC/HDL-C ratio remained significant. High TG/HDL-C ratio correlated positively (r = 0.152) and significantly with central obesity (p = 0.010).

Table 8. Partial correlation between central obesity and comorbidities.

Variables	Correlation	*p* Value
Diabetes	0.162	0.006
Hypertension	0.170	0.004
High ACR	0.105	0.077
Low eGFR	−0.015	0.797
Kidney disease	0.097	0.103
High LDL-C/HDL-C	0.228	<0.001
High TC/HDL-C	0.072	0.122
High TG/HDL-C	0.152	0.010

Table 9 presents multivariate regression analysis between comorbidities and general obesity, after adjusting for age, gender, marital status, highest level of education, current smoker, and current alcohol consumption. Central obesity was associated with hypertension (aOR = 2.664, 95% CI: 1.963–3.606), diabetes (aOR = 4.926, 95% CI: 2.386–10.168), LDL/HDL-C (aOR = 3.150, 95% CI: 2.312–4.291), TC/HDL-C (aOR = 1.844, 95% CI: 1.226–2.773), and TG/HDL-C (aOR = 4.220, 95% CI: 1.147–15.532). There was no association between central obesity and kidney disease.

Table 9. Multivariate logistic regression between comorbidities and central obesity.

Variables	1st Model (OR 95% CI) (Unadjusted)	*p* Value	Last Model (OR 95% CI) (Adjusted)	*p* Value
Hypertension	2.795 (1.983;3.940)	<0.001	2.664 (1.968;3.606)	<0.001
Diabetes	4.471 (2.171;9.208)	<0.001	4.926 (2.386;10.168)	<0.001
Kidney disease	1.152 (0.749;1.772)	0.520		
High LDL/HDL-C	3.029 (2.214;4.145)	<0.001	3.150 (2.312;4.291)	<0.001
High TC/HDL-C	2.363 (1.379;4.051)	0.002	1.844 (1.226;2.773)	0.003
High TG/HDL-C	4.707 (1.176;18.851)	0.029	4.220 (1.147;15.532)	0.030

The comorbidities were the dependent variable, adjusted for age, gender, marital status, the highest level of education, current smoker, and current alcohol consumption.

4. Discussion

This study included significantly more women (69.4%) than men (30.4%). This may be because women are more likely to pursue and utilize healthcare services that improve their health [27]. In addition, they tend to comply with healthcare programs and watch out for the wellbeing of others as well as themselves, as demonstrated by previous research [28,29]. Another reason for this difference may be the fact that the majority of the men are either day workers in the study area settings or have official jobs in urban areas and, hence, are not able to partake throughout the day [30].

Increased BMI values, as well as a rise in overweight and obesity among Black South Africans, have been reported in South African studies [31,32]. In the present study, the mean BMI was 28.01 ± 8.05. Corroborating previous findings in Limpopo Province [32,33], excess weight, among other health concerns, exists in Black South Africans. This study reported the mean BMI to be significantly higher in women as compared to men, which is in agreement with the findings by Smith et al. [8]. Women had a significantly higher mean waist circumference than men. Similar findings were reported by Gaziano et al. [34] in their study of cardiometabolic risk in a population of older adults with multiple comorbidities in rural South Africa. Similar results were also observed for subcutaneous and visceral adipose tissue mass. These results suggest that fat deposition is more common among women compared to men. Several reasons explaining high fat deposition among women have been described. Firstly, the increased fat deposition among women may be attributed to hormonal (i.e., oestrogen) imbalances largely affecting women, particularly older women [35]. When oestrogen levels are high enough during childbearing age, it can inhibit lipid synthesis in adipocytes, assisting in the regulation of energy expenditure and food consumption [35]. Its deficiency after menopause, which is more common in older people, enhances fat deposition in adipose tissue, resulting in obesity [35,36]. Secondly, most women available for the present study were older and poor, forcing them to eat high-carbohydrate foods like pap [37].

