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Use of the biomarker score in determining the risk of heart failure in diabetics in Goma, North Kivu in the Democractic Republic of the Congo



ARTICLE INFO	A B S T R A C T					
Handling editor: D Levy	- Background: The use of biomarkers, such as N-terminal pro-brain natriuretic peptide (NTpBNP), high-sensitivity					
<i>Keywords</i> Using biomarker score Risk of heart failure Asymptomatic diabetic	— C-reactive protein (hs-CRP) and high-sensitivity troponin (hs-TnI) is an alternative approach to detect the risk of heart failure (HF), but data on this approach are fragmentary in sub-Saharan Africa. The objective of this study is to determine the correlation between the risk of heart failure and the score of biomarkers in the population of asymptomatic diabetics in the city of GOMA.					
	<i>Methods:</i> Asymptomatic diabetics in the city of Goma were cross-sectionally recruited at the Center of the Association of Diabetics in Congo (ADIC) in Goma, DRC during the period from February 5 to 19, 2023. The risk of insufficiency heart rate at 5 years was determined using pulse pressure. The biomarker score was calculated using NTproBNP, hs-CRP, hs-troponin and left ventricular hypertrophy (LVH). The association between the risk of heart failure and the biomarker score was evaluated using the logistic regression test at the threshold of $p < 0.05$. <i>Results:</i> Of a total of 408 diabetic patients examined, 29.9% had presented a risk of heart failure. The risk of heart failure was higher in patients with a high biomarker score (57.7%), in patients with type 1 diabetes (60%) and in patients with type 2 diabetes (57.1%). Independent risk of biomarker score on occurrence of heart failure. The risk of heart failure was multiplied by 2 if the biomarker score was intermediate (OR: 2.19, 95% CI: 1.11–4.34) and by 5 if the biomarker score is associated with the risk of heart failure in our study via the increase in the score elements as reported in European studies.					

1. Introduction

Non-communicable diseases (NCDs) are a major public health problem worldwide due to the socio-economic burden they cause. Each year, more than 40 million people die worldwide and 70% of these deaths are attributed to NCDs such as diabetes, hypertension and obesity [1].

Due to the increasing prevalence of diabetes worldwide, an increase in the incidence of premature death attributable to both diabetes and its complications is to be expected [2,3]. Cardiovascular (CV) complications related to diabetes mellitus include stroke, diabetic retinopathy, heart failure (HF), coronary artery disease, and peripheral artery disease [3,4].

The prevalence of HF in patients with diabetes mellitus is twice as high as in those without [5,6]. The Reykjavík study showed an overall prevalence of diabetes mellitus and HF of 0.5% in men and 0.4% in women [7]. Therefore, the odds ratio (OR) for the association between diabetes and CI is 2.8 (95% CI = 2.2–3.6) [7]. The prevalence of diabetes, reported in a few small studies in the Democratic Republic of Congo (DRC), varies between 3.5 and 14%. The synergistic burdens of diabetes mellitus and heart failure are well elucidated in the literature by projections the International Diabetes Federation (IDF) predicts an

increase in the prevalence of diabetic patients to more than 500 million in 2040 and especially in populations low income. Mortality linked to heart failure is around 26% while that linked to HF in diabetic patients is double that of diabetic patients without HF around 48% [4-7].

Standard ultrasound is not affordable for most patients in low- and middle-income countries, so biomarker scoring may be an alternative screening process for early detection of HF and associated risks [8]. The biomarker score offers an option to identify the risk of HF in asymptomatic diabetics. The use of biomarkers, such as N-terminal pro-brain natriuretic peptide (NTpBNP), high-sensitivity C-reactive protein (hs-CRP), and high-sensitivity troponin (hs-TnI) is an alternative approach to detect subclinical HF. Previous studies have shown the impact of biomarkers in determining the risk of HF in diabetics [9–12]. Thus learned societies, in particular the American Diabetes Association (ADA) and the European Society of Cardiology (ESC), suggest measuring NTpBNP, hs-CRP and hs-TnT in all asymptomatic diabetics to determine the need for cardiac imaging and prevent heart failure [13,14].

In the Democratic Republic of Congo, studies on biomarkers in determining the risk of FH are rare, despite scientific recommendations. Thus, this study aims to determine the risk of heart failure using the biomarker score among asymptomatic diabetics in Goma.

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2. Patients and methods

2.1. Study population and design

The study was carried out at the Center of the Association of Diabetics in Congo (ADIC) in Goma, DRC. This site was selected on the basis of a reasoned choice based on the age of the center and the number of diabetic patients seen in one week. This was a cross-sectional and analytical study including all known asymptomatic diabetics in the city of Goma during the period from February 5 to 19, 2023.

