Sleep Quality Assessment in Correlation to Autonomic Nerve Function in Type 2 Diabetic Patients

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Background and Objective

Diabetes mellitus negatively impacts the quality of life of its patients. Autonomic dysfunction may disturb sleep quality by negatively affecting multiple systems, including, but not limited to, the cardiovascular, respiratory, and genitourinary tracts. The current study aims to assess sleep quality and examine the degree of correlation with autonomic nervous function in relatively well-controlled type 2 diabetic patients.

Methods

This study uses a cross-sectional design to assess sleep quality in 88 type 2 diabetic patients via the Pittsburg Sleep Quality Index (PSQI), accordingly dividing them into two groups: good or poor sleepers. Subsequently, the study evaluates autonomic nerves’ conductivity by measuring electrical skin conductance using Sudoscan for the hands and feet.

Results

Of 88 recruited patients, 53% showed poor sleep quality, with higher incidence in females. Autonomic nerve conductivity showed moderate damage in poor sleepers with 59.53 ± 13.35 μS and 59.68 ± 16.91 μS of hand and foot electrodes, respectively. Autonomic damage induces sleep disturbance mainly through increased nighttime voiding in 91.49% of the poor sleepers group compared to 41.46% in the good sleepers group. PSQI score was found to strongly and inversely correlate with autonomic nerve conductivity via hand electrodes, with a correlation coefficient of -0.62 and a determination coefficient of 0.39.

Conclusion

Poor sleep quality seems to be a significant problem even in relatively well-controlled type 2 diabetic patients with no diagnosed micro- or macrovascular complications. Autonomic dysfunction negatively affects the quality of sleep and leads to sleep disturbance by increasing nighttime micturition as one of its complications.

INTRODUCTION

The prevalence of type 2 diabetes mellitus (T2DM) has increased globally by at least fourfold in the last 35 years based on World Health Organization (WHO) reports [1]. The Middle East, including Iraq, is one of the worldwide hotspots of diabetes, with an incidence of 8.2% of the population in 2017, and this number is expected to rise to 9.4% in 2045 [2].

A bidirectional influence seems to control the relationship between diabetes and poor sleep quality. On the one hand, diabetes mellitus is a leading factor for developing nocturia. Nocturia, in turn, induces osmotic diuresis in response to sustained elevation of urine glucose to a level that exceeds the reabsorption capacity of the proximal renal tubules [3]. Consequently, nocturia prolongs sleep latency, reduces sleep duration, and intensifies daytime exhaustion [4]. The greater the number of nighttime voids, the poorer the sleep quality index score; that’s why nocturia is considered as an independent driver of general poor sleep.

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quality in about 71% of patients with concomitant overactive bladder [5].

Poor sleep quality, on the other hand, contributes to the progression of diabetes probably by increasing peripheral insulin resistance. Reduction of sleep duration down to 4 hours for a single night only is associated with an increase of peripheral insulin resistance by 19%–25% [6]. This is probably because sleep disturbance is a significant trigger for stress hormones like cortisol, a hyperglycemic agent and appetite inducer that induces white adipose tissue deposition deep in the abdomen [7]. Additionally, sleep disturbance significantly reduces glucagon-like peptide-1 (GLP1), an incretin hormone that reduces the appetite by reducing gastric emptying and intestinal motility, optimizing nutrient absorption, and mitigating postprandial metabolic disruption [8].

Autonomic neuropathy is one of the early microvascular complications of diabetes. Sustained hyperglycemia is a leading factor in the generation of advanced glycation end products (AGEs). The loss of normal endothelial vascular function occurs when AGEs bind to their specific receptors distributed among these endothelial cells. The activation of the receptors for AGEs (RAGEs) triggers a low-grade inflammatory response by reducing the production of proinflammatory mediators like TNF-α and NF-kB. RAGE activation also increases the levels of reactive oxygen species, which are another trigger of proinflammatory mediators [9]. Over time, sustained inflammation at the level of the microvasculature results in the degeneration of the neurons supplied by those capillaries [10]. AGEs themselves may have a direct degenerative neuronal effect, whereby the induced inflammatory process at the neuronal level ends with epineuria, perineuria, and endoneuria [11-13].