The proportion of hypertension in the total population was 28.7%, and substantially more women than men were hypertensive. In accordance with the current study, a study conducted in the USA by Cook et al. [38] reported similar findings. The high proportion of hypertension in women may be attributed to hormonal changes. A study by Pimenta [39] reported that the prevalence of hypertension in postmenopausal women was more than twice that in premenopausal women. This could be the reason for the high proportions of hypertension in women in the present study given that women in the present study were of menopausal age. Although the present study found hypertension proportions to be high in women, a study conducted amongst Americans with the aims of investigating racial differences in hypertension found that hypertension was more prevalent in both men and women Black Americans compared to other races [40]. However, differences in geographical setting, lifestyle, and socioeconomic status may have contributed to the inconsistencies between the present study and that of Lackland et al. [40]. In addition, cardiovascular demographic differences exist between Africans and Caucasians, particularly regarding conditions such as hypertension, diabetes mellitus (DM), and coronary artery disease (CAD) [41–43]. These variations in prevalence may significantly influence how obesity impacts individuals within this specific geographical region [44]. Understanding these disparities is crucial for tailoring healthcare strategies and interventions to address the unique cardiovascular risks associated with each population groups.

The proportion of hypertension was significantly higher in obese participants than in the nonobese. Bivariate correlation analysis revealed that hypertension was positively and significantly associated with obesity. The correlation remained unchanged even after controlling for age and gender in a partial correlation analysis. In addition, after controlling for age, gender, and other covariates, multivariate logistic regression confirmed the positive relationship between hypertension and obesity even further. A study by Jiang et al. [10] reported similar findings. In agreement with the present study, Mollan et al. [45] reported

a positive relationship between obesity and hypertension. Moreover, Maimela et al. [31] found obesity to be associated with hypertension.

The proportion of hypertension was significantly higher in central obesity participants than in noncentral obesity. Bivariate correlation analysis revealed that hypertension was positively and significantly associated with central obesity in the current study. The correlation remained unchanged even after controlling for age and gender in a partial correlation analysis. In addition, after age, gender, and other covariates, in multivariate logistic regression, the results confirmed the positive relationship between hypertension and central obesity even further, which were in line with those of Jiang et al. [10].

There are several ways that obesity causes hypertension. Adipose tissue in excess raises cardiac output and blood volume, which places additional strain on artery walls [46,47]. Furthermore, greater insulin levels are linked to obesity and might result in blood pressure elevation and sodium retention [2,46,47]. Vascular alterations and elevated blood vessel resistance may also result from the production of inflammatory cytokines and hormones from adipose tissue [2,10]. Ultimately, obesity frequently throws off the renin–angiotensin–aldosterone system's balance, which raises blood pressure even more [2,10]. In addition, plasma aldosterone has been reported to be high, especially in central obesity participants [48]. An increase in aldosterone can lead to vasocontraction and ultimately lead to hypertension [49,50]. It increases blood pressure in obese people by acting on mineralocorticoid receptors found in various tissues such as the kidney, vasculature, and brain [51,52].

There was no difference in the prevalence of diabetes between nonobesity and obesity participants. Partial correlation analysis revealed that diabetes participants were negatively but not significantly associated with obesity. The correlation remained insignificant even after controlling for age and gender in a partial correlation analysis. Multivariate logistic regression further confirmed that there is no relationship between obesity and diabetes. In contrast with the present study findings, Dai and Jiang [53] reported a positive association between obesity and diabetes. The inconsistencies between the present study and the study by Dai and Jiang may be due to the difference in race, ethnic groups, diet, and different geographical locations. Another reason could be that the other study focused on mitochondrial dysfunction, which is reported to be the key regulator in the pathophysiology of obesity and diabetes. Furthermore, mutations in certain mitochondrial genes have been shown to be the primary causes of these metabolic diseases [54].

Compared to general obesity, the prevalence of diabetes was significantly higher in central obesity in the present study. Bivariate correlation analysis in the current study found a positive and significant association between diabetes and central obesity. Even after controlling for age and gender in partial correlation analysis, the association did not change. Additionally, multivariate logistic regression supported the association between diabetes and central obesity. The findings of the present study are in agreement with those of a study by Jiang et al. [10]. Compared to general obesity, visceral fat was reported to be more metabolically active and to release more inflammatory markers and free fatty acids into the bloodstream [51]. In addition, the proximity of visceral fat to internal organs means that it has a more direct impact on their function, leading to more severe health issues such as obesity [55]. In particular, visceral fat has been associated with fatty pancreas, a condition that has been linked with insulin resistance, an indicator of diabetes [56].