The study population consisted of all diabetics who consulted the ADIC Center and those from other hospitals during the study period; after raising awareness in their WhatsApp groups and on the radio.

2.2. Sample size

The sample size was calculated from Fisher's formula: $n \ge$ where n = Sample size, z = 1.96 (confidence coefficient), p = previous prevalence, d = 0.05 (margin of error or range of imprecision reflecting the desired degree of absolute precision).

The probability of the risk of IC in diabetics being not yet elucidated in our country, we prefer to take 50% which is the median where the phenomenon is better distributed. So p = 0.5 was the prior prevalence used in all studies to calculate the sample size. So the calculated sample size is $n \ge 2 = 384$. By incorporating the 10% of non-respondents, we obtain 422 diabetics to be included. During the data collection period in this Center, 408 diabetics met the inclusion criteria.

The selection was made on free informed consent and recorded in writing.

The inclusion criterion was any diabetic with controlled diabetes mellitus who consented to participate in the study. The criteria for noninclusion included diabetic complications including heart disease undergoing treatment, diabetics on dialysis, with serious complications related to diabetes mellitus.

2.3. Data collection and procedure

Data collection was done using a form pre-established by our research group. The parameters of interest were retained and collected by way of questioning for the ethnic group, sex, age and by way of clinical and paraclinical examinations for the anthropometric, biochemical and hemodynamic parameters measured by the equipment indicated in following paragraphs.

2.4. Anthropometric data

Anthropometric data were measured for weight and height using the scale coupled with the Health O meter® brand height rod, model 500KL, SN 5000155271, DATE CODE: 3718, Made in China. A millimeter tape measure was used to measure waist circumference (TT) and hip circumference (TH). Systolic (PAS) and diastolic (PAD) arterial pressures (BP) were measured using an OMRON model M2 Basic electronic blood pressure monitor (HEM-7120-E).

2.5. Electrocardiogram data

The electrocardiogram (ECG) was recorded by a Comen brand device, Model: CM 1200B, SN 92190522018B, manufactured on May 22, 2019, connected to the electric generator, after explaining the technique to the patient. The electrocardiograms were interpreted by a single cardiologist for better and uniform results.

2.6. Biological data

Biological data included blood data. The venous blood was taken at the level of the fold of the elbow on dry tubes and EDTA tubes for the various analyses, 5ml of venous blood taken was put on an EDTA tube for the analyzes of glycated hemoglobin, BNP, hs-Troponine and hs-CRP. The dry tubes were used for analyzes such as NT-proBNP, Creatinine, Total cholesterol, Cholesterol-HDL, Triglycerides and LDLcholesterol. The tubes for biochemical analyzes (total-cholesterol, HDL, LDL and, triglycerides) were made on a spectrophotometer brand RAYTO 9200 Semi-auto chemistry Analyzer, SN: 602321157 IE (Rayto, Guangming in China). For glycemia was carried out after ring finger disinfection with a brand glucometer 2019TRUE METRIX®Meter, SE-RIAL:T07123273,LOT:KX0747,EXP DATE:2025-02-06. The tubes for immunoserological analyzes (BNP, NT-proBNP, hs-CRP and hs-Troponine) were made on the Biotime type machine, BioT-YG-IFIA, Analyzer, REFBT0104, SN: BY IZ220101002E (Biotime, Xiamen in China).

The dosage of glycated hemoglobin is carried out on whole blood collected on EDTA K2 anticoagulant by nephelometry method on Genrui PA120 Fully-auto Specific Protein Analyzer.SN: 1141030201223, REF 31000003. The results were interpreted according to the threshold of reference values containing reagents on each kit as follows: blood sugar had a reference value of 70–110mg/dL, total cholesterol <200mg/dL, HDL-c >55mg/dL for men and >65mg/dL for women, LDL-c <100mg/dL, Triglycerides <150mg/dL. Regarding the immunoserology results: NT-proBNP <125pg/ml; BNP \leq 100pg/ml, hs-CRP: 0–1mg/L, hs-CTnI: 0.0–0.3ng/ml.

In order to guarantee the accuracy of the results, the CIMAK laboratory carried out a commercial internal quality control (IQC) (freezedried serum to be reconstituted). These checks were carried out and validated each morning, and adapted to regulatory requirements. ELITROL I (normal references) and ELITROL II (pathological references) control sera were used. CQI results were interpreted taking into account Westgard rules and Levey-Jennings charts. Appropriate corrective measures were taken whenever the values fell outside the defined limits. Depending on the case, these measurements concerned the IQC serum and/or the automaton and/or the reagents and/or the calibration.

