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Excessive daytime sleepiness and associated factors in patients with type 2 diabetes mellitus: a cross-sectional study

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Abstract

***Aim*:** This study aimed to assess the prevalence and associated factors of excessive daytime sleepiness (EDS) in patients with type 2 diabetes mellitus (T2DM).
***Methods*:** We conducted a cross-sectional study in Beijing, China, from November 2015 to October 2016, and patients with T2DM were invited to participate. Structured questionnaires were used to collect data. EDS was assessed using the Epworth Sleepiness Scale (ESS), and anxiety and depression were assessed using the Hospital Anxiety and Depression Scale (HADS). Logistic regression analysis was used to evaluate factors associated with EDS.
***Results*:** Of the 224 patients with T2DM, 24 (10.7%) had EDS. The proportions of anxiety, depression, and comorbid anxiety and depression were significantly different between the EDS and non-EDS groups. ESS scores, anxiety scores, and depression scores were positively correlated. Logistic regression analysis showed that mild anxiety (OR 11.055; 95% CI 2.272–53.785; P = 0.003), moderate to severe anxiety (OR 33.223; 95% CI 4.896–225.440; P < 0.001), and mild depression (OR 6.227; 95% CI 1.319–29.399; P = 0.021) were associated with EDS in patients with T2DM. These associations remained significant after adjustment for age, sex, body mass index, diabetes duration, and apnoea–hypopnoea index. In addition, the severity of obstructive sleep apnoea–hypopnoea syndrome was not significantly associated with EDS, anxiety, depression, and comorbid anxiety and depression in this study.
***Conclusions*:** The prevalence of EDS was high in patients with T2DM, and anxiety and depression were significantly associated with EDS. When developing interventions to improve EDS in patients with T2DM, healthcare providers may need to consider interventions that target anxiety and depression.

Keywords: *type 2 diabetes mellitus*; *excessive daytime sleepiness*; *cross-sectional study*; *anxiety*; *depression*

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Excessive daytime sleepiness (EDS), one of the daily manifestations of obstructive sleep apnoea–hypopnoea syndrome (OSAHS), refers to an individual's inability to stay awake and alert during the main waking hours of the day, resulting in uncontrolled sleep.[1] Patients with OSAHS suffer from recurrent apnoea and hypopnea during nighttime sleep, often accompanied by snoring, frequent arousals, and sleep fragmentation, which can lead to daytime sleepiness.[2] People with EDS are prone to experiencing excessive sleepiness, unintentional falling asleep, long periods of sleep, and repeated naps.[3] These sleep-related symptoms are associated with cognitive impairment, decreased quality of life, poor work performance, and a high incidence of traffic accidents, posing a serious threat to patients' work and life.[4]

By 2021, approximately 537 million people worldwide suffer from diabetes.[5] China is a major epicentre of the growing global diabetes epidemic, with approximately 140 million people, more than 90% of whom have type 2 diabetes mellitus (T2DM).[5,6] Observational studies have reported that the prevalence of OSAHS in patients with T2DM in China is 60–80%, which is high.[7–10] Considering the adverse effects of EDS, the high prevalence of OSAHS in patients with T2DM and the large number of patients with T2DM in China, it is necessary for healthcare providers to focus on daytime sleepiness in patients with T2DM.

In the past decade, several studies have reported the prevalence of EDS in patients with T2DM. Ramtahal et al. reported an EDS rate of 11.3% in 291 patients with

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T2DM in Trinidad and found that the body mass index (BMI) of patients in the EDS group was significantly higher than that of patients in the non-EDS group (P = 0.04).[11] According to Arosemena Coronel et al., the prevalence of EDS was 56.8% in 290 participants with T2DM with severe hypoglycaemic episodes in Guayaquil, Ecuador, and a higher proportion of EDS was found in individuals aged 56–75 years (P < 0.001).[12] Raj et al. reported a prevalence of EDS of 17.5% in 102 patients with T2DM in Erode, Tamil Nadu, India.[13] However, these previous studies lacked the consideration of exploring factors associated with EDS in patients with T2DM. In addition, to the best of our knowledge, EDS in Chinese patients with T2DM remains to be explored.

Therefore, this study aimed to evaluate the prevalence and associated factors of EDS in patients with T2DM in China and to provide a reference for healthcare providers to develop appropriate interventions to improve EDS in patients with T2DM.

