Original Article

Sleep Quality in Parkinson Disease: Clinical Insights and PSQI Reliability Assessment

Julia Valle Pezzini Dante Diniz Trevisan Victor Henrique Dominiak Soares Luís Eduardo Gauer Marcelo M. S. Lima

Neurophysiology Laboratory, Department of Physiology, Biological Sciences Sector, Universidade Federal do Paraná, Curitiba, PR, Brazil

Address for correspondence Marcelo M. S. Lima, PhD (e-mail: mmslima@ufpr.br; marcelomslima.neuro@gmail.com).

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Abstract

Sleep disturbances are prevalent in Parkinson disease (PD), encompassing a spectrum from parasomnias like REM sleep behavior disorder to symptoms of sleep-wake cycle dysregulation, such as insomnia and daytime sleepiness. This research investigates sleep quality in PD patients compared with a matched healthy control group and explores the relationships between PD clinical characteristics and sleep parameters. Additionally, the study assesses the reliability of the Pittsburgh Sleep Quality Index (PSQI) for PD patients by examining internal consistency. The study comprises 52 participants, 27 in the PD group and 25 in the healthy control group, matched for sex and age. Sleep quality revealed that PD patients experienced significantly poorer sleep quality than the control group ($p = 0.009$). Weak correlations were found between PSQI scores and the modified Hoehn and Yahr scale ($p = 0.062$), with no correlation observed with the daily equivalent dose of levodopa (L-DOPA). The prevalence of poor sleep quality (PSQI score > 5) was 85.1% for PD patients and 68% for the control group. The internal consistency analysis of the PSQI yielded a Cronbach's α of 0.588 for the PD group. While the PSQI demonstrates utility in detecting general sleep abnormalities and gauging patient perceptions of sleep quality in PD, its limitation as a global score is emphasized. The index prioritizes sleep habits and may not fully capture important sleep disorders in this population. These findings underscore the complex relationship between PD and sleep quality, suggesting the need for a comprehensive approach to assess and address sleep disturbances in PD patients.

Keywords

- Parkinson disease
- sleep quality
- sleep-wake disorders

Introduction

The prevalence of sleep disorders in patients with idiopathic Parkinson disease (PD) is known to be higher than in the general population, at 60.3% and 30.3%, respectively.[1] These disorders are generally associated with nocturnal manifestations such as impaired control of motor activity during sleep, leading to parasomnias, as well as impairments in sleep and wakefulness regulation, resulting in insomnia and daytime sleepiness.[2] In this regard, electroencephalographic recordings in patients with or without treatment predominantly show a significant reduction in sleep efficiency, percentage, and duration of rapid eye movement (REM) sleep, as well as decrease in the latency to non-REM sleep, and sleep fragmentation.[3]

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Physiologically, it is believed that the mechanism of both REM and non-REM sleep regulation in PD is influenced by disruptions in the dopaminergic system, caused by degenerative changes in regions such as the substantia nigra pars compacta and the ventral tegmental area, negatively impacting the regulation of REM sleep.[4,5] Moreover, PD patients' sleep can be affected by concomitant psychiatric disorders and also by antiparkinsonian medications.[6] This complex array of causes make the investigation of the role of the dopaminergic system in the regulation of different sleep stages and their cyclic dynamics much more challenging.

In light of these issues, the present study aimed to evaluate the effects of PD in sleep quality as measured by the Pittsburgh Sleep Quality Index (PSQI), as well as to examine the relationships between different clinical parameters of PD, such as motor impairment measured by the modified Hoehn and Yahr scale (HY-M), time since diagnosis, and the levodopa (L-DOPA) equivalent daily dose (LED), with the sleep parameters obtained through the application of the PSQI. Additionally, we explored the reliability of the PSQI for assessing sleep quality in PD patients through measures of internal consistency, namely Cronbach α and Pearson correlations between the global score and the component scores of the questionnaire.

