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Cognitive impairment and sleep disturbances after minor ischemic stroke

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Abstract

Purpose Post-stroke cognitive impairment (PSCI) is common among stroke survivors, although its risk factors are not well understood. Here, we assessed cognitive function in patients within 14 days after minor stroke and investigated the risk factors of PSCI, including sleep-related factors.

Methods Patients with minor acute ischemic stroke ($n = 86$) were continuously recruited from November 2015 to October 2016. Demographic and clinical data were collected, and cognitive assessment and polysomnography were performed. Based on their cognitive performance, stroke patients were divided into PSCI and no PSCI groups. Age-, sex-, and education-matched participants ($n = 36$) were included as a healthy control (HC) group.

Results Stroke patients showed impairments in multiple cognitive domains relative to HC participants ($p < 0.01$). Among stroke patients, the prevalence of PSCI and obstructive sleep apnea was 81.4 and 74.4%, respectively. Impairments in attention and working memory (87.1%) and executive function (84.3%) were the most common among stroke patients. Compared with no PSCI patients, PSCI patients showed a higher prevalence of obstructive sleep apnea (50.0 vs. 80.0%, $p = 0.030$) and shorter total sleep time (435.1 ± 104.0 vs. 347.3 ± 98.1 min, $p = 0.002$). Logistic regression analysis showed that education duration, total sleep time, and lowest SaO_2 were independent risk factors for PSCI.

Conclusions The prevalence of PSCI is high after minor ischemic stroke. In particular, attention and working memory and executive function are most commonly impaired. Although the risk factors for PSCI are numerous, shorter total sleep time and degree of hypoxia at night warrant further attention.

Keywords Stroke · Cognitive impairment · Sleep

Introduction

Post-stroke cognitive impairment (PSCI), including both dementia and cognitive impairment not fulfilling criteria for dementia, is common among stroke survivors and is associated with poor quality of life [1]. Cohort studies report a prevalence of PSCI ranging from 25 to 81% [2–4], with the wide variation in estimates likely due to differences among studies in patient characteristics, neuropsychological testing methods, sample sizes, and analytical methods. Although most previous studies used the Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA) to assess cognition [4–11], these tests were not designed to detect minor or specific changes in cognition, and fewer studies have employed more sensitive neuropsychological testing methods [12–15].

Impairments in cognition can be seen even after minor stroke or transient ischemic attack. For instance, one study reports declines in executive function, psychomotor processing speed, and memory 90 days after minor stroke or transient ischemic attack [15], and other studies provide evidence of mild cognitive decline in 42% of patients after minor stroke or transient ischemic attack [16, 17]. Furthermore, although obstructive sleep apnea (OSA) and changes in sleep duration are risk factors of stroke and are common in stroke patients [18, 19], little is known about

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the relationship between sleep disturbances and cognitive impairment in stroke patients [20].

The aim of this study was to assess cognitive performance in patients within 14 days after minor stroke using a five-domain neuropsychological test battery and to identify risk factors for PSCI, including sleep-related factors.

Methods

Study design

In this prospective single-center observational study, patients with minor acute ischemic stroke were continuously recruited from November 2015 to October 2016 at the Neurology Department of the Second Affiliated Hospital of Soochow University. Minor acute ischemic stroke was defined by a score of 5 or less than 5 on the National Institute of Health Stroke Scale (NIHSS) at admission. At the same period, the partner or friends of patients with minor acute ischemic stroke were continuously recruited as healthy control (HC) participants. Demographic data and clinical characteristics were collected, and patients underwent cognitive assessment and polysomnography (PSG) within 14 days after minor stroke. All participants provided informed consent, and the study protocol was approved by the Research Ethics Committee of the Second Affiliated Hospital of Soochow University.

Participants

Patients were included if they had a clinically confirmed diagnosis of ischemic stroke based on computed tomographic and/or magnetic resonance imaging within the previous 7 days, were at least 18 years of age, and with a score of ≤ 5 on the NIHSS at admission. Patients were excluded if they had severe circulatory or respiratory disease, a history of subjective cognitive impairment before the stroke, a history of stroke, or right limb weakness and aphasia. HC participants were included if they were at least 18 years of age. HC participants were excluded from the study if they had a history of stroke, subjective cognitive impairment, or snoring.

