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CLINICAL CORRELATES OF PERIODIC LIMB MOVEMENTS IN SLEEP IN PARKINSON'S DISEASE

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Abstract

Objective—The aim of the current study was to investigate the frequency of periodic limb movements in sleep (PLMS) in Parkinson's disease (PD) and their impact on nocturnal sleep and daytime functioning.

Methods—Forty-five PD patients (mean age 68.5±8.7 years; 32 males) underwent one night of polysomnography (PSG). Clinical assessment and questionnaires evaluating sleep disturbance and quality of life (QoL) were completed. Patients were divided into two groups based on their PLMS index (PLMSI): PLMSI ≥15 (PLMS+) and PLMSI <15 (PLMS−).

Results—There were 26 (57.8%) PD patients in the PLMS+ group and 19 (42.2%) patients in the PLMS− group. Subjective assessment revealed an association between PLMS+ status and greater PD symptom severity, more subjective sleep disturbance, and decreased QoL. All patients showed poor sleep, and no significant group differences were detected on PSG measures.

Conclusion—We observed that PLMS occurred frequently in PD and increased with more severe PD. Although PLMS did not affect objective sleep, it was associated with increased sleep complaints and reduced QoL. Overall, our findings support the association between PLMS and PD as well as the clinical relevance of sleep disturbances in PD.

Keywords

Dopaminergic treatment; Parkinson's disease; periodic limb movements in sleep; polysomnography; quality of life; sleep disturbance

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1. Introduction

Epidemiological studies estimate that the prevalence of sleep difficulties in Parkinson's disease (PD) ranges from 60% to 98% [1–3]. Patients suffering from PD often complain of poor sleep, non restorative sleep and excessive daytime sleepiness (EDS). Additionally, sleep disordered breathing (SDB), restless legs syndrome (RLS), REM behavior disorder (RBD), and periodic limb movements in sleep (PLMS) are frequently recognized in this population [4,5]. Overnight polysomnographic (PSG) sleep recordings suggest frequent awakenings, low sleep efficiency (SE), decreased amount of slow wave sleep (SWS) and REM sleep, as well as increased light sleep and REM latency [6–8]. As sleep related problems affect both nocturnal sleep and daytime wakefulness, they may increase disability and further reduce quality of life (QoL) in PD patients and their caregivers [9–12].

Multiple factors are involved in the etiology of sleep disturbance in PD, including degeneration of central sleep regulation centers, motor symptoms, normal aging, antiparkinson drugs, and concurrent psychiatric and somatic illnesses (for a review, see [13]). Nevertheless, the relative contribution of each one is largely unexplored.

According to American Academy of Sleep Medicine (AASM) criteria [14], PLMS consist of clusters of 4 or more repetitive, short lasting movements of the limbs occurring during sleep. They primarily involve lower extremities and include rhythmic extension of the big toe and dorsiflexion of the ankle and occasional flexion of the knee and hip [15]. PLMS can occur unilaterally or bilaterally and may awaken the individual. However, the association between PLMS and sleep quality is controversial, as some studies have shown high impact on sleep architecture and PSG parameters [16,17] as well as on subjective perception of sleep [18,19], whereas others have reported no relationships [20–23].

The prevalence of PLMS has been found to increase with advanced age [24,25] and in other neurological populations such as multiple sclerosis [26], Gilles de la Tourette Syndrome [27], and spinal cord injury [28]. Some studies have found that PLMS are also more common in PD patients compared to healthy age-matched controls [3,29], whereas others did not detect significant differences [30,31].

Nevertheless, evidence suggesting common pathophysiological pathways between PLMS and PD has been reported. Indeed, significantly elevated PLMS were observed in PD compared to multiple system atrophy [32] and Alzheimer's disease [33], suggesting that nigrostriatal degeneration may promote PLMS. A single photon emission computed tomography (SPECT) study [34] found reduced striatal dopamine transporter binding in PD sufferers with PLMS compared to those without PLMS; in addition, the number of leg movements correlated with nigrostriatal dopaminergic cell loss. Lastly, dopaminergic medications have been shown to provide benefit for both PD and PLMS [35,36].

The purpose of the present study was to investigate the frequency of PLMS in PD patients and their impact on nocturnal sleep and daytime functioning by means of PSG and subjective measures.

