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Dynamics in the Prevalence of Insulin Resistance between 2005 and 2023 in Type 2 Diabetics in South Kivu in the East of the Democratic Republic of Congo: Cross-Sectional Studies

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Abstract

Aim: Sub-Saharan Africa is undergoing an epidemiological transition responsible for a change in the metabolic profile in favour of insulin resistance. The aim of this study was to assess the dynamics of the prevalence of insulin resistance and associated risk factors in diabetic patients in the Democratic Republic of Congo between 2005 and 2023. **Method:** We measured fasting blood glucose and insulin levels and looked for metabolic syndrome parameters (2009 criteria) in type 2 diabetes patients in 2005-2008 (n = 176) and in 2018-2023 (n = 303). The HOMA model was used to measure insulin sensitivity and islet β-cell secretory function. **Results:** Between 2005 and 2013, the trend was towards an increase in the prevalence of insulin resistance (from 13.1% to 50.8%; $p < 0.0001$), obesity (from 17.0% to 31.7%; $p = 0.0005$) and abdominal obesity (from 32.4% to 49.8%; $p = 0.0002$). Diabetes mellitus without insulin resistance remained more prevalent in rural than in urban areas (60.2% vs. 24.5%; $p < 0.0001$). Finally, the Triglycerides/HDL-C ratio [AUC = 0.513 (0.46 - 0.56); $p = 0.64$] and the metabolic syndrome [AUC = 0.539 (0.49 - 0.58), $p = 0.12$] were not predictive of insulin resistance. **Conclusion:** This present study shows an increase in insulin resistance in Congolese urban areas and a persistence of atypical diabetes mellitus in Congolese

rural areas, confirming the particularity of the pathophysiology of the disease in African areas currently influenced by the epidemiological transition. Further studies using an appropriate methodology are required.

Keywords

Prevalence, Diabetes Mellitus, Insulin Resistance, Metabolic Syndrome, South Kivu

1. Introduction

Insulin resistance is an alteration in the biological response to insulin stimulation in target tissues (liver, skeletal muscle, fatty tissue) [1]. Besides beta-cell dysfunction, insulin resistance is the main pathophysiological mechanism of type 2 diabetes mellitus (T2DM) in European populations [2]. It is also associated with atherogenic dyslipidaemia, essential arterial hypertension and, ultimately, cardiovascular disease [3], which is responsible for more than 18 million deaths worldwide each year [4]. It is therefore important to detect insulin resistance and treat it. In clinical practice, the usual biological and clinical measures are Homa-S, Triglycerides/HDL-C ratio, body mass index (BMI), metabolic syndrome (MetS), waist circumference and waist-to-height ratio (W/H), the two latter as proxi measures of abdominal obesity [1].

However, in sub-Saharan Africa (SSA), the profile of diabetics is not typical. Mapatano M. A *et al.* observed a very low prevalence of obesity (8.1%) among 4967 diabetic patients in the Democratic Republic of Congo, while 26.4% were malnourished [5]. Also in this country, Katchunga B. P *et al.* observed a very low frequency of insulin resistance of 5% among diabetics in the South Kivu region [6]. In South Africa, Goedecke J. M *et al.* noted that black women had less visceral adipose tissue than white women [7]. Thus, the pathophysiology of T2DM appears to be atypical in the majority of cases [8]. High proportion of diabetics in this region is not obese. In addition, SSA women often have significantly more subcutaneous accumulation of abdominal and gluteal fat than perivisceral and hepatic accumulation of adipose tissue. Furthermore, beta-cell function loss appears to be the main driver of these atypical T2DM rather than insulin resistance [8]. Yet these observations date back several years. The general SSA population is currently undergoing a cultural and epidemiological transition, which is bound to have an impact on its metabolic phenotype [9]. It also underlies a marked urban-rural disparity in this region [8].

An update of the data on insulin resistance in an African diabetic population south of the Sahara is therefore advisable.