Methods

Study design

This was a cross-sectional study to investigate the prevalence and associated factors of EDS in patients with T2DM.

Ethical approval and informed consent

This study followed the Helsinki Declaration and was reviewed and approved by the Ethics Committee of Beijing Pinggu Hospital. All participants signed a written informed consent before participating in the study.

Setting and participants

This study was conducted at the Department of Endocrinology, Pinggu District Hospital, Beijing, from November 2015 to October 2016, and the participants were hospitalised patients. Inclusion criteria: 1) T2DM according to WHO 1999 criteria; 2) age ≥ 18 years; 3) no history of mental illness and no sedatives were used; 4) able to communicate with the researchers. Patients with serious physical illnesses such as malignant tumours and stroke who were unable to complete the survey were excluded. Information on history of mental illness was obtained by reviewing the electronic medical record (EMR) system and interviewing the patient.

Measurements

Assessment of demographic and clinical characteristics

The demographic and clinical information considered in this study consisted of age, gender, height, weight, BMI, systolic blood pressure (SBP), diastolic blood pressure (DBP), waist circumference (WC), smoking status, drinking status, duration of diabetes, glycosylated haemoglobin (HbA1c), fasting plasma glucose (FPG), serum creatinine (sCr), cholesterol (CHOL), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), urine microalbumin (uMA), lipid accumulation product (LAP), apnoea–hypopnoea index (AHI), oxygen desaturation index (ODI), lowest pulse oxygen saturation (LS_pO_2), hypertension, dyslipidaemia, coronary heart disease, cerebrovascular disease, and snoring. Patients enrolled in the study were invited for sleep monitoring and sleep-related data were recorded by the investigator. Demographic and clinical information was obtained from the EMR system.

BMI was calculated by dividing the weight (kg) by the square of height (m). Blood pressure was measured with an Omron electronic blood pressure monitor. FPG, sCr, CHOL, TG, LDL, and HDL were detected by the automatic biochemical analyser AU5400 (Beckman, USA). HbA1c was evaluated by automatic saccharification analyser D10 (Bio-Rad, USA), and uMA was detected by multifunctional quantitative gold standard detector NycoCard Reader II (Axis-shield, Norway). AHI, ODI, and LS_pO_2 were measured by overnight polysomnography using the Alice PDx portable sleep diagnostic system (Philips, USA). LAP (male) = (WC-65)*TG, and LAP (female) = (WC-58)*TG.

Assessment of daytime sleepiness

Daytime sleepiness was measured using the 8-item Epworth Sleepiness Scale (ESS).[14,15] Each item on the ESS is scored from 0 to 3, and the total score ranges from 0 to 24. The higher the total score, the greater the degree of daytime sleepiness. In this study, an ESS score ≥ 9 was defined as EDS. The Cronbach's alpha for the ESS in this sample was 0.808.

Assessment of anxiety and depression

The Hospital Anxiety and Depression Scale (HADS) has 14 items that can assess anxiety and depression, respectively.[16,17] Items 1, 3, 5, 7, 9, 11, and 13 assess anxiety, and items 2, 4, 6, 8, 10, 12, and 14 assess depression. Each item is scored from 0 to 3, and total scores for both the anxiety and depression subscales range from 0 to 21. A score of 0 to 7 indicates normal, 8 to 10 indicates mild anxiety or depression, 11 to 14 indicates moderate anxiety or depression, and 15 to 21 indicates severe anxiety or depression. In this study, the Cronbach's alpha of the anxiety subscale was 0.791 and the Cronbach's alpha of the depression subscale was 0.776.

Procedure

Data were collected between November 2015 and October 2016. All investigators received training prior to data collection. Structured questionnaires were used to collect

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information on demographic and clinical characteristics, anxiety, depression, and daytime sleepiness. The first two patients admitted to the hospital each day who met the inclusion criteria were invited to participate in the study. If only one case met the inclusion criteria that day, only that case would be recruited. Patients would not be recruited on that day if neither of the first two cases met the inclusion criteria. Patients took approximately 25 min to complete the questionnaire, which was collected on site by the investigator upon completion.

