

Sleep Regularity and Migraine Burden: A Simulated Prospective Cohort Study

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Abstract

Background: Migraine and disturbed sleep frequently coexist, but improvement in both does not establish a causal relationship. This educational study used synthetic observations to demonstrate how sleep regularity and headache burden could be examined prospectively without presenting invented observations as clinical evidence.

Methods: A simulated cohort comprised 80 adults aged 18–55 years with episodic migraine, divided equally between structured sleep counseling and routine migraine advice. Counseling emphasized consistent waking times, adequate sleep opportunity, and reduced evening caffeine consumption. Outcomes were monthly migraine days, Pittsburgh Sleep Quality Index scores, and days using acute headache medication. Baseline and eight-week summaries were constructed to be clinically plausible. Analyses were descriptive; no participants were recruited, and no hypothesis tests were performed.

Results: Mean monthly migraine days decreased from 7.6 to 5.1 in the counseling group and from 7.4 to 6.6 in the comparison group. The corresponding reductions were 2.5 and 0.8 days. Mean sleep-quality scores declined by 3.3 and 1.0 points, respectively. Acute medication use decreased by 2.2 days with counseling and 0.7 days with routine advice. These differences reflect the specified synthetic dataset rather than measured treatment effects.

Conclusion: The simulation illustrates a transparent framework for studying behavioral sleep management in migraine. Its numerical patterns cannot establish efficacy, safety, or clinical recommendations. A randomized study with verified diaries, adequate follow-up, and prespecified adjustment for relevant confounders would be required to evaluate whether sleep counseling reduces migraine burden.

Keywords: migraine, sleep regularity, headache diary, sleep quality, behavioral counseling, synthetic data

1. Introduction

Cerebral small vessel disease is a common neurological condition characterized by injury to the brain's small penetrating arteries, arterioles, capillaries, and venules. Its magnetic resonance imaging manifestations include white matter hyperintensities, lacunes, cerebral microbleeds, and enlarged perivascular spaces. Although these abnormalities may initially occur without overt neurological

symptoms, their cumulative burden is associated with stroke, gait disturbance, mood changes, and cognitive impairment. As populations age and vascular risk factors remain prevalent, identifying clinically meaningful consequences of small vessel disease has become an important priority for preventive neurology.

Hypertension is a major modifiable contributor to cerebral microvascular injury. Sustained elevation of blood pressure may promote vascular remodeling, endothelial dysfunction, impaired autoregulation, and disruption of the blood–brain barrier. These processes can compromise tissue perfusion and contribute to chronic white matter damage. However, the relationship between hypertension, structural brain abnormalities, and cognitive performance is not uniform. Individuals with similar blood pressure measurements or apparently comparable imaging findings may differ substantially in cognitive functioning, suggesting that demographic characteristics, vascular comorbidities, and cognitive reserve influence clinical expression.

Cognitive impairment associated with cerebral small vessel disease frequently involves processing speed, attention, and executive functioning. Memory difficulties may also occur, particularly when vascular injury coexists with neurodegenerative pathology. In routine outpatient practice, subtle cognitive changes can remain unrecognized because patients may retain independence in basic daily activities and present primarily for headache, dizziness, or blood pressure management. Brief cognitive assessment may therefore provide useful information beyond the standard neurological examination. Nevertheless, screening scores require careful interpretation because education, language, mood symptoms, and sensory limitations can affect performance independently of cerebral vascular injury.

White matter hyperintensity severity offers a practical imaging marker for investigating these associations. Visual rating scales are relatively accessible and can be applied to routine magnetic resonance imaging without advanced computational infrastructure. Their clinical usefulness, however, depends on whether increasing lesion severity corresponds to measurable cognitive differences after relevant confounding factors are considered. White matter hyperintensities are also not specific to a single disease mechanism, and their location may influence cognitive outcomes in ways that global severity ratings cannot fully capture. Consequently, imaging findings should be interpreted alongside clinical and neuropsychological information rather than treated as direct evidence of vascular cognitive impairment.

Despite growing recognition of these relationships, uncertainty remains regarding their relevance in hypertensive adults attending general neurology services, particularly those without previous clinically apparent stroke or diagnosed dementia. Studies restricted to highly selected research cohorts may not fully reflect the heterogeneity of outpatient populations. Conversely, analyses that combine patients with major cerebrovascular events and those with incidental imaging changes may obscure associations relevant to earlier detection. Examining a clearly defined outpatient cohort could help clarify whether routine imaging findings identify patients who may benefit from more detailed cognitive evaluation.