The following definitions were used in this work.

2.7. Operational definitions

Hypertension was defined, for some studies, by blood pressure (BP) taken in the office, including systolic \geq 140 mmHg and/or diastolic \geq 90 mmHg and/or the presence of a personal history of hypertension.

Diabetes mellitus was defined by the following criteria: a fasting blood glucose level \geq 126 mg/dl (7.0 mmol/l) and/or a personal history of known diabetes mellitus and/or a glycated hemoglobin level \geq 6.5% (48 mmol/mol). The pathological value of high sensitivity troponin T was a level \geq 6 ng/l [9]. The pathological value of NT-proBNP will be a level \geq 125pg/ml and the pathological value of B-type natriuretic peptide (BNP) > 50 pg/ml [9]. The pathological value of high sensitivity CRP was a level \geq 3 mg/l [9] Dyslipidemia was defined as an HDL cholesterol level <1.03 mmol/L for men or <1.04 mmol/L for women. women, LDL cholesterol level \geq 3.38 mmol/L, total cholesterol level \geq 5.17 mmol/L and/or triglyceride level \geq 1.69 mmol/[15].Left ventricular hypertrophy was defined with the Peguero index \geq 28mm in men and \geq 23mm in women [16]. Biomarker Risk Score: 1 Point per elevated biomarker (troponin T, CRP and HVG and Maximum Score = 4.

- Very Low risk if Biomarker score=0
- Low risk if Biomarker score=1
- Intermediate risk if Biomarker score=2
- High risk if Biomarker score=3–4 [9].

High risk of heart failure in diabetics was defined with pulse pressure \geq 65 mmHg [9].

2.8. Statistical analyzes

After validity, we exported the database to SPSS for Windows software version 24.

Qualitative data were represented in absolute and relative frequency (%) and quantitative data as means \pm standard deviation (SD) (if normal distribution) or median & interquartile range (if asymmetric distribution). The Mantel-Haenzel Chi-square test was used to compare the proportions with application of Ficher's exact test and Yates' corrected test, if necessary. Student's t-test was used to respectively compare the means of two or more groups with normal distributions. Man Whitney's U test was applied in case of asymmetric distribution. The normal distribution of each variable was assessed by the Kolmogorov-Smirnov test. Logistic regression was used to assess the association between risk of heart failure and biomarker score, with calculation of OR with confidence intervals to estimate the degree of association. p < 0.05 was the threshold for statistical significance.

2.9. Ethical considerations

The protocol was submitted for approval by the Medical Ethics Committee of the University of Goma at No. UNIGOM/CEM/09/2022.

3. Results

Fig. 1 illustrates the flow of participants in the present study.

The sociodemographic characteristics of the patients are summarized in Table 1. It shows that the mean age of the patients was 53.9 ± 14.5 years, this age was more significantly higher in patients with DS type 2 (p < 0.001). The majority of patients were female (58.6%, sex ratio of 10 M/14F), the majority of patients had no occupation (45.3%), married marital status (66.4%) and secondary school level (38.2%). The majority of patients had a medium and low socioeconomic level respectively in the proportions of 42.6% and 34.3% (Tableau 1).

This table illustrates the clinical and biological characteristics of the patients. It shows that the majority of patients had abdominal obesity (67.4%), hypertension (43.6%) and consumed more alcohol (42.2%). The frequency of abdominal obesity, hypertension, overweight and obesity were significantly higher in patients with DS type 2 (p < 0.05). Regarding biological parameters, no statistically significant difference was found between patients with type 1 diabetes mellitus and those with type 2 diabetes, except for the frequency of BNP \geq 100pg/mL which was higher. in patients with type 2 diabetes mellitus (p = 0.038). This table

Table 1

Sociodemographic and biological characteristics of the study population.