Statistical analysis

SPSS software (version 23.0) was used for statistical analysis. Participants were divided into EDS (ESS ≥ 9) and non-EDS (ESS < 9) groups based on their ESS scores. Continuous variables were expressed as mean and standard deviation (SD). The Kolmogorov–Smirnov test indicated that all continuous variables were not normally distributed, so the Mann–Whitney *U* test was used to compare group differences. Categorical variables were described using frequencies and percentages. Pearson's χ^2 test, continuity-corrected χ^2 test, and Fisher's exact test were used to compare group differences and post hoc analyses were performed with Bonferroni correction. Spearman correlation analysis was used to evaluate the correlation between variables. Logistic regression analysis was used to examine factors associated with EDS. A two-tailed $P < 0.05$ was considered statistically significant.

Results

Demographic and clinical characteristics of the participants

A total of 237 patients with T2DM were enrolled, of whom 13 were excluded due to incomplete data, and 224 were included in the final analysis. The mean age of the patients was 52.88 ± 12.90 years old, and 118 (52.7%) patients were male. The demographic and clinical characteristics of the patients with T2DM are shown in Table 1. There were 24 (10.7%) patients with anxiety, including 11 (4.9%) patients with mild anxiety, and 13 (5.8%) patients with moderate to severe anxiety. There were 29 (12.9%) patients with depression, including 16 (7.1%) patients with mild depression and 13(5.8%) patients with moderate to severe depression. In addition, 11(4.9%) patients had a comorbid anxiety and depression, and 24 (10.7%) patients had EDS.

Comparison of characteristics between EDS and non-EDS groups

Table 2 compares the demographic and clinical characteristics of the EDS and non-EDS groups. The prevalence of EDS in patients with different levels of anxiety was significantly different ($P < 0.001$). Post hoc multiple comparisons showed that the prevalence of EDS was significantly higher in the mild ($P = 0.003$, Bonferroni correction $P = 0.0167$) and moderate-to-severe ($P < 0.001$, Bonferroni correction $P = 0.0167$) anxiety groups compared to the normal group. The level of depression was also significantly correlated with the prevalence of EDS ($P = 0.001$). Post hoc multiple comparisons showed that the prevalence of EDS was significantly higher in the mild depression group than in the normal group ($P = 0.001$, Bonferroni correction $P = 0.0167$). Patients with both anxiety and depression had significantly higher rates of EDS than those who didn't have both conditions ($P < 0.001$). Other participant characteristics were not significantly different between the EDS and non-EDS groups ($P > 0.05$). The Bonferroni correction is the correction for *P*-values. For example, if three groups are compared, three pairwise comparisons are needed to see if there is a difference between each group. If one comparison is made, the *P*-value is 0.05; if three comparisons are made, the *P*-value is 0.167.

Correlation analysis of anxiety scores, depression scores, ESS scores, and AHI

Table 3 shows the results of the correlation analysis of anxiety scores, depression scores, ESS scores, and AHI. ESS scores were positively correlated with anxiety scores ($r = 0.185$, $P < 0.01$) and depression scores ($r = 0.282$, $P < 0.001$), and anxiety scores were also positively correlated with depression scores ($r = 0.602$, $P < 0.001$). There was no significant correlation between AHI and ESS scores, anxiety scores, and depression scores ($P > 0.05$).

Logistic regression analysis of factors associated with EDS

Variables with $P < 0.05$ in the univariate analysis were included in the logistic regression analysis. The results of the logistic regression analysis of factors associated with EDS are shown in Table 4. Mild anxiety (OR 11.055; 95%CI 2.272–53.785; $P = 0.003$), moderate to severe anxiety (OR 33.223; 95%CI 4.896–225.440; $P < 0.001$), and mild depression (OR 6.227; 95% CI 1.319–29.399; $P = 0.021$) were associated with EDS in patients with T2DM. These associations remained after further adjustment for age, gender, BMI, diabetes duration, and AHI.

Comparison of OSAHS severity with EDS, anxiety, depression, and comorbid anxiety and depression

Table 5 shows the relationship between OSAHS severity and EDS, anxiety, depression, and comorbid anxiety and depression. EDS, anxiety, depression, and comorbid anxiety and depression were not significantly associated with OSAHS severity ($P > 0.05$).

