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Depression, poor sleep quality, and diabetic control in type 2 diabetes patients at Sunyani Regional Hospital, Ghana: a case–control study

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Abstract

Background Diabetes patients are at risk of developing mental health comorbidities such as depression and poor quality of sleep. These conditions can affect diabetic management, including glycemic and plasma lipid control to optimal levels. We investigated the association between diabetic control and the presence of depression and poor sleep quality in type 2 diabetes (T2DM) patients at Sunyani Government hospital in Ghana. Using a case–control design, we recruited 200 T2DM patients and compared them to 160 non-diabetic controls. The presence of depression was assessed using the Patient Health Questionnaire (PHQ)-9 and sleep quality using the Pittsburgh Sleep Quality Instrument (PSQI). Blood samples were collected to measure glycated hemoglobin (HbA1c) levels and plasma lipid profiles. Poor glycemic control was defined as HbA1c > 7%, depression as PHQ-9 score > 9 and poor sleep quality as PSQI score ≥ 5.

Results T2DM patients had a higher prevalence of depression (31.5% vs 10.6%, $p < 0.001$) and poor sleep quality (64% vs 40%, $p < 0.001$) compared to non-diabetic controls. Glycemic control was not associated with depression and poor sleep quality in T2DM patients. Depression was associated with increased odds of hypercholesteremia [OR (95% CI) = 10.71 (2.64–43.41), $p < 0.001$] in non-diabetic controls and poor sleep quality was associated with increased odds of low HDL cholesterol in T2DM patients [3.2 (1.38–7.48), $p = 0.007$] and hypertriglyceridemia in non-diabetic controls [2.54 (1.15–8.51), $p < 0.001$].

Conclusion In our study population, depression and poor sleep quality were common in T2DM patients compared to non-diabetic controls. Depression and poor sleep quality were associated with abnormalities in serum lipid levels, but not glycemic control.

Keywords Type 2 diabetes, Depression, Sleep quality, Glycemic control, Dyslipidemia

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic debilitating disease that poses a public health problem worldwide, especially in middle- and low-income countries, where about two-thirds of the disease is expected to occur. According to the International Diabetes Federation, 463 million people worldwide (adults 20–79 years old) were living with T2DM in 2019, and this number is expected to increase to 700 million by 2045 [1]. In Ghana, a recent meta-analysis reported that the prevalence of diabetes was 6.46% in the adult population [2]. T2DM is a systemic disease that affects almost every organ of the

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body, notably the cardiovascular and nervous systems, resulting in a progressive deterioration of quality of life, a high cost of treatment and an excessive burden on the under-resourced healthcare systems [3]. One major complication of T2DM that is usually neglected at various healthcare facilities in sub-Saharan Africa is mental health disorders.

Several studies have reported that T2DM patients have poor mental health, particularly depression and poor sleep quality [4–8]. The World Health Organization describes depression as one of the leading causes of health deterioration and progression toward disability [9]; with the occurrence of depression associated with a higher risk of diabetes complications and increased utilization of health care services [3]. Depression is often associated with poor sleep health [10] and both conditions are common in T2DM patients [3, 8, 11]. Depression and poor sleep quality are associated with poor adherence to diabetic medication and self-care management protocols, thus increasing the risk of serious short- and long-term diabetic complications such as blindness, amputations, stroke, cognitive decline, decreased quality of life, and premature death. When mental health comorbidities of diabetes are not diagnosed and treated, the financial cost to society and health care systems is catastrophic, and the human suffering that results is profound [12].

Diabetes alters carbohydrate and lipid metabolism, and these metabolites affect cognition and mental health [13]. There are conflicting studies on the association between diabetic control, such as glycemic and lipid control, and depression and quality of sleep; some studies report a significant association, while others found no association. In many of these studies, glycemic control was associated with either depression or poor quality of sleep [5, 14]. Therefore, we compared the prevalence of depression and poor sleep quality between T2DM patients and non-diabetic controls. In addition, we investigated the association between glycemic control and serum lipid levels versus depression and poor sleep quality. We hypothesize that T2DM patients would have a higher burden of depression and poor sleep quality, and these would be associated with poor glycemic and dyslipidemia.