The aim of this study was to analyze trends in insulin resistance and the validity of insulin resistance markers in a group of African patients with T2DM in South Kivu, East of the Democratic Republic of Congo, between 2005 and 2023.

2. Subjects, Materials and Methods

This cross-sectional, multi-centre study involved hospitals in the city of Bukavu in the east of the Democratic Republic of Congo (DRC) and in the rural area of Kaziba, 75 km from the city of Bukavu.

Data were first collected between 2005 and 2008. The mimimum sample size was calculated using the formula $N \geq p(1 - p)\ Z\infty 2/i^2$ where p = 5% [10], Z∞ = 1.96 and a precision i of ±4%. Thus, N was ≥ 114 diabetic patients. Sampling was by convenience.

The patients concerned were black ancestry patients with T2DM, all native Bantus from sub-Saharan Africa. They were recruited as they attended the hospitals for medical check-up. Informed verbal consent was obtained beforehand. The data were collected anonymously and confidentially. The privacy and personal details of patients were secured according to the Helsinki Declaration.

Between 2018 and 2023, a second round of data collection was carried out in the city of Bukavu and in the rural area of Kaziba. The same methodology and ethical considerations were applied. The protocol for this study was accepted by the Ethics Committee of the Official University of Bukavu (UOB/CEM/013/2023).

2.1. Data Collection

During the first medical visit, for each diabetic patient, demographic parameters (age, sex) and medical history of the disease and other cardiometabolic risk factors were obtained. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured according to ESC/ESH recommendations [10]. Weight and body height were also measured using validated equipment. Waist circumference (WC) at the umbilicus was measured at the end of expiration using a tape measure.

The patient was then sent to the laboratory for fasting measurements of the following biological parameters: glycaemia, insulinemia, total cholesterol (TC), HDL-c, and triglycerides (TG). LDL-C was calculated using Friedewald's formula. Patients on insulin therapy were excluded. A 24-hour washout period of oral glucose-lowering drugs was also applied.

Body mass index was calculated as the ratio of weight (in kilograms) to the square of height (in metres).

The HOMA model, presented as a software program based on the glucose/insulin regulatory loop, was used to measure insulin sensitivity (HOMA S, expressed as % of normal (100%)) and islet β-cell secretory function (HOMA β, also expressed as % of normal (100%)).

2.2. Operational Definitions

The T2DM patient was one with a history of the disease and/or fasting blood glucose > 126 mg/dl on multiple occasions and/or Hb A1c ≥ 6.5% [11].

In the present study, insulin resistance was considered when Homa-S < 50%.

Diabetes with normal insulin sensitivity was considered for Homa-S ≥ 100% [12].

A body mass index between 25.0 and 29.9 Kg/m^2 or ≥ 30 Kg/m^2 defined overweight or obesity, respectively [13].

The presence of a metabolic syndrome was defined according to the 2009 harmonised criteria [14] as the co-occurrence of at least 3 of 5 of the following metabolic abnormalities: fasting glycaemia ≥ 100 mg/dl or previous diagnosis of T2DM, blood pressure ≥ 130/85 mmHg and/or treated hypertension, HDL-cholesterol levels < 50 mg/dl and< 40 mg/dl in women and men, respectively, TG ≥ 150 mg/dl in both sexes, and abdominal circumference ≥ 80 cm and ≥ 94 cm in women and men, respectively.

Other markers of insulin resistance associated with abdominal obesity were: a W/H ratio > 0.5 [15] and a TG/HDL-C ratio > 5.0 [16].

2.3. Statistical Analysis

The distribution of the variables was tested for normality using the Kolmogorov-Smirnov test. As appropriate, data are presented as medians (interquartile range) or frequency (percent).

The non-parametric Kruskal-Wallis test was utilized to compare medians between groups.

The chi-square test was applied to compare difference in categorical variables between groups.

Finally, the predictive value of insulin resistance determined as Homa-S < 50% (gold-standard) for different clinical markers of insulin resistance was studied by constructing an Receiver Operating Characteristic (ROC) curve.

