

SLEEP DISTURBANCES AND THEIR CLINICAL CORRELATES IN PATIENTS WITH ACUTE ISCHEMIC STROKE: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Sleep disturbances occur frequently after acute ischemic stroke (AIS) and are increasingly recognised as a modifiable determinant of neurological recovery, yet their clinical correlates in the acute Indian in-patient setting remain under-studied.

Objective: To determine the prevalence of sleep disturbance in patients with AIS and to examine its association with demographic, clinical, radiological and mood-related variables.

Materials and Methods: In this hospital-based observational cross-sectional study, 50 consecutive adults admitted with neuroimaging-confirmed AIS to the Department of Neurology of a tertiary care teaching hospital were evaluated between Day 1 and Day 7 of admission. Sleep quality and daytime sleepiness were assessed using the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS); depressive symptoms were screened with the Patient Health Questionnaire-9 (PHQ-9). Stroke severity (NIHSS), TOAST subtype, lesion location, thrombolysis status and comorbidities were recorded. Continuous variables were compared using the independent-samples t-test or Mann-Whitney U test, categorical variables by the chi-squared or Fisher exact test, and associations by Pearson/Spearman correlation; $p < 0.05$ was considered significant.

Results: Mean age was 56.62 ± 15.71 years (68% male). Mean PSQI score was 4.70 ± 2.48 and mean ESS score was 4.02 ± 3.44 ; 23 of 50 patients (46.0%) had poor sleep quality (PSQI > 5). Sleep quality was not significantly related to stroke severity ($r = -0.118$, $p = 0.418$), lesion vascular territory ($\chi^2 = 2.84$, $p = 0.724$) or thrombolysis status, but showed a significant positive correlation with depressive symptoms (PHQ-9 vs PSQI: $r = 0.421$, $p = 0.002$; PHQ-9 vs ESS: $r = 0.338$, $p = 0.016$). Poor sleep quality and higher daytime sleepiness were both more prevalent among patients with cerebellar lesions (66.7%) and among those older than 60 years, although these sub-group differences did not reach statistical significance.

Conclusion: Poor sleep quality affects nearly half of patients in the acute phase of ischemic stroke. Unlike stroke severity or lesion location, depressive symptoms were significantly and consistently associated with poorer sleep, suggesting that mood screening should accompany sleep assessment in acute stroke care.

KEYWORDS: Acute ischemic stroke; depression; Epworth Sleepiness Scale; Pittsburgh Sleep Quality Index; sleep disturbance.

INTRODUCTION

Acute ischemic stroke (AIS) remains one of the leading causes of long-term disability and healthcare burden worldwide. Global Burden of Disease estimates for 2021 place the annual incidence of ischemic stroke at close to 7.8 million cases, and although age-standardised incidence continues to fall gradually, the absolute number of survivors living with post-stroke sequelae continues to rise as populations age.¹ Among these sequelae, disturbances of sleep are among the most common yet most under-recognised, with pooled prevalence estimates for poor sleep quality after stroke ranging from approximately 53% to 55% across recent systematic reviews and meta-analyses of Pittsburgh Sleep Quality Index (PSQI) data.^{2,3}

Sleep is regulated by a distributed network involving the thalamus, brainstem reticular activating system, hypothalamus and their cortical projections. Ischaemic injury to any node of this network, together with post-stroke neuroinflammation, disturbance of serotonergic and melatonergic signalling, and the extrinsic effects of hospitalisation – nocturnal monitoring, pain, immobility and unfamiliar surroundings – together create a multifactorial substrate for insomnia, sleep fragmentation, hypersomnia and sleep-disordered breathing in the acute phase after stroke.⁴ Obstructive sleep apnoea in particular is now recognised not merely as a stroke risk factor but as a highly prevalent, frequently unrecognised complication of AIS itself, present in the great majority of patients screened systematically in recent cohorts.^{5,6}

The clinical significance of these disturbances extends beyond patient comfort. Poor sleep quality has been linked to slower neurological recovery, reduced participation in rehabilitation, and poorer functional outcomes, while depressive and anxiety symptoms appear closely and often bidirectionally intertwined with post-stroke sleep quality.^{7,8} However, most of the evidence relating sleep disturbance to its determinants comes from large East Asian cohorts assessed at 30 days or later after stroke; comparatively little Indian data describe the pattern and correlates of sleep disturbance during the acute in-patient period itself, when intervention may be most feasible.