Methods

Setting, design, and participants

The study was a case–control design, conducted at the Sunyani Regional Hospital in Ghana from November 2019 to March 2020. The Sunyani Regional Hospital is a primary healthcare facility with a 300-bed capacity that serves various regional administrative towns in the middle belt of Ghana. The hospital provides ambulatory medical care and consultation, dietetics, diabetes education, and eye services for diabetes patients. We recruited 200 T2DM patients, aged between 30 and 65 years, into the study by systematic random sampling of every fourth consenting patient visiting the diabetic clinic. Afterwards, 160 non-diabetic controls were conveniently invited from the surrounding communities into the study. T2DM status was determined clinically as patients with late diagnosis of diabetes (after 30 years) who were managed initially on lifestyle modification or antidiabetic drugs. Patients with type 1 diabetes, pregnant women and those aged less than 30 years at diagnosis or patients older than 75 years were excluded from the study. In addition, patients with other causes of depression such as the loss of a close family member within the past month and medication/history of depression or manic/hypomanic episodes were excluded from the study. A structured questionnaire was used to collect data on socio-demographic and clinical characteristics such as age, gender, education, employment, alcohol and smoking status, duration of diabetes, and diabetes medication. We also collected information on any diagnosed cardiovascular comorbidities such as hypertension, hyperlipidemia, angina, coronary heart disease, heart attack, stroke, and gout by asking patients or taking that information from the case notes in their hospital folders.

Depression and sleep quality

Depression was evaluated using the Patient Health Questionnaire-9 (PHQ-9). The PHQ-9 is an instrument that has been validated in the Ghanaian population [15, 16] and patients with diabetes [17] for the detection of major depressive symptoms. The PHQ-9 is a multiple-choice self-report questionnaire designed to establish the diagnosis of depression according to the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV criteria [18]. The Twi (popular Ghanaian language) version of this questionnaire has been validated, with a diagnostic validity comparable to the original English version, and this was used for patients with limited formal education [15]. This PHQ-9 instrument has 9 items, each of which is scored from 0 (not at all) to 3 (nearly every day), providing a severity score. The cut-off points 5, 10, and 15 represent mild, moderate, and severe levels of depressive symptoms, respectively; a cut-off point of 10 or higher allows the diagnosis of major depression [16, 18].

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). The PSQI, a validated questionnaire, was used to measure sleep quality in the past month. The first four questions asked participants to report the time they went to bed (not necessarily the time they fell asleep), the number of minutes it took to fall asleep when they woke up, and the hours of sleep per night. The next 10 questions asked how often the

participant had trouble sleeping for reasons such as having to get up to use the restroom, feeling too hot or too cold, having pain, or waking up in the middle of the night, the questions answered on a four-point scale ranging from 'never' to 'three times or more a week'. Participants were also rated on the same four-point scale about their use of medication to fall asleep, how often they have had trouble staying awake during social activities, and if enthusiasm for completing tasks has been reduced. Lastly, participants provided a subjective rating of their sleep quality on a four-point scale from 'very good' to 'very bad'. The PSQI questions were combined into seven different scores ranging from 0 (no difficulty) to 3 (severe difficulty) on the topics of sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, sleep medication and daytime dysfunction per the PSQI scoring guidelines. The seven component scores were summed for a final PSQI score that ranged from 0 to 21, with sleep quality declining with each increase in the score. PSQI score > 5 is indicative of poor sleep quality.

Anthropometry and biochemical analyses

Blood pressure was measured after 5 min rest using an automated digital blood pressure monitor (Omron 907XL pro, Healthcare, Inc., Vernon Hills, IL), with the patients seated comfortably with back support and arm resting on a table. Body weight and height were measured with a Seca 740 scale and a stadiometer respectively, and the body mass index (BMI) was computed using the formula: weight in kilograms divided by the height in meters squared. Blood samples were drawn in the morning after 8–12 h of overnight fasting into plain vacuum tubes to measure plasma lipids and fluoride oxalate tubes for glucose levels. Plasma lipid profiles were analyzed by enzymatic assays using a biochemistry analyzer (Contec BC 400, China) and commercial reagents (Randox Laboratory Reagents, UK). Glycated haemoglobin (HbA1c) levels were measured using the boronate affinity chromatography method on PDQ Plus HPLC autoanalyzer (Primus Diagnostics, Trinity Biotech, Ireland). The definition of parameters was as follows: poor glycemic control as HbA1c > 7%; high levels of triglycerides, total and LDL cholesterol were defined as ≥ 1.7 mmol/l, ≥ 5.2 mmol/l, and > 3.4 respectively, and low HDL cholesterol levels were defined as < 1 mmol/l for males and < 1.3 mmol/l for females. Dyslipidemia was defined as having at least one of the lipid abnormalities.

Ethical considerations

Ethical approval was sought and obtained from the Ethics and Protocol Review Committee of the College of Health Sciences of the University of Ghana (Protocol ID number: CHS-Et/M.2–4.11/2018–2019) and each patient provided written voluntary informed consent after the rationale and procedure of the study were thoroughly explained. Patients found to be depressed were referred to the Department of Psychiatry for further assessment and possible treatment.