Autonomic neuropathy negatively affects several organs, including the cardiovascular, gastrointestinal, and genitourinary tracts. The relationship between autonomic neuropathy and sleep quality can be attributed to several mechanisms. The respiratory system is under the control of the autonomic nervous system during sleep. Autonomic neuropathy is considered as an independent factor affecting sleep quality by inducing sleep apnea [14]. Vagal nerves inhibit heart rate and blood pressure and control the non-rapid eye movement phase of the sleep cycle [15]. The damage of parasympathetic reflexes, which occurs first in autonomic dysfunction, significantly contributes to the development of obstructive sleep apnea through the dominating effect of the sympathetic tone, which induces arousal, hypoxemia, and hypercapnia [16,17].

The negative effect on the genitourinary tract represents another mechanism that potentially correlates autonomic neuropathy with sleep quality. Sympathetic, parasympathetic, and somatic innervation are involved in controlling the filling, storage, and emptying of the urinary bladder. Bladder filling is coordinated by a urethral reflex called the guarding reflex. The guarding reflex primarily involves the activation of the sympathetic tone, which serves to maintain the urethral sphincter contraction by activating α1 receptors and inducing relaxation of the detrusor muscle by activating β3 receptors. Parasympathetic tone of the smooth detrusor muscles is also inhibited during bladder filling [18]. Bladder emptying is partially under parasympathetic control, which is mediated via the contraction of the detrusor muscle via the activation of M3 receptors. The process is also accompanied by a reduction of the sympathetic tone, which facilitates external sphincter relaxation [19]. Voluntary control of micturition is mediated via somatic innervation, which activates the nicotinic receptor in the striated muscles of the urethral sphincter to induce muscle contraction and keep the sphincter closed [20]. Somatic innervation delays the urgency to void; however, when the bladder is full, reflexive voiding becomes the dominant means to induce micturition [21].

In a fashion similar to its effect on the respiratory system, vagal innervation of the bladder is the first to be affected by sustained hyperglycemia as a result of sacral parasympathetic damage, associated with a progressive dominance of sympathetic activity [22]. Parasympathetic damage is characterized by urinary bladder hypo- or areflexia associated with increased voiding residual volume and urinary retention [23]. Twenty-three percent of diabetic patients suffer from impaired detrusor contractility, and 10% complain of urinary areflexia. On the other hand, 55% of diabetic patients suffer from detrusor hyperreflexia, which occurs due to either somatic or sympathetic neuronal damage [24]. In this case, excessive sweating, hypertension, and reflex bradycardia can be noticed as clinical manifestations [25]. Neurogenic bladder negatively affects patients’ quality of life, especially in terms of anxiety and sleep disturbance [26-28].

Based on that, this research aims to assess sleep quality in T2DM patients using the Pittsburg Sleep Quality Index (PSQI), a trustworthy questionnaire that has an assured sensitivity score of 89.6%, specificity of 86.5%, and reliability of 0.87 [29,30]. The research then tries to find out the correlation between the sleep quality of the whole group of surveyed patients and their autonomic function, independently of their glycemic state and other major cofactors that may interfere with the quality of sleep (as mentioned in the inclusion and exclusion criteria). Evaluation of autonomic nerve function was conducted using Sudoscan (Impeto Medical Corp., Paris, France), a non-invasive, easily accessible, software-controlled apparatus. Sudoscan quantitatively assesses autonomic nervous conductivity by applying low-voltage current (lower than 4 V) to the hands and feet through four nickel electrodes. Sweat glands’ small, unmyelinated C fibers in both palms and soles are transdermally affected by the voltage gradient applied through reverse iontophoresis, triggering a restricted movement of chloride ions via the sweat ducts since the stratum corneum acts as an isolative barrier [31]. The generated electrochemical reaction is detected by Sudoscan’s four electrodes. The level of autonomic fiber damage is proportionally
associated with a reduction of the detected electrochemical conductance. Sudoscan has been shown to have an experimentally approved sensitivity of 92% and a specificity of 78% [32].

METHODS

The research was a prospective, observational, cross-sectional, comparative study that involved screening 88 T2DM patients and distributing them into two groups based on their sleep quality: good vs. poor sleepers. Autonomic nerves’ conductivity was then quantitatively measured, and its mean values in the two study groups were comparatively assessed. The comparison involves other parameters like anthropometric measurements, age, and the duration of disease. A correlation and regression plot was subsequently applied to understand the relationship between sleep quality and autonomic nerves’ functionality.

The study was conducted in a private diabetic center in Baghdad, Iraq. Patients’ recruitment was performed over a period of four consecutive months from January 20 to April 16, 2023.