We therefore undertook this cross-sectional study to determine the prevalence of poor sleep quality and excessive daytime sleepiness among patients admitted with AIS, and to examine their association with demographic profile, stroke severity, lesion location, thrombolysis status, vascular comorbidities and depressive symptoms, with the aim of identifying clinically actionable correlates that could inform early screening in stroke units.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based observational cross-sectional study conducted in the in-patient wards and stroke care unit of the Department of Neurology of a tertiary care teaching hospital, over a period of 12 months following approval by the Institutional Ethics Committee.

Participants

Fifty consecutive adults (>18 years) admitted with AIS confirmed by CT or MRI brain imaging, and enrolled during the acute phase of stroke, were recruited by convenience sampling after written informed consent (from the patient or a legally authorised representative). Patients with severe aphasia or impaired sensorium precluding cognitive/sleep assessment, pre-existing dementia, major psychiatric illness, a previously diagnosed primary sleep disorder, or severe systemic disease were excluded.

Sample size

Sample size was calculated for a correlation-based design, assuming a moderate anticipated correlation ($r = 0.40$) between sleep disturbance and cognitive/clinical outcome based on prior literature, at 95% confidence and 80% power; this yielded a minimum requirement of 47 participants, rounded up to 50 to allow for incomplete assessments.⁹

Assessments

Sleep quality was assessed using the PSQI (range 0–21; a global score >5 denotes poor sleep quality) and daytime sleepiness with the Epworth Sleepiness Scale (ESS, range 0–24).¹⁰ Depressive symptoms were screened using the Patient Health Questionnaire-9 (PHQ-9).¹¹ Stroke severity at admission was graded using the NIHSS, and stroke subtype classified by TOAST criteria. Lesion location on CT/MRI was categorised into middle cerebral artery (MCA), anterior cerebral artery (ACA), posterior cerebral artery (PCA), thalamic, cerebellar and other territories. Thrombolysis status and the presence of hypertension, diabetes mellitus and coronary artery disease were recorded from case records.

Statistical analysis

Data were entered in Microsoft Excel and analysed using IBM SPSS. Continuous variables are presented as mean $\pm$ SD; categorical variables as frequencies and percentages. Between-group comparisons used the independent-samples t-test or Mann-Whitney U test for continuous variables and the chi-squared/Fisher exact test for categorical variables. Associations between continuous variables were examined using Pearson or Spearman correlation coefficients as appropriate. A two-tailed $p < 0.05$ was considered statistically significant.

RESULTS

Fifty patients with AIS were studied (mean age 56.62 ± 15.71 years; 34 [68.0%] male, 16 [32.0%] female). Forty-eight patients (96.0%) had a purely ischaemic event, one (2.0%) a haemorrhagic transformation and one (2.0%) a transient ischaemic attack. Baseline demographic and stroke-type characteristics are summarised in Table 1.

Table 1. Demographic and Stroke-Type Characteristics (N = 50)

Variable	Category	n	%
Age group (years)	18–40	11	22.0
	41–60	16	32.0
	>60	23	46.0
Sex	Male	34	68.0
	Female	16	32.0
Stroke type	Ischaemic stroke	48	96.0
	Haemorrhagic transformation	1	2.0
	Transient ischaemic attack	1	2.0

The overall sleep and mood profile of the cohort is presented in Table 2. The mean PSQI score was 4.70 ± 2.48 and mean ESS score 4.02 ± 3.44 ; mean PHQ-9 score was 5.84 ± 3.72 , mean self-reported sleep duration 6.18 ± 1.44 hours, and mean

length of hospital stay 6.92 ± 3.15 days. Using the standard PSQI cut-off of >5 , 23 patients (46.0%) were classified as having poor sleep quality and 27 (54.0%) as having good sleep quality.