Partial correlation analysis showed that kidney disease was positively and not significantly associated with obesity in the present study. In addition, after controlling for age, gender, and other covariates, multivariate logistic regression confirmed that there was no relationship between kidney disease and obesity. There was no association between high ARC, eGFR, and obesity. Compared to general obesity, in the present study, the proportion of kidney disease was significantly higher in central obese participants than in noncentral obese. In the present study, bivariate correlation and partial correlation showed a positive association between high ACR and central obesity; the findings of the present study are in agreement with Du et al. [46], who reported that central obesity is positively associated with an increased urinary ACR. In addition, a study by Quin et al. [57] reported similar findings:

participants with central obesity had a higher risk of elevated urinary ACR. Bivariate correlation and partial correlation showed a positive association between kidney disease and central obesity. In agreement with the present study, Silvar Junior [58] reported central obesity to be a major cause of abnormal kidney disease. Increased visceral adiposity leads to a cascade of events affecting renal function: enhanced renal sodium reabsorption due to activation of renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system, impaired renal-pressure natriuresis, reducing the ability of kidneys to excrete sodium in response to high blood pressure. Expansion of extracellular fluid volume due to sodium and water retention contributes to hypertension and cardiovascular strain [59]. In addition, elevated blood pressure coupled with renal vasodilation and glomerular hyperfiltration, sympathetic nervous system (SNS) and RAAS activation, inflammation, and metabolic derangements eventually cause renal injury [60].

The proportion of high LDL-C/HDL-C ratio and TC/HDL-C ratio was significantly higher in obese participants than in nonobese in the present study. Bivariate correlation analysis revealed that a high LDL-C/HDL-C ratio correlated positively and significantly with obesity. The correlation remained unchanged even after controlling for age and gender in a partial correlation analysis. The findings of the present study are in agreement with Nadeem et al. [61], who reported increased body weight to be associated with a high LDL-C/HDL-C lipid ratio.

The proportion of high LDL-C/HDL-C ratio and TC/HDL-C ratio was significantly higher in the central obese participants than in the noncentral obese ones. Bivariate correlation analysis revealed that high LDL-C/HDL-C ratio and TC/HDL-C ratio were positively and significantly associated with central obesity in the current study. The correlation remained unchanged even after controlling for age and gender in a partial correlation analysis. Multivariate logistic regression confirmed the association between central obesity and lipoid rations. In agreement with the present study, Rysz et al. [62] reported a positive association between central obesity and LDL-C/HDL-C ratio. Central obesity is significantly associated with higher levels of TC/HDL-C ratios [63,64]. This suggests that waist circumference plays an important role in classifying the risk of lipid deposition in adipose tissue [61].

Obesity has a negative effect on HDL metabolism because it increases chylomicron and very-low-density lipoprotein (VLDL) remnants and impairs lipolysis [65,66]. Increased LDL-C and total cholesterol lipoprotein levels result in increased cholesteryl ester-transfer-protein (CETP) activity, which exchanges HDL cholesterol esters for VLDL and LDL TG [66]. Furthermore, hepatic lipase lipolyses these TC-rich HDL, producing small HDL with a lower affinity for apo A-I, resulting in apo A-I detachment from HDL. This will eventually result in lower HDL-C levels, a decrease in circulating HDL particles, impaired reversed cholesterol transport, and an increase in LDL-C and total cholesterol levels in the blood [66].

Study Limitations

Firstly, this study used a convenient sampling method, so the results cannot be generalized to the entire population of Limpopo Province or the larger community outside of this group. Secondly, causal relationships could not be evaluated because the study was cross-sectional rather than longitudinal. Nevertheless, we believe that the present study provides insight into the association between obesity and its comorbidities among the rural Black population.

5. Conclusions

In the present study, obese participants were more likely to be hypertensive and have increased LDL-C/HDL-C levels; however, this was not associated with diabetes and kidney diseases. Central obese participants were more likely to be diabetic, hypertensive, have chronic kidney disease, and have increased LDL-C/HDL-C, TG/HDL-C, and TC/HDL-C. These findings underscore that central obesity is more closely linked to comorbidities than general obesity. In light of this, healthcare professionals are advised to focus more intently

on individuals with central obesity, as they may need more thorough monitoring and tailored intervention strategies to effectively manage the associated health risks. Additionally, educational programs should highlight the importance of regular check-ups, dietary changes, and physical activity in managing and reducing the risk of these comorbidities. Furthermore, increasing awareness about the specific health risks tied to central obesity can empower individuals to take proactive measures in managing their health, potentially lowering the occurrence of related conditions.

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Data Availability Statement: The data presented in this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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