PSG

All patients underwent an overnight PSG in our sleep laboratory with an E-Series System (Compumedics Company, Australia), which lasted for more than 7 h within 14 days after stroke. All data were analyzed according to criteria from the American Academy of Sleep Medicine's Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications [21]. A registered polysomnographic technician who was blind to the results of cognitive assessment analyzed PSG data. The apnea-hypopnea index (AHI), oxygen desaturation index, lowest SaO_2 (L-SaO_2), percentage of total time with oxygen saturation level < 90%, average SaO_2, total sleep time (TST), sleep efficiency, sleep latency, and proportions of different sleep stages were recorded. OSA was defined as AHI ≥ 5 events/h. Before PSG, all patients completed the Epworth Sleepiness Scale [22], Hamilton Anxiety Scale [23] and Hamilton Depression Scale [24].

PSG test, the Hamilton Anxiety Scale and Hamilton Depression Scale were not performed on the HC participants.

Clinical and cognitive assessment

Head magnetic resonance imaging examinations were performed within 7 days after stroke. Magnetic resonance imaging including T1-weighted imaging (T1WI), T2-weighted imaging T2 (T2WI), fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and apparent diffusion coefficient (ADC) sequences was performed on a 3 T GE scanner. The severity of leukoaraiosis was rated according to the visual rating scale proposed by Fazekas [25], with grades ranging from 0 to 3 (0, no lesion; 1, punctuate foci; 2, beginning confluent foci; 3, confluent changes). We categorized the location of infarction as cortical, deep, cortical and deep, or infratentorial. We also collected demographic data (i.e., education duration, smoking, age, and sex) and past medical history of disease (i.e., hypertension, diabetes mellitus, atrial fibrillation, and dyslipidemia). Body mass index was calculated by dividing body weight by height squared (kg/m^2).

All patients underwent neuropsychological assessment on the second day of PSG examination. Tests of global cognition included the MMSE and MoCA (Beijing Version) [26, 27]. Five domains of cognitive function were assessed [28]: (1) attention and working memory using the Trail Making Test A, forward digit span, and Symbol Digit Modalities Test; (2) executive function using the Trail Making Test B, backward digit span, and Stroop Color-Word Test; (3) language function using a semantic verbal fluency test of animal naming within 1 min; (4) memory function using the Auditory Verbal Learning Test (word list learning with immediate and delayed recall and recognition conditions) and Rey-Osterrieth complex figure recall test; and (5) visuospatial function using the Rey-Osterrieth complex figure copying test and clock-drawing test. Cognition was also assessed in age-, sex-, and education-matched HC participants.

Grouping

Stroke patients were divided into PSCI and no PSCI groups. PSCI was defined as impairment (≥ 1.5 standard deviations) in at least one cognitive domain compared with age- and education-matched norms [17].

Statistical analysis

Data were analyzed with SPSS software (version 17.0). Continuous variables that were normally distributed were expressed as mean ± standard deviation, and continuous variables that were not normally distributed were expressed as median (interquartile range). Comparisons between groups were performed using independent Student's *t* tests when continuous variables were normally distributed and rank tests when continuous variables were not normally distributed. Distributions of categorical variables were expressed as percentages, and comparisons between groups were performed using chi-square or Fisher's exact tests. Multivariate logistic regression was used to determine predictors of PSCI. All variables with $p < 0.1$ in the univariate analysis were included in multivariate analyses. Data are reported as odds ratios (ORs) with 95% confidence intervals (CIs). Two-tailed p values < 0.05 were considered statistically significant.