Sample size calculation

The minimum sample size required was computed with the IBM SPSS ver 28. We assumed that the minimum prevalence of depression or poor sleep quality among non-diabetic controls would be 10% [4, 19] and the T2DM patients would have an increased odds ratio of 2.5 for depression [11, 12] and poor sleep quality [20]. At 95% significant level and 90% power, at least 131 participants were required for each group. We, therefore, recruited 200 T2DM patients and 160 non-diabetic controls for the study.

Statistical analysis

Data were analyzed using IBM SPSS version 27. Data were presented as mean with standard deviation for continuous variables and as proportions for categorical variables. The differences between T2DM patients and non-diabetic control regarding their sociodemographic, clinical and biochemical variables were analyzed using a chi-squared (χ^2) test for the comparison of categorical variables and the Student t test for continuous measures. Logistic regression models were performed to determine the odds ratio of depression and poor sleep quality among T2DM patients and non-diabetic controls, as well as the change in the odds of depression and glycemic control after adjusting for confounding variables. The level of significance was set at $p < 0.05$.

Results

General characteristics of participants

In this study, T2DM patients were older than non-diabetic individuals (52 ± 7.9 vs 48.6 ± 10.6 years, $p < 0.001$). There were no significant differences in the gender distribution of participants between T2DM patients and non-diabetic controls. Smoking status and alcohol intake were not significantly different among study participants. The mean BMI was significantly higher in T2DM patients compared to non-diabetic individuals. For blood pressure (BP), T2DM patients had higher systolic, diastolic, mean and pulse BPs, and heart rate when compared to non-diabetes individuals (Table 1).

Compared to non-diabetes controls, T2DM patients had higher levels of plasma triglycerides, total and LDL cholesterol, and lower levels of HDL cholesterol. The prevalence of, depression and poor sleep quality was high in T2DM patients compared to non-diabetic controls (Table 2).

Table 1 General characteristics of study participants

	T2DM ($n=200$)	Non-diabetes ($n=160$)	*P*
Gender, *n* (%)			0.404
Male	76 (38)	54 (33.8)	
Female	124 (62)	106 (66.2)	
Married	113 (56.5)	98 (61.3)	0.363
Living with relatives	139 (69.5)	109 (68.1)	0.779
Hypertension, *n* (%)	132 (66)	16 (10)	< 0.001
Age decades, *n* (%)			0.001
< 40	20 (10)	38 (23.7)	
40–49	42 (21)	40 (25)	
50–59	108 (54)	64 (40)	
60 +	30 (15)	18 (11.3)	
Educational levels, *n* (%)			0.005
None	16 (8)	18 (11.3)	
Elementary	108 (54)	60 (37.5)	
SHS/vocational	54 (27)	68 (42.5)	
Tertiary	22 (11)	14 (8.8)	
Previous smoker, *n* (%)	74 (37)	15 (9.4)	< 0.001
Alcohol intake, *n* (%)	86 (42)	46 (28.8)	0.005
BMI, kg/m^2	30.8 ± 7.1	25.9 ± 5.9	< 0.001
Systolic BP, mmHg	145 ± 27	133 ± 11	< 0.001
Diastolic BP, mmHg	89 ± 19	82 ± 14	< 0.001
Mean BP, mmHg	117 ± 13	111 ± 10	< 0.001
Pulse BP, mmHg	61 ± 12	52 ± 11	< 0.001
Heart rate, beats/min	72 ± 16	67 ± 8	< 0.001

SHS senior high school, *BMI* body mass index, *BP* blood pressure

Table 2 Biochemical, depression, and sleep quality among study participants

	T2DM ($n=200$)	Non-diabetes ($n=160$)	*p*
Glycated haemoglobin, %	7.8 ± 3.5	5.1 ± 0.8	< 0.001
Total cholesterol, mmol/l	5.6 ± 1.2	3.9 ± 1.1	< 0.001
Triglycerides, mmol/l	2.4 ± 1.1	1.4 ± 0.8	< 0.001
HDL cholesterol, mmol/l	1.1 ± 0.5	1.4 ± 0.4	< 0.001
LDL cholesterol, mmol/l	2.3 ± 1.1	2 ± 1.2	0.015
vLDL cholesterol, mmol/l	0.48 ± 0.22	0.28 ± 0.09	< 0.001
PHQ-9 score	7.9 ± 5.7	5.1 ± 3.8	< 0.001
PHQ-9 score > 9, *n* (%)	63 (31.5)	17 (10.6)	< 0.001
Global PSQI score	7.8 ± 3.9	5.5 ± 3	< 0.001
PSQI score > 5, *n* (%)	148 (74)	64 (40)	< 0.001