The study sample involved 51 male and 37 female patients. The average age of the total sample is 50.83 ± 8.58 years. The inclusion and exclusion criteria were designed to limit the confounding factors as much as possible. Inclusion criteria included:
- Restricting the recruited patients’ ages to between 35 and 65 years to avoid age-dependent sleep pattern disturbances.
- HbA1C levels of less than 8% to limit the effects of sleep-disturbing nocturia due to uncontrolled hyperglycemia.
- Patients with a disease duration of ≥ 2 years, as this is the time frame when autonomic neuropathy starts to develop.

Additionally, the study excluded several factors that may negatively affect sleep quality, including peripheral neuropathy, diabetic foot, benign prostatic hyperplasia, hypothyroidism, certain medications like antidepressants and β-blockers, inflammatory pelvic diseases, and stress.

The PSQI was used to subjectively assess sleep quality in the enrolled patients. The questionnaire was intentionally designed to obtain quantitative outcomes describing patients’ sleep quality. While the patients with a final PSQI score of ≤ 5 are classified as good sleepers, those with a final score of > 5 are classified with poor sleep quality.

Sudoscan was used to quantitatively measure the autonomic nerves’ conductivity. The procedure is fast, limited to only 3–5 minutes. The results are displayed on a monitor, where the conductivity of the unmyelinated autonomic neurons is measured in microsiemens (μS), where ESC ≥ 60 μS is classified as functionally normal, 40–60 μS is considered as moderately dysfunctional, and if ESC was < 40 μS, the autonomic nerves are considered to suffer from severe dysfunction.

Statistical Analysis
Microsoft Office Professional Plus 2016 and XLSTAT 2023 were used to organize, summarize, and analyze the collected data. All research variables were quantitatively measured, and their symmetrical distribution in terms of central tendency and dispersion was defined using mean and standard deviation, respectively.

Good and poor sleep quality were compared by using a two-sample t-test assuming unequal variance. Meanwhile, a correlation coefficient was used to measure how the variables were linearly associated with each other, where linear regression described the magnitude of association between the variables, and to derive a mathematical formula to predict the value of the dependent variable through the explanatory variables.

Ethics Approval
The study design was reviewed and accredited by the Institutional Review Board (IRB), College of Medicine, Al-Nahrain University, Iraq on January 10, 2023: No. 87/3/2. Enrolled patients signed an informed consent as a part of the commitment to transparency and ethics.

RESULTS

Eighty-eight T2DM patients were strictly recruited to match a deliberate set of inclusion and exclusion criteria. The average age of the total sample was 50.83 ± 8.58 years, the average duration of diabetes was 7.22 ± 4.93 years, and the average HbA1C was 6.93% ± 0.09%. Anthropometric parameters in the current study indicated an average weight of the total sample of 86.39 ± 14.69 kg, an average height of 1.67 ± 0.08 m, an average waist circumference of 101.94 ± 13.60 cm, and finally an average body mass index (BMI) of 31.04 ± 5.81 kg/m². Accordingly, the mean BMI of the total recruited patients in the current study falls within the obesity range (Table 1).

The number of male patients was 51, representing 58% of the sample; female patients numbered 37, representing 42% of the total population. Female patients were significantly older and shorter, with a greater waist circumference and a BMI mean value of 34.23 ± 5.99 kg/m², falling in the obesity range, unlike that of the male patients, which fell in the overweight range with a mean of 28.59 ± 4.63 kg/m². The mean duration of diabetes, patients’ weight, and HbA1C showed insignificant differences based on gender. Table 1 describes differences in general characteristics between males and females.

Sleep Quality Assessment
Out of 88 enrolled T2DM patients, 41 (47%) exhibited good sleep quality with a PSQI score ≤ 5. The other 47 patients (53%) were classified as having poor sleep quality, since their total PSQI score was > 5. Women in the sample showed worse sleep habits, with 59% categorized as poor sleepers (22 out of 37 female patients). Men, on the other hand, showed better outcomes, with

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49% named as poor sleepers (25 out of 51 male patients). Comparing the good sleep quality group to the poor sleep group shows insignificant differences in the anthropometric measurements, duration of disease, age, and HbA1C, with the exception of waist circumference (Table 2).