A value of $p \leq 0.05$ defined the threshold of statistical significance. MedCalc® software (version 18.11) was used for statistical analyses.

3. Results

3.1. General Characteristics of Diabetic Patients

Table 1 shows the general characteristics of the population studied. A total of four hundred and seventy-nine (479) T2DM patients were enrolled. The first group included 176 (36.7%) T2DM patients recruited between 2005 and 2008 and the second group included 303 T2DM patients recruited between 2018 and 2023. In the whole group, the median age was 59.0 (53.0 - 68.0) years and the median disease duration was 5.0 (1.0 - 11.0) years. 201 (42.0%) were male. There was no statistically significant difference between the two groups (p = ns).

3.2. Dynamics Changes in T2DM Phenotype between over 18 Years

Table 1 and Figure 1 show the dynamic changes of the T2DM phenotype over time. Between 2005 and 2023, the trend was towards a significant decrease in insulin sensitivity (Homa-S) from 113.0 (71.5 - 159.0)% to 45.3 (23.8 - 102.7)%

Table 1. Type 2 diabetes mellitus patients characteristics in 2005 and 2013.

	All n= 479 (100%)	2005 n= 176 (36.7%)	2023 N = 303 (63.3)	p
Male sex, n (%)	201 (42.0)	65 (36.9)	136 (44.9)	0.08
Median (interquartile range)				
T2DM duration (years)	5.0 (1.0 - 11.0)	4.0 (2.0 - 9.0)	5.0 (0.0 - 12.0)	0.54
Age (years)	59.0 (53.0 - 68.0)	58.0 (52.0 - 66.0)	60.0 (53.0 - 68.0)	0.27
SBP (mmHg)	139.0 (125.0 - 157.0)	140.0 (124.0 - 159.5)	138.0 (125.0 - 156.0)	0.60
DBP (mmHg)	86.0 (78.0 - 96.0)	88.0 (79.0 - 97.0)	84.0 (77.0 - 94.0)	0.006
BMI (Kg/m^2)	25.5 (22.2 - 30.2)	24.5 (21.3 - 28.3)	26.4 (22.7 - 31.1)	0.0003
WC (cm)	92.0 (82.0 - 101.0)	89.5 (79.0 - 96.0)	94.3 (83.0 - 103.0)	<0.0001
W/H	0.57 (0.51 - 0.63)	0.56 (0.50 - 0.61)	0.58 (0.52 - 0.64)	0.002
TC (mg/dl)	185.0 (151.4 - 211.7)	170.5 (142.0 - 201.5)	208.0 (162.1 - 224.0)	<0.0001
HDL-C (mg/dl)	52.0 (41.0 - 73.3)	41.0 (35.0 - 49.0)	61.7 (46.3 - 81.0)	<0.0001
LDL-C (mg/dl)	110.4 (90.0 - 146.7)	104.1 (81.9 - 128.5)	115.0 (96.3 - 152.0)	<0.0001
Non-HDL-C (mg/dl)	127.4 (100.3 - 158.2)	125.5 (103.0 - 154.5)	127.4 (100.3 - 162.1)	0.84
TG (mg/dl)	126.0 (88.4 - 163.5)	103.5 (73.5 - 140.5)	160.0 (154.0 - 166.0)	0.001
TG/HDL-C	2.3 (1.5 - 3.4)	2.6 (1.7 - 3.8)	2.1 (1.52 - 3.16)	0.01
Albumin (g/L)	-	-	3.53 (32.7 - 38.0)	-
Homa S (%)	75.9 (32.2 - 128.1)	113.0 (71.5 - 159.0)	45.3 (23.8 - 102.7)	<0.0001
Homa B (%)	38.1 (18.5 - 68.6)	28.0 (12.6 - 53.2)	46.6 (24.6 - 83.8)	<0.0001
Frequency, n (%)				
T2DM without IR	204 (42.6)	100 (56.8)	104 (34.3)	<0.0001
T2DM with IR	177 (37.0)	23 (13.1)	154 (50.8)	<0.0001
MetS score ≥ 3/5	350 (73.1)	125 (71.0)	225 (74.3)	0.44
Overall obesity	126 (26.3)	30 (17.0)	96 (31.7)	0.0005
Abdominal obesity	208 (43.4)	57 (32.4)	151 (49.8)	0.0002
W/H > 0.5	196 (55.4)	57 (32.4)	139 (78.1)	<0.0001
TG/HDL-C ratio > 5.0	55 (11.6)	21 (11.9)	34 (11.4)	0.85