Study Sample and Methods

Inclusion and Exclusion Criteria

Our initial sample consisted of 60 participants from the Neurology Outpatient Clinic of Complexo do Hospital de Clínicas da Universidade Federal do Paraná (CHC-UFPR), including 34 patients with PD (PD group) and 26 individuals without a diagnosis or suspicion of PD (control group), sampled by convenience in the same outpatient clinic.

The inclusion criteria for the PD group consisted of patients of any sex aged 18 years or older, diagnosed with idiopathic PD by physicians from the Neurology service at CHC-UFPR, at disease stages 2 to 3 according to the HY-M scale, capable of ambulating without assistance and on stable therapeutic conditions, that is, not having experienced changes in antiparkinsonian medication or dosage in the 10 days prior to study recruitment. In the control group, participants of both sexes aged 40 years or older and capable of ambulating without assistance were included, as long as they had no clinical suspicion or diagnosis of PD or any other form of Parkinsonism. In conformity, the exclusion criteria dismissed participants of both groups with severe cognitive impairment—defined as a score of 17 or less on the Mini-Mental State Examination (MMSE), as proposed by Tombaugh and McIntyre[7]—as well as those with severe neuropsychiatric, visual, cardiovascular, rheumatological, and/or musculoskeletal impairments, as these comorbidities could introduce bias in the sleep analysis of these individuals.

Interviews

The interviews were conducted by the researchers at the Neurology Outpatient Clinic of CHC-UFPR, ranging from 15 to 40 minutes of duration. In these interviews, identification data such as name, age, sex, height, weight, and level of schooling were collected, as well as comorbidities and continuous-use medications. For the PD group, the time since PD diagnosis, stage of motor progression according to the HY-M scale, and dosage of antiparkinsonian medication were obtained directly from medical records.

The L-DOPA equivalent daily doses (LEDs) were calculated using the following formula: (LED = [conventional L-DOPA dose + dispersible L-DOPA dose + amantadine dose] × 1 + [extended-release L-DOPA dose] × 0.75 + [conventional L-DOPA dose with concurrent use of entacapone 200 mg] × 1.33 + [pramipexole dose + rasagiline dose] × 100 + [selegiline dose] × 10), as described by Tomlinson and colleagues.[8]

Sleep Assessment – PSQI

The PSQI is a self-rated questionnaire composed of 19 items organized into 7 different components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime dysfunction. Each component is scored from 0 to 3, resulting in a final score ranging from 0 to 21. The PSQI is a tool with good sensitivity and specificity for assessing sleep disorders, and it has been well validated in the literature and has good test-retest reliability.[9] Furthermore, this questionnaire has been widely applied in other studies with PD patients.[10,11]

Statistical Analysis

An analysis of covariance (ANCOVA) was performed to assess whether the differences observed in the mean PSQI scores between groups and sexes were statistically significant after controlling for the effects of age and level of schooling. The normality criterion of the dependent-variable "PSQI score" was met according to the Kolmogorov-Smirnov test with a significance level of 0.05, and the homogeneity of variances criterion was met using the Levene test with a significance level of 0.05. Other continuous variables, such as level of schooling, LED, HY-M scale stage, and time since PD diagnosis, were studied using Pearson correlations to identify factors that might be correlated with sleep quality in individuals with PD, considering a p-value ≤ 0.05 as statistically significant.

The internal consistency of the PSQI for each group, as well as for the entire sample, was evaluated using the Cronbach α.[12] This coefficient provides a measure of how well the items on a scale measure the same concept, and is, therefore, widely used as an index of scale reliability.[13] Furthermore, Pearson correlations were calculated between the component scores and the global score of the PSQI, both for the entire sample and for each group separately, with the aim of complementing the internal consistency and reliability analyses of the questionnaire. The data analyses were performed using the software Jamovi version 2.3.21 for Windows.