Table 3 summarizes the differences in the seven components of the PSQI between the good and poor sleeper groups. All PSQI functions revealed statistically significant differences, with the exception of the “use of medications” component. It also clarifies how the seven components of the questionnaire unequally contribute to the final PSQI score. “Sleep latency” acted as a main driver, representing 24% of the total points scored in the PSQI questionnaires filled by the 88 enrolled patients in the current cross-sectional study. The second driver was “sleep disturbances,” with a 21% contribution to the total points calculated from the answers of the surveyed sample. “Sleep duration” had

### Table 1. Comparison of general characteristics of the recruited T2DM patients based on gender

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total (n = 88)</th>
<th>Male (n = 51, 58%)</th>
<th>Female (n = 37, 42%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of diabetes (yr)</td>
<td>7.22 ± 4.93</td>
<td>6.80 ± 4.75</td>
<td>7.78 ± 5.10</td>
<td>0.370</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>50.83 ± 8.58</td>
<td>49.24 ± 8.43</td>
<td>53.03 ± 8.30</td>
<td>0.041*</td>
</tr>
<tr>
<td>HbA1C (%)</td>
<td>6.93 ± 0.09</td>
<td>6.96 ± 0.87</td>
<td>6.88 ± 0.92</td>
<td>0.690</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86.39 ± 14.69</td>
<td>85.04 ± 14.30</td>
<td>88.24 ± 15.01</td>
<td>0.320</td>
</tr>
<tr>
<td>Anthropometric measurements</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.67 ± 0.08</td>
<td>1.72 ± 0.06</td>
<td>1.61 ± 0.04</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>101.94 ± 13.60</td>
<td>97.43 ± 9.48</td>
<td>108.16 ± 15.79</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>31.04 ± 5.81</td>
<td>28.59 ± 4.63</td>
<td>34.23 ± 5.99</td>
<td>&lt; 0.001**</td>
</tr>
</tbody>
</table>

Data are presented as mean ± standard deviation.
* p < 0.05; ** p < 0.001.
T2DM, type 2 diabetes mellitus.

### Table 2. Comparison of general characteristics of good and poor sleepers

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Good sleepers (n = 41, 47%)</th>
<th>Poor sleepers (n = 47, 53%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>M/F</td>
<td>26/15</td>
<td>25/22</td>
<td>-</td>
</tr>
<tr>
<td>Duration of diabetes (yr)</td>
<td>7.85 ± 4.98</td>
<td>6.66 ± 4.81</td>
<td>0.263</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>51.98 ± 9.13</td>
<td>49.83 ± 7.94</td>
<td>0.251</td>
</tr>
<tr>
<td>HbA1C (%)</td>
<td>6.91 ± 1.02</td>
<td>6.94 ± 0.76</td>
<td>0.890</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>84.59 ± 13.17</td>
<td>87.96 ± 15.73</td>
<td>0.282</td>
</tr>
<tr>
<td>Anthropometric measurements</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.67 ± 0.08</td>
<td>1.67 ± 0.07</td>
<td>0.961</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>97.8 ± 11.37</td>
<td>105.6 ± 15.34</td>
<td>0.007*</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>30.29 ± 5.81</td>
<td>31.55 ± 6.07</td>
<td>0.326</td>
</tr>
</tbody>
</table>

Data are presented as mean ± standard deviation.
* p < 0.05.

### Table 3. PSQI components’ contributions and scores in good and poor sleep quality groups

<table>
<thead>
<tr>
<th>PSQI component</th>
<th>Function contribution (%)</th>
<th>Good sleep quality (n = 41)</th>
<th>Poor sleep quality (n = 47)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjective sleep quality</td>
<td>16</td>
<td>0.34 ± 0.47</td>
<td>1.45 ± 0.92</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Sleep latency</td>
<td>24</td>
<td>0.59 ± 0.58</td>
<td>2.15 ± 0.77</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Sleep duration</td>
<td>19</td>
<td>0.83 ± 0.49</td>
<td>1.32 ± 0.99</td>
<td>0.004*</td>
</tr>
<tr>
<td>Habitual sleep efficiency</td>
<td>2</td>
<td>0 ± 0</td>
<td>0.26 ± 0.44</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Sleep disturbances</td>
<td>21</td>
<td>1.05 ± 0.38</td>
<td>1.38 ± 0.58</td>
<td>0.001*</td>
</tr>
<tr>
<td>Use of medication</td>
<td>3</td>
<td>0.1 ± 0.43</td>
<td>0.21 ± 0.58</td>
<td>0.296</td>
</tr>
<tr>
<td>Daytime dysfunction</td>
<td>15</td>
<td>0.71 ± 0.55</td>
<td>1.06 ± 0.52</td>
<td>0.003*</td>
</tr>
<tr>
<td>Final PSQI score</td>
<td>100</td>
<td>3.61 ± 1.03</td>
<td>7.83 ± 2.31</td>
<td>&lt; 0.001**</td>
</tr>
</tbody>
</table>

