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RESEARCH

Incorporation of suboptimal health status as a potential risk assessment for type II diabetes mellitus: a case-control study in a Ghanaian population

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Abstract Due to a paradigm shift in lifestyles, there is growing concern that type 2 diabetes mellitus (T2DM) will reach epidemic proportions in Ghana. However, specific characteristics of the disease are under explored in this region. More challenging are those yet to be diagnosed or who complain of poor health in the absence of a diagnosed disease—suboptimal health status (SHS). We conducted a study to examine various factors that characterise SHS and T2DM. Using a cross-sectional design, we recruited 264 people as controls and 241 T2DM patients from January to June 2016. The controls were categorised into high and low SHS based on how they rated on an SHS questionnaire-25 (SHSQ-25). Anthropometric and biochemical parameters: body mass index (BMI); blood pressure (BP); fasting plasma glucose (FPG); glycated haemoglobin (HbA1c); serum lipids [(total cholesterol, triglycerides (TG), high- and low-density lipoprotein-cholesterol (HDL-c and LDL-c)] were measured. The male to female ratio for T2DM and controls were 99:142 and 98:166, respectively, whilst the mean ages were 55.89 and 51.52 years. Compared to controls, T2DM patients had higher FPG (8.96 ± 4.18 vs. 6.08 ± 1.79; $p < 0.0001$) and HbA1c (8.23 ± 2.09 vs. 5.45 ± 1.00; $p < 0.0001$). Primarily sedentary [adjusted odds ratio (aOR) = 2.97 (1.38–6.39); $p = 0.034$)], systolic blood pressure (SBP) ($p = 0.001$) and diastolic blood pressure (DBP) ($p = 0.001$) significantly correlated with high SHS. After adjusting for age and gender, central adiposity [aOR = 1.74 (1.06–2.83); $p = 0.027$)], underweight [aOR = 5.82 (1.23–27.52); $p = 0.018$)], high SBP [aOR = 1.86 (1.14–3.05); $p = 0.012$)], high DBP [aOR = 2.39 (1.40–4.07); $p = 0.001$)] and high TG [aOR = 2.17 (1.09–4.33); $p = 0.029$)] were found to be independent risk factors associated with high SHS. The management of T2DM in Ghana is suboptimal and undiagnosed risk factors remain prevalent. The SHSQ-25 can be translated and applied as a practical tool to screen at-risk individuals and hence prove useful for the purpose of predictive, preventive and personalised medicine.

Keywords Chronic diseases · Biomarkers · Predictive · Preventive and personalised medicine

Introduction

The rising prevalence of diabetes mellitus (DM) is a major health threat worldwide. Presently, DM affects more than 422 million people with an enormous proportion (≈ 90%) of these being type II diabetes mellitus (T2DM) [1]. Data from the World Health Organisation (WHO) [1] and the International Diabetes Federation (IDF) [2] suggest that T2DM is directly related to urbanisation, mechanisation, physical inactivity and unhealthy diet and because these characteristics are still being exhibited by many adults, the prevalence of T2DM is likely to increase. In fact, the projected trajectory of the prevalence in the years 2025–2030 is 500 million worldwide [3]. More disconcerting is the increasing prevalence of the disease among adolescents and young

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adults. These individuals are likely to spend more on medical costs and have more time to suffer from both microvascular and macrovascular complications of the disease than older adults [4, 5].

T2DM affects multiple organs in the human body and people with the disease have an elevated risk of blindness, cognitive decline, kidney failure, cardiovascular diseases, fractures, brain damage, depression and consequently, premature death [6–11]. Many of these complications may be averted or delayed with timely health education and intervention [10, 12–15]. Unfortunately, the majority of people, especially those residing in less healthcare-resourced and low-income developing countries such as Ghana are not aware of their risk status.

In Ghana, up to 440,000 people were documented to have T2DM in 2013 but the number of those with prediabetes have not been recorded or identified to date [16]. These individuals can remain undiagnosed for a long period, even for many decades of their life. Effective intervention for such people can only commence following the manifestation of clinical symptoms. This, from the perspective of a preventive, predictive and personalised medicine (PPPM) standpoint, is a delayed response [3]. PPPM is defined as 'an integrative concept that enables the prediction of an individual's predisposition before the onset of a disease, to provide targeted preventive measures and create personalised treatment algorithms tailored to a person' [17]. Over the past few years, PPPM has made a significant impact on the prevention and treatment of diseases because it adopts a holistic approach (e.g. environmental, behavioural and traditional factors) to solving health problems [3, 18, 19].

As with many chronic diseases, screening for prediabetes or T2DM is central in PPPM and it provides the stimulus for initiating treatment and delaying long-term complications. Most often, screening is performed in a health care facility in order to allow health care providers to perform appropriate follow-up testing and institute quality health care [20]. However, with recent developments in public health research, there are robust screening tools that are non-invasive, inexpensive and can be applied both in a health care setting and in the field or the wider community. One such tool is the suboptimal health status questionnaire (SHSQ-25) [21, 22].