HDL high-density lipoprotein, *LDL* low-density lipoprotein, *vLDL* very low-density lipoprotein, *PHQ-9* Patient's Health Questionnaire-9, *PSQI* Pittsburgh Sleep Quality Instrument

As expected, most of the T2DM patients had a previous diagnosis of CVD compared to non-diabetic controls (85% vs. 27%, $p<0.001$). The most common comorbidity was hypertension (66% vs 8%, $p<0.001$), followed by gout (14% vs 4%, $p=0.005$), with hyperlipidemia (10%), angina (6%), heart attack (5%), stroke (4%), CHD (2%), and amputation (2%) found only in T2DM patients.

Depression, sleep quality, and glycemic control

Among T2DM patients, 122 (61%) had poor glycemic control (HbA1c > 7%). There was no association between glycemic control and depression status ($\chi^2=0.199$, $p=0.655$), as well as poor sleep quality ($\chi^2=0.568$, $p=0.451$) (Fig. 1). In correlational analyses, HbA1c positively correlated with PHQ-9 scores ($r=0.43$, $p<0.001$) and PSQI scores ($r=0.2$, $p=0.011$) in non-diabetic controls, but no correlation was found between HbA1c and PHQ-9 scores ($r=-0.09$, $p=0.201$) or PSQI scores ($r=-0.07$, $p=0.295$) in T2DM patients (Fig. 2). For lipid abnormalities, the presence of depression was associated with low HDL cholesterol ($\chi^2=4.47$, $p=0.035$) in T2DM patients and high total cholesterol ($\chi^2=29$, $p<0.001$) in non-diabetic controls (Fig. 3). Poor sleep quality was associated with low HDL cholesterol in T2DM patients and high total cholesterol ($\chi^2=10.1$, $p=0.002$), hypertriglyceridemia ($\chi^2=13.8$, $p<0.001$) and dyslipidemia ($\chi^2=10.5$, $p=0.001$) in non-diabetic controls (Fig. 4).

Univariate and multivariable logistics regression analyses

In unadjusted logistic regression analysis, depression was associated with increased odds of low HDL cholesterol and high LDL cholesterol in all study participants, increased odds of low HDL cholesterol in T2DM patients and increased odds of hypercholesteremia non-diabetic controls. After adjustment of confounders in multivariable logistic regression models, hypercholesteremia was significantly associated with depression in the entire study participants and non-diabetic controls (Table 3).

Poor sleep quality was associated with increased odds of hypertriglyceridemia in all study participants, increased odds of low HDL cholesterol in T2DM patients, and increased odds of hypertriglyceridemia and dyslipidemia in non-diabetic controls in unadjusted logistic regression models. After adjustments for confounders in multivariable regression models, low HDL cholesterol status was associated with poor sleep quality in T2DM patients and hypertriglyceridemia was associated with poor sleep quality in non-diabetic controls (Table 4).

Discussion

The main findings of the study

We found that the prevalence of depression and poor quality of sleep was higher in T2DM patients compared