Results

Of the 60 individuals interviewed (►**Table 1**), 7 patients from the experimental group were excluded according to the exclusion criteria, and 1 subject from the control group

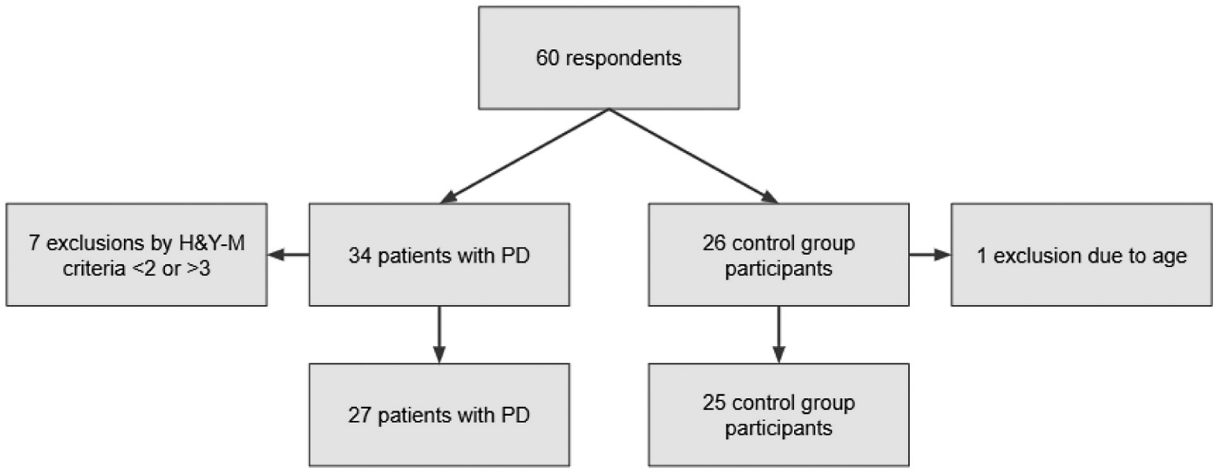

Fig. 1 Characterization of the sample.

was excluded for being out of the age range. The sample consisted of 52 participants, with 27 individuals with PD and 25 control individuals (►**Fig. 1**).

Regarding the clinical aspects of the PD group, most participants had stage 2 PD, according to the HY-M scale (44.4%), followed by stages 3 (37%) and 2.5 (18.5%), resulting in an average score of 2.46 ± 0.46 (►**Table 2**). The diagnosis time varied from 3 to 20 years, with an average value of 9.78 ± 4.89. The antiparkinsonian medications used by patients included L-DOPA combined with benserazide hydrochloride in conventional, dispersible, and extended-release formulations, as well as entacapone; selegiline; rasagiline; amantadine; and pramipexole. The identified minimum LED was 200 mg, and the maximum was 1,825 mg, with an average of 900 ± 393 mg.

Regarding the sleep evaluation, among individuals with PD, 85.1% had a score > 5 and 40.7% had a score > 10 on the PSQI, compared with 68.0% and 8.0% in control individuals, respectively (►**Table 3**). An ANCOVA was performed with the PSQI score as the dependent variable and sex and group as the independent variables, considering level of schooling and age as covariates, in a sample of 52 individuals. The results indicated a significant effect only for the group variable ($F = 7.46$; $p = 0.009$), as the Parkinson group obtained a significantly higher average PSQI score (10.00 ± 4.16) than the control group (6.48 ± 2.92) (►**Fig. 2**).

Table 1 Demographic characterization of the groups.

		Parkinson		Control	
		n	%	n	%
Gender	Feminine	14	51.9%	14	56.0%
	Masculine	13	48.2%	11	44.0%
	Total	***27***		***25***	
Age	< 65 years	15	55.6%	19	76.0%
	65–69 years	4	14.8%	4	16.0%
	70–74 years	2	7.4%	1	4.0%
	75–79 years	4	14.8%	1	4.0%
	≥ 80 years	2	7.4%	0	0.0%
	Total	***27***		***25***	
Level of schooling	Incomplete elementary school	11	40.7%	3	12.0%
	Complete elementary school	2	7.4%	1	4.0%
	Incomplete high school	0	0.0%	1	4.0%
	Complete high school	7	25.9%	5	20.0%
	Incomplete college education	2	7.4%	0	0.0%
	Complete college education	5	18.5%	15	60.0%
	Total	***27***		***25***	

Table 2 Clinical staging and LED of patients with PD.

		n	%	Mean LED
Modified (►**Table 1**) scale score	2	12	44.4%	789 ± 273
	2.5	5	18.5%	984 ± 578
	3	10	37.0%	990 ± 421
	Total	*27*		900 ± 393

Abbreviations: LED, levodopa equivalent daily dose; PD, Parkinson disease.