SHSQ-25 identifies persons that complain of poor health in the absence of any diagnosable condition [23]. It explores human health from five domains: fatigue, cardiovascular, immune, digestive and mental, and over the years, it has been successfully applied for screening purposes among Caucasians [24] and Chinese [21–23, 25, 26]. In these studies, it is apparent that a high SHS (i.e. SHS score > median score) is associated with chronic disease risk factors and that these scores are largely under the influence of external factors such as employment type, lifestyle, socioeconomic, cultural and climatic conditions.

Here, we extend our previous research by examining SHS in a Ghanaian population, and in parallel, we examine the anthropometric, clinical and biochemical parameters among Ghanaian T2DM patients. Understanding these factors in both healthy and T2DM participants will be instrumental in the pursuit of PPPM.

Methods

Study design

This cross-sectional study was conducted from January to June 2016. Recruitment for the study was based on purposeful sampling where T2DM patients, who reported at the Diabetic Centre, Komfo Anokye Teaching Hospital (KATH), were invited to participate. KATH is a referral hospital with over 1200 beds with not less than 100 diabetic/hypertensive patients attending the hospital every fortnight. Utilising a convenient sampling method, we recruited 264 control participants from three suburbs (Ash-town, Pankrono and Abrepo) within the Kumasi metropolis. The Committee on Human Research, Publication and Ethics (CHRPE), Kwame Nkrumah University of Science and Technology (KNUST), Kumasi, and the Human Research Ethics Committee (HREC), Edith Cowan University (ECU), Australia, reviewed and approved the study protocol. Written informed consent was obtained from all participants.

Data collection

The SHSQ-25 questionnaire was used to measure SHS. The SHSQ-25 comprises 25 items, which are categorised into five domains: fatigue (9 items), cardiovascular system (3 items), digestive system (3 items), immune system (3 items) and mental health (7 items). Each participant was asked to rate a statement on a 5-point Likert type scale, based on how often they had experienced a particular complaint in the previous 3 months: (1) never or almost never, (2) occasionally, 3) often, (4) very often and (5) Always. The raw scores of 1 to 5 on the SHSQ-25 were recoded as 0–4. SHS score was calculated by summing the ratings for the 25 items. A high SHS score represents poor health [21–23, 27]. To test for reliability of the SHSQ-25, we determined the Cronbach's α coefficient which was found to be 0.91.

Anthropometric examination

Weight (kg) and height (cm) were measured with a standard stadiometer (SECA, Hamburg, Germany). These data were used to determine the body mass index (BMI), calculated as BMI = weight (kg)/height $(m)^2$. Waist and hip circumference were measured in centimetres using a tape measure and waist

to hip ratio (WHR) was calculated as WHR = waist (cm)/hip (cm). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using a standard sphygmomanometer (Omron HEM711DLX, UK).

Clinical data

Fasting blood samples were collected from the antecubital vein of each participant into tubes containing EDTA, gel separator and fluoride oxalate. Fasting plasma glucose (FPG) in fluoride tubes and glycated haemoglobin (HbA1c) in EDTA tubes were measured on the automated chemistry analyser (Roche Diagnostics, COBAS INTEGRA 400 Plus, USA). Likewise, serum total cholesterol, triglycerides (TG), low-density lipoproteins (LDL)-cholesterol and high-density lipoprotein (HDL)-cholesterol were measured on the automated chemistry analyser. Non-HDL was calculated as non-HDL = total cholesterol-HDL.

Inclusion and exclusion criteria

Cases

The study included only participants who were diagnosed as having T2DM, based on the international classification of diabetes (ICD 10) criteria. Participants on insulin medication or injections were considered to be suffering from type I diabetes mellitus and hence excluded. Of the 260 T2DM participants recruited for the study, 19 were excluded from the analysis due to missing biochemical data.

Controls

In order to screen for individuals with undiagnosed risk factors, we excluded all participants who had been previously diagnosed with diabetes and/or hypertension. In addition, individuals who were suffering from other chronic diseases related to the genitourinary, digestive, respiratory and haematological systems were excluded. We included participants aged 18–80 years.

Statistical analysis

All continuous data were recorded as mean ± standard deviation and percentages for categorical variables. Between-group comparisons for continuous variables were determined using student *t* tests, whilst intergroup comparisons of categorical variables were done with chi-square tests and analysis of variance (ANOVA). Association between SHS and cardiovascular risk factors were performed using linear regression and multiple logistic regression models. Odds ratios (ORs) at 95% confidence intervals (95% CI) were recorded for logistic regression analysis. All statistical analysis was performed on the Statistical Package for Social Sciences (SPSS), version 22. A two-sided $p < 0.05$ was considered significant.

Results

The characteristics of the 505 participants comprising 264 controls and 241 cases are shown in Table 1. Over 44% of all T2DM patients had hypertension, male to female ratio (98:142), were overweight (33.19%), were obese (18.26%), had tertiary education (14.52%), had moderate activity (67.21%), were employed (55.17%) and had smoking (14.10%) and alcohol (42.32%) histories.