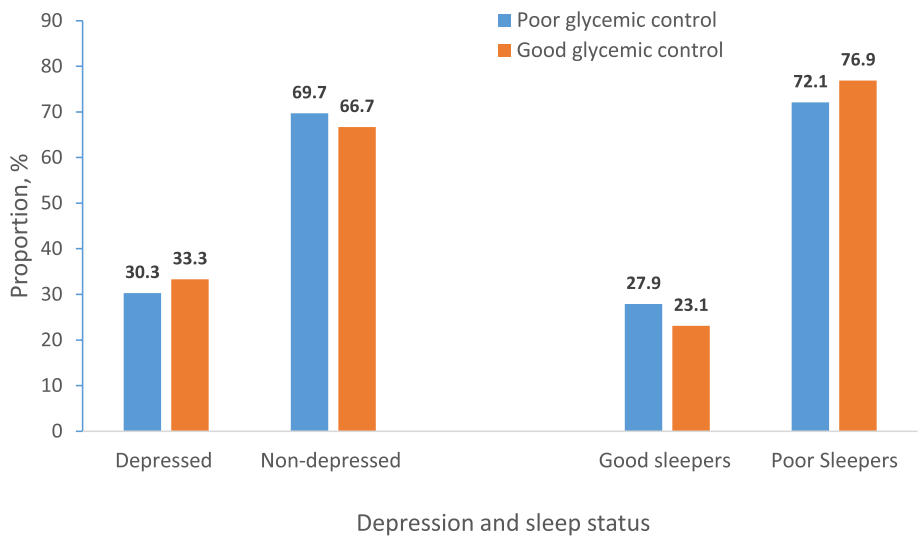

Fig. 1 Glycemic control in T2DM patients with depression and poor sleep quality

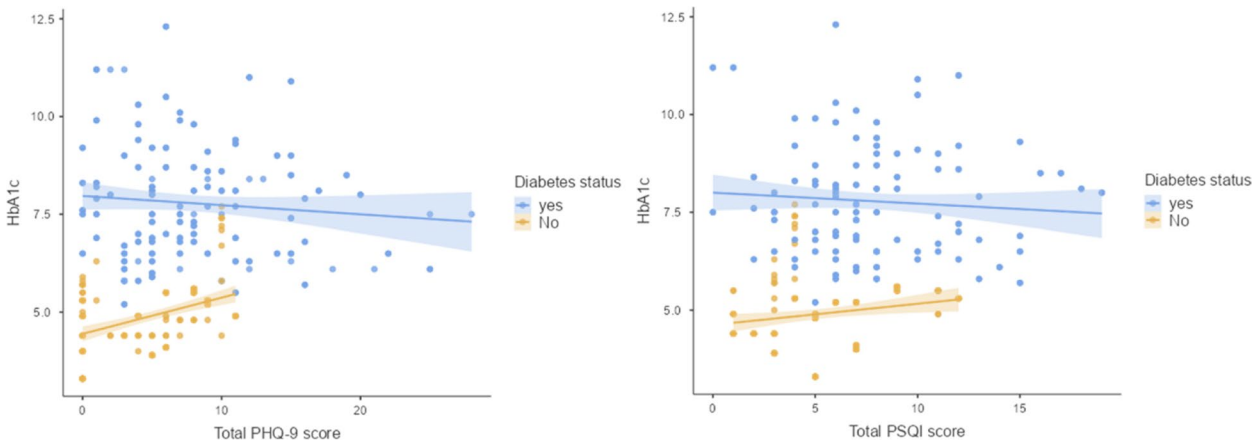

Fig. 2 Correlation between HbA1c versus PHQ-9 scores and PSQI scores

to non-diabetic controls. There was no association between glycemic control versus depression and poor sleep quality. Depression was associated with an increased odds of hypercholesteremia in non-diabetic controls and poor sleep quality was associated with low HDL cholesterol in T2DM patients and hypertriglyceridemia in non-diabetic controls.

Prevalence of depression

In this study, depression was screened in study participants using the Patient Health Questionnaire (PHQ)-9 instrument. The PHQ-9 instrument is among the acceptable psychiatric tools widely used for screening and monitoring the treatment of depression. In Ghana, a report on studies conducted on pregnant women in Kintampo showed that the PHQ-9 instrument performs better than other questionnaire-based instruments used to detect depression [15]. The items in the PHQ-9 instrument are easily understood and the time for administration is short. Therefore, patients give reliable responses to items in PHQ-9, which leads to high internal validity [21].

The findings of the present study indicate that the prevalence of depression was higher in diabetes than in non-diabetes participants. This is consistent with a case–control design study conducted in Calabar, Nigeria, that reported a higher prevalence of depression in diabetes patients compared to non-diabetes controls (23 vs 9%) [22]. The prevalence of depression in this study is similar to the findings of Akpalu et al., who reported that the prevalence of depression was 31.3% in a tertiary facility in Ghana. However, their study design did not include non-diabetic controls [12]. In Cameroon, a study conducted at Douala General Hospital reported the prevalence of depression to be 29% among T2DM patients