This cross-sectional study therefore aimed to evaluate the association between white matter hyperintensity severity and cognitive performance among adults with hypertension undergoing brain magnetic resonance imaging in a neurology outpatient setting. A secondary objective was to assess whether this association persisted after adjustment for age, education, and selected vascular risk factors. We hypothesized that greater lesion severity would be associated with poorer cognitive performance, while recognizing that a cross-sectional design cannot establish temporal sequence or causality.

2. Materials and Methods

This prospective observational cohort study will examine the association between sleep quality one month after ischemic stroke and cognitive performance three months after stroke. Participants will be recruited consecutively from the neurology service of a tertiary hospital over an anticipated 18-month period. Assessments will be conducted during hospitalization, at 30 ± 7 days, and at 90 ± 14 days after stroke onset. The protocol will receive institutional ethics committee approval before recruitment, and written informed consent will be obtained from each participant or an authorized representative when

permitted by local regulations.

Eligible participants will be adults aged 40–80 years with a first-ever ischemic stroke confirmed by computed tomography or magnetic resonance imaging. Participants must be medically stable and able to complete assessments in the study language using locally validated instruments. Exclusion criteria will include previously diagnosed dementia, a progressive neurodegenerative disorder, major psychiatric illness requiring hospitalization within the preceding year, and severe aphasia or sensory impairment that prevents valid questionnaire administration. Patients experiencing delirium at the one-month assessment will have testing deferred within the permitted window; those with persistent delirium will not undergo the primary exposure assessment. These criteria will limit generalizability to patients with severe stroke.

A trained research nurse will collect demographic characteristics, educational attainment, smoking status, alcohol use, vascular risk factors, and medication history using interviews and medical records. Stroke severity will be measured using the National Institutes of Health Stroke Scale within 48 hours of admission. Lesion location and laterality will be abstracted from routine imaging reports by a neurologist unaware of subsequent sleep and cognitive results. Functional status will be recorded using the modified Rankin Scale. Prestroke cognitive decline will be screened through an informant questionnaire when a knowledgeable informant is available, with informant availability documented.

The primary exposure will be the Pittsburgh Sleep Quality Index global score at the one-month visit, reflecting sleep experiences during the preceding month. Higher scores indicate worse perceived sleep quality. The score will be analyzed continuously, with a score greater than five used only for descriptive classification of poor sleep. Questionnaire completion will be supervised without suggesting responses. Depressive symptoms will be measured using the Patient Health Questionnaire-9, and risk of obstructive sleep apnea will be assessed using STOP-Bang. STOP-Bang findings will be interpreted as screening results rather than diagnostic evidence. Hypnotic use, antidepressant treatment, and rehabilitation participation will also be recorded.

The primary outcome will be the Montreal Cognitive Assessment score at three months, administered by a trained assessor blinded to the one-month sleep score. Administration and education-related scoring adjustments will follow the validated instrument instructions. Testing will occur in a quiet room, with corrective glasses and hearing aids available when required. Cognitive scores will be interpreted as screening measures rather than diagnoses of dementia. Secondary outcomes will include three-month functional status and change in sleep quality between the one- and three-month visits.

A target of 180 participants will allow approximately 153 evaluable participants assuming 15% attrition. This sample would provide approximately 80% power at a two-sided significance level of 0.05 to detect an unadjusted correlation of approximately 0.23. This calculation is an approximate planning basis; precision of adjusted estimates will depend on covariate distributions and missingness. Reasons for withdrawal, missed visits, recurrent stroke, and death will be documented separately.

Continuous variables will be summarized using means and standard deviations or medians and interquartile ranges, as appropriate. Categorical variables will be presented as counts and percentages. The primary analysis will use multivariable linear regression to estimate the difference in three-month cognitive score per one-point increase in the one-month sleep score. Prespecified covariates will include age, sex, education, admission stroke severity, depressive symptom score, and sleep apnea screening score. Model assumptions will be examined using residual diagnostics, and robust standard errors will be used if heteroscedasticity is evident.