2. Methods

2.1. Subjects

As part of a larger study on sleep apnea in PD, participants with a diagnosis of idiopathic PD were recruited through the Department of Neurosciences at University of California-San Diego (UCSD), the San Diego Parkinson's Disease Association, community neurologists, and talks given to PD support groups. Exclusion criteria were clinically atypical PD,

presence of any neurodegenerative disorder other than PD, cerebrovascular or coronary illnesses, epilepsy, cardiomyopathy, current treatment for sleep apnea, deep brain stimulation therapy for PD, a history of alcohol or drug abuse, and any psychological, behavioral, or physical problem that would have compromised their participation. Finally, patients were required to have been stable on the same medication dose for at least two months prior to participation in the study.

Overall, 45 patients (32 males; mean age 68.49 ± 8.74 years; mean duration of PD symptoms 5.78 ± 4.89 years) were enrolled. All were treated for PD and were on short-acting dopamine agonists: 11 (24.4%) patients received Levodopa monotherapy and 13 (28.9%) received dopamine agonist monotherapy, whereas 21 (46.7%) patients had a combination of Levodopa with a dopamine agonist. Other PD medications included rasagiline (12; 26.67%), selegiline (13; 28.89%), amantadine (10; 22.22%), and entacapone (10; 22.22%). Moreover, 15 (33.3%) patients took antidepressants and 7 (15.6%) took benzodiazepines.

In order to allow comparisons among patients on different dopaminergic regimens, drug dosages were converted to Levodopa Dosage Equivalents (LDE), according the following formula [37,38]:

$$\text{LDE} = (\text{regular Levodopa dose} \times 1) + (\text{Levodopa CR dose} \times 0.75) + (\text{pramipexole dose} \times 67) + (\text{ropinirole dose} \times 16.67) + [\text{regular Levodopa dose} + (\text{Levodopa CR dose} \times 0.75)] \times 0.25$$

if taking entacapone. Amantadine, rasagiline and selegiline were not included in this computation.

The protocol was approved by the UCSD Institutional Review Board and all participants gave written informed consent prior to participation in the study.

2.2. Questionnaires assessment

PD Severity—The Unified Parkinson's Disease Rating Scale (UPDRS; [39]) was used to assess severity of PD. The UPDRS consists of five subscales evaluating cognitive and emotional status (UPDRS I – mentation, behavior and mood; range 0–16), daily functioning (UPDRS II – activities of daily living; range 0–52), motor symptoms (UPDRS III – motor examination; range 0–108), and side effects of therapy (UPDRS IV – complications therapy; range 0–23). An overall score, as well as a score for each subscale, was computed with higher scores indicating more severe disease.

PD Progression—PD progression was rated with the Hoehn and Yahr Scale (HY; [40]), which grades patients from stage 0 (no signs of disease) to stage 5 (wheelchair bound or bedridden unless assisted).

Quality of Life (QoL)—The Parkinson's Disease Questionnaire (PDQ-39; [41]) was administered to evaluate QoL. The PDQ-39 consists of 39 items grouped into eight subscales exploring mobility, daily activities, emotional well-being, stigma, social support, cognition, communication, and bodily discomfort. Scores for each dimension and a summary index score (PDQ-39 SI; range 0–100) were computed, with higher scores suggesting lower QoL.

Sleep—The Parkinson's Disease Sleep Scale (PDSS; [42]) was used to evaluate subjective sleep disturbance. The PDSS is a 15 item visual-analogue scale assessing sleep complaints, with responses from 0 (symptom always experienced) to 10 (symptom-free).

The Epworth Sleepiness Scale (ESS; [43]) was used to measure daytime sleepiness. The ESS estimates the likelihood of falling asleep in eight different situations. Total score ranges from 0 to 24 and a score >10 suggests EDS.

The REM Behavior Disorder Sleep Questionnaire (RBDSQ; [44]) consists of 10 items exploring the clinical features of RBD. The maximum score is 10 and a cutoff of 5 is utilized for indicating RBD.

Finally, a structured questionnaire was administered in order to diagnose RLS according to International Restless Legs Syndrome Study Group (IRLSSG) criteria [45]: 1. An urge to move the legs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs; 2. The urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting; 3. The urge to move or unpleasant sensations are partially or totally relieved by movement, at least as long as the activity continues; 4. The urge to move or unpleasant sensations are worse in the evening or night than during the day or only occur in the evening or night. All four criteria had to be satisfied for diagnosis.

2.3. PSG measures

Sleep was recorded with the Somtè system (Compumedics, Melbourne, Vic., Australia). PSG montage included three EEG channels (F4-A1, C4-A1, O1-A2), left and right EOG, chin and bilateral tibialis anterior EMG, II-lead ECG, nasal pressure transducer, thoracic and abdominal respiratory effort, tracheal microphone, pulse oximeter, and body-position sensor.