Table 3 Pittsburgh Sleep Quality Index scores.

		Parkinson		Control	
		n	%	n	%
Pittsburgh Sleep Quality Index score	Good sleep quality (0–5 points)	4	14.8%	8	32.0%
	Bad sleep quality (6–10 points)	12	44.4%	15	60.0%
	Sleep disturbance (> 10 points)	11	40.7%	2	8.0%
	Total	*27*		*25*	

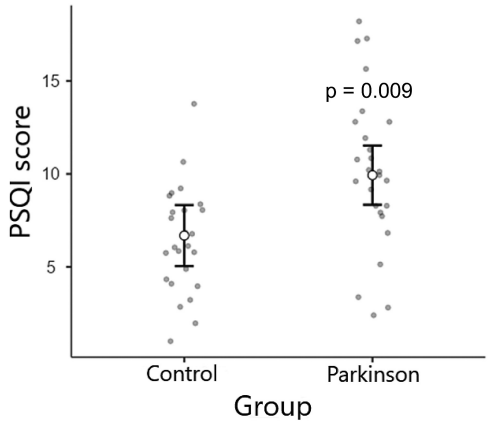

Fig. 2 Mean Pittsburgh Sleep Quality Index score (white dots) and 95% confidence interval (bars) in the control and Parkinson groups. Abbreviation: p = Pearson coefficient obtained by the analysis of covariance test.

Furthermore, when dividing the PSQI score into its components, ANCOVA revealed a statistically significant effect of the group only on the scores of components 1 ($F = 4.31$; $p = 0.043$) and 7 ($F = 5.23$; $p = 0.027$), namely, subjective sleep quality and daytime dysfunction, respectively (►**Table 4**). The means were higher in the PD group for all components of the PSQI.

According to our data from Pearson correlations between variables related to clinical aspects of PD and sleep quality, it was found that the HY-M scale score had a weak correlation with the overall PSQI score, but without statistical significance ($r = 0.364$; $p = 0.062$). Moreover, the time since diagnosis was positively correlated with LED ($r = 0.444$; $p = 0.020$) and with HY-M score ($r = 0.529$; p¼0.005), and component 3 of the PSQI (sleep duration) showed a positive correlation with LED ($r = 0.443$; $p = 0.021$) and a borderline correlation with HY-M score ($r = 0.365$; $p = 0.061$) (►**Table 5**).

Table 4 Global Pittsburgh Sleep Quality Index score of the groups

Components	Mean ± σ*		ANCOVA
	Control ($n = 25$)	Parkinson ($n = 27$)	F (p)
1. Subjective sleep quality	0.92 ± 0.86	1.41 ± 0.89	4.314 (0.043)
2. Sleep latency	0.92 ± 0.76	1.26 ± 1.09	0.243 (0.625)
3. Sleep duration	0.76 ± 0.88	1.22 ± 1.09	3.035 (0.062)
4. Habitual sleep efficiency	1.56 ± 1.53	2.22 ± 1.34	0.645 (0.426)
5. Sleep disturbances	1.36 ± 0.57	1.70 ± 0.67	1.771 (0.190)
6. Use of sleep medication	0.80 ± 1.29	1.26 ± 1.48	1.230 (0.273)
7. Daytime dysfunction	0.56 ± 0.71	0.96 ± 0.98	5.234 (0.027)
Global score	6.48 ± 2.92	10.0 ± 4.16	7.463 (0.009)

Abbreviation: ANCOVA, analysis of covariance.
Notes: Components and global score of Pittsburgh Sleep Quality Index in the control and Parkinson groups. *σ = standard deviation.

Table 5 Pearson correlations between clinics and sleep quality.