Results

Demographic data and clinical characteristics

The participant flowchart is illustrated in Fig. 1. A total of 91 stroke patients were enrolled, but three patients with no PSG examination and two patients with no cognitive assessment were excluded. Therefore, 86 stroke patients (66 males and 20 females) were included in the analysis. The mean age of patients was 60.3 ± 12.1 years (range, 29–82 years), and the average duration of education was 8.9 ± 3.5 years (range, 3–15 years). OSA was present in 74.4% (64/86) of CI patients, with most patients (60.9%, 39/64) exhibiting mild to moderate OSA. Thirty-six HC participants (25 males and 11 females) also underwent cognitive assessment. The mean age of HC participants was 62.0 ± 7.7 years (range, 47–79 years), and the average duration of education was 10.2 ± 2.7 years (range, 5–17 years) (Table 1).

Differences in cognitive function between stroke patients and HC participants

Age and education duration were not significantly different between stroke patients and HC participants. Compared with HC participants, stroke patients showed impairments in attention and working memory, executive function, memory, visuospatial function, and language function (Table 2).

Among stroke patients, the prevalence of PSCI was 81.4% (70/86). Across the five cognitive domains, impairments in attention and working memory (87.1%, 61/70) and executive function (84.3%, 59/70) were the most common, whereas language impairment was the least common (28.6%, 20/70). The prevalence of visuospatial function and memory impairment was 61.3 and 83.8%, respectively.

Differences in clinical and sleep characteristics between PSCI and no PSCI patients

Compared with no PSCI patients, PSCI patients were older ($p = 0.014$) and had shorter education durations ($p = 0.009$). PSCI patients were also more likely to have a history of hypertension ($p = 0.015$), hyperlipidemia ($p = 0.047$), and OSA ($p = 0.030$) than no PSCI patients. However, there were no differences in sex, body mass index, Hamilton Anxiety Scale score, or Hamilton Depression Scale score between groups. The largest proportion of no PSCI patients had grade 0 leukoaraiosis (62.5%), whereas the largest proportion of PSCI patients had grade 1 leukoaraiosis (41.4%), which was a significant difference between groups ($p = 0.015$; Table 3).

PSCI patients had a shorter TST than no PSCI patients ($p = 0.002$), whereas there were no differences between groups in AHI ($p = 0.629$), L-SaO2 ($p = 0.065$), or average SaO2 ($p = 0.065$; Table 4).

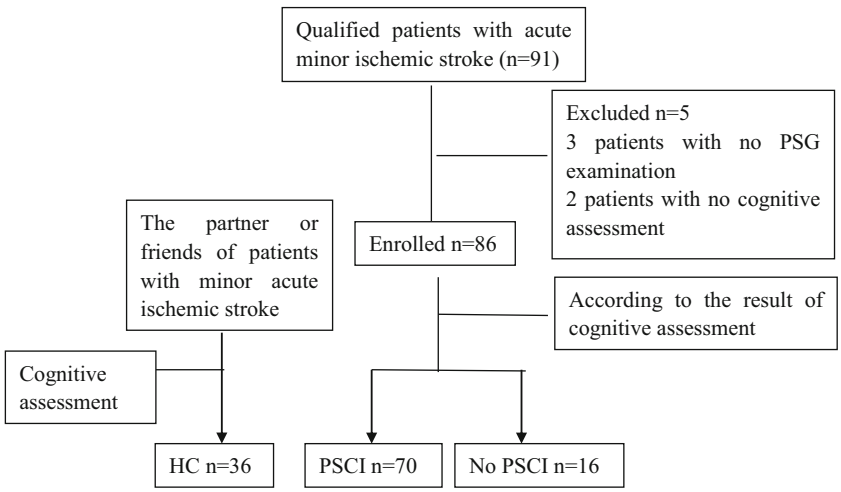

Fig. 1 Participant flowchart. Polysomnography (PSG), post-stroke cognitive impairment (PSCI), healthy control (HC)

Table 1 Demographic and clinical characteristics of stroke patients and HC participants