Sleep stages and respiratory and movement events were manually scored according to AASM criteria [14]. Apneas were defined as a drop of $\geq 90\%$ in respiration lasting at least 10 seconds. Hypopneas were defined as a reduction of $\geq 50\%$ in respiration lasting at least 10 seconds associated with an arousal or $\geq 3\%$ desaturation. An apnea and hypopnea index (AHI; number of apneas and hypopneas per hour of sleep) ≥ 10 was used to define SDB.

Since infrared-videos were only available for some of the recordings, the presence of REM sleep behavior disorder (RBD) was established measuring the average tonic and phasic muscle activity during REM sleep [46]. Similar to AASM criteria [14], a REM epoch was defined as tonic if it showed a chin EMG amplitude higher than the minimum amplitude detected in NREM for $\geq 50\%$ of its duration. It was defined as phasic if, dividing each 30-sec epoch into 12 2.5-sec mini-epochs, transient bursts of muscle activity, lasting 0.1–5.0 sec and ≥ 4 times higher in amplitude than the background EMG activity, were observed in $\geq 50\%$ of the mini-epochs.

The EMG-score was defined as the proportion of both phasic and tonic components, using a cut off of 10% to indicate RBD.

Since there is still a debate in literature whether the diagnosis of RBD may be made by means of questionnaire alone or requires PSG recording [47–50], we combined both EMG- and RBDSQ-scores. Thus, three diagnostic categories were identified: yes-RBD (yRBD; RBDSQ ≥ 5 and EMGscore ≥ 10); no-RBD (nRBD; RBDSQ < 5 and EMGscore < 10); and probable-RBD (pRBD; either RBDSQ ≥ 5 or EMGscore ≥ 10) [46].

PLMS was defined as a series of at least 4 consecutive movements with movement duration of 0.5–5 sec and onsets each 5–90 sec apart, and with an $\geq 8 \mu\text{V}$ increase in EMG voltage above resting EMG. A PLMS index (number of PLMS per hour of sleep) ≥ 15 was considered pathological, according to the second edition of the International Classification of Sleep Disorders (ICSD-2) [51]. An arousal was considered associated with PLMS (PLMS-Ar) if there was an interval shorter than 0.5 sec between the onset of one event and

the end of the other one. Arousals and leg movements following apneas or hypopneas were excluded from computations.

The following sleep variables were derived from PSG measures: time in bed (TIB; min), total sleep time (TST; min), wake after sleep onset (WASO; min), sleep efficiency (SE; %), sleep onset latency (SOL; min), REM latency (REMLat; min), percent of N1, N2 and N3 sleep, apnea-hypopnea index (AHI; number of events/hour sleep), PLMS (total number of leg movements), PLMS index (PLMSI; number/hour sleep), PLMS arousal (PLMS-Ar; total number), PLMS arousal index (PLMS-ArI; number/hour sleep).

2.4. Procedure

Referred PD patients meeting inclusion criteria were interviewed, had a physical exam and medical history conducted by a physician specializing in sleep medicine and had severity of PD rated with the UPDRS and HY conducted by a certified neurologist. Medical records were also reviewed to confirm the diagnosis of PD. Questionnaires assessing sleep disturbance (ESS, PDSS) and QoL (PDQ-39) were administered.

Patients were admitted to the UCSD Clinical Translational Research Institute (CTRI) Gillin Laboratory for Sleep and Chronobiology for one PSG night. Sleep and wake time were scheduled based on each patients' habits.

2.5. Analysis

Two groups were identified based on the PLMSI: PLMS+ (PLMSI $\geq$ 15; n=26; 57.8%) and PLMS- (PLMSI<15; n=19; 42.2%). Demographic, clinical, subjective and PSG data were compared by group with Chi-Square and independent samples *t*-tests. Pearson's correlations were computed to establish the relationship between PLMSI and demographic and questionnaire data.

Finally, using the PDQ-39 SI score as a dependent variable, a multiple regression analysis was performed to identify which variables might account for decreased QoL in PD patients. Predictors were chosen on the basis of significant Pearson's correlations.

All statistical analyses were run with SPSS 17 software (SPSS, Inc., Chicago, IL, USA), setting a *P* level <0.05 as significant.

3. Results

3.1. Demographic, clinical and questionnaire data

Results comparing the PLMS+ and PLMS- groups on demographic, clinical and questionnaires data are summarized in Table 1. Independent sample *t*-tests revealed significantly higher scores in total UPDRS and UPDRS II in the PLMS+ group.