Correlation	r (*p*)
Educational level and MMSE	0.305 (0.028)
HY-M and time since diagnosis	0.529 (0.005)
LED and time since diagnosis	0.444 (0.020)
HY-M and PSQI	0.364 (0.062)
HY-M and PSQI (C3*)	0.365 (0.061)
LED and PSQI (C3*)	0.442 (0.021)

Abbreviations: HY-M, Hoehn and Yahr scale; LED, levodopa equivalent daily dose; MMSE, Mini-Mental State Examination; PSQI, Pittsburgh Sleep Quality Index.
Notes: Correlations related to clinical aspects of Parkinson disease and sleep quality. *C3 = compound 3 of the PSQI (sleep duration).

Considering the analysis of the internal consistency of the PSQI, the Cronbach α was 0.588 for PD patients, 0.499 for healthy controls, and 0.618 for the total sample. Also, Pearson correlations between the scores of the 7 components and the overall PSQI score in the PD group ranged from $r = 0.342$ ($p = 0.081$) for component 6 to $r = 0.746$ ($p = < 0.001$) for component 1. The complete description of these correlations is presented in ►**Table 6**.

Discussion

The prevalence of poor sleep quality found in the present study, defined as a score > 5 on the PSQI, was 85.1% for PD patients and 68% for individuals in the control group, showing that individuals with PD have poorer sleep quality than their control counterparts. These values are similar to the prevalence found in the literature, in which 46.6% and 73.3% of patients present poor sleep (PSQI > 5) before and after treatment with dopaminergic agonists, respectively.[14] Similar percentages were obtained by Fabbrini et al. (2002), with 60% of PD patients and 56% of control individuals scoring above 5 on the PSQI.[15]

Worsening of Sleep Quality in PD

The relationship between PD and sleep disturbances is well established in the literature. It is believed that these alterations are partially related to the neurodegenerative processes of the disease,[16] and may even manifest as risk factors for the development of PD: in a 6-year follow-up of healthy individuals with a mean age at the beginning of the study of 65.4 ± 10.3, worsening sleep quality and shortened sleep duration were associated with the risk of developing PD.[17] Furthermore, motor and non-motor nighttime symptoms, dopaminergic treatment, and comorbid sleep disorders, such as RBD, restless leg syndrome, and obstructive sleep apnea, also contribute to the sleep impairment experienced by PD patients.[18]

Regarding the worsening of sleep quality in PD, a statistically significant difference was found between the subjective sleep quality and daytime dysfunction components' scores of the PSQI when comparing the PD with the control group. Excessive daytime sleepiness (EDS) is a common phenomenon in PD – in a recent meta-analysis, the calculated prevalence of EDS in PD patients was 35%. This study also found associations between EDS and increasing age, longer disease duration, greater severity of motor and autonomic symptoms, higher LED, reduced autonomy, and more severe psychiatric symptoms.[19] Moreover, in a cohort study, the prevalence of EDS in PD patients increased over time, rising from 5.6% in the 1st year of follow-up to 44.9% in the 8th year, with a total prevalence of 54.2% over the 8 years.[20] The authors also reported that EDS was a persistent disorder for most patients, being related to age, sex, and the use of dopaminergic agonists.[20]

Table 6 Pearson correlations between the components and the global score of the Pittsburgh Sleep Quality Index.

Component	All (*n* = 52)	Control (*n* = 25)	Parkinson (*n* = 27)
	r (p)	r (p)	r (p)
1. Subjective sleep quality	0.669 (< 0.001)	0.480 (0.015)	0.746 (< 0.001)
2. Sleep latency	0.602 (< 0.001)	0.545 (0.005)	0.615 (< 0.001)
3. Duration of sleep	0.524 (< 0.001)	0.307 (0.136)	0.578 (0.002)
4. Usual sleep efficiency	0.566 (< 0.001)	0.638 (< 0.001)	0.482 (0.011)
5. Sleep disturbances	0.595 (< 0.001)	0.243 (0.242)	0.724 (< 0.001)
6. Use of sleeping medications	0.334 (0.016)	0.215 (0.303)	0.342 (0.081)
7. Daily dysfunction	0.580 (< 0.001)	0.507 (0.010)	0.567 (0.002)