The mean age for T2DM only and T2DM with hypertension were 55.89 ± 11.27 and 60.07 ± 9.93, respectively. BMI was not different between T2DM and hypertensive T2DM patients ($p = 0.158$). WHR was higher among T2DM patients with hypertension (0.92 ± 0.55 vs. 0.94 ± 0.061; $p < 0.0001$) However, FPG, HbA1c, total cholesterol (TC), TG, HDL-c, LDL-c and coronary risk were not different in T2DM and T2DM with hypertension ($p > 0.05$). T2DM patients were generally older than controls ($p < 0.0001$), had a higher WHR (0.94 ± 0.061 vs. 0.88 ± 0.08; $p < 0.0001$), higher FPG (8.96 ± 4.18 vs. 6.08 ± 1.79; $p < 0.0001$) and higher HbA1c (8.23 ± 2.09 vs. 5.45 ± 1.00; $p < 0.0001$). However, compared with non-hypertensive T2DM patients, the controls had higher SBP (143.69 ± 25.82 vs. 122.17 ± 11.86; $p < 0.0001$), DBP (84.27 ± 15.37 vs. 89.16 ± 12.62, $p < 0.0001$) and coronary risk (5.37 ± 1.49 vs. 4.90 ± 1.52; $p < 0.011$). There were no differences in TC, TG, LDL-c and very-LDL-c (VLDL-c) between controls and non-hypertensive T2DM patients. Similarly, compared to controls, hypertensive T2DM patients were older ($p < 0.0001$), had higher WHRs (0.94 ± 0.061 vs. 0.88 ± 0.08; $p < 0.0001$), higher SBP (160.48 ± 18.24 vs. 84.27 ± 15.37; $p < 0.0001$) and higher DBP (89.16 ± 12.62 vs. 84.27 ± 15.37; $p < 0.0001$) (Table 2).

The mean age of control participants was 51.67 ± 11.45 years with a male to female ratio of 98:166. A high proportion had at least a basic education (35.2%), were married (65.9%) and employed (40.5%). Women were generally obese compared to men when BMI (33.1% vs. 2.0%; $p = 0.001$) and central adiposity (68.7% vs. 5.1%; $p = 0.001$) were used, respectively, as an obesity index. A higher proportion of men than women were smokers (15.3% vs. 1.2%; $p = 0.001$) and had a history of alcohol intake (41.8% vs. 25.6%; $p = 0.005$). There was a significantly higher DBP ($p = 0.034$), HbA1c ($p = 0.043$), TC ($p = 0.001$), HDL-c ($p = 0.011$), non-HDL-c ($p = 0.004$) and LDL-c ($p = 0.006$) among women compared to men. Levels of SBP, FPG, TG, VLDL-c, coronary risk and WHR among

Table 1 Characteristics of study participants with or without T2DM or hypertension

Variable	Control	DM2 only	DM2 + HPT	x^2	*p* value
Age groups (years)				27.75	*0.001*
31–40	14 (5.3)	12 (9.1)	2 (1.9)		
41–50	31 (11.7)	32 (24.2)*	17 (15.7)		
51–60	74 (28.0)	45 (34.1)	36 (33.3)		
61–70	87 (33.0)	31 (23.5)	37 (34.3)		
71–80	58 (22.0)	12 (9.1)*	16 (14.8)		
Gender				0.985	0.611
Male	98 (37.1)	52 (39.4)	46 (42.6)		
Female	166 (62.9)	80 (60.6)	62 (57.4)		
BMI				15.39	*0.017*
Underweight	13 (4.9)	8 (6.1)	1 (0.9)		
Normal weight	107 (40.5)	67 (51.1)	39 (36.1)		
Overweight	87 (33.0)	32 (24.4)	48 (44.4)*		
Obese	57 (21.6)	20 (18.5)	24 (18.3)		
Marital status				23.77	*0.003*
Married	174 (65.9)	91 (68.9)	72 (66.7)		
Never married	29 (11.0)	3 (2.3)*	1 (0.9)*		
Divorced	24 (9.1)	12 (9.1)	13 (12.1)		
Widowed	37 (14.0)	26 (19.7)	22 (20.4)		
Education				15.66	*0.048*
Tertiary	36 (13.6)	17 (12.9)	18 (16.7)		
Senior high school	82 (31.1)	38 (28.8)	19 (17.6)		
Junior high school	93 (35.2)	38 (28.8)	40 (37.0)		
Lower primary	31 (11.7)	26 (12.1)	12 (11.1)		
No formal education	22 (8.3)	23 (17.4)	19 (17.6)		
Occupation				69.88	*0.0001*
Employed	107 (40.5)	82 (62.1)	51 (47.2)		
Retired	23 (8.7)	12 (9.1)	22 (20.4)		
Unemployed	32 (12.2)	28 (21.2)	23 (21.3)		
Informal employment	102 (38.6)	10 (7.6)	12 (11.1)		
Physical activity				11.07	0.086
Primarily sedentary	87 (33.0)	35 (26.5)	43 (39.8)		
Moderate activity	177 (67.5)	97 (73.5)	65 (60.1)		
Family history				54.59	*0.0001*
Yes	121 (46.0)	97 (73.5)	85 (78.7)		
Smoking history				11.09	*0.026*
Yes	17 (6.5)	17 (12.9)	17 (15.7)		
History of alcohol intake				9.57	*0.048*
Yes	83 (31.7)	54 (40.9)	48 (44.4)		

All statistical analyses were performed using SPSS and tests of significance were two-tailed ($p < 0.05$)

women were not significantly different from men ($p > 0.05$) (Table 3).