Variables	All patients n = 408 (%)	DM T1 n = 120 (%)	DM T2 n = 288 (%)	р
Age (years)	53.9 ± 14.5	$\textbf{37.8} \pm \textbf{10.8}$	60.6 ± 9.7	< 0.001
Sex				0.483
Male	169(41.4)	49(40.8)	120(41.7)	
Feminine	239(58.6)	71(59.2)	168(58.3)	
Occupation				0.248
Unemployed	185(45.3)	49(40.8)	136(47.2)	
Formal	91(22.3)	25(20.8)	66(22.9)	
Informal	132(32.4)	46(38.3)	86(29.9)	
Civil statute				< 0.001
Married	271(66.4)	76(63.3)	195(67.7)	
Bachelor	35(8.6)	31(25.8)	4(1.4)	
Divorce	16(3.9)	7(5.8)	9(3.1)	
Widower	86(21.1)	6(5.0)	80(27.8)	
Level of studies				< 0.001
Primary	146(35.8)	28(23.3)	118(41.0)	
Secondary	156(38.2)	45(37.5)	111(38.5)	
University	106(26.0)	47(39.2)	59(20.5)	
Socioeconomic				0.476
level				
Low	140(34.3)	41(34.2)	99(34.4)	
Middle	174(42.6)	47(39.2)	127(44.1)	
High	94(23.0)	32(26.7)	62(21.5)	
Fasting glucose	172.0	176.0	172.0	0.684
mg/dL	(163.0–179.5)	(137.5–211.0)	(162.0–179.0)	
HbA1C (%)	9.1 (8.8–9.4)	9.3 (8.8–10.0)	9.0 (8.7–9.3)	0.136
NT-ProBNP	40.0	37.2 (32.7-	41.3	0.223
(pg/mL)	(37.3-42.0)	10.7)	(37.7–44.0)	
BNP (pg/mL)	44.6	44.1	45.6	0.184
	(41.9-48.9)	(37.9–50.4)	(42.0–50.0)	
hs-CRP (mg/L)	1.6 (1.3–1.9)	1.3 (1.0–2.4)	1.7 (1.4–2.0)	0.546
LDLc (mg/dL)	112.7	104.9	115.9	0.346
	(105.7–119.7)	(94.6–120.2)	(106.9–123.3)	
Triglyceride	96.1	85.2	101.3	0.414
(mg/dL)	(89.5–103.6)	(71.4–96.1)	(92.5–107.4)	
HDLc (mg/dL)	50.4	49.6	51.7	0.063
	(49.5–52.6)	(47.9–53.5)	(49.6–53.0)	
TC (mg/dL)	186.1	179.9	186.2	0.066
	(182.5–194.7)	(167.8–200.9)	(182.8–197.5)	

Abbreviations: DM: diabetes mellitus,DMT1:diabetes mellitus type1, DMT2: diabetes mellitus type 2,HbA1c: Glycosylated hemoglobin, eGFR: Estimated glomerular filtration rate; NT-ProBNP: N-Terminal pro-brain natriuretic peptide, BNP: B-type natriuretic peptide, Hs CRP: high sensitivity c-reactive protein, LDLc: Low density lipoproteins cholesterol, HDLc: Hight density lipoproteins cholesterol, TC:Total cholesterol.



Fig. 1. Participant flowchart.

Table 2

Clii	iical	characteristics	of	the	study	population.
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Variables	All patients n = 408 (%)	DM T1 n = 120 (%)	DM T2 n = 288 (%)	р
Abdominal obesity	275(67.4)	60(50.0)	215(74.7)	< 0.001
hypertension	178(43.6)	23(19.2)	155(53.8)	< 0.001
Alcohol	172(42.2)	48(40.0)	124(43.1)	0.324
Overweight	142(34.8)	34(28.3)	108(37.5)	0.048
Obesity	127(31.1)	27(22.5)	100(34.7)	0.009
Tobacco	30(7.4)	9(7.5)	21(7.3)	0.544
$HbA1C \ge 7\%$	297(87.4)	91(90.1)	206(86.2)	0.210
NT-ProBNP ≥125	35(8.6)	7(5.8)	28(9.7)	0.138
pg/mL	(0(15.0)	10(10.0)	50(15.4)	
BNP≥100pgmL	62(15.2)	12(10.0)	50(17.4)	0.038
hc-CRP>1mgmL	270(66.2)	73(60.8)	197(68.4)	0.088
CRP≥5mg/L	83(20.3)	24(20.0)	59(20.5)	0.514
LDLc >135 mg/dL	121(29.7)	34(28.3)	87(30.2)	0.400
TG > 150 mg/dL	105(25.7)	30(25.0)	75(26.0)	0.465
HDL-c<40F and	127(31.1)	40(33.3)	87(30.2)	0.306
<50H				
TC > 185 mg/dL	213(52.2)	60(50.0)	153(53.1)	0.320
LVH	113(27.7)	30(25.0)	83(28.8)	0.255
Biomarker score				0.007
Very low	76(18.6)	30(25.0)	46(16.0)	
Low	213(52.2)	63(52.5)	150(52.1)	
Intermediate	93(22.8)	22(18.3)	71(24.7)	
High	26(6.4)	5(4.2)	21(7.3)	

Abbreviation: NT-ProBNP: N-Terminal pro-brain natriuretic peptide, BNP: Btype natriuretic peptide, Hs CRP: high sensitivity c-reactive protein, LDLc: Low density lipoproteins cholesterol, HDLc: Hight density lipoproteins cholesterol, TG: Triglycerides, CT:Total cholesterol.

also shows that 22.8% and 6.4% of patients had an intermediate and high biomarker score (Table 2).