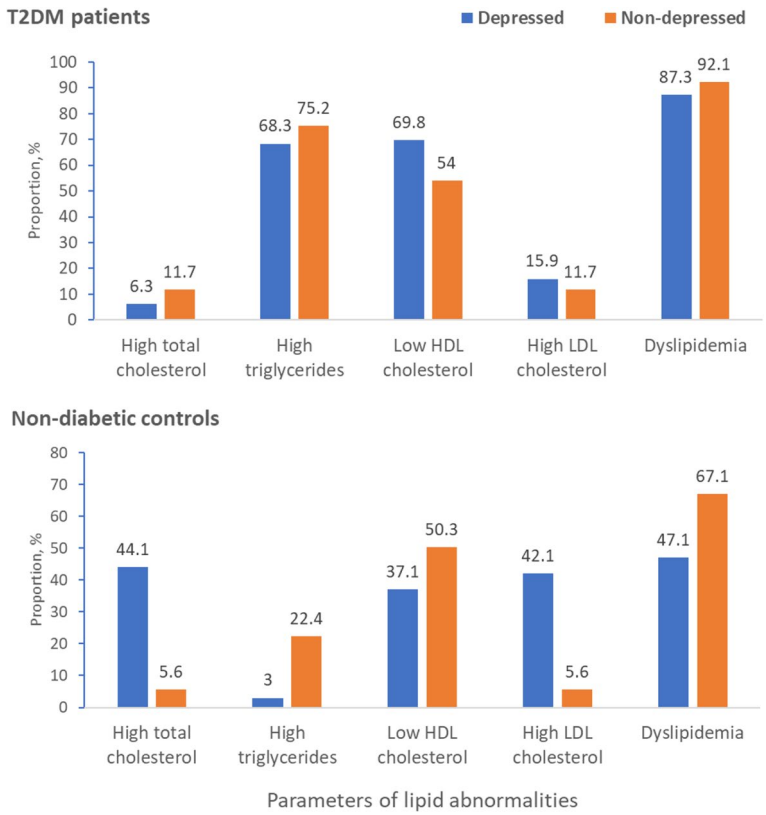

Fig. 3 Prevalence of abnormal plasma lipids by depression status among study participants

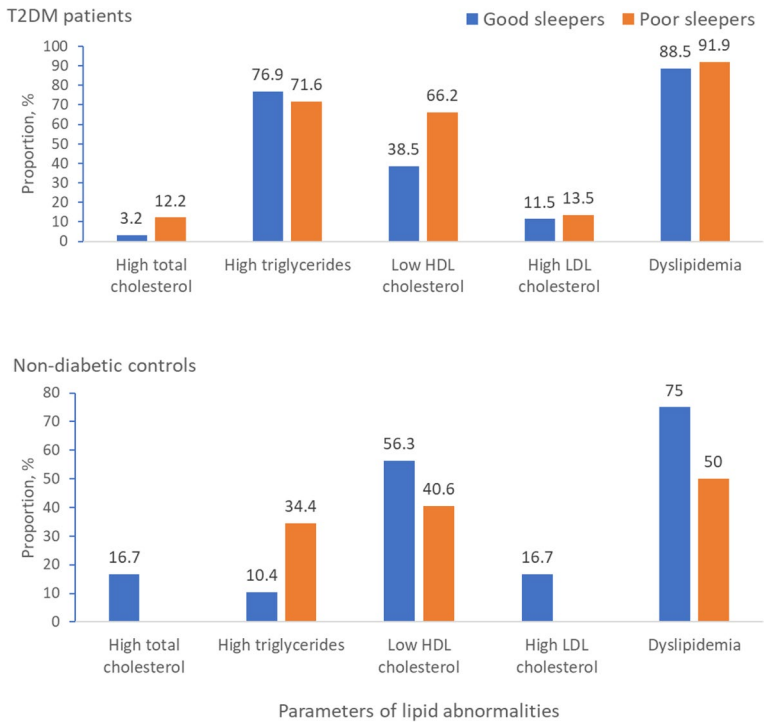

Fig. 4 Prevalence of abnormal plasma lipids by sleep status among study participants

Table 3 Association between depression diabetes control and depression

	Unadjusted OR (95% CI)	*P*	Adjusted OR (95% CI)	*P*
All participants				
Glycemic control	1.49 (0.74–3)	0.269		
High total cholesterol	1.88 (0.9–3.96)	0.095		
High triglycerides	1.25 (0.76–2.05)	0.383		
Low HDL cholesterol	1.71 (1.02–2.86)	0.043	0.87 (0.45–1.66)	0.665
High LDL cholesterol	3.1 (1.58–6.06)	< 0.001	4.81 (1.98–11.71)	< 0.001
Dyslipidemia	0.95 (0.52–1.74)	0.862		
T2DM patients				
Glycemic control	0.87 (0.47–1.6)	0.655		
High total cholesterol	0.51 (0.16–1.6)	0.25		
High triglycerides	0.71 (0.37–1.37)	0.306		
Low HDL cholesterol	1.97 (1.05–3.72)	0.036	1.36 (0.62–2.97)	0.446
High LDL cholesterol	1.43 (0.61–3.35)	0.414		
Dyslipidemia	0.54 (0.2–1.45)	0.221		
Non-diabetic controls				
High total cholesterol	15 (4.56–49.3)	< 0.001	10.71 (2.64–43.41)	< 0.001
Low HDL cholesterol	0.88 (0.32–2.4)	0.798		
High LDL cholesterol	15 (4.56–49.3)	< 0.001	10.71 (2.64–43.41)	< 0.001
Dyslipidemia	0.44 (0.16–1.2)	0.108		