Data are presented as mean ± standard deviation.
* p < 0.05; ** p < 0.001.
PSQI, Pittsburg Sleep Quality Index.
a vital impact of 19% of the total calculated PSQI score. “Subjective sleep quality” and “daytime dysfunction” contributed 16% and 15% of the score, respectively. Finally, “use of medication” and “habitual sleep efficiency” were the weakest contributors to the final PSQI score, with contributions limited to 3% and 2%, respectively.

Analyzing the “sleep disturbance” function of PSQI, which is composed of nine questions, revealed that 25% (22 out of 88 patients) experienced nighttime breathing problems/symptoms at least once a week, of whom 31.91% (15 out of 47 patients) were poor sleepers and 17.07% (7 out of 41 patients) were good sleepers. On the other hand, sleep disturbance due to the need to use the bathroom was much more common: 68.18% (60 out of 88 patients) confirmed their need to use the bathroom at night. Of these, 91.49% (43 out of 47 patients) were poor sleepers, and 41.46% were good sleepers (17 out of 41 patients) (Table 4).

Sleep Quality Assessment and Autonomic Nerve Function

While the reported mean hands score of the total number of patients surveyed was 62.42 ± 11.92 μS, the mean feet score was 64.93 ± 15.33 μS. Patients in the good sleep category illustrated good autonomic nerve conductivity in both the hand and foot electrodes, with 65.39 ± 8.55 μS and 70.95 ± 10.43 μS, respectively. On the other hand, poor sleep quality patients exhibited significantly moderate autonomic nerve damage, with slower conductivity of both hands and feet with 59.53 ± 13.35 μS and 59.68 ± 16.91 μS, respectively.

Fig. 1 compares autonomic nerve conductivity in the whole sample, good sleepers, and poor sleepers measured through Sudoscan’s hand and foot electrodes, respectively.

Correlations and Regression

The whole study sample showed a strong inverse correlation between PSQI score and autonomic nerves’ conductivity measured through hand electrodes, with a correlation coefficient of -0.62 (p = 0.016). On the other hand, a moderate inverse statistical correlation controlled the relationship between the feet’s autonomic nerve conductivity and patients’ PSQI score, with a correlation coefficient of -0.54 (p < 0.001).

Fig. 2 illustrates the linear regression plots and the formulae that control the study variables.

DISCUSSION

The current study showed that 53% of the patients experienced poor sleep quality, with a PSQI score ≤ 5. Incidence of poor sleep quality in females was greater than in males: 59% compared to 49%, respectively. It should be mentioned that the recruited women in the current cross-sectional study were significantly older and shorter, with higher waist circumference and BMI.

The high incidence of poor sleep quality based on PSQI score is compatible with the results of a study on diabetic patients in Myanmar, where 48.4% of the patients suffered from poor sleep quality. However, the current study recruited better-controlled diabetic patients with no macro- or microvascular complications, which means worse sleep habits in Iraqi patients compared

| Table 4. PSQI: the rates of nighttime breathing problems and nighttime micturition in the different study groups |
|-----------------------------------------------|-----------------|-----------------|
|                   | Whole sample   | Good sleepers   | Poor sleepers   |
|                   | (n = 88)       | (n = 41)        | (n = 47)        |
| Inability to breathe comfortably at least once a week | 22 (25)         | 7 (17.07)       | 15 (31.91)      |
| Need to use bathroom at night at least once a week   | 60 (68.18)      | 17 (41.46)      | 43 (91.49)      |

Data are presented as number (%).