3.1. Heart failure risk frequency at 5 years

Of a total of 408 diabetic patients examined, 122 presented a risk of heart failure, a frequency of 29.98%.

The comparison of patients with an Hb1AC level of less than 7% with those with an Hb1AC level greater than or equal to 7% had shown that despite the low Hb1AC level, these patients had presented a high frequency of risk of heart failure (44.2% vs 27.9% in those with Hb1AC \geq 7%).

3.2. Risk of heart failure according to biomarker score

The risk of heart failure was higher in patients with a high biomarker score in the whole group (57.7%), in patients with type 1 diabetes (60%)

and in patients with type 2 diabetes (57.1%) (Fig. 2).

3.3. Independent risk of biomarker score on occurrence of heart failure

In all patients, the risk of heart failure is multiplied by 2 if the biomarker score is intermediate (OR: 2.19 95% CI: 1.11–4.34) and by 5 if the biomarker score is high (OR: 4.73, 95% CI: 1.84–6.20). for type 1 and type 2 diabetic patients, this risk was respectively 3 (OR: 2.50, 95% CI: 1.28–4.01) and 3 (OR: 2.82, 95 M CI: 1.85–3.92) in the event of a biomarker score of intermediate and by 4 (OR: 4.35, 95% CI: 2.99–6.33) and 5 (OR: 4.50, 95% CI: 2.87–7.19) if the biomarker score is high (Table 3).

4. Discussion

This study aimed to determine the correlation between the risk of heart failure and the biomarker score in the population of asymptomatic diabetics in the city of GOMA. The salient results of this study showed that the risk of heart failure is multiplied by 2 if the biomarker score is intermediate and by 5 if the biomarker score is high. For type 1 and type 2 diabetic patients, this risk was respectively 3 in the event of a biomarker score of intermediate and by 4 and 5 if the biomarker score is high.

In the asymptomatic diabetics retained in this study, the biomarker score constituted by neurohormonal stress, systemic inflammation and left ventricular hypertrophy (LVH), are independently associated with a higher risk of heart failure. Moreover, from the score of these biomarkers, it was demonstrated a significant and independent association in predicting the risk of HF. Diabetics with biomarker scores equal to 0 and 1 had no risk or had a very low risk of HF compared to those with a biomarker score greater than or equal to 2. Thus, the risk of HF was considerably higher in subjects with high biomarker scores (3-4). In terms of measurement of association, these results report that diabetics with a biomarker score equal to 2 had a risk of heart failure multiplied by 3, while those with a biomarker score between 3 and 4 had a risk of heart failure multiplied. by 5. Similar results were already reported by Pandy in 2021 who found an association between the biomarker score at 2 and 3-4, multiplying the risk of heart failure within 5 years by 2 and by 5 [9]. Therefore, considering this more serious risk, patients with a biomarker score of 1-4 had benefited from a prescription of sodium-glucose co-transporter type 2 (iSGLT-2) inhibitors such as Dapagliflozin and Empagliflozin available in our environment. to prevent progression of risk to heart failure. The mechanisms by which iSGLT-2 provide cardiovascular protection include increased urinary glucose excretion and reduced water and sodium retention, reduced insulin levels and insulin response to food intake, modulation of the



Fig. 2. Risk of heart failure according to biomarker score.

Table 3

Independent risk of biomarker score on occurrence of heart failure.

Score	All patients	All patients		DM type 1		DM type 2	
Variable	р	OR	р	OR	р	OR	
Very low, score $= 0$		1		1		1	
Low, score $= 1$	0.604	1.18 (0.63–2.20)	0.413	1.38 (0.82–2.57)	0.790	1.10(0.45–1.83)	
Intermediate, score $= 2$	0.024	2.19 (1.11–4.34)	0.023	2.50 (1.28-4.01)	0.012	2.82 (1.85–3.92)	
High, score $= 3-4$	0.001	4.73 (1.84–6.20)	0.006	4.35 (2.99–6.33)	0.001	4.50(2.87–7.19)	

Abbreviation: OR: Odd Ratio.

renin-angiotensin-aldosterone system, reduction in weight and blood pressure without an increase in sympathetic nervous activity [17]. Therefore, in view of the results recorded, it is potentially useful to integrate the biomarker approach to predict the risk of HF in asymptomatic diabetic patients. It is a more cost-effective approach and could help target preventive interventions to diabetic subjects most at risk of HF [18–21].