T2DM = Type 2 diabetes mellitus, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, BMI = Body mass index, WC = Waist circumference, W/H = waist-to-height ratio, TC = Total cholesterol, HDL-C = High density lipoprotein, LDL-C = Low desnsity lipoprotein, TG = Triglyceride, TG/HDL-C = Triglycerides/HDL-C ratio, MetS = Metabolic syndrom.

($p < 0.0001$) and a significant increase in DM with insulin resistance from 13.1% to 50.8 % ($p < 0.0001$).

There was also a significant increase in obesity frequency (from 17.0% to 31.7%; $p = 0.0005$), abdominal obesity (from 32.4% to 49.8%; $p = 0.0002$), the proportion of patients with W/H ratio > 0.6 (from 32.4% to 45.9%; $p = 0.003$) and median total cholesterol (from 170.5 mg/dl to 208.0 mg/dl; $p < 0.0001$), LDL-C (from 104.1 mg/dl to 115.0 mg/dl; $p < 0.0001$) and triglycerides (from

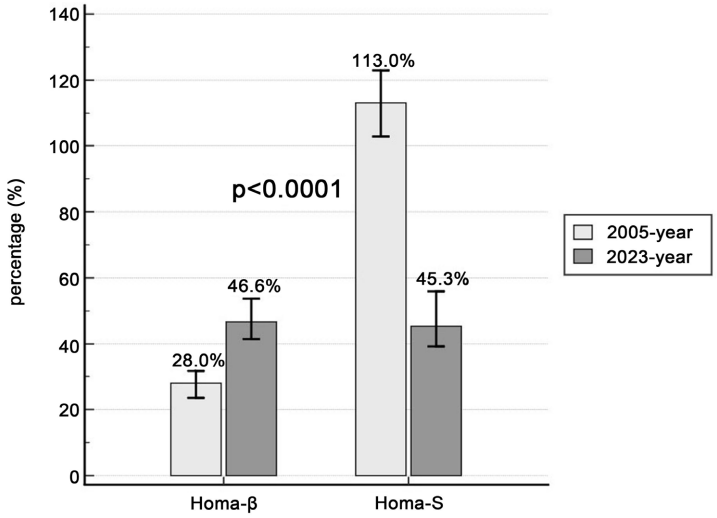


Figure 1. Dynamics changes in T2DM phenotype (Homa-S and Homa-) between 2005 and 2023.

103.5 mg/dl to 160.0 mg/dl; p = 0.001) respectively. However, the increase in metabolic syndrome > 3/5 (p = 0.44) and in Tg/HDL-C ratio > 5.0 (p = 0.85) was not significant.

3.3. Urban/Rural Differences in T2DM Phenotype in 2023

The urban-rural differences in T2DM phenotype as of 2023 are showed in **Table 2** and **Figure 2**. In the rural area, median insulin sensitivity (Homa-S) was 123.7 (53.5 - 200.2)%. 60.2% of patients had DM without insulin resistance compared with 21.7% who had DM with insulin resistance (p < 0.0001). Furthermore, only 6.0% were obese, 19.3% had abdominal obesity, 18.1% had W/H > 0.5 and 57.8% had metabolic syndrome.