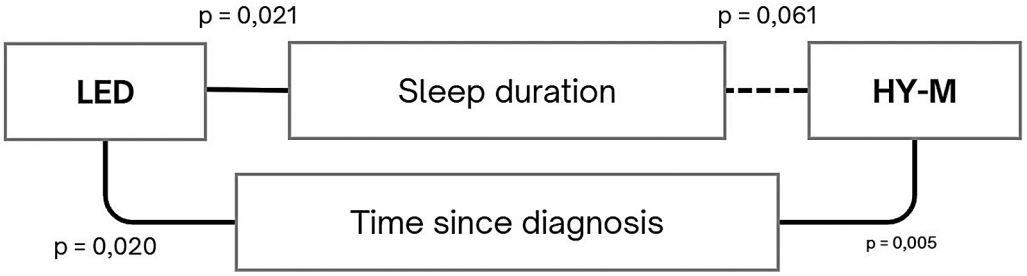

Fig. 3 Correlations between LED, levodopa equivalent daily dose, and HY-M, modified Hoehn-Yahr scale score, with sleep duration and time since diagnosis of PD patients. P-values were obtained by the Pearson correlation coefficient between variables.

LED and Sleep in PD

We found that LED significantly correlated with the sleep duration component of the PSQI, which was also associated with the HY-M scale score. Considering the correlations between LED and the time of diagnosis, which is related to the disease progression measured by the HY-M scale, it is not clear whether the higher dose of antiparkinsonian medication or the neurological progression of disease, or a combination of both, were responsible for the impairment of the sleep parameters evaluated by our study, as shown in ►**Fig. 3**.

Accordingly, Qiu et al. (2022) found, through a multivariate logistic regression analysis, that an increased dose of L-DOPA ($\geq$ 600 mg) is a risk factor for the occurrence of sleep disorders in individuals with PD.[21] Furthermore, LED is considered a predictive factor for sleep quality in these patients.[22] Also, the LED in individuals with PD who experienced poor sleep was twice as high as that in individuals with PD who reported good sleep quality, with the classification into these two groups determined by the Parkinson Disease Sleep Scale-2.[23] In addition, such increase in LED may be interpreted as a result of increased severity of PD, given that the scale results were positively correlated with disease duration.[23]

Furthermore, it is known that the progression of PD is associated with the development of several sleep disorders. In a cohort study following 218 PD patients and 102 controls for 5 years, the sleep problems with the highest increases in frequency reported were insomnia (endpoint prevalence of 44.5%), EDS (32.1%), and REM sleep behavior disorder (31.2%), while the frequency of sleep disorders in control individuals remained stable, with insomnia being the most common problem during the 5 years.[24]

Use of PSQI in the PD Population

In the current study, the PSQI demonstrated a Cronbach's of 0.588, contrasting to the English version of the PSQI, which obtained a Cronbach α ranging from 0.80 to 0.83, with the exception of some studies with non-clinical populations or with chronic fatigue syndrome.[25,26] It is possible to hypothesize that such discrepancy may be due to factors such as the small sample size, as well as the variability in sleep alterations both in PD patients and in elderly individuals.[27]

In addition, while some studies demonstrated unidimensionality, factor analyses that determined two to four distinct factors were also found for the PSQI.[28] Thus, although the Cronbach's alone is not sufficient to determine the internal consistency of the PSQI, the coefficient reported in our study is consistent with other findings. Furthermore, it has been described that the removal of component 6, which was not statistically significant (use of sleeping medications - control: 0.215 [0.303]; Parkinson: 0.342 [0.081]) led to an increase in Cronbach α to 0.699 in the PD group, suggesting that the score of this particular component demonstrates less interrelation with the scores of the other components.[28]