Discussion

This study investigated the prevalence and associated factors of EDS in patients with T2DM. The results showed that approximately one tenth (10.7%) of patients with T2DM had

Table 1. Demographic and clinical characteristics of patients with T2DM

Variable	(*N* = 224) Mean ± SD or *n* (%)
Age (years)	54.88 ± 12.90
Gender	
Male	118 (52.7%)
Female	106 (47.3%)
Height (cm)	164.70 ± 7.78
Weight (kg)	72.95 ± 14.78
BMI (kg/m^2)	26.75 ± 4.28
SBP (mmHg)	129.67 ± 14.58
DBP (mmHg)	78.39 ± 10.03
Diabetes duration (months)	113.14 ± 89.77
HbA1c (%)	9.57 ± 2.20
FPG (mmol/L)	10.40 ± 3.97
sCr (μmol/L)	63.55 ± 20.40
uMA (mg/L)	59.91 ± 102.18
CHOL (mmol/L)	4.86 ± 1.16
TG (mmol/L)	2.46 ± 1.93
LDL (mmol/L)	2.57 ± 0.85
HDL (mmol/L)	1.20 ± 0.31
WC (cm)	95.39 ± 10.45
LAP	87.22 ± 82.64
AHI (times/h)	14.60 ± 15.57
ODI (times/h)	13.57 ± 15.13
LS_PO_2 (%)	81.18 ± 9.41
Smoking	
Yes	109 (48.7)
No	115 (51.3)
Drinking	
Yes	76 (33.9)
No	148 (66.1)
Hypertension	
Yes	127 (56.7)
No	97 (43.3)
Dyslipidaemia	
Yes	169 (75.4)
No	55 (24.6)
Coronary heart disease	
Yes	26 (11.6)
No	198 (88.4)
Cerebrovascular disease	
Yes	33 (14.7)
No	191 (85.3)
Snoring	
Never	30 (13.4)
Occasionally	68 (30.4)
Frequently	91 (40.6)
Not known	35 (15.6)
Comorbid anxiety and depression	
Yes	11 (4.9)
No	213 (95.1)
Anxiety	
Normal	200 (89.3)
Mild	11 (4.9)
Moderate/severe	13 (5.8)
Depression	
Normal	195 (87.1)
Mild	16 (7.1)
Moderate/severe	13 (5.8)
EDS	
Yes	24 (10.7)
No	200 (89.3)

EDS, excessive daytime sleepiness; T2DM, type 2 diabetes mellitus; SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HbA1c, glycosylated haemoglobin; FPG, fasting plasma glucose; sCr, serum creatinine; CHOL, cholesterol; TG, triglycerides; LDL, low-density lipoprotein; HDL, high-density lipoprotein; WC, waist circumference; uMA, urine microalbumin; LAP, lipid accumulation product; AHI, apnoea–hypopnoea index; ODI, oxygen desaturation index; LS_PO_2, lowest pulse oxygen saturation.

EDS, which is consistent with the results reported by Ramtahal et al. and Raj et al. (11.3 and 17.5%, respectively).[11,13] Given the high prevalence of EDS, healthcare providers should promptly assess daytime sleepiness in patients with T2DM to further develop strategies for improvement.

The significant relationship between anxiety and EDS has been reported in many groups, such as patients with OSAHS, medical students, and adults of different ages.[18–21] This study found that anxiety was associated with EDS in patients with T2DM, which identified a significant relationship between anxiety and EDS in the field of T2DM. Previous studies have shown that patients with anxiety are prone to sleep disturbances due to excessive anxiety and worry, such as insomnia, difficulty staying asleep, and poor sleep quality.[22–24] These sleep problems interfere with the normal nocturnal sleep process and may lead to increased daytime sleepiness.[25,26] Therefore, the co-occurrence of anxiety and sleep problems may explain the significant association between anxiety and EDS in T2DM patients. Furthermore, studies have shown that sleep problems may trigger or further exacerbate anxiety, which means that the relationship between anxiety and sleep problems may be bidirectional.[22,27,28] As reported by Luo et al., EDS at baseline could predict new-onset anxiety at follow-up in rural Chinese adolescents.[29] Future longitudinal studies are needed to further investigate the relationship between daytime sleepiness and anxiety in patients with T2DM.