With a median SHS score of 21, participants were grouped into high SHS (≥ 21) and low SHS (< 21). Gender ($p = 0.023$), age ($p = 0.020$), education ($p = 0.001$), marital status ($p = 0.019$), occupation ($p < 0.0001$) and physical activity ($p = 0.006$) were significantly associated with high SHS. Meanwhile, being female [adjusted odds ratio (aOR) = 1.7 (1.04–2.85); $p = 0.034$)], elderly [aOR = 10.8 (1.69–68.97); $p = 0.018$)], illiterate [aOR = 5.34 (1.61–17.77); $p = 0.007$)], having lower primary education [aOR = 3.14 (1.14–8.65); $p = 0.029$)], widowed [aOR = 2.75 (1.28–5.91); $p = 0.011$)], retired [aOR = 7.0 (2.40–20.40); $p = 0.0001$)], unemployed [aOR = 4.28 (1.83–9.99); $p = 0.0009$)], having informal

Table 2 Clinical data of study participants with or without T2DM or hypertension

Variables	Controls	T2DM Only	T2DM HPT	*p* value
Age	51.62 ± 11.92	55.89 ± 11.27†	60.07 ± 9.93*¥	< *0.0001*
BMI	25.86 ± 5.06	25.60 ± 5.38	26.80 ± 4.72	0.158
WHR	0.88 ± 0.08	0.92 ± 0.55†	0.94 ± 0.061*¥	< *0.0001*
SBP	143.69 ± 25.82	122.17 ± 11.86†	160.48 ± 18.24*¥	< *0.0001*
DBP	84.27 ± 15.37	75.45 ± 11.29†	89.16 ± 12.62*¥	< *0.0001*
FPG	6.08 ± 1.79	8.96 ± 4.18†	9.49 ± 4.68*	< *0.0001*
HbA1c	5.45 ± 1.00	8.23 ± 2.09†	8.35 ± 2.09*	< *0.0001*
Total cholesterol	4.57 ± 1.25	4.71 ± 1.17	4.76 ± 1.39	0.342
Triglycerides	1.32 ± 0.91	1.22 ± 0.57	1.33 ± 0.55	0.484
HDL-c	1.23 ± 0.31	1.37 ± 0.35†	1.33 ± 0.29*	< *0.0001*
LDL-c	2.77 ± 1.06	2.77 ± 1.11	2.81 ± 1.23	< *0.0001*
VLDL-c	0.59 ± 0.35	0.55 ± 0.26	0.60 ± 0.25	0.928
Coronary risk	5.37 ± 1.49	4.90 ± 1.52†	5.05 ± 1.53	*0.011*
Creatinine	91.41 ± 27.75	100.84 ± 33.37†	112.70 ± 49.85*¥	< *0.0001*

Values are presented as mean ± SD. One-way ANOVA followed by Tukey post hoc multiple comparison. BMI: $p < 0.05$ was considered statistically significant. All statistical analyses were performed using SPSS and tests of significance were two-tailed ($p < 0.05$)

† *p* value is significant (comparison between control and DM only)

**p* value is significant (comparison between control and DM + HTN)

¥ *p* value is significant (comparison between DM only and DM + HTN)

employment [aOR = 2.68 (1.52–4.68); $p = 0.0008$)] and primarily sedentary [aOR = 2.97 (1.38–6.39); $p = 0.034$)] were significant independent risk factors for high SHS after adjusting for age and gender. Participants with high SHS had a significantly higher mean SBP ($p = 0.004$) and DBP ($p = 0.001$) compared to those with low SHS. However, there were no significant differences between the mean lipid profile among participants with high SHS compared to low SHS ($p > 0.05$) (Table 4).

After adjusting for age and gender, central adiposity [aOR = 1.74 (1.06–2.83); $p = 0.027$)], underweight [aOR = 5.82 (1.23–27.52); $p = 0.018$)], high SBP [aOR = 1.86 (1.14–3.05); $p = 0.012$)], high DBP [aOR = 2.39 (1.40–4.07); $p = 0.001$)] and high TG [aOR = 2.17 (1.09–4.33); $p = 0.029$)] were found to be significant independent risk factors associated with high SHS (Table 5).

After controlling for age and gender, significant positive linear relationships were observed between SHS score and SBP, DBP and CR irrespective of gender ($p < 0.05$). There were inconsistent correlations between other risk factors and high SHS. SHS scores were significantly associated with LDL-c in men whilst FPG, TC and non-HDL-c were significantly associated with SHS in women ($p < 0.05$) (Table 6). There was no significant linear relationship between BMI, HbA1c, TG, HDL-c, VLDL-c and high SHS in either men or women ($p > 0.05$) (Table 6).