In the urban area, median insulin sensitivity (Homa-S) was 35.8 (23.1 - 74.9)%. 61.8% of patients had diabetes mellitus with insulin resistance compared with 24.5% with diabetes mellitus without insulin resistance (p < 0.0001). 41.4% of patients were obese, 61.4%, 56.4% and 80.5% had abdominal obesity, W/H > 0.5 and metabolic syndrome respectively.

Finally, serum albumin was significantly lower in rural than in urban areas: 3.45 (3.13 - 3.72) gr/L vs. 3.58 (3.29 - 3.80) gr/L; p = 0.04.

3.4. Validity of Insulin Resistance Markers

ROC curve results (**Table 3** and **Figure 3**) show that for estimation of insulin resistance as HOMA-S < 50% (reference model), the area under the curve (AUC) was 0.629 (0.579 - 0.679) for BMI [Predictive value > 24. 7 Kg/m^2, sensitivity = 70.6%, specificity = 51.6%], 0.629 (0.58 - 0.67) for WC [Predictive value > 91 cm, sensitivity = 67.2%, specificity = 56.9%], and 0.603 (0.55 - 0.64) for W/H ratio [Predictive value > 0.58, sensitivity = 60.4%, specificity = 59.9%], respectively. The AUC was not significant for the TG/HDL-C ratio [0.513 (0.46 - 0.56); p = 0.64] nor for MetS [0.539 (0.49 - 0.58), p = 0.12].

Table 2. Rural-urban differences in Type 2 diabetes mellitus phenotype as of 2023.

	Rural-2023 n = 83 (27.4)	Urban-2023 n = 220 (72.6)	p
Male sex, n (%)	40 (48.2)	96 (43.6)	0.47
Median (interquartile range)			
T2DM duration (years)	3.0 (0.0 - 8.0)	6.0 (1.0 - 14.0)	<0.0001
Age (years)	63.0 (53.0 - 73.0)	59.0 (53.0 - 66.0)	0.05
SBP (mmHg)	132.0 (120.2 - 157.7)	139.0 (128.0 - 156.0)	0.13
DBP (mmHg)	84.0 (77.0 - 95.7)	84.0 (77.0 - 94.0)	0.64
BMI (Kg/m^2)	21.8 (19.1 - 24.3)	28.5 (24.9 - 32.4)	<0.0001
WC (cm)	80.0 (74.0 - 88.7)	99.0 (91.0 - 105.0)	<0.0001
W/H	0.51 (0.46 - 0.57)	0.61 (0.55 - 0.66)	<0.0001
TC (mg/dl)	208.4 (162.1 - 247.1)	196.8 (160.0 - 210.0)	0.0006
HDL-C (mg/dl)	73.3 (54.0 - 88.8)	61.0 (46.3 - 78.5)	0.0001
LDL-C (mg/dl)	138.9 (96.5 - 161.1)	111.9 (92.6 - 147.7)	0.04
TG (mg/dl)	159.2 (99.5 - 238.9)	135.2 (97.3 - 171.0)	0.008
TG/HDL-C	2.1 (1.3 - 3.3)	2.1 (1.5 - 3.0)	0.99
Serum albumin (g/L)	3.45 (3.13 - 3.72)	3.58 (3.29 - 3.80)	0.04
Homa S (%)	123.7 (53.5 - 200.2)	35.8 (23.1 - 74.9)	<0.0001
Homa B (%)	31.3 (9.3 - 45.1)	58.6 (32.0 - 100.3)	<0.0001
Frequency, n (%)			
T2DM without IR	50 (60.2)	54 (24.5)	<0.0001
T2DM with IR	18 (21.7)	136 (61.8)	<0.0001
MetS score ≥ 3/5	48 (57.8)	177 (80.5)	0.0001
Overall obesity	5 (6.0)	91 (41.4)	<0.0001
Abdominal obesity	16 (19.3)	135 (61.4)	<0.0001
W/H > 0.6	15 (18.1)	124 (56.4)	<0.0001
TG/HDL-C > 5.0	11 (13.3)	23 (10.6)	0.52

T2DM = Type 2 diabetes mellitus, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, BMI = Body mass index, WC = Waist circumference, W/H = waist-to-height ratio, TC = Total cholesterol, HDL-C = High density lipoprotein, LDL-C = Low desnsity lipoprotein, TG = Triglyceride, TG/HDL-C = Triglycerides/HDL-C ratio, MetS = Metabolic syndrom.