	Stroke	HC
Subjects, *n*	86	36
Age, years	60.3 ± 12.1	62.0 ± 7.7
Education duration, years	8.9 ± 3.5	10.2 ± 2.7
Male, *n* (%)	66 (76.7%)	25 (69.4%)
Hypertension, *n* (%)	55 (64.0%)	18 (50%)
Diabetes, *n* (%)	32 (37.2%)	8 (22.2%)
Atrial fibrillation, *n* (%)	6 (7.0%)	0 (0.0%)
Hyperlipidemia, *n* (%)	29 (33.7%)	9 (25.0%)
Smoking, *n* (%)	45 (52.3%)	7 (19.4%)
OSA, *n* (%)	64 (74.4%)	–
Mild to moderate OSA, *n* (%)	39 (60.9%)	–
Severe OSA, *n* (%)	25 (39.1%)	–
AHI, events/h	20.7 ± 18.5	–
NIHSS score	1.8 ± 1.5	–
Cerebral infarction location		
Cortical, *n* (%)	11 (12.8%)	–
Deep, *n* (%)	38 (44.2%)	–
Cortical and deep, *n* (%)	16 (18.6%)	–
Infratentorial, *n* (%)	21 (24.4%)	–

HC, healthy control; *OSA*, obstructive sleep apnea; *AHI*, apnea-hypopnea index; *NIHSS*, National Institute of Health Stroke Scale; *SD*, standard deviation

Associations between clinical characteristics and PSCI

In univariate analyses, age ($p = 0.020$), education duration ($p = 0.018$), hypertension ($p = 0.019$), TST ($p = 0.004$), and leukoaraiosis grade ($p = 0.004$) were associated with PSCI. In multivariate analyses, education level (OR 0.600; 95% CI 0.446–0.807; $p = 0.001$), L-SaO2 (OR 1.138; 95% CI 1.040–1.246; $p = 0.005$), and TST (OR 0.981; 95% CI 0.971–0.991; $p < 0.001$) were independently associated with PSCI (Table 5).

Discussion

Previous studies report a relatively high incidence of PSCI, with one third of stroke survivors exhibiting a significant degree of cognitive impairment within the first month after the event [29]. In the present study, we assessed the cognitive function of patients in the acute phase after mild acute cerebral infarction and found that the prevalence of PSCI was 81.4%. However, if we consider only a MoCA score of < 26 as indicative of cognitive impairment, the prevalence of PSCI would be 67.4%, suggesting that some PSCI patients cannot be detected through the use of the MoCA or MMSE alone. Therefore, a more comprehensive cognitive assessment is necessary to facilitate early detection of and

Table 2 Cognitive function in stroke patients and HC participants

	Stroke ($n = 86$) ($n = 86$)	HC ($n = 36$) ($n = 36$)	*p*
Age, years	60.3 ± 12.1	62.0 ± 7.7	0.354
Education duration, years	8.9 ± 3.5	10.2 ± 2.7	0.053
MMSE	27.0 (24.8, 29.0)	28.5 (28.0, 29.0)	< 0.001
MoCA	21.2 ± 4.4	24.8 ± 2.5	< 0.001
Attention and working memory			
TMT-A, time	86.9 ± 33.0	44.5 ± 10.7	< 0.001
Digit span forward	11.0 (9.0, 12.0)	13.0 (12.0, 13.8)	< 0.001
SDMT	26.6 ± 11.2	42.9 ± 9.6	< 0.001
Executive function			
TMT-B, time	145.2 ± 79.3	70.4 ± 20.0	< 0.001
TMT-B, errors	0.0 (0.0, 3.0)	0.00 (0.00–0.00)	< 0.001
Digit span backward	4.0 (3.0, 5.0)	7.0 (5.0, 8.8)	< 0.001
SCWT, time	10.0 (3.0, 15.2)	11.6 (7.7, 17.2)	0.139
SCWT, errors	0.0 (0.0, 3.0)	0.0 (0.0, 1.0)	0.123
Language			
SVFT, 1 min animals	13.9 ± 4.3	16.1 ± 3.1	0.006
Memory			
AVLT, immediate recall	13.3 ± 4.1	20.6 ± 3.8	< 0.001
AVLT, delayed recall	3.0 (1.0, 4.0)	7.0 (6.0, 8.0)	< 0.001
AVLT, recognition	8.0 (6.0, 10.0)	11.5 (10.3, 12.0)	< 0.001
Rey-O figure, recall	9.6 ± 6.6	19.7 ± 4.0	< 0.001
Visuospatial function			
Rey-O figure, copy	26.0 (22.5, 28.1)	31.0 (28.3, 32.0)	< 0.001
CDT	22.5 ± 5.3	26.4 ± 2.8	< 0.001