Discussion

T2DM is largely a consequence of accumulated metabolic damage due to increasing urbanisation, physical inactivity, unhealthy eating and sedentary lifestyle [7, 10, 28, 29]. Early diagnosis remains the blueprint for preventing T2DM and promoting better health outcomes [12, 30–32]. This study is premised on the hypothesis that cardiometabolic risk factors are prevalent in Kumasi, an urban city in Ghana [14]. As such, we have explored modifiable risk factors in both T2DM sufferers and healthy controls (Tables 1, 2 and 3).

Among the controls, we used a simple and inexpensive tool (SHSQ-25) to reveal highly at-risk individuals. Participants were classified into two groups based on how they rated the SHSQ-25. Here, a median score < 21 represents low SHS (good health) whereas a median score > 21 represents high SHS (poor health). Ideally, filling this short questionnaire alone should encourage individuals who obtain a high SHS score to have their clinical/biochemical indicators measured. Such persons could be advised by health providers on dietary/lifestyle modifications that will enable them to live healthier and delay the onset of T2DM. Alternatively, a person with a high SHS may have undiagnosed, asymptomatic T2DM, or its related co-morbidities and may need immediate intervention or therapy.

In this study, undiagnosed hypertension is prevalent among the participants, and similar to our previous

Table 3 Characteristics of controls stratified by gender

Characteristics	Total	Men (n = 98)	Women (n = 166)	p value
Age (years)	51.67 ± 11.45	51.09 ± 12.02	51.44 ± 11.89	0.761
Anthropometric data				
Waist to hip ratio	0.88 ± 0.07	0.89 ± 0.06	0.87 ± 0.08	0.148
Body mass index (kg/m^2)				*< 0.0001*
Underweight	13 (4.9)	8 (8.2)	5 (3.0)	
Normal weight	107 (40.5)	60 (61.2)	47 (28.3)	
Overweight	87 (33.0)	28 (28.6)	59 (35.5)	
Obese	57 (21.6)	2 (2.0)	55 (33.1)	
Central obesity				*< 0.0001*
Normal	145 (54.9)	93 (94.9)	52 (31.3)	
Obese	119 (45.1)	5 (5.1)	114 (68.7)	
Socioeconomic data				
Education				*< 0.0001*
Tertiary	36 (13.6)	26 (26.5)	10 (6.0)	
Senior high school	82 (31.1)	26 (26.5)	56 (33.7)	
Junior high school	93 (35.2)	35 (35.7)	58 (34.9)	
Lower primary	31 (11.7)	6 (6.1)	25 (15.1)	
No formal education	22 (8.3)	5 (5.1)	17 (10.2)	
Marital status				*0.001*
Married	174 (65.9)	75 (76.5)	99 (59.6)	
Never married	29 (11.0)	14 (14.3)	15 (9.0)	
Divorced	24 (9.1)	3 (3.0)	21 (12.6)	
Widowed	37 (14.0)	6 (6.1)	31 (18.7)	
Occupation				*< 0.001*
Employed	107 (40.5)	52 (53.1)	55 (33.1)	
Retired	23 (8.7)	13 (13.3)	10 (6.0)	
Unemployed	32 (12.2)	1 (1.0)	31 (18.6)	
Informal employment	102 (38.6)	32 (32.7)	70 (42.2)	
Biochemical data				
Systolic blood pressure (mmHg)	144.12 ± 26.61	145.82 ± 30.96	142.43 ± 22.25	0.305
Diastolic blood pressure (mmHg)	83.74 ± 15.70	81.66 ± 18.02	85.81 ± 13.38	*0.034*
Fasting Plasma Glucose (mmol/l)	6.08 ± 1.79	6.04 ± 1.78	6.11 ± 1.79	0.751
Glycated haemoglobin (%)	5.41 ± 0.98	5.28 ± 0.91	5.54 ± 1.04	*0.043*
Total cholesterol (mmol/l)	4.50 ± 1.17	4.24 ± 1.00	4.76 ± 1.33	*0.001*
Triglycerides (mmol/l)	1.29 ± 0.89	1.19 ± 0. 81	1.39 ± 0.96	0.105
HDL-c (mmol/l)	1.12 ± 0.30	1.16 ± 0.28	1.26 ± 0.32	*0.011*
NonHDL-c (mmol/l)	3.23 ± 1.09	3.07 ± 0.91	3.50 ± 1.26	*0.004*
VLDL-c (mmol/l)	0.58 ± 0.35	0.54 ± 0.36	0.61 ± 0.34	0.133
LDL-c (mmol/l)	2.73 ± 2.03	2.54 ± 0.91	2.91 ± 1.12	*0.006*
Coronary risk	5.33 ± 2.87	5.22 ± 1.28	5.45 ± 1.59	0.236
Family history and activity				
Diabetes family history (yes)	121 (46.0)	43 (43.9%)	78 (47.3%)	0.343
Smoking (yes)	17 (6.5)	15 (15.3)	2 (1.2)	*< 0.001*
Drinking (yes)	83 (31.7)	41 (41.8)	42 (25.6)	*0.005*
Physical activity				*0.037*
Primarily sedentary	87 (33.0)	29 (29.6)	58 (34.9)	
Moderate activity	135 (51.1)	46 (46.9)	89 (53.6)	
Primarily physical	42 (16)	23 (23.4)	19 (11.4)	