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Table 2. Comparison of characteristics between EDS and non-EDS groups in patients with T2DM

Variable	EDS (*n* = 24) Mean ± SD or *n* (%)	Non-EDS (*n* = 200) Mean ± SD or *n* (%)	Z/χ^2 value	*P*
Age (years)	52.96 ± 13.67	55.11 ± 12.82	-0.622	0.534
Gender				
Male	13 (54.2)	105 (52.5)	0.024	0.877
Female	11 (45.8)	95 (47.5)		
Height (cm)	166.04 ± 8.88	164.54 ± 7.65	-0.557	0.577
Weight (kg)	76.19 ± 17.06	72.57 ± 14.48	-0.797	0.425
BMI (kg/m^2)	27.44 ± 4.62	26.67 ± 4.24	-0.665	0.506
SBP (mmHg)	131.25 ± 14.31	129.49 ± 14.63	-0.377	0.706
DBP (mmHg)	79.58 ± 13.01	78.25 ± 9.65	-0.196	0.845
Diabetes duration (months)	127.79 ± 81.64	111.38 ± 90.73	-1.050	0.294
HbA1c (%)	9.42 ± 2.46	9.59 ± 2.17	-0.757	0.449
FPG (mmol/L)	10.04 ± 5.14	10.44 ± 3.82	-1.147	0.252
sCr (μmol/L)	65.50 ± 20.98	63.32 ± 20.37	-0.557	0.578
uMA (mg/L)	84.91 ± 164.22	56.91 ± 92.19	-0.474	0.635
CHOL (mmol/L)	4.83 ± 1.20	4.87 ± 1.15	-0.195	0.845
TG (mmol/L)	2.36 ± 1.30	2.47 ± 2.00	-0.220	0.826
LDL (mmol/L)	2.65 ± 0.89	2.56 ± 0.84	-0.572	0.568
HDL (mmol/L)	1.17 ± 0.29	1.21 ± 0.32	-0.400	0.689
WC (cm)	98.02 ± 11.38	95.07 ± 10.32	-0.959	0.338
LAP	88.57 ± 66.38	87.05 ± 84.52	-0.527	0.598
AHI (times/h)	19.38 ± 20.66	14.03 ± 14.81	-0.842	0.400
ODI (times/h)	16.67 ± 18.13	13.20 ± 14.74	-0.532	0.594
LS_pO_2 (%)	81.79 ± 7.66	81.10 ± 9.61	-0.447	0.655
Smoking				
Yes	10 (41.7)	99 (49.5)	0.526	0.468
No	14 (58.3)	101 (50.5)		
Drinking				
Yes	11 (45.8)	65 (32.5)	1.699	0.192
No	13 (54.2)	135 (67.5)		
Hypertension				
Yes	15 (62.5)	112 (56.0)	0.369	0.544
No	9 (37.5)	88 (44.0)		
Dyslipidaemia				
Yes	17 (70.8)	152 (76.0)	0.309	0.578
No	7 (29.2)	48 (24.0)		
Coronary heart disease				
Yes	5 (20.8)	21 (10.5)	1.337	0.248
No	19 (79.2)	179 (89.5)		
Cerebrovascular disease				
Yes	6 (25.0)	27 (13.5)	1.433	0.231
No	18 (75.0)	173 (86.5)		
Snoring				
Never	3 (12.5)	27 (13.5)	5.752	0.116
Occasionally	5 (20.8)	63 (31.5)		
Frequently	15 (62.5)	76 (38.0)		
Not known	1 (4.2)	34 (17.0)		
Comorbid anxiety and depression				
Yes	6 (25.0)	5 (2.5)	18.663	<0.001
No	18 (75.0)	195 (97.5)		

Table 2. (Continued)

Variable	EDS (*n* = 24) Mean ± SD or n (%)	Non-EDS (*n* = 200) Mean ± SD or *n* (%)	Z/χ^2 value	*P*
Anxiety				
Normal	13 (54.2)	187 (93.5)	25.454	<0.001
Mild	4 (16.7)	7 (3.5)		
Moderate/severe	7 (29.2)	6 (3.0)		
Depression				
Normal	15 (62.5)	180 (90.0)	12.878	0.001
Mild	6 (25.0)	10 (5.0)		
Moderate/severe	3 (12.5)	10 (5.0)		

Note: Mann–Whitney U test, Pearson's χ^2 test, continuity corrected χ^2 test, and Fisher's exact test were used to compare differences between groups. EDS, excessive daytime sleepiness; T2DM, type 2 diabetes mellitus; SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HbA1c, glycosylated haemoglobin; FPG, fasting plasma glucose; sCr, serum creatinine; CHOL, cholesterol; TG, triglycerides; LDL, low-density lipoprotein; HDL, high-density lipoprotein; WC, waist circumference; uMA, urine microalbumin; LAP, lipid accumulation product; AHI, apnoea–hypopnoea index; ODI, oxygen desaturation index; LS_pO_2, lowest pulse oxygen saturation.