HC, healthy control; *MMSE*, Mini-Mental State Examination; *MoCA*, Montreal Cognitive Assessment; *TMT-A*, Trail Making Test A; *SDMT*, Symbol Digit Modalities Test; *TMT-B*, Trail Making Test B; *SCWT*, Stroop Color-Word Test; *SVFT*, semantic verbal fluency test; *AVLT*, Auditory Verbal Learning Test; *Rey-O figure*, Rey-Osterrieth complex figure; *CDT*, clock-drawing test

Table 3 Clinical characteristics of no PSCI and PSCI patients

	No PSCI ($n = 16$)	PSCI ($n = 70$)	p
n	16	70	
Age, years	53.6 ± 13.8	61.8 ± 11.3	0.014
Education duration, years	10.5 (9.0, 13.0)	8.0 (5.8, 12.0)	0.009
BMI	23.7 (23.6, 28.7)	24.2 (22.1, 27.5)	0.809
HAMA	3.0 (2.0, 4.8)	3.0 (2.0, 5.0)	0.479
HAMD	4.5 (2.0, 6.0)	3.0 (1.0, 5.0)	0.061
MMSE	28.5 ± 1.6	25.5 ± 3.4	< 0.001
MoCA	25.1 ± 3.0	20.3 ± 4.2	< 0.001
Male gender, n (%)	12 (75%)	54 (77.1%)	1.000
Hypertension, n (%)	6 (37.5%)	49 (70.0%)	0.015
Diabetes, n (%)	4 (25.0%)	28 (40.0%)	0.263
Hyperlipidemia, n (%)	2 (12.5%)	27 (38.6%)	0.047
Smoking, n (%)	8 (50.0%)	37 (52.9%)	0.836
Atrial fibrillation, n (%)	2 (12.5%)	4 (5.7%)	0.309
OSA, n (%)	8 (50.0%)	56 (80.0%)	0.030
Cerebral infarction location			0.899
Cortical, n (%)	2 (12.5%)	9 (12.9%)	
Deep, n (%)	7 (43.8%)	31 (44.3%)	
Cortical and deep, n (%)	2 (12.5%)	14 (20.0%)	
Infratentorial, n (%)	5 (31.3%)	16 (22.9%)	
Leukoaraiosis grade			0.015
0, n (%)	10 (62.5%)	19 (27.1%)	
1, n (%)	6 (37.5%)	29 (41.4%)	
2, n (%)	0 (0.0%)	18 (25.7%)	
3, n (%)	0 (0.0%)	4 (5.7%)	

PSCI, post-stroke cognitive impairment; *BMI*, body mass index; *HAMA*, Hamilton Anxiety Scale; *HAMD*, Hamilton Depression Scale; *OSA*, obstructive sleep apnea

Leukoaraiosis grade 0, no lesion; 1, punctuate foci; 2, beginning confluent foci; 3, confluent changes

intervention for PSCI. It is also notable that we found a higher prevalence of PSCI than that reported by previous studies. This may be because previous studies did not perform detailed cognitive assessment, we assessed cognitive impairment in the acute phase (i.e., < 2 weeks) after stroke, or our sample had a lower educational level.

The traditional diagnosis of mild cognitive impairment emphasizes an impairment in memory function, but not necessarily in stroke patients. Previous studies show that cognitive impairment in post-stroke patients manifests in various cognitive domains, with executive function impairment and aphasia being most common [30, 31]. We found declines in attention and working memory, executive function, memory function, and visuospatial function in stroke patients compared with HC participants. Of these, impairments in attention and work memory and executive function were most prevalent. Execution function, which is the ability to sort, plan, organize, initiate a task, and flexibly address other events that occur in the process, is associated with frontal subcortical loops, suggesting that PSCI involves frontal subcortical regions, consistent with the view of Moorhouse [32]. Moorhouse also proposes that macrovascular disease is important to but not common in the pathogenesis of vascular cognitive impairment, with small blood vessel disease being the most common pathological type of vascular cognitive impairment. In the present study, we found that PSCI patients exhibited more severe leukoariosis, providing further evidence of the involvement of frontal subcortical regions. Also, our finding that language impairment was the least common cognitive impairment may be due to the fact that we selected patients who had mild stroke without aphasia.