Missing data patterns will be examined before analysis. Multiple imputation will be considered when a missing-at-random assumption is clinically defensible, with complete-case estimates reported as a sensitivity analysis. Cognitive outcomes will not be imputed after death. Sensitivity analyses will additionally exclude participants with recurrent stroke before follow-up and explore adjustment for hypnotic use. Results will be reported with 95% confidence intervals. Secondary analyses will be considered exploratory, and associations will not be interpreted as causal.

3. Results

In this simulated prospective cohort of adults with first-ever ischemic stroke, 276 patients met the eligibility criteria, of whom 20 declined participation and 16 did not complete the 3-month cognitive assessment. The final analytical sample therefore comprised 240 participants. All numerical findings presented here are hypothetical and illustrate a plausible observational study rather than findings from an actual patient dataset. Participants completing follow-up had a median age of 66 years, and 138 (57.5%) were men. The median admission National Institutes of Health Stroke Scale (NIHSS) score was 5, consistent with a predominantly mild-to-moderate stroke population.

At 3 months, 88 participants (36.7%) met the prespecified screening definition of post-stroke cognitive impairment, based on an education-adjusted Montreal Cognitive Assessment (MoCA) score below 26. The remaining 152 participants (63.3%) scored at or above this threshold. The median MoCA score was 22 in the cognitively impaired group and 28 in the group without impairment. This classification indicated performance below a screening threshold and did not establish a diagnosis of dementia. No participant had a documented dementia diagnosis before the index stroke, although previously unrecognized cognitive decline could not be excluded.

Baseline demographic, clinical, and imaging characteristics are summarized in Table 1. Participants with cognitive impairment were older, had fewer years of formal education, and had greater neurological deficit at admission. Hypertension and diabetes were numerically more frequent in this group. Moderate-to-severe white matter hyperintensity burden, defined as a Fazekas score of 2–3, was present in 51.1% of participants with impairment compared with 25.0% of those without impairment.

Table 1. Baseline characteristics according to cognitive status at 3 months

Characteristic	Cognitive impairment (n = 88)	No cognitive impairment (n = 152)
Age, years, median (IQR)	71 (64–77)	63 (56–70)
Male sex, n (%)	48 (54.5)	90 (59.2)
Education, years, median (IQR)	10 (8–12)	12 (10–15)
Hypertension, n (%)	66 (75.0)	99 (65.1)
Diabetes mellitus, n (%)	30 (34.1)	36 (23.7)
Admission NIHSS score, median (IQR)	7 (4–10)	4 (2–7)
Atrial fibrillation, n (%)	20 (22.7)	28 (18.4)
Fazekas score 2–3, n (%)	45 (51.1)	38 (25.0)
Acute infarct volume, mL, median (IQR)	12.4 (5.6–24.8)	8.1 (3.2–17.5)

Across the complete sample, 83 participants (34.6%) had moderate-to-severe white matter hyperintensities. Cognitive impairment occurred in 45 of these participants (54.2%), compared with 43 of 157 participants (27.4%) with absent or mild white matter hyperintensities. The corresponding absolute difference was 26.8 percentage points. The unadjusted odds ratio was 3.14 (95% confidence interval, 1.81–5.46). These proportions describe an association between imaging burden and subsequent screening performance, without determining whether white matter abnormalities directly contributed to the observed cognitive deficits.

In the prespecified multivariable logistic regression model, moderate-to-severe white matter hyperintensities remained associated with cognitive impairment after adjustment for age, sex, education, admission NIHSS score, and diabetes. The adjusted odds ratio was 2.18 (95% confidence interval, 1.16–4.10). Older age and greater admission stroke severity also showed positive associations with impairment, whereas additional years of education were associated with lower odds. The estimates for male sex and diabetes were less precise, with confidence intervals spanning the null value. Full adjusted estimates are presented in Table 2.

Table 2. Multivariable associations with cognitive impairment at 3 months

Predictor	Adjusted odds ratio (95% confidence interval)	P value
Fazekas score 2–3 versus 0–1	2.18 (1.16–4.10)	0.016
Age, per 10-year increase	1.62 (1.16–2.26)	0.005
Male versus female sex	0.88 (0.47–1.64)	0.687
Education, per additional year	0.87 (0.79–0.96)	0.006
Admission NIHSS, per additional point	1.12 (1.04–1.21)	0.004
Diabetes mellitus, present versus absent	1.38 (0.70–2.72)	0.353

The adjusted association for white matter hyperintensity burden was attenuated relative to the unadjusted estimate. This pattern was consistent with shared variation involving age, educational attainment, and stroke severity, although the analysis could not separate confounding from overlapping biological pathways. No strong collinearity was detected among the included predictors; all variance inflation factors were below 2.0. The model had an apparent area under the receiver operating characteristic curve of 0.79, decreasing to 0.76 after bootstrap correction for optimism. These internal performance estimates did not constitute external validation.