Table 3. Correlation analysis of anxiety scores, depression scores, ESS scores, and AHI

	Mean ± SD	Anxiety scores	Depression scores	ESS scores	AHI
Anxiety scores	3.16 ± 3.79	1			
Depression scores	3.13 ± 3.91	0.602***	1		
ESS scores	2.97 ± 3.93	0.185**	0.282***	1	
AHI	14.60 ± 15.57	-0.091	-0.082	0.114	1

Note: **$P < 0.01$; ***$P < 0.001$.
SD, standard deviation; AHI, apnoea–hypopnoea index; ESS, Epworth Sleepiness Scale.

Table 4. Logistic regression analysis of factors associated with EDS in patients with T2DM

Variable	Model 1		Model 2	
	OR (95%CI)	*P*	OR (95%CI)	*P*
Anxiety				
Mild (ref: normal)	7.925 (1.755–35.779)	0.007	11.055 (2.272–53.785)	0.003
Moderate/severe (ref: normal)	16.881 (3.171–89.858)	0.001	33.223 (4.896–225.440)	<0.001
Depression				
Mild (ref: normal)	5.092 (1.171–22.141)	0.030	6.227 (1.319–29.399)	0.021
Moderate/severe (ref: normal)	1.287 (0.147–11.292)	0.820	1.452 (0.142–14.898)	0.753
Comorbid anxiety and depression	0.641 (0.060–6.899)	0.714	0.407 (0.032–5.104)	0.486

Note: Logistic regression analysis was performed to investigate the factors associated with EDS. Model 1: variables with $P < 0.05$ in univariate analysis were included; Model 2: variables with $P < 0.05$ in univariate analysis were included and adjusted for age, gender, BMI, diabetes duration, and AHI.
EDS, excessive daytime sleepiness; T2DM, type 2 diabetes mellitus; OR, odds ratio; CI, confidence interval; BMI, body mass index; AHI, apnoea–hypopnoea index.

In conclusion, it is necessary for healthcare providers to assess the anxiety levels in patients with T2DM and interventions targeting anxiety may be helpful for EDS.

We also found that depression was associated with EDS in patients with T2DM. Previous studies have focused on the association between EDS and depression in different populations, such as schoolchildren, adolescents, college students, patients with myotonic dystrophy, and older adults with DM.[25,30–33] This study confirmed the significant association between depression and EDS in the context of T2DM. Previous studies have reported that more than half of people with depression have sleep disturbances, such as insomnia, leading to impaired concentration and increased daytime sleepiness.[34–36] According to the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5), insomnia is also one of the diagnostic criteria for the major depressive disorder.[37] Thus, depression is most often associated with sleep problems,

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Table 5. Comparison of OSAHS severity with EDS, anxiety, depression, and comorbid anxiety and depression in patients with T2DM

Variable	Non-OSAHS AHI < 5 (*n* = 64)	Mild OSAHS AHI 5–15 (*n* = 87)	Moderate/severe OSAHS AHI > 15 (*n* = 73)	χ^2 value	*P*
EDS					
Yes	7 (10.9)	7 (8.0)	10 (13.7)	1.330	0.514
No	57 (89.1)	80 (92.0)	63 (86.3)		
Anxiety					
Normal	54 (84.4)	79 (90.8)	67 (91.8)	3.548	0.475
Mild	4 (6.3)	5 (5.7)	2 (2.7)		
Moderate/severe	6 (9.4)	3 (3.4)	4 (5.5)		
Depression					
Normal	54 (84.4)	79 (90.8)	62 (84.9)	6.087	0.181
Mild	6 (9.4)	2 (2.3)	8 (11.0)		
Moderate/severe	4 (6.3)	6 (6.9)	3 (4.1)		
Comorbid anxiety and depression					
Yes	4 (6.3)	3 (3.4)	4 (5.5)	0.835	0.737
No	60 (93.8)	84 (96.6)	69 (94.5)		