Some previous studies suggest that PSCI is associated with the volume and location of cerebral infarction, with particularly risky regions being the left carotid artery, paraventricular white matter, basal ganglia, cortex, small penetrating arteries in deep brain structures, and the dominant hemisphere [7, 8, 12]. By contrast, other neuroimaging studies suggest that the area of infarction (i.e., cortical, subcortical), type of infarction (i.e., single, multiple), and size of lesion do not correlate with frontal executive dysfunction or cognitive status [15, 33, 34]. We found no difference in cerebral infarction location between PSCI and no PSCI patients, but this may be due to small numbers for each individual location. However, we found that patients with PSCI were more likely to have more severe leukoaraiosis, consistent with previous studies showing that leukoaraiosis is closely related to PSCI [35–38]. Therefore, we speculate that leukoaraiosis is more closely associated with cognitive decline than infarction site, suggesting that we should focus on the degree of leukoaraiosis existing before stroke when assessing the prognosis of stroke patients. There are many risk factors for leukoaraiosis including age, hypertension, and tobacco use, with hypertension being the most important risk factor [39]. Studies on the relationship between sleep and leukoaraiosis are scarce. A cross-sectional investigation found that moderate-to-severe OSA is an independent risk factor for leukoaraiosis in the general population [40]. Moreover, a recent finding indicated that moderate-to-severe OSA is an independent predictor of moderate-to-severe leukoaraiosis in patients with acute cerebral ischemia [41]. However, one of the studies could not establish a correlation between OSA and leukoaraiosis [42]. In this study, we found that patients with PSCI were more likely to have more severe leukoaraiosis and OSA. However, we did not find that OSA and leukoaraiosis were independent risk factors of PSCI in the univariate analysis. The relationship among OSA, leukoaraiosis, and PSCI is complex; many common characteristics such as hypertension and age are shared, which is difficult to be classified by a cross-sectional study.

Although PSCI has received increasing attention, its risk factors are not yet fully understood. A review of risk factors for PSCI indicates that age, level of education, history of stroke, diabetes mellitus, hypertension, type of

Table 4 Sleep characteristics of no PSCI and PSCI patients

	No PSCI ($n = 16$)	PSCI ($n = 70$)	p
n	16	70	
ESS	4.5 (1.8, 6.5)	4.0 (2.0, 8.0)	0.944
AHI	22.7 ± 24.6	20.2 ± 17.0	0.629
ODI	9.2 (2.0, 43.6)	11.5 (2.4, 22.7)	0.876
L-SaO2	82.1 ± 8.1	85.9 ± 7.0	0.065
Average SaO2	94.5 (93.3, 95.8)	96.0 (94.0, 96.0)	0.065
TS90%	1.7 (0.2, 13.9)	0.6 (0.0, 3.3)	0.144
TST	435.1 ± 104.0	347.3 ± 98.1	0.002
Sleep efficiency	70.4 ± 18.9	66.4 ± 16.8	0.431
Number of awakenings	29.0 ± 20.3	32.0 ± 16.3	0.526
REM%	16.3 ± 6.5	15.6 ± 7.3	0.708
Respiratory-related microarousals	3.7 (0.6, 15.7)	5.1 (1.1, 15.1)	0.498
Sleep latency	17.0 (6.1,42.4)	17.5 (6.0, 40.5)	0.722