Adjusted for age, gender, BMI, mean blood pressure, marital status, smoking, and drinking status in all participants and T2DM patients. In non-diabetic controls, adjustments were made for age, gender mean blood pressure, and BMI to maintain model stability (tolerance and valence inflation factor). No logistic regression model was performed for high triglyceride levels in non-diabetic controls due to the low number of events in that group

HDL high-density lipoprotein, *LDL* low-density lipoprotein

Table 4 Association between diabetic control and poor sleep quality

	Unadjusted OR (95% CI)	*p*	Adjusted OR (95% CI)	*P*
All participants				
Glycemic control	4.01 (0.48–23.78)	0.201		
High total cholesterol	0.67 (0.34–1.34)	0.67		
High triglycerides	2.99 (1.93–4.63)	< 0.001	0.58 (0.22–1.51)	0.264
Low HDL cholesterol	1.41 (0.92–2.15)	0.112		
High LDL cholesterol	0.6 (0.31–1.14)	0.117		
Dyslipidemia	0.97 (0.58–1.63)	0.911		
T2DM patients				
Glycemic control	0.78 (0.4–1.5)	0.452		
High total cholesterol	3.46 (0.78–15.46)	0.104		
High triglycerides	0.76 (0.36–1.58)	0.46		
Low HDL cholesterol	3.14 (1.63–6.03)	< 0.001	3.2 (1.38–7.48)	0.007
High LDL cholesterol	1.2 (0.45–3.17)	0.716		
Dyslipidemia	1.48 (0.53–4.16)	0.459		
Non-diabetic controls				
High triglycerides	4.51 (1.96–10.37)	< 0.001	2.54 (1.15–8.51)	< 0.001
Low HDL cholesterol	0.53 (0.28–1.01)	0.054		
Dyslipidemia	0.93 (0.37–1.65)	0.401		

Adjusted for age, gender, BMI, mean blood pressure, marital status, smoking, and drinking status in all participants and T2DM patients. In non-diabetic controls, adjustments were made for age, gender mean blood pressure, and BMI to maintain model stability (tolerance and valence inflation factor). No logistic regression model was performed for high total and LDL cholesterol levels in non-diabetic controls due to the low number of events in that group

HDL high-density lipoprotein, *LDL* low-density lipoprotein

[23]. Similarly, the prevalence of depression was reported to be 34% in Ugandan diabetes patients [24]. Contrary to the findings of this study, a low prevalence of depression (18%) was reported in Ethiopian diabetes patients [11]. The high burden of depression in diabetes patients could be explained by two causal pathways. One hypothesis postulates that depression occurs before T2DM, causing increased secretion of counter-regulatory hormones to insulin, alterations in glucose transport function, and increased immuno-inflammatory activation. These physiological alterations are believed to contribute to insulin resistance and beta-islet cell dysfunction, leading to the development of T2DM [25]. The second hypothesis is that depression in diabetes patients may be due to chronic psychosocial stressors of having a chronic medical condition [26].

Prevalence of poor sleep quality

The tool for assessing sleep quality in this study, PSQI, has been shown to have high internal consistency and validity in several groups of patients [27] and the general population [28]. The result of this study shows a high burden of poor sleep quality in T2DM patients compared to non-diabetic controls. Few studies have evaluated sleep quality in diabetes patients in African populations. In a case–control study conducted in Jimma, Ethiopia, the prevalence of poor sleep quality in T2DM patients was found to be 55.6%, higher than that in non-diabetes controls, which was 32.3% [29]. A cross-sectional study conducted in Nairobi, Kenya, reported a prevalence of poor sleep quality of 53% in T2DM patients [30], lower than what was found in the current study. Compared to the prevalence of poor quality of sleep found in this study, studies conducted in developed countries such as the USA [31], Italy [32], Taiwan [33, 34], and China [35] reported a similar pattern of poor quality of sleep in diabetes patients compared to non-diabetes controls. Poor quality of sleep may be associated with metabolic dysregulation in diabetes patients through activation of the hypothalamic–pituitary–adrenal (HPA) axis, which deregulates neuroendocrine parameters such as cortisol, leading to downstream increases in glucose and insulin and decreases in adiponectin levels [36]. The HPA axis pathway, along with an increase in the sympathetic nervous system and inflammatory responses, has been implicated in the relationship between short sleep duration and an increased risk of hypertension, coronary heart disease, recurrent acute coronary syndrome, and heart failure [37].

Diabetic control, depression, and poor sleep quality.