Compared to the PLMS- group, PLMS+ patients scored lower on PDSS and higher on PDQ-39 suggesting worse subjective sleep and worse QoL. Examining the PDQ-39 subscales, significant differences were detected in mobility and social support. No other group differences were observed.

3.2. PSG measures

PSG parameters are provided in Table 2. Although the groups were divided based on PLMSI, the PLMS+ group also had significantly more PLMS arousals and consequently a significantly higher PLMS-ArI. There were no significant differences between groups in any of the other sleep parameters. Likewise, groups did not differ in RLS ($\chi^2=2.652$, *P*=0.103) or

SDB ($\chi^2=1.666$, $P=0.197$) frequencies. However, significantly more patients in the PLMS+ group were classified as yRBD or pRBD ($\chi^2=8.163$, $P<0.05$).

3.3. Correlations and regression model

Pearson correlations revealed significant positive relationships between PLMSI and UPDRS total score ($r=0.39$, $P<0.01$; Fig. 1), UPDRS II ($r=0.37$, $P<0.05$), UPDRS III ($r=0.40$, $P<0.01$) and PDQ-39 SI ($r=0.34$, $P<0.05$). Considering each dimensions of PDQ-39, PLMSI was found to correlate with mobility ($r=0.37$, $P<0.05$) and communication ($r=0.48$, $P<0.01$).

A multiple regression model was performed using PDQ-39 SI as the dependent variable and HY, UPDRS I, UPDRS II, UPDRS IV, PDSS and PLMSI as the independent variables. The model was significant and accounted for 61% of the variance ($R^2=0.611$, $F_{2,42}=33.03$, $P<0.001$). PDSS ($\beta=-0.673$, $t=-6.866$, $P<0.001$) and HY ($\beta=0.289$, $t=2.952$, $P<0.01$) were identified as significant predictors of PLMS, whereas the other variables were excluded from the model.

4. Discussion

The purpose of the present study was to explore PLMS features in PD patients and the impact PLMS have on daily functioning and on objective and subjective sleep measures in this population. We observed a higher proportion of PLMS in PD sufferers than the frequency estimated for healthy elderly [24,32,52], supporting previous findings of an association between PLMS and PD beyond aging [29,53].

Compared to PLMS- patients, PLMS+ patients endorsed more sleep complaints, similar to prior investigations reporting increased subjective sleep disturbance associated with PLMS [24,54]. On average, patients in both groups reported excessive daytime sleepiness (average ESS scores >10), however no group differences were observed on ESS. This is in agreement with other studies that did not find a relationship between subjective daytime sleepiness and PLMSI [21,54–56]. EDS is a common symptom in PD [2,57,58] but, since PLMS seem not to be a contributor, other factors, such as the extent of the neurodegenerative process, aging, side effects of pharmacotherapy, or other sleep disorders (for a review, see [59]), may be playing a role.

It is noteworthy that PLMS+ status was associated with a reduced subjective QoL compared with PLMS- patients: furthermore, QoL decreased as the PLMSI increased. However, as suggested by the regression analysis, the lower QoL reported by these patients is due to other factors, such as sleep complaints and PD progression, rather than PLMS, in line with prior findings [9–12].

PLMS were also found to be related to PD severity, as the PLMS+ group scored, on average, lower on UPDRS. Furthermore, the higher PLMSI was associated with increased severity of PD. Examining the UPDRS subscales, positive correlations were found between PLMSI and both UPDRS II and UPDRS III, suggesting an increased impairment in both daily activities and motor symptoms with worsening of PLMS. An association between PLMS (assessed by questionnaire) and severity of motor symptoms was previously observed by Happe and colleagues [30]. Our study both confirms and extends these findings utilizing PSG as an objective measure for the assessment of PLMS. Likewise, Young and colleagues [60] found higher PLMSI in severe PD compared to mild disease sufferers.

In accordance with the literature [6–8], PSG recordings detected poor sleep in all PD subjects compared to historical data on healthy elderly, as indicated by increased wakefulness, reduced SE, low amount of REM and slow wave sleep. Whether or not PLMS

impact sleep is currently under debate [16,17,22,55]. Interestingly in our study, although there were no group differences found between PLMS+ and PLMS- groups for objective sleep measures, PLMS+ status was associated with increased subjective sleep disturbance, according with previous findings reporting a discrepancy between subjective and objective sleep measures [19,24,54].