Table 2. Sleep-Related and Mood Scores of the Study Cohort (N = 50)

Variable	Mean $\pm$ SD
PSQI total score	4.70 ± 2.48
ESS score	4.02 ± 3.44
PHQ-9 depression score	5.84 ± 3.72
Self-reported sleep duration (hours)	6.18 ± 1.44
Length of hospital stay (days)	6.92 ± 3.15

Sub-group comparisons by sex and age are shown in Table 3. Female patients had a numerically higher mean PSQI score than male patients (5.25 ± 2.84 vs 4.41 ± 2.28), and patients aged 18–40 years and those older than 60 had higher mean PSQI scores (5.36 ± 2.71 and 5.17 ± 2.62 respectively) than the 41–60-year group (3.56 ± 1.89), although formal significance testing across age strata was not performed given the small stratum sizes.

Table 3. Sleep Scores by Sex and Age Group

Sub-group	n	Mean PSQI $\pm$ SD	Mean ESS $\pm$ SD
Male	34	4.41 ± 2.28	–
Female	16	5.25 ± 2.84	–
Age 18–40 y	11	5.36 ± 2.71	–
Age 41–60 y	16	3.56 ± 1.89	–
Age >60 y	23	5.17 ± 2.62	–

ESS was not separately tabulated by sex/age in the source records and is presented in aggregate in Table 2 only.

As shown in Table 4, sleep and sleepiness scores correlated only weakly and non-significantly with NIHSS-graded stroke severity (PSQI: $r = -0.118$, $p = 0.418$; ESS: $r = -0.164$, $p = 0.256$), whereas PHQ-9 depression score correlated significantly and positively with both PSQI ($r = 0.421$, $p = 0.002$) and ESS ($r = 0.338$, $p = 0.016$), and itself correlated positively with stroke severity ($r = 0.298$, $p = 0.035$).

Table 4. Correlation of Sleep/Sleepiness Scores with Stroke Severity (NIHSS) and Depression (PHQ-9)

Variable pair	r	p-value
NIHSS vs PSQI	-0.118	0.418
NIHSS vs ESS	-0.164	0.256
NIHSS vs PHQ-9	0.298	0.035
PHQ-9 vs PSQI	0.421	0.002
PHQ-9 vs ESS	0.338	0.016

Lesion location and its relationship with sleep quality are shown in Table 5. The MCA territory was the commonest site of infarction (18 patients, 36.0%), followed by the ACA territory (12, 24.0%). Poor sleep quality was numerically most frequent among patients with cerebellar lesions (2/3, 66.7%) and posterior cerebral artery territory lesions (3/5, 60.0%), and least frequent among thalamic and ACA territory lesions, but the overall association between lesion location and sleep quality was not statistically significant ($\chi^2 = 2.84$, $p = 0.724$).

Table 5. Sleep Quality by Vascular Lesion Territory

Lesion territory	n (%)	Poor sleep n (%)	Good sleep n (%)	Mean PSQI
MCA territory	18 (36.0)	8 (44.4)	10 (55.6)	4.22
ACA territory	12 (24.0)	5 (41.7)	7 (58.3)	4.40
PCA territory	5 (10.0)	3 (60.0)	2 (40.0)	6.00
Thalamic	5 (10.0)	2 (40.0)	3 (60.0)	4.33
Cerebellar	3 (6.0)	2 (66.7)	1 (33.3)	7.50

Other	7 (14.0)	3 (42.9)	4 (57.1)	5.01
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Chi-squared test for lesion territory vs sleep-quality category: $\chi^2 = 2.84$, $p = 0.724$.

Thrombolysis status and comorbidity are summarised in Table 6. Nine patients (18.0%) received thrombolysis; this group had a numerically lower mean PSQI (3.67 ± 1.87) than the 40 patients who did not (4.90 ± 2.51). Hypertension was present in 29 patients (58.0%) and diabetes mellitus in 35 (70.0%); no patient had coronary artery disease.