These results also show that in diabetics, even those who reach target levels of conventional risk factors, including glycosylated hemoglobin <7%, face a high risk of HF. These results are similar to those of Pandy et al., and Rawshani A et al. who demonstrated that the risk of heart failure was higher in diabetics with a glycosylated hemoglobin level <7% [9,22].

The mechanisms by which the elements of the biomarker score appear to predict the risk of HF is the subject of controversy in the scientific world [23–27]. Moreover, HF in diabetics is characterized by inflammation, alteration of metabolic reserves, hyperglycemia, insulin resistance, alteration of the calcium transporter, increased oxidative stress, action of advanced glytation products, atherosclerosis, myocardial ischemia, diabetic autonomic neuropathy, sympathetic stimulation of the renin-angiotensin-aldosterone system (RAAS), diabetic cardiomyopathy, obesity, hypertension, renal failure, insulin therapy, thiazo-lidinedione, epigenetics and genetics [22,28–39].

The role of biomarkers in determining HF in asymptomatic diabetics involves increasing afterload with Laplace's law for LVH. hs-CRP is associated with oxidative stress, atherosclerosis and insulin resistance affecting the myocardium. While NT-ProBNP reflects myocardial aggression by volume overload or increased intraventricular pressure [40–42].

The analysis of the use of biomarkers in determining the risk of heart failure in diabetics in Goma did not individualize the elements of the biomarker score and could underestimate the group of patients at high risk of heart failure, despite its cross-cutting nature. This aspect will be taken into account in subsequent work and requiring longitudinal studies. Apart from these limitations, the present study is the first of its kind in our field and showed that the biomarker score in diabetics is strongly associated with the risk of heart failure at 5 years but ignored.

5. Conclusion

The present study showed despite the nature of the study that the biomarker score is an independent factor in determining the risk of heart failure in asymptomatic diabetic patients in the DRC and Sub-Saharan Africa like European studies.

Ethics approval and consent to participate

The study protocol was approved by the ethics committee of the University of Goma (UNIGOM/CEM/09/2022) and the study was conducted in accordance with the Helsinki principles. All participants signed written informed consent forms before enrollment.

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CRediT authorship contribution statement

Ferdinand Ng'ekieb Mukoso: Conceptualization, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing. Aliocha Natuhoyila Nkodila: Conceptualization, Methodology, Resources, Software, Writing – original draft, Writing – review & editing. Hippolyte Nani tuma Situakibanza: Conceptualization, Resources, Supervision. Stannislas Okitotsho Wembonyama: Conceptualization, Methodology, Resources, Supervision. Zacharie Kibendelwa Tsongo: Conceptualization, Methodology, Resources, Supervision.

Declaration of competing interest

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

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References

- [1] A.C. Mwana-waben, S.M. Lwamushi, C.M. Eboma, M.B. Pacifique, B.C. Lyab, H. Karemere, et al., Choix thérapeutiques des hypertendus et diabétiques en milieu rural : une étude mixte dans deux zones de santé de l'Est de la République Démocratique du Congo, African Journal of Primary Health Care & Family Medicine (2022) 1–9.
- [2] N. Cho, J. Shaw, S. Karuranga, Y. Huang, J.D.R. Fernandes, A. Ohlrogge, B. Malanda, IDF Diabetes Atlas: global estimates of diabetes prevalence for 2017 and projections for 2045, Diabetes Res. Clin. Pract. 138 (2018) 271–281.
- [3] M. Lichtenauer, P. Jirak, V. Paar, B. Sipos, K. Kopp, A.E. Berezin, Heart failure and diabetes mellitus: biomarkers in risk stratification and prognostication, Appl. Sci. 11 (10) (2021) 4397, https://doi.org/10.3390/app11104397.
- [4] D. Glovaci, W. Fan, N.D. Wong, Epidemiology of diabetes mellitus and cardiovascular disease, Curr. Cardiol. Rep. 21 (2019) 21.
- [5] B. Ziaeian, B.Z.G.C. Fonarow, Epidemiology and aetiology of heart failure, Nat. Rev. Cardiol. 13 (2016) 368–378.
- [6] M. Lehrke, N. Marx, Diabetes mellitus and heart failure, Am. J. Cardiol. 120 (2017) S37–S47.
- [7] I.S. Thrainsdottir, T. Aspelund, G. Thorgeirsson, V. Gudnason, T. Hardarson, K. Malmberg, G. Sigurdsson, L. Rydén, The association between glucose abnormalities and heart failure in the population-based reykjavik study, Diabetes Care 28 (2005) 612–616.
- [8] A. Halabi, E. Potter, H. Yang, L. Wright, J.W. Sacre, J.E. Shaw, et al., Association of biomarkers and risk scores with subclinical left ventricular dysfunction in patients with type 2 diabetes mellitus, Halabi et al. Cardiovascular Diabetology 21 (2022) 278.
- [9] A. Pandey, M. Vaduganathan, K.V. Patel, C. Ayers, C.M. Ballantyne, M. N. Kosiborod, et al., Biomarker-based risk prediction of incident heart failure in pre-diabetes and diabetes, JACC (J. Am. Coll. Cardiol.): Heart Fail. 9 (3) (2021) 2 1 5–2 3.
- [10] J.-G. Dillinger, C. Patin, P. Bonnin, T. Vidal-Trecan, E. Paven, J.-F. Gautier, et al., Elevated brain natriuretic peptide and high brachial pulse pressure in patients with diabetes, Am. J. Hypertens. 35 (5) (2022) 414–422.