All statistical analysis was performed using SPSS. Data is expressed as mean ± standard deviation or (n %) and tests of significance were two-tailed ($p < 0.05$)

findings, high SHS is significantly associated with both DBP and SBP (Tables 4 and 6). This also confirms the findings of another community-based study in the sub-region that showed that a high proportion of adults in sub-Saharan Africa (SSA) (44–93%) who have high blood pressure are unaware of their condition [33, 34]. Another study in a peri-urban community in Ghana showed the prevalence of undiagnosed hypertension as 28.7% [34]. This is disturbing because high BP is by far the main risk factor for T2DM and CVD [34–36]. High BP, for example, causes 42% of all ischaemic heart diseases [37] and one third of all heart failures [38]. As such, there is an

Table 4 Distribution of risk factors with or without SHS

Variables	Total *n* (%)	SHS score ≥ 21 *n* (%)	SHS score < 21 *n* (%)	*p* value	x^2	aOR (95% CI)	*p* value
Gender				*0.023*	4.49		
Male	98 (37.3)	42 (31.1)	56 (43.8)			1.0[#]	
Female	165 (62.7)	93 (68.9)	72 (56.3)			1.7 (1.04–2.85)	*0.034*
Age (years)				*0.02*	13.34		
21–30	14 (5.3)	5 (3.7)	9 (7.0)			1.0[#]	
31–40	30 (11.4)	17 (12.6)	13 (10.2)			2.35 (0.63–8.73)	0.332
41–50	74 (28.1)	41 (32.0)	33 (24.4)			1.45 (0.44–4.72)	0.574
51–60	87 (33.1)	47 (36.7)	40 (29.6)			1.53 (0.47–4.94)	0.569
61–70	44 (16.7)	28 (20.7)	16 (12.5)			3.15 (0.89–11.04)	0.119
71–80	14 (5.3)	12 (8.9)	2 (1.6)			10.8 (1.69–68.97)	*0.018*
Education				*0.001*	19.81		
Tertiary	36 (13.7)	14 (10.4)	22 (17.2)			1.0[#]	
Senior high school	82 (31.2)	30 (22.2)	52 (40.6)			0.91 (0.40–2.03)	0.838
Junior high school	93 (35.4)	54 (40.0)	39 (30.5)			2.17 (0.99–4.78)	0.076
Lower primary	30 (11.4)	20 (14.8)	10 (7.8)			3.14 (1.14–8.65)	*0.029*
No education	22 (8.4)	17 (12.6)	5 (3.9)			5.34 (1.61–17.77)	*0.007*
Marital status				*0.019*	11.76		
Married	173 (68.5)	80 (59.3)	93 (72.7)			1.0[#]	
Never married	29 (11.0)	13 (9.6)	16 (12.5)			0.94 (0.42–2.08)	0.999
Divorced/separated	24 (9.1)	16 (11.8)	8 (6.2)			2.32 (0.94–5.72)	0.081
Widowed	37 (14.1)	26 (19.3)	11 (8.6)			2.75 (1.28–5.91)	*0.011*
Occupation				*< 0.001*	27.09		
Employed	106 (40.3)	36 (26.7)	70 (54.7)			1.0[#]	
Retired	23 (8.7)	18 (13.3)	5 (3.9)			7.00 (2.40–20.40)	*0.0001*
Unemployed	32 (12.2)	22 (16.3)	10 (7.8)			4.28 (1.83–9.99)	*0.0009*
Informal employment	102 (38.8)	59 (43.7)	43 (33.6)			2.68 (1.52–4.68)	*0.0008*
Physical activity				*0.006*	12.35		
Primarily sedentary	87 (33.1)	57 (42.2)	30 (23.4)			2.97 (1.38–6.39)	*0.007*
Moderate activity	135 (51.3)	62 (45.9)	73 (57)			1.32 (0.65–2.71)	0.476
Primarily physical	41 (15.6)	16 (11.9)	25 (19.6)			1.0[#]	
Biochemical data							
SBP (mmHg)	143.69 ± 25.82	148.33 ± 24.05	139.20 ± 26.58	*0.004*			
DBP (mmHg)	84.27 ± 15.37	87.33 ± 15.35	81.24 ± 14.7	*0.001*			
FPG (mmol/l)	6.08 ± 1.78	6.15 ± 1.75	6.01 ± 1.84	0.544			
HbA1c (%)	5.45 ± 0.99	5.44 ± 0.94	5.44 ± 1.06	0.997			
TC(mmol/l)	4.57 ± 1.25	4.66 ± 1.3	4.47 ± 1.18	0.217			
TG (mmol/l)	1.32 ± 0.91	1.41 ± 1.01	1.22 ± 0.79	0.099			
HDL-c (mmol/l)	1.23 ± 0.31	1.25 ± 0.32	1.20 ± 0.3	0.177			
VLDL-c (mmol/l)	0.59 ± 0.35	0.62 ± 0.35	0.56 ± 0.36	0.155			
LDL-c (mmol/l)	2.77 ± 1.06	2.81 ± 1.09	2.73 ± 1.02	0.554			
Coronary risk	5.37 ± 1.49	5.40 ± 1.5	5.35 ± 1.47	0.805			

Multivariate regression model was adjusted for age and gender

aOR adjusted odds ratio, *CI* confidence interval

[#] Reference, $p < 0.05$. Tests of significance were two tailed ($p < 0.05$)

overarching need to identify these individuals and begin treatment to avoid complicated health outcomes.