4. Discussion

In SAA, studies of the trend in insulin resistance between two periods are rare [17]. This study is the first to have analyzed the dynamics of change over time in insulin resistance and other components of the cardiometabolic phenotype among T2DM patients in the Democratic Republic of Congo.

This analysis produced three major findings.

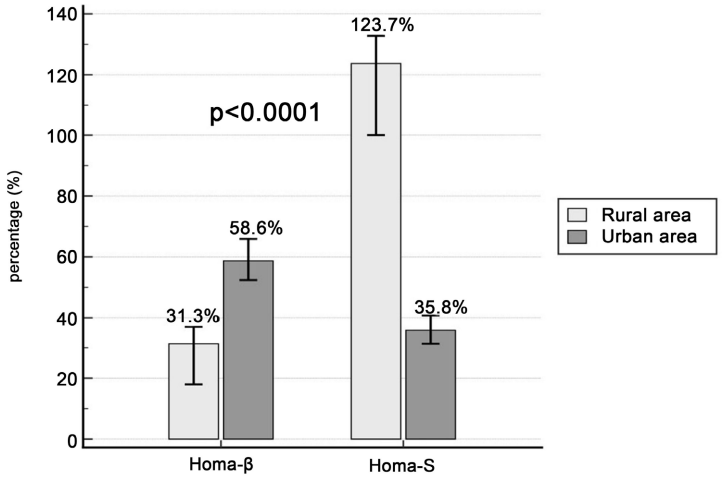
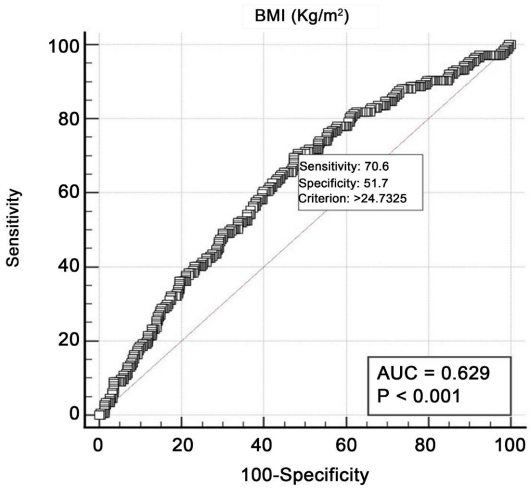


Figure 2. Urban/rural differences in T2DM phenotype (Homa-S and Homa-) in 2023.

Table 3. Receiver Operating Characteristic (ROC) curves of insulin resistance markers.

Predictive value	AUC (99% CI) p-value	Sensitivity (%)	Specificity (%)
BMC > 24.7	0.629 (0.579 - 0.679) p < 0.0001	70.6	51.6
WC > 91 cm	0.629 (0.58 - 0.67) p < 0.0001	67.2	56.9
W/H > 0.58	0.603 (0.55 - 0.64) p < 0.0001	60.4	59.9
MetS score ≥ 3/5	0.539 (0.49 - 0.58) p = 0.12	79.1	30.5
TG/HDL-C < 2.90	0.513 (0.46 - 0.56) p = 0.64	71.0	37.1

AUC = the area under the curve.