PSCI, post-stroke cognitive impairment; *ESS*, Epworth Sleepiness Scale; *AHI*, apnea-hypopnea index; *ODI*, oxygen desaturation index; *L-SaO$_2$*, lowest SaO_2; *TS90%*, percentage of time with $SaO_2 < 90\%$; *TST*, total sleep time; *REM*, rapid eye movement

stroke, affected region, size and location of infarction, depressive symptoms, and physical function may influence the cognitive status of stroke survivors [43]. At present, however, there are few studies of the relationship between sleep and PCSI, with most focusing on sleep apnea after stroke, and their results are inconsistent. As studies of the relationships between sleep time and structure and cognitive function after stroke are scarce [15, 20], we examined sleep-related parameters, including sleep apnea, TST, and sleep structure, in the present study. We found that older age, less education, hypertension, shorter TST, and more severe leukoaraiosis were risk factors for PSCI in univariate analysis, whereas less education, greater degree of hypoxia, and shorter TST were independent risk factors for PSCI in multivariate analysis.

Concerning the relationship between sleep characteristics and PSCI, we found that patients with PSCI showed a higher prevalence of OSA and shorter TST than patients without PSCI. However, there were no differences between PSCI and no PSCI patients in sleep structure or

Table 5 Univariate and multivariate binary logistic regression analyses of factors associated with PSCI

	Univariate analysis			Multivariate analysis		
	β	p	OR (95% CI)	β	p	OR (95% CI)
Gender	0.118	0.855	1.125 (0.319–3.973)			
Age	0.052	0.020	1.053 (1.008–1.100)			
Education duration	− 0.207	0.018	0.813 (0.685–0.966)	− 0.512	0.001	0.600 (0.446–0.807)
Hypertension	1.358	0.019	3.889 (1.251–12.0860			
Diabetes	0.693	0.269	2.000 (0.585–6.832)			
Smoking	0.114	0.836	1.121 (0.378–3.323)			
Hyperlipidemia	1.481	0.062	4.395 (0.926–20.871)			
Atrial fibrillation	− 0.857	0.349	0.424 (0.071–2.548)			
OSA	1.399	0.301	1.490 (0.700–3.170)			
L-SaO2	0.062	0.078	1.064 (0.993–1.140)	0.129	0.005	1.138 (1.040–1.246)
Average SaO2	0.181	0.203	1.198 (0.907–1.582)			
ESS	0.011	0.890	1.011 (0.870–1.173)			
TST	− 0.010	0.004	0.990 (0.983–0.997)	− 0.019	< 0.001	0.981 (0.971–0.991)
Leukoaraiosis grade	1.367	0.004	3.925 (1.533–10.051)			

OSA, obstructive sleep apnea; *L-SaO$_2$*, lowest SaO_2; *ESS*, Epworth Sleepiness Scale; *TST*, total sleep time

number of awakenings, consistent with a previous finding by Aaronson et al. that daytime sleepiness and sleep quality are not related to cognitive function [20]. We also found no difference in AHI between PSCI and no PSCI patients, with both groups showing mild to moderate AHI. Furthermore, univariate analysis showed that shorter TST was a risk factor for PSCI, and multivariate analysis showed that greater degree of hypoxia and shorter TST were independent risk factors for PSCI. Together, these results suggest that total TST and hypoxia at night may be more closely related to PSCI than sleep apnea.

The strengths of present study include its use of five cognitive domain scales and PSG to detect PSCI and assess sleep disturbances, respectively, and its exploration of the relationship between sleep disturbances and PSCI. However, some limitations should also be considered. First, our sample sizes were relatively small. Second, we did not measure infarct size or brain volume. Third, we excluded the patients with cognitive impairment before stroke based on the verbal history, rather than on a multi-domain neuropsychological test battery, which may have led to the enrollment of some patients with mild cognitive impairment who had no subjective cognitive impairment in self-evaluation; this might have resulted in higher false-positive rate of PSCI. Fourth, our study had a cross-sectional design that lacked long-term follow-up.

Conclusion

In conclusion, we found a high prevalence of PSCI after minor ischemic stroke. Although multiple cognitive domains were affected by stroke, impairments in attention and working memory and executive function were the most common, suggest that PSCI involves the frontal subcortex. Leukoaraiosis also appeared to be associated with cognitive decline after stroke. Although the risk factors for PSCI are numerous, shorter TST and degree of hypoxia at night warrant further attention.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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