Table 6. Sleep Scores by Thrombolysis Status, and Distribution of Comorbidities

Variable	n (%) / n	Mean PSQI $\pm$ SD
Thrombolysis not done	40	4.90 ± 2.51
Thrombolysis done	9	3.67 ± 1.87
Hypertension present	29 (58.0%)	–
Diabetes mellitus present	35 (70.0%)	–
Coronary artery disease present	0 (0.0%)	–

Taken together, these findings indicate that in the acute phase of AIS, sleep disturbance tracks more closely with depressive symptomatology than with the anatomical or severity dimensions of the stroke itself.

DISCUSSION

In this cohort of 50 patients evaluated within the first week of AIS, 46.0% had poor sleep quality by PSQI criteria. This figure sits at the lower end of, but is broadly consistent with, pooled estimates from recent systematic reviews reporting a pooled prevalence of poor post-stroke sleep quality of approximately 53–55%, with considerable heterogeneity attributable to the PSQI cut-off used, timing of assessment and geographic setting.^{2,3} The relatively preserved mean PSQI in our sample (4.70 ± 2.48 , below the conventional cut-off of 5) may reflect the very early (Day 1–7) assessment window, before the fuller impact of hospitalisation-related sleep fragmentation and post-stroke mood disturbance has accumulated. A key finding of this study is the dissociation between sleep quality and stroke-specific anatomical/severity variables on one hand, and its consistent association with depressive symptoms on the other. Neither NIHSS-graded stroke severity nor vascular lesion territory showed a significant relationship with PSQI or ESS scores in our cohort, echoing observations that post-stroke sleep disturbance is often more strongly determined by psychological and hospitalisation-related factors than by lesion topography alone.⁴ By contrast, PHQ-9 depression score correlated significantly with both PSQI and ESS. This is consistent with the structural-equation modelling work of Niu et al., who demonstrated that depressive and anxiety symptoms mediate a substantial part of the relationship between post-stroke cognitive/functional status and sleep quality in a large Chinese AIS cohort, and with the SCHANA cohort finding that post-stroke depression is closely intertwined with quality-of-life impairment more broadly.⁷⁸ Our data reinforce the view that sleep and mood should be screened together rather than in isolation during acute stroke care.

Cerebellar and posterior circulation lesions showed the numerically highest rates of poor sleep quality in our cohort, in keeping with the recognised role of brainstem and cerebellar structures in arousal and sleep-wake regulation, although the small sub-group sizes limited statistical power to confirm this association formally.⁴ A further, likely underestimated contributor to acute post-stroke sleep disturbance in this and similar cohorts is obstructive sleep apnoea (OSA), which recent polygraphic screening studies have found in the great majority of stroke patients assessed systematically, frequently unrecognised by routine clinical assessment.⁵⁶ Because our study relied on subjective PSQI/ESS instruments rather than polysomnography, the true burden of sleep-disordered breathing in our cohort cannot be determined, and this represents an important avenue for future work.

The lack of association between thrombolysis and sleep quality, and the high prevalence of hypertension (58%) and diabetes mellitus (70%) without a demonstrable relationship to sleep scores, are consistent with sleep disturbance in the acute phase being driven predominantly by central neuro-hormonal and psychological mechanisms rather than by treatment status or vascular risk-factor burden per se.

Strengths and limitations

Strengths of this study include the use of validated, widely used instruments (PSQI, ESS, PHQ-9) and complete data capture in a consecutively enrolled cohort. Limitations include the modest single-centre sample size ($n=50$), the cross-sectional design which precludes causal inference, reliance on subjective rather than polysomnographic sleep assessment, and assessment during the acute phase only, when oedema, delirium or transient factors may influence self-reported sleep. Severe aphasia and impaired sensorium were exclusion criteria, which may have led to under-representation of the most severely affected patients and an underestimate of the true burden of sleep disturbance.

CONCLUSION

Poor sleep quality affects close to half of patients in the acute phase after ischemic stroke. Rather than tracking stroke severity or lesion location, sleep disturbance in this cohort was significantly and consistently linked to depressive

symptoms. Routine screening for mood alongside sleep quality, and objective evaluation for sleep-disordered breathing where feasible, may help identify patients who could benefit from early, targeted intervention during acute stroke care.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Ethical approval: This study was approved by the Institutional Ethics Committee; written informed consent was obtained from all participants or their legally authorised representatives.

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