F. Ng'ekieb Mukoso et al.

- [11] D. D'Amario, S. Migliaro, J.A. Borovac, A. Restivo, R. Vergallo, M. Galli, et al., Microvascular dysfunction in heart failure with preserved ejection fraction, Front. Physiol. 10 (2019) 1347.
- [12] T. Padro, O. Manfrini, R. Bugiardini, J. Canty, E. cenko, G. De Luca, et al., ESC working Group on Coronary pathophysiology and Microcirculation position paper on "coronary microvasculaire dysfunction in cardiovascular disease", Cardiovasc. Res. 116 (2022) 741–755.
- [13] R. Pop-Busui, J.L. Januzzi, D. Bruemmer, S. Butalia, J.B. Green, W.B. Horton, C. Knight, M. Levi, N. Rasouli, C.R. Richardson, Heart failure: an underappreciated complication of diabetes a consensus report of the American Diabetes Association, Diabetes Care 45 (2022) 1670–1690.
- [14] P. Ponikowski, Adriaan A. Voors, Stefan D. Anker, H. Bueno, J.G.F. Cleland, A.J. S. Coats, et al., 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC, Eur. Heart J. 37 (27) (2016) 2129–2200.
- [15] L. Wu, K.G. Parhofer, Diabetic dyslipidemia, Metabolism 63 (12) (2014) 1469–1479.
- [16] F.N. Mukoso, A.N. Nkodila, T.-S.M. Muhemed, T.M. Kashinde, S.J. Ananas, S. N. Myatsi, et al., Performance of the peguero-lo presti index in diagnosis of left ventricular hypertrophy at CIMAK hospital center in north Kivu, democratic republic of the Congo, World J. Cardiovasc. Surg. 13 (2023) 11–25.
- [17] M.C.R. Giuseppe, V. Cristiana, S. Petar, Heart failure in patients with diabetes mellitus, Card. Fail. Rev. 3 (1) (2017) 52–55.
- [18] C. Iribarren, A.J. Karter, A.S. Go, et al., Glycemic control and heart failure among adult patients with diabetes, Circulation 103 (2001) 2668–2673.
- [19] M.W. Segar, K.V. Patel, M. Vaduganathan, et al., Association of long-term change and variability in glycemia with risk of incident heart failure among patients with type 2 diabetes: a secondary analysis of the ACCORD trial, Diabetes Care 43 (2020) 1920–1928.
- [20] K. Matsushita, S. Blecker, A. Pazin-Filho, et al., The association of hemoglobin a1c with incident heart failure among people without diabetes: the Atherosclerosis Risk in Communities study, Diabetes 59 (2010) 2020–2026.
- [21] J. Wang, K. Sarnola, S. Ruotsalainen, et al., The metabolic syndrome predicts incident congestive heart failure: a 20-year follow-up study of elderly Finns, Atherosclerosis 210 (2010) 237–242.
- [22] A. Rawshani, A. Rawshani, S. Franzen, et al., Risk factors, mortality, and cardiovascular outcomes in patients with type 2 diabetes, N. Engl. J. Med. 379 (2018) 633–644.
- [23] D. Aguilar, B. Bozkurt, K. Ramasubbu, A. Deswal, Relationship of haemoglobin A1C and mortality in heart failure patients with diabetes, J. Am. Coll. Cardiol. 54 (2009) 422–428, https://doi.org/10.1016/j.jacc.2009.04.049. PMID: 19628117; PMCID:PMC2753214.
- [24] S.L. Jeffcoate, Diabetes control and complications: the role of glycated haemoglobin, 25 years on, Diabet. Med. 21 (2004) 657–665, https://doi.org/10.1046/ j.1464-5491.2003.01065.x. PMID: 15209755.
- [25] C. Antonio, C. Doina, C. Chanchal, C. Francesco, C.D. Annique, I. Baruch, et al., Heart failure in type 2 diabetes: current perspectives on screening, diagnosis and management, Cardiovasc. Diabetol. 20 (2021) 218, https://doi.org/10.1186/ s12933-021-01408-1.
- [26] P.B. Rodica, L.J. Januzzi, D. Bruemmer, S. Butalia, J.B. Green, W.B. Horton, et al., Heart failure: an underappreciated complication of diabetes. A consensus report of the American diabetes association, Diabetes Care 45 (2022) 1670–1690, https:// doi.org/10.2337/dci22-0014.
- [27] M. Correale, F. Fioretti, L. Tricarico, F. Croella, N.D. Brunetti, R.M. Inciardi, The role of congestion biomarkers in heart failure withReduced ejection fraction, J. Clin. Med. 12 (2023) 3834, https://doi.org/10.3390/jcm12113834.
- [28] A. Groenewegen, F.H. Rutten, A. Mosterd, A.W. Hoes, Epidemiology of heart failure, Eur. J. Heart Fail, 22 (8) (2020) 1342–1356.
- [29] S. Yoon, G.H. Eom, Heart failure with preserved ejection fraction: present status and future directions, Exp. Mol. Med. 51 (12) (2019) 1–9.
- [30] S. Nirengi, Valgas da Silva C. Peres, K.I. Stanford, Disruption of energy utilization in diabetic cardiomyopathy; a mini review, Curr. Opin. Pharmacol. 54 (2020) 82–90.
- [31] Marion Lacout, Adélaide Demiralp, Erwan Donal, Insuffisance Cardiaque chez le patient diabetique:faut-il la dépister?Université de Rennes, CHU de Rennes, INSERM, 2022. LTSI-UMR 1099,Rennes.