PSQI, Pittsburg Sleep Quality Index.
to those in Myanmar [33]. Several other studies’ outcomes align with the outcomes of the current study regarding poor sleep quality: These studies were conducted in Ethiopia (47.2%) [34], Kenya (53.4%) [35], Iran (50.7%) [36], and Korea (49%) [37]. The prevalence of poor sleep quality in T2DM was even worse in a cross-sectional study in Chile, with 75% of patients complaining of poor sleep quality. Among the Chilean diabetic patients, 77.3% were classified with poor glycemic control and 46% suffered from comorbidities. The study also revealed that females in the whole study sample exhibited a higher incidence of poor sleep quality, with 80.2% compared to 64% in males, which agrees with the results of the current study [38]. The gender-based difference may be attributed to hormonal distribution [39]. Premenopausal women have been diagnosed with higher incidence of insomnia than postmenopausal women [40,41]. Significant age, waist circumference, and BMI differences in the current study may also play a role in such variation.

Examples of other studies that indicated worse sleep quality were conducted in Sudan (97.1%) [42], the United States (84%) [42], and Saudi Arabia (72%) [43]. The wide differences in sleep quality may be attributed to the type of diabetes, anthropometric parameters, lifestyle, or psychological differences [29].

All PSQI questionnaire functions indicate a significant difference between the good and poor sleep quality groups with the exception of the “use of medication” function, where the p-value was 0.296.

PSQI functions unequally contribute to the final PSQI score. Sleep latency is the main driver affecting total PSQI score in the current study, followed by sleep disturbances. Sleep duration was the third factor. Subjective assessment of sleep quality represents the fourth force affecting PSQI score, followed by daytime dysfunction as a result of poor sleep quality. Finally, use of medication and habitual sleep efficiency minimally contributed to the PSQI score with 3% and 2%, respectively.

Certain components of PSQI proved a similar magnitude of effect on sleep quality in various studies in the Middle East. The current study agreed with Khosravan et al. [36] in confirming the powerful impact of sleep latency and sleep duration on the final PSQI score. In the same context, the weak effect of the use of sleep medication was noticed in both studies. However, subjective sleep quality had a greater impact in the Iranian study compared to ours. Additionally, daytime dysfunction had a weaker impact in the study by Khosravi et al. [29] than in the current one. The impact of PSQI components on Jordanian T2DM patients was quite similar to the results of Iraqi patients in this research [44].

The autonomic nerve function of both the hands and feet of the whole group of patients surveyed in the present study was found to be in the normal range, with a mean value over 60 μS [45]. However, while good sleepers experienced normal autonomic function, poor sleepers showed moderate autonomic nerve damage, with significantly lower nerve conductivity compared to good sleepers.

Hands’ autonomic nerve function demonstrated a strong negative correlation with the PSQI score of the surveyed patients in this study; feet’s autonomic nerve conductivity, on the other hand, moderately correlated with sleep quality in the negative direction.

According to the regression model, 39% of the variance in PSQI scores is explained by hands’ autonomic nerve records and 29% by feet’s results. That means other factors affect the quality of sleep in T2DM patients, in spite of the significant role of autonomic nerve dysfunction. Obesity is a highly possible risk factor; specifically, the mean BMI of the surveyed sample in the present study fell in the obesity range.

Sleep disturbance function is more affected by nighttime voiding than by breathing problems during sleep; the current study outcomes tend to explain the impact of autonomic neuropathy on sleep quality through bladder autonomic nerve dysfunction. Poor sleepers showed higher incidence of night micturition and autonomic dysfunction than good sleepers. The results concur with Li et al. [46], who confirmed worse sleep quality and health-related quality of life in T2DM females suffering from urinary incontinence with more than three episodes of nighttime voiding. The results are also consistent with Moningi et al. [47], who found that neurogenic bladder may be a result of impaired detrusor function and reduced bladder sensation, which in turn
could be a result of the damage to parasympathetic or sympathetic nerve fibers or both, leading to urinary retention, increased voiding frequency, and increased post-voiding residual volume of more than 150 mL, which may increase the incidence of urinary tract infection. Wang et al. [48] concurred with the above conclusions. They found that urinary urgency in T2DM female patients appeared not to be influenced by glycemic control. The authors attributed urinary urgency to the irreversible neurovascular damage mediated by several years of diabetes.

In conclusion, the Iraqi T2DM patients in this study showed a predominance of poor sleep quality. Females in the study group were significantly older, shorter, more obese, and poorer sleepers than males. Poor sleepers exhibited moderate autonomic dysfunction, while good sleepers exhibited normal autonomic function. Autonomic nerve function is strongly and inversely correlated with sleep quality.

Availability of Data and Material
The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Author Contributions

Conflicts of Interest
The authors have no potential conflicts of interest to disclose.

Funding Statement
None

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