(a)

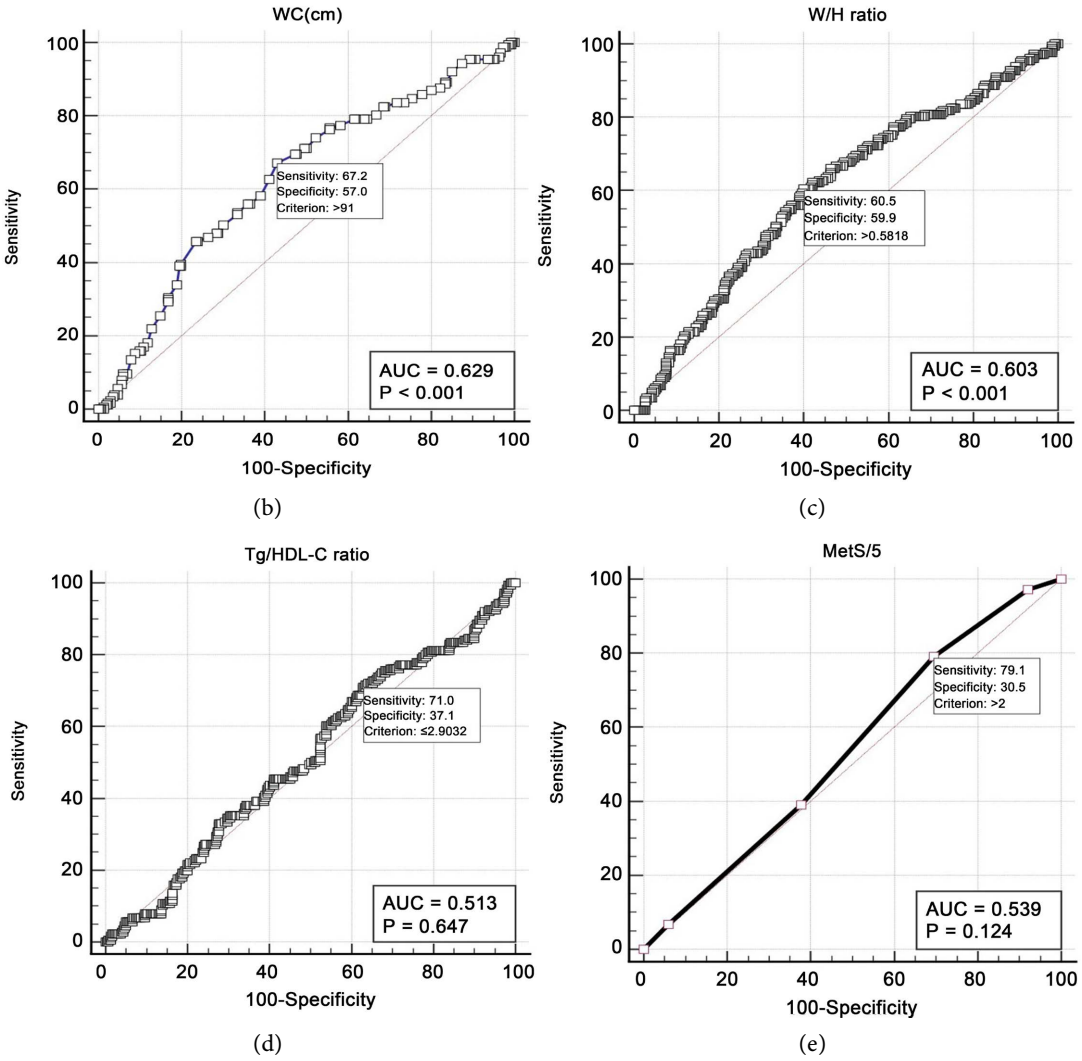


Figure 3. ROC curve for body mass index (a), waist circumference (b), waist circumference to height ratio(c), triglyceride to HDL-C ratio (d) and metabolic syndrome score (e) respectively to predict insulin resistance defined by Homa-S < 50%.