International Journal of Cardiology Cardiovascular Risk and Prevention 21 (2024) 200263

- [32] Beauloye Christophe, Traitement du diabete de type 2 en 2019 :un meme rapport cout-benefices, Louv. Med. 138 (3) (2019) 162–165.
- [33] B.C. Field, R. Gordillo, P.E. Scherer, The role of ceramides in diabetes and cardiovascular disease regulation of ceramides by adipokines, Front. Endocrinol. 11 (2020) 569250.
- [34] Salvatore De Rosa, Biagio Arcidiacono, Eusedio Chiefari, Antonio Brunetti, Liro Indolfi, et al., Type 2Diabetes mellitus and cardiovascular disease:genetic and epigenetic links, Freontiers in Endocrinology 9 (2018). /Article2.
- [35] P. Valensi, Autonomic nervous system activity changes in patients with hypertension and overweight: role and therapeutic implications, Cardiovasc. Diabetol. 20 (1) (2021) 170.
- [36] W. Mullens, K. Damman, J.M. Testani, P. Martens, C. Mueller, J. Lassus, et al., Evaluation of kidney function throughout the heart failure trajectory – a position statement from the Heart Failure Association of the European Society of Cardiology, Eur. J. Heart Fail. 22 (4) (2020) 584–603.
- [37] C.A. Lawson, S. Seidu, F. Zaccardi, G. McCann, U.T. Kadam, M.J. Davies, et al., Outcome trends in people with heart failure, type 2 diabetes mellitus and chronic kidney disease in the UK over twenty years, EClin Med 32 (2021) 9.
- [38] Y. Li, B. Liu, Y. Li, X. Jing, S. Deng, Y. Yan, et al., Epicardial fat tissue in patients with diabetes mellitus: a systematic review and meta-analysis, Cardiovasc. Diabetol. 18 (1) (2019) 3.
- [39] A. Ceriello, Chanchal Chandramouli DoinaCatrinolu, Francesco Cosentino, et al., Heart failure in type 2 diabetes :current perspectives on creening, diagnosis and management, Cardiovasc. Diabetol. 20 (2021) 218.
- [40] P. Valensi, Autonomic nervous system activity changes in patients with hypertension and overweight: role and therapeutic implications, Cardiovasc. Diabetol. 20 (1) (2021) 1–12.
- [41] S. De Rosa, B. Arcidiacono, E. Chiefari, A. Brunetti, C. Indolfi, D.P. Foti, Type 2 diabetes mellitus and cardiovascular disease: genetic and epigenetic links, Front. Endocrinol. 9 (2018) 2.
- [42] T. Ohkuma, Y. Komorita, S.A. Peters, M. Woodward, Diabetes as a risk factor for heart failure in women and men: a systematic review and meta-analysis of 47 cohorts including 12 million individuals, Diabetologia 62 (2019) 1550–1560.

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