In this study, we did not find any association between glycemic control and depression. There are conflicting reports on the association between glycemic control and depression in T2DM patients. In agreement with the findings of this study, a previous cross-sectional study in a tertiary healthcare facility in Ghana reported no association between depression and glycemic control in T2DM patients after the adjustment of confounders [12]. Another cross-sectional study in the United States likewise reported no association between depression and glycemic control [38]. Some longitudinal studies even found no association between glycemic control and depression in T2DM patients [4, 39]. Contrary to the findings of this study, systematic reviews and meta-analyses have reported that the presence of depression increases the likelihood of poor glycemic control in the longitudinal [5, 14] and cross-sectional study design [6]. We also did not find an association between poor sleep quality and glycemic control in T2DM patients in this study, similar to the findings in Sudanese diabetic patients [7]. Likewise, Behan et al. reported no association between poor sleep quality and glycemic control [40]. However, in contrast to our findings, studies conducted in Japan [8], Turkey [41], and China [33, 42] reported an association between glycemic control and poor sleep quality.

We found that hypercholesteremia, hypertriglyceridemia, and low HDL cholesterol are associated with depression and poor quality of sleep. Several studies have reported contradictory findings on the association between depression and serum cholesterol levels. Consistent with the findings in our non-diabetic controls, Tedders et al. reported that depression was associated with an increased odds of having low HDL cholesterol in the general population of the USA, but LDL cholesterol showed a U-shaped relationship with depression, with both lower and higher levels of LDL cholesterol associated with an increased likelihood of depression compared to normal levels [43]. Furthermore, a recent meta-analysis reported that patients with the first episode of major depressive disorder had increased odds of hypertriglyceridemia and low HDL cholesterol levels [44]. However, the Multi-Ethnic Study of Atherosclerosis reported that an increase in total and LDL cholesterol levels was associated with a decrease in depressive symptoms [45], and this was supported by earlier studies as reported in a meta-analysis conducted more than a decade ago [46]. A study in a sub-Saharan African population also found no association between serum lipids and depression [19, 47].

We found that poor quality of sleep was associated with low HDL cholesterol in T2DM patients and hypertriglyceridemia in non-diabetic controls. In the Japanese [48] and Taiwanese populations [49], both short and long durations of sleep were associated with hypertriglyceridemia and low HDL cholesterol. In T2DM patients, frequent snoring which is an indication of poor quality

of sleep was associated with increased serum triglyceride levels and decreased HDL cholesterol levels [20]. The association between abnormal serum lipid levels and depression and poor sleep quality of sleep can be explained by the fact that lipid metabolism is linked to myelin formation, synaptic plasticity, and receptor function in the central nervous system [50].

Limitations of the study

The findings of this study are limited by it being conducted in a single secondary healthcare facility, and hence, the results cannot be generalized to the entire Ghanaian population. Also, the one-time data collection prevents any inference of causality. Therefore, we cannot conclude that depression and poor sleep quality are responsible for poor glycemic control. Although we encouraged the study participants to answer all items of the questionnaire truthfully to their best ability, there might be recall biases in the response they might have provided. We did not assess factors that can affect depression and sleep such as cultural or religious beliefs, available forms of social support systems, individual resilience, self-care practises and medication adherence.

Conclusion

In our study population, we found a high prevalence of depression and poor sleep quality among T2DM patients compared to non-diabetic individuals. There was no association between glycemic control and depression as well as poor quality of sleep. However, depression and poor sleep quality were associated with abnormal serum lipid levels. This suggests that depression and sleep status should be taken into account when managing diabetes, especially preventing or treating complications associated with lipid abnormalities.

Abbreviations

BMI	Body mass index
BP	Blood pressure
HDL	High-density lipoprotein
LDL	Low-density lipoprotein
PHQ	Patient Health Questionnaire
PSQI	Pittsburgh Sleep Quality Index
T2DM	Type 2 diabetes

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Authors' contributions

KY conceptualized the study, analyzed the data, and drafted the manuscript. TG collected the data and revised the manuscript. JAA analyzed the data and made scientific contributions to the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

The dataset supporting the conclusions of this paper is available and can be requested from the corresponding author.

Declarations

Ethics approval and consent to participate

The study was conducted in conformity with the Helsinki Declaration on Human Experimentation, 1964, with subsequent revisions, latest Seoul, October 2008. Ethical approval was obtained from the Ethics and Protocol Review Committee of the College of Health Sciences of the University of Ghana (Protocol ID number: CHS-Et/M.2–4.11/2018–2019) and each patient provided written voluntary informed consent after the rationale and procedure of the study were thoroughly explained.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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