The first is the trend towards a significant increase in the prevalence of T2DM with insulin resistance (the common form of T2DM worldwide) between 2005 and 2023, from 13.1% to 50.8%, alongside a significant decrease in the prevalence of atypical T2DM (*i.e.* without insulin resistance), from 56.8% to 34.3%. These changes were accompanied by an increase in overall obesity and in abdominal obesity. These results corroborate recent findings in the field. Thus, Katchunga B.P *et al.* reported a significant increase in both overall obesity and abdominal obesity in the general population of the East of the DRC, from 7.9% to 9.8% and from 12.6% to 14.6%, respectively [18]. Similarly, Muyer M. T at al noted an increase in the prevalence of diabetes mellitus from 4.0% in 2012 [19] to 5.3% in 2019 [20] in rural areas in the Western DRC. The literature reports that populations in developing countries such as the DRC are under the influence of psycho-social, economic and cultural factors (globalization of Western

culture, rapid urbanisation, ageing, poverty, stress, etc.) which lead them to adopting unhealthy lifestyles (poor diet, sedentary lifestyle) responsible for the increase in intermediate cardiometabolic risk factors (hypertension, T2DM, obesity, dyslipidemia) [21] associated with insulin resistance and compensatory hyperinsulinemia [3].

The second observation is the urban-rural difference in T2DM phenotype as of 2023. Insulin resistance, overall obesity and abdominal obesity were significantly more prevalent in urban than in rural areas. Such urban-rural difference were reported previously [8]. It is most likely the result of more exposure to unhealthy lifestyles (poor diet, sedentarity, mental stress) in urban areas [8] [22]. The high prevalence of atypical T2DM in the rural area studied could be ascribed, at least partly, to higher prevalence of food insecurity and/or undernutrition, as evidenced by a significantly lower serum albumin level, and a significantly lower prevalence of overall obesity and abdominal obesity in this environment. These results corroborate those of other authors [8] [22]. It is also possible that lower socio-economic level combined with higher incidence of communicable diseases may have contributed to some phenotypic differences. These determinants were not investigated in this study, but have been identified in other studies from the same region. Overall, this study confirms the pathophysiology of atypical African T2DM, marked by short-lived hyperinsulinaemia followed by early exhaustion of insulin secretion [8]. The extra-burden of insulin resistance can be superimposed once overall and abdominal obesity develop, usually as a result of individuals adopting unhealthy lifestyles.

This study shows that the area under the curve for the TG/HDL-C ratio and the metabolic syndrome score in predicting insulin resistance were not statistically significant as explanatory variables. These results corroborate those of other studies. Indeed, Katchunga B.P *et al.* showed that the metabolic syndrome was weakly predictive of insulin resistance in diabetics from this region and that arterial hypertension was linked to determinants other than insulin resistance [6]. Saad *et al.* noted that the association between hypertension and insulin resistance was limited to population of European ancestry, whereas it was absent among Pima Indians, who have a very high prevalence of T2DM and obesity, and among black subjects [23]. Finally, the literature reports a naturally high frequency of hypotriglyceridaemia and hyperHDL-cholesterolaemia among blacks, the cause of which is unknown but likely related to ethnic differences in TG-rich lipoproteins metabolism [24].

Finally, the results of this study must be interpreted in the light of their limitations. The first limitation is the cross-sectional nature of the various studies, which makes it impossible to establish a causal link between exposure to risk factors and the incidence of insulin resistance. Nor does this methodology make it possible to estimate the incidence of diabetes mellitus in the region. The second limitation is that various determinants of atypical African diabetes mellitus were not investigated (HHV8, HIV, hepatitis C virus, ferritinemia, inflam-

matory markers, etc.). Finally, the sample size was relatively small, thus reducing the statistical power of this study.

5. Conclusion

This study shows a trend towards increased insulin resistance frequency in urban areas against a background of persistent atypical type 2 diabetes mellitus in rural areas of South Kivu Province, confirming the particularity of the pathophysiology of the disease in an African environment currently influenced by the epidemiological transition. The study also shows that the metabolic syndrome and the TG/HDL-C ratio were not predictive of insulin resistance in this region. Further studies using a suitable methodology are required.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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