Possible explanations are that the use of dopaminergic medication may be related to an improvement of other aspects of sleep quality or the finding that many elderly individuals without PD who have poor sleep quality, in part, do not use drug therapies for this purpose.[24] In addition, moderate correlations were found in the PD patient group in relation to the overall score for components 2, 3, 4, and 7, as well as strong correlations for components 1 and 5, indicating that the latter two factors (subjective sleep quality and sleep disturbances, respectively) were more impacted by PD. In view of these aspects, the application of the PSQI in PD patients seems to have a good utility in assessing the presence and severity of general sleep abnormalities, encompassing the patient's perception of their sleep quality. However, it is important to note the index itself, as a global score, is somewhat limited considering the inconclusive value of Cronbach's ($\alpha = 0.588$) and its focus on sleep habits, thus, not covering important sleep disorders in this population, such as nighttime motor problems, restless leg syndrome, REM sleep behavior disorder, and EDS, in addition to its intrinsic ambiguity in some itens, potentially confusing the patient.[25,30] Furthermore, significant correlations between PSQI and sleep diary parameters have been described, such as sleep latency, sleep quality, and ease of falling asleep, which, in turn, showed several correlations with metrics derived from polysomnography in individuals with PD, suggesting that a sleep diary could also be a useful tool for this purpose in these patients.[31]

Although the PSQI provides data that assist in clinical decision-making and allow for external comparisons through standardized measurement of sleep quality, it cannot replace a detailed sleep complaint history from the patient, including input from caregivers, partners, or others. In specific cases, when possible, it is recommended to also apply tools for objective sleep evaluation, such as polysomnography, to better detect the extent of sleep disorders in PD patients.

Study Limitations

This study followed an observational cross-sectional design, and it has limitations regarding inferences about the causality of the observed effect. Additionally, no blinding was adopted during the evaluation, meaning that the researchers themselves conducted the interviews, data entry, and statistical analyses. The statistical analysis itself may have been restricted by the limited number of participants.

Moreover, the differences between the PD and control groups may have been maximized or minimized by the lenght bias. This is a type of bias that arises when there is a diagnosed population and a non-diagnosed one, leading to treatments, interventions, and behavioral changes of those who are diagnosed impacting certain effects being evaluated in the study. For instance, a patient diagnosed with PD, being under neurologists' follow-up, is more likely to be receiving treatment for their sleep disturbances compared with a control group individual who is not under the same follow-up, influencing their sleep quality.

Lastly, as in any questionnaire-based study, this study is also subject to information bias, such as memory bias, which occurs when participants are asked about retrospective data, as well as interviewer bias, when participants are evaluated by different interviewers, and social desirability bias, when patients tend to respond in a socially more accepted or desirable manner.

Conclusion

Parkinson disease is associated with worsened sleep quality, possibly related to disease progression as measured by the HY-M scale. Interestingly, the LED was shown to have an effect only on the sleep duration component of the PSQI; however, the further associations of LED with disease progression, a confounding factor for the worsening of sleep quality, made the relationship unclear. More studies assessing the isolated effects of antiparkinsonian medication on sleep quality are warranted to understand its real impact in PD patients.

In the internal consistency analysis, although the Cronbach's value for the PSQI in PD patients did not reach the conventional threshold for clinical validity (> 0.70), factors such as the potential multidimensionality of the questionnaire and the relatively small sample size should be considered. Furthermore, considering the increase in this coefficient when excluding the medication use component of the PSQI from the analysis, as well as its weak correlation with the overall score, it is suggested that the results of the PSQI be cautiously examined by its seven components, not just the final score. Statistical tests with larger and more representative samples are recommended to better assess the reliability and internal consistency of the PSQI for the PD population.

The management of PD patients can benefit from the application of the PSQI, especially in identifying and grading the severity of general sleep disturbances, encompassing the patient's perception of their sleep quality. However, it is important to note that it does not replace a comprehensive and targeted sleep and sleepiness complaint history, which is essential for identifying the various sleep disorders affecting this population, such as REM sleep behavior disorder, restless legs syndrome, and EDS.

Ethics

All the experiments were approved by the Ethics Committee in Human Research from the CHC-UFPR - approval number 57813722.0.0000.0096. All study participants signed the consent form to express their agreement to participate in the study.

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Conflict of Interests

The authors have no conflict of interests to declare.

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