Similar to our previous findings, age was associated with high SHS (Table 4). This is not surprising since ageing is

Table 5 Association between SHS and metabolic risk factors

Variables	Total (*n* %)	SHS score ≥ 21 (*n* %)	SHS score < 21 (*n* %)	x^2	*p* value	OR (95% CI)	*p* value
Central obesity				4.88	*0.018*		
Normal	144 (54.8)	65 (48.1)	79 (61.7)			1.0[#]	
Obese	119 (45.2)	70 (51.9)	49 (38.3)			1.74 (1.06–2.83)	*0.027*
BMI				6.75	0.08		
Underweight	13 (4.9)	11 (8.1)	2 (1.6)			5.82 (1.23–27.52)	*0.018*
Normal weight	107 (40.7)	52 (38.5)	55 (43.0)			1.0[#]	
Overweight	86 (32.7)	41 (30.4)	45 (35.2)			0.96 (0.54–1.70)	0.987
Obese	57 (21.7)	31 (11.8)	26 (20.3)			1.26 (0.66–2.40)	0.514
Blood pressure							
Systolic blood pressure							
Normal systolic BP	121 (46.0)	52 (38.5)	69 (53.9)	6.26	*0.009*	1.0[#]	
High systolic BP	142 (54.0)	83 (61.5)	59 (46.1)			1.86 (1.14–3.05)	*0.012*
Diastolic blood pressure							
Normal diastolic BP	176 (66.9)	78 (57.8)	98 (76.6)	10.47	*0.001*	1.0[#]	
High diastolic BP	87 (33.1)	57 (42.2)	30 (23.4)			2.39 (1.40–4.07)	*0.001*
Fasting plasma glucose				2.87	0.090		
Normal	113 (43.1)	51 (38.1)	62 (48.4)			1.0[#]	
High	149 (56.9)	83 (61.9)	66 (51.6)			0.65 (0.40–1.07)	0.105
Glycated Haemoglobin				1.93	0.164		
Normal	195 (75.6)	98 (73.7)	97 (77.6)			1.0[#]	
High	63 (24.4)	38 (29.2)	25 (19.5)			1.50 (0.84–2.86)	0.192
Cholesterol				0.03	0.489		
Normal	186 (72.1)	96 (71.6)	90 (72.6)			1.0[#]	
High	72 (27.9)	38 (28.4)	34 (27.4)			1.05 (0.61–1.81)	0.867
Triglycerides				4.97	*0.03*		
Normal	215 (83.3)	105 (78.4)	110 (88.7)			1.0[#]	
High	43 (16.7)	29 (21.6)	14 (11.3)			2.17 (1.09–4.33)	*0.029*
HDL-c							
Normal	151 (58.5)	78 (58.2)	73 (58.9)	0.12	0.508	1.0[#]	
Low	107 (41.5)	56 (41.8)	51 (41.1)			1.03 (0.63–1.69)	0.999
Non-HDL-c				1.66	0.123		
Normal	137 (53.1)	66 (49.3)	71 (57.3)			1.0[#]	
High	121 (46.9)	53 (42.7)	68 (50.7)			1.38 (0.85–2.25)	0.532
LDL-c							
Normal	126 (48.8)	65 (48.5)	61 (49.2)	0.12	0.506	1.0[#]	
High	132 (51.2)	69 (51.5)	63 (50.8)			1.03 (0.63–1.68)	0.999

aOR adjusted odds ratio, *CI* confidence interval

[#] Reference, $p < 0.05$. Tests of significance were two-tailed ($p < 0.05$)

associated with less physical activity and sedentary lifestyle; making it a high-order independent risk factor for T2DM [39]. From the perspective of metabolism, this ageing is accompanied by an imbalance in the production of reactive oxygen species (ROS) and inflammation that together lead to metabolic dysregulation. Metabolic dysregulation will lead to insulin resistance and consequently T2DM [40].