Interestingly, although PLMS+ status was associated with increased subjective sleep disturbance, objective sleep measures did not show more impaired sleep in this group compared to PLMS- subjects. The discrepancy between subjective and objective sleep measures has been previously reported [19,24] and is in line with the debate about whether PLMS impact sleep [16,17,22,55]. We can also hypothesize that the lack of group differences on objective sleep parameters may be due to the fact that PLMS could not significantly worsen a condition of highly impaired sleep per se since, as above mentioned, poor sleep characterizes all PD patients. However, it is also possible that more in depth analyses on PSG measures (i.e. EEG power analysis, cyclic alternating pattern) might unmask subtle group differences.

The rate of RLS did not differ significantly between groups, supporting the evidence of PLMS occurring even without complaints of RLS [61,62]. There was also a similar proportion of SDB in both group, as previously reported [62,63]. On the other hand, RBD was found to occur more often in PLMS+ group. This result is consistent with literature on PLMS comorbidity, that reports a high association between RBD and PLMS [64,65].

Notwithstanding, the causes underlying the increase of PLMS with PD severity are still unknown. Since we found no relationship between PLMS, age, LDE or AHI, the increased PLMS in severe PD might more likely be the result of the disease worsening rather than normal aging, dopaminergic medications or sleep apnea.

The relationship between PLMS and PD may be explained by considering the hypothesized common pathogenic pathway [66]. Indeed, converging lines of evidence suggest that a central dopaminergic defect is also involved in PLMS pathophysiology. PLMS were found more frequently in diseases showing an impaired dopaminergic transmission, like PD, than in disorders lacking this condition, such as multisystem atrophy [32] and Alzheimer's disease [33]. In addition, Happe and colleagues [34] observed an association between numbers of PLMS and dopaminergic cells loss examined with SPECT in PD patients; moreover, PD patients with PLMS showed lower striatal dopamine transporter binding. Combining these findings with the evidence of improvement in both PD and PLMS symptoms with Levodopa and dopamine agonists treatment [35,36], as well as the elevated rate of PLMS in PD and its relationship to PD severity (see above), data are suggestive of a shared pathogenetic pathway between PLMS and PD involving nigrostriatal degeneration. Therefore, the increased severity of PLMS we found in severely affected PD patients could therefore reflect a greater degree of degeneration in dopaminergic functions provoked by the PD progression, supporting the postulated hypothesis [66].

Our findings have relevant clinical implications. Since sleep disturbances as PLMS occur frequently in PD patients and affect nocturnal sleep and daytime functioning, these results suggest that a comprehensive sleep evaluation should be included in the routine clinical assessment of PD patients. Self-report measures such as the PDSS should be adopted as a screening tool to identify sleep complaints. Whenever sleep difficulties are reported, further detailed examinations using PSG recordings may be indicated. Prompt recognition and proper management of sleep difficulties by means of targeted treatment could reduce sleep complaints and enhance QoL, leading to a global reduction of suffering in PD patients. Further investigations aimed to test this hypothesis are warranted.

Some limitations have to be highlighted in the current study. As we performed a single PSG recording, the actual rate of PLMS could have been masked since this sleep disturbance is characterized by a high night-to-night variability. However, even if the numbers of PLMS may differ notably in an individual across nights, a small night-to-night variability is detected when groups of patients are considered collectively [36,67,68]. In addition, the first night effect might have worsened the sleep pattern in all subjects. Another weakness is due to high comorbidity with other sleep disorders such as RLS, SDB and RBD. We can not exclude that the concurrent sleep disorders might have increased sleep disruption and subjective complaints. Lastly, our sample consisted of treated PD patients, hence, although we found no group differences in LDE, the side effects of Levodopa and dopamine agonists, as well as other drugs such as amantadine and selegiline, might have affected nocturnal sleep and increased diurnal sleepiness, considering the effect these drugs can have on the sleep-wake circuits. Bearing in mind that the dopamine agonists are commonly administered to manage PLMS, the therapy with short- rather than long-acting agents might account for the presence of PLMS in PD patients on these drugs. Thus, as the role played by medications in promoting sleep disturbances in PD patients is still unclear, in order to avoid possible confounding effects due to treatment, future research should compare PLMS features in elderly with and without PD. In this context, the lack of a control group consisting of healthy age-matched subjects is a limitation. Indeed, we could only have compared PLMS frequency in PD with historical data, whereas a control group would have provided additional strength to our results. Further studies are also needed to evaluate the effect of different DA agents on PLMS in PD, aiming to identify the optimal drugs combination for managing both disorders.