Note: Data expressed as *N* (%). Pearson's χ^2 test, continuity corrected χ^2 test, and Fisher's exact test were used to compare differences between groups. EDS, excessive daytime sleepiness; T2DM, type 2 diabetes mellitus; AHI, apnoea–hypopnoea index; OSAHS, obstructive sleep apnoea–hypopnoea syndrome.

which may explain the significant association between depression and EDS in patients with T2DM. Furthermore, insomnia increases the risk of depression,[25,38] suggesting that there may also be a bidirectional relationship between sleep problems and depression. Future longitudinal studies are also needed to further investigate the causal relationship between daytime sleepiness and depression in patients with T2DM. Healthcare providers should promptly assess depression levels in patients with T2DM, and treatment of depression may help to reduce EDS.

Zhang et al. and Shao et al. reported that among patients with OSAHS, the AHI of patients with daytime sleepiness was significantly higher than that of patients without daytime sleepiness.[39,40] Patients with OSAHS have sleep disturbances due to recurrent apnoea and hypopnoea during nocturnal sleep, and daytime sleepiness is a common clinical symptom.[2] AHI is an important diagnostic indicator of OSAHS and the basis for assessing the severity of OSAHS.[41] The higher the AHI, the more severe the patient's daytime sleepiness. However, in patients with T2DM, we did not observe a significant difference in AHI between the EDS and non-EDS groups. We also grouped patients with T2DM according to AHI and compared the group differences in EDS. The results showed no significant difference in EDS between the non-OSAHS group, the mild OSAHS group, and the moderate to severe OSAHS group in patients with T2DM. Interestingly, the relationship between daytime sleepiness and AHI was also insignificant in chronic kidney disease, coronary heart disease, and heart failure.[42–44] The body's response to these conditions, such as the impact and damage to the nervous system, may affect an individual's subjective perception of sleepiness caused by apnoea and hypopnoea.[42] Therefore, the mechanism of daytime sleepiness in patients with T2DM needs further investigation. In addition, we found that anxiety, depression, and comorbid anxiety and depression were not significantly associated with different levels of AHI in patients with T2DM, similar to previous studies.[45,46] However, other studies reported that anxiety and depression were significantly associated with OSAHS.[47,48] Considering the possible influence between anxiety, depression and AHI, we controlled for AHI in the logistic regression model. Therefore, the effects of anxiety and depression on EDS in our sample were independent of AHI.

This study has several strengths. Firstly, the study reported a high prevalence of EDS in patients with T2DM, which helps to promote the attention of healthcare providers to EDS in patients with T2DM. Secondly, the finding that anxiety and depression are factors associated with EDS in patients with T2DM provides concrete guidance for healthcare providers to develop strategies to improve EDS in patients with T2DM. For example, interventions for anxiety and depression may benefit EDS in patients with T2DM. Thirdly, this study found that the relationship between EDS and AHI in patients with T2DM was not significant, which provides a basis for further research into the potential mechanism of EDS in patients with T2DM.

Limitations

The study also has several limitations. Firstly, the cross-sectional study design limited the causal inference ability of

the results of this study and the results of logistic regression could only show a significant association between anxiety and depression and EDS. Secondly, only hospitalised patients with T2DM were included in this study, which may limit the applicability of these results to non-hospitalised patients with T2DM. Compared to non-hospitalised patients with T2DM, hospitalised patients with T2DM may be less healthy and the neurological impact of the disease may be more severe, which may be associated with EDS. Thirdly, although there are many objective indicators in this study, anxiety, depression, and EDS were all assessed by patient self-report questionnaires, which may lead to inevitable recall and reporting bias.

Conclusions

Patients with T2DM have a high prevalence of EDS, and anxiety and depression are factors associated with EDS. Healthcare providers should pay more attention to anxiety, depression, and daytime sleepiness in patients with T2DM and implement timely interventions to promote mental and physical health and reduce daytime sleepiness. Interventions for anxiety and depression may be beneficial in improving daytime sleepiness levels in patients with T2DM. Future studies should use more rigorous designs to investigate the causal relationship between anxiety, depression and EDS.

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Conflicts of interest and funding

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Data availability statement

The datasets generated during and/or analysed in this study are available from the corresponding author upon reasonable request.

Authors' contributions

Mingzi Li designed the study and revised the manuscript, Xiangshuang Kong collected the data, and Jing Huang performed the statistical analysis and wrote the manuscript.

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