In addition, gender, education, marital status, occupation and physical activity were associated with high SHS (Table 4). However, we could not validate the association between high SHS and higher FPG, HbA1c, TC, LDL and low HDL (Table 5). In part, this observation could be attributed to the low sample size used for this investigation. All our previous investigations involved large cohorts in China, i.e. 2799

Table 6 Multivariate linear regression model for SHS score in relation to metabolic risk factors stratified by gender

	Male		*p* value	Female		*p* value
	β	SE		β	SE	
BMI	0.52	0.44	0.237	0.16	0.22	0.454
SBP	2.09	0.51	*0.035*	1.52	0.05	*0.046*
DBP	2.16	0.84	*0.012*	2.11	0.07	*0.005*
FPG	0.67	0.86	0.442	2.09	0.62	*0.0009*
HbA1c	0.13	1.68	0.936	1.17	1.1	0.118
TC	1.33	1.52	0.387	1.75	0.85	*0.043*
TG	2.07	1.89	0.276	1.18	1.2	0.329
HDL-c	−9.27	5.32	0.085	−1.42	3.64	0.696
Non-HDL	2.56	1.68	0.131	2.08	0.91	*0.024*
VLDL	4.60	4.16	0.272	4.19	3.39	0.219
LDL-c	3.31	1.66	*0.049*	2.02	1.02	0.051
CR	2.79	1.16	*0.019*	1.45	0.72	*0.049*

$p < 0.05$. Tests of significance were two-tailed ($p < 0.05$)

B regression coefficient, *SE* standard error

participants in 2009 [23], 3019 in 2012 [26], 3405 in 2012 [41] and 4313 in 2016 [21]. Cohorts from geographically distinct populations are exposed to different stressors (e.g. variation in job types, lifestyles, socioeconomic, environmental and cultural factors). For example, whilst the majority of the Ghanaian participants are primarily sedentary and engage in less energy-demanding jobs, the Chinese cohorts are mainly industry workers who spend long hours at work and therefore more likely to be stressed. Subsequently, stressful conditions, especially in the hours preceding testing, may affect biochemical assessments. Further, it is possible that the biochemical assessments of this study are somewhat influenced by laboratory conditions or equipment used [42]. Therefore, other highly sensitive and state-of-the art health facilities should be available for validation.

Among T2DM sufferers, hypertension was high and this agrees with a previous study reported from the Kumasi region (Table 2) [43]. Further, the results of the present study show that the majority of T2DM patients had FPG and HbA1c levels higher than the recommended targets (i.e. > 7 and > 7.2, respectively), many of whom are on the path to developing complications and co-morbidities. Surprisingly, all these individuals have been using blood pressure- and lipid-lowering medications long before the start of this project. On the one hand, this could be attributed to delayed intervention, ineffective treatments, untargeted medications, drug response and drug resistance [14, 43]. On the other hand, the suboptimal management could be due to other factors including (1) institutional (e.g. health care policies, facilities and resources); (2) environmental, dietary and lifestyles; (3) genetic and epigenetics and (4) individual factors (physical, mental, social and spiritual wellbeing). In order to address such a complex situation, there must be a transition from the current medical practise to PPPM. PPPM holds the key to revolutionising T2DM care by promoting adequate patient stratification, disease modelling, surveillance, optimal diagnosis and prediction of adverse drug-drug interaction [3, 17–19]. Taken together, this will lead to better health outcomes, delay the onset of complications, improve quality of life and promote longevity.

Overall, it is clear that modifiable risk factors are prevalent among T2DM sufferers but importantly, we have shown that SHSQ-25 could be a risk stratification tool for T2DM. Compared to many survey instruments and risk prediction models [44–48], the SHSQ-25 is simple, inexpensive and can be self-completed prior to, or administered during, a consultation. The scoring system is easy and data interpretation/analysis does not require special expertise to perform.

Whilst recognising this, this tool is a subjective health measure and it should be supported with advanced objective biomarkers. These days, highly sophisticated and powerful analytical tools are available for measuring, detecting and characterising important biomarkers [49–51]. This will help in the understanding of the molecular intricacies that underpin the disease' pathogenesis. For example, it is possible to determine transcriptional regulation, post-translational modifications, protein expression and interaction and altered enzyme activity [49, 50]. Our team have commenced such research where we examined *N*-glycosylation profiles in metabolic syndrome (MetS) [47]. Here, we showed that nine *N*-glycan traits were associated with DBP, SBP, FPG and BMI and these could be potential biomarkers for MetS [52]. Moreover, another investigation of the *N*-glycosylation profiles in the plasma samples of participants in this present Ghana study (T2DM and controls) is ongoing.

As interesting as the study is, a few limitations need to be mentioned. The major one is related to the cross-sectional design. We were unable to determine the proportion of participants in the high SHS group who will develop T2DM over time. The study tried to perform age-gender matching but the recruited controls were still generally younger than cases. However, this does not invalidate the significance of the findings of this study since potential confounding was to an extent addressed by logistic regression and multivariate analyses. The sample size of the study does not allow a generalisation to be made. Moreover, metabolic risk factors such as blood pressure, blood glucose and lipid profiles, particularly among the controls, were limited to only one measurement and therefore the prevalence of risk factors may be either under or overestimated.

Conclusion

There is poor management of risk factors among T2DM patients in this region of Ghana. More disturbing is the fact that the majority of people who are at risk, particularly those with

hypertension, are undiagnosed. This underscores the need for novel screening tools that can identify such individuals. The SHSQ-25 represents an instrument of choice and in turn sets the platform for prediction, prevention and treatment of T2DM, which is vital, particularly for a region where laboratory-based measures are not routinely available.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

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