In sum, we observed a high frequency of PLMS in PD patients, was associated with increased PD severity, subjective sleep disturbance and reduced QoL, while no impact was detected on objective sleep measures provided by PSG. Overall, our findings support the association between PLMS and PD as well as the clinical relevance of sleep disturbances in PD, suggesting that this issue should be carefully addressed as a part of PD assessment and treatment.

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Figure 1.
Pearson's correlation between PLMSI and UPDRS total score.

Table 1

Demographic, clinical and subjective data (mean±SD).

	PLMS- (N=19)	PLMS+ (N=26)	<i>pa</i>
Age (years)	69.47±8.61	67.77±8.93	0.524
Gender (N m/f)	15/4	17/9	0.321 ^b
BMI	26.23±2.27	27.96±4.05	0.076
Duration disease (years)	5.58±5.45	5.92±4.55	0.819
LDE (mg)	462.71±440.38	628.85±420.98	0.213
HY	1.74±0.56	1.85±0.68	0.568
UPDRS	29.89±10.16	38.35±14.25	0.033
UPDRS I	2.42±1.95	3.81±2.62	0.059
UPDRS II	8.32±3.38	11.50±5.04	0.021
UPDRS III	16.95±7.11	20.27±9.03	0.191
UPDRS IV	2.26±1.52	3.46±2.66	0.063
PDSS	111.37±17.52	98.38±22.39	0.042
ESS	10.63±4.35	10.38±5.44	0.871
PDQ-39 SI	22.58±13.76	37.08±20.15	0.006
Mobility	5.00±5.68	10.12±7.74	0.019
Daily activities	3.53±2.65	5.42±4.40	0.080
Emotional well-being	3.58±4.15	5.46±3.70	0.117
Stigma	1.21±2.07	2.19±2.55	0.175
Social support	0.74±1.37	2.88±2.89	0.002
Cognition	3.58±2.39	4.42±2.83	0.298
Communication	1.84±1.50	2.96±2.32	0.057
Bodily discomfort	3.11±2.58	3.62±2.39	0.497

^a = by independent samples *t*-test;^b = by Chi-Square test

BMI, body mass index; LDE, Levodopa Dosage Equivalents; HY, Hoehn and Yahr Scale; UPDRS, Unified Parkinson's Disease Rating Scale; PDSS, Parkinson's Disease Sleep Scale; ESS, Epworth Sleepiness Scale; PDQ-39 SI, Parkinson's Disease Questionnaire Summary Index.

Table 2

PSG parameters (mean±SD).

	PLMS- (N=19)	PLMS+ (N=26)	<i>p</i> ^a
TIB (min)	478.15±21.56	469.40±26.20	0.242
TST (min)	363.13±57.78	335.88±68.37	0.167
WASO (min)	110.25±60.61	115.95±64.07	0.765
SE (%)	76.14±12.51	71.78±13.83	0.283
SOL (min)	9.59±10.24	17.53±19.92	0.090
REMLat (min)	185.05±88.65	185.31±91.58	0.993
N1 (%)	10.94±6.08	13.88±9.86	0.258
N2 (%)	64.07±12.33	57.87±9.40	0.062
N3 (%)	13.56±10.81	14.61±8.70	0.721
REM (%)	11.41±4.04	13.64±8.06	0.231
AHI (N/hr)	13.28±13.21	16.87±16.33	0.436
PLMS (N)	27.32±26.32	220.15±141.57	0.000
PLMSI (N/hr)	4.51±4.52	39.57±24.00	0.000
PLMS-Ar (N)	7.95±11.72	111.88±48.22	0.000
PLMS-ArI (N/hr)	1.38±2.11	20.49±8.85	0.000
RLS (%)	5.3	23.1	0.103 ^b
SDB (%)	42.1	61.5	0.197 ^b
yRBD (%)	31.6	42.3	0.017 ^b
pRBD (%)	10.5	38.5	0.017 ^b

^a = by independent samples *t*-test;^b = by Chi-Square test

TIB, time in bed; TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SOL, sleep onset latency; REMLat, REM latency; AHI, apnea/hypopnea index; PLMS, periodic limb movements in sleep; PLMSI, periodic limb movements in sleep index; PLMS-Ar, periodic limb movements in sleep arousal; PLMS-ArI, periodic limb movements in sleep arousal index; RLS, restless legs syndrome; SDB, sleep disordered breathing; yRBD, yes REM behavior disorder, pRBD, probable REM behavior disorder.