A sensitivity analysis using a more restrictive education-adjusted MoCA threshold below 24 classified 61 participants (25.4%) as cognitively impaired. Under this definition, moderate-to-severe white matter hyperintensities remained associated with impairment, with an adjusted odds ratio of 2.06 (95% confidence interval, 1.03–4.12). The wider interval reflected the smaller number of outcome events. Adding log-transformed acute infarct volume to the primary model produced a similar, slightly attenuated estimate for white matter burden; adjusted odds ratio 2.02 (95% confidence interval, 1.06–3.86).

Functional outcomes also differed by cognitive screening status. At 3 months, a modified Rankin Scale score of 0–2 was recorded in 51 participants with cognitive impairment (58.0%) and 126 without impairment (82.9%). Because cognition and functional status were assessed concurrently, this difference did not establish temporal direction. Among the 16 enrolled participants without cognitive follow-up, median admission NIHSS was 8, compared with 5 among completers, indicating possible selection toward less severe stroke in the analytical sample. Overall, the simulated findings showed an association between greater white matter disease burden and poorer cognitive screening performance that persisted across the specified adjusted analyses.

4. Discussion

The findings of this cross-sectional study suggest that poorer sleep quality is associated with greater headache burden among adults with episodic migraine. Participants reporting more disturbed sleep also tended to report more frequent headache days and greater migraine-related disability. This pattern supports considering sleep quality alongside conventional measures of migraine severity during clinical assessment. However, the observed associations should not be interpreted as evidence that sleep disturbance causes migraine progression, because sleep and headache characteristics were measured within the same observation period.

The relationship is biologically plausible and likely bidirectional. Sleep disruption may influence pain sensitivity through changes in descending pain modulation, autonomic regulation, and hypothalamic function. These systems also participate in migraine susceptibility and the regulation of circadian rhythms. Conversely, migraine attacks can delay sleep onset, interrupt sleep continuity, and encourage compensatory daytime sleep. Anticipatory concern about subsequent attacks may further interfere with restorative sleep. The present findings therefore fit a model of interacting sleep and headache processes rather than a simple pathway in which one condition consistently precedes the other.

An important interpretive issue is the contribution of psychological symptoms. Anxiety and depressive

symptoms can influence both perceived sleep quality and the functional impact of migraine. Their presence may partly account for the observed relationship, while also representing clinically meaningful components of the same symptom network. Statistical adjustment for these factors can reduce some confounding, but it cannot fully distinguish confounders from mediators in a cross-sectional analysis. Consequently, any association remaining after adjustment should be understood as a conditional relationship within the measured variables, not as proof of an independent biological effect.

The clinical relevance of the findings lies primarily in identifying potentially overlooked problems during routine migraine care. A brief sleep history may reveal irregular sleep schedules, insomnia symptoms, excessive daytime sleepiness, or features suggesting sleep-disordered breathing. These conditions require different assessment pathways and should not be grouped under a single assumption of poor sleep hygiene. Incorporating sleep questions into headache consultations may help clinicians characterize symptom burden more comprehensively. Nevertheless, this study does not establish that treating sleep disturbance will reduce migraine frequency, and it does not justify substituting sleep-focused care for established migraine management.

Several alternative explanations deserve consideration. Participants with more disabling migraine may have used acute medication more frequently, altered caffeine consumption, reduced physical activity, or changed work schedules. Each of these factors could affect sleep and potentially distort the observed association. Preventive medications may also influence sleep architecture or daytime alertness, making medication exposure particularly relevant to interpretation. Where exposure information is incomplete, residual confounding remains possible. Moreover, self-reported sleep and disability measures may share reporting influences, including current distress, which could strengthen associations without a corresponding increase in objectively measured sleep disruption.

Measurement timing is also important. Migraine burden can fluctuate across weeks, whereas sleep questionnaires often summarize experiences over a defined retrospective interval. Imperfect alignment between these intervals may obscure temporal relationships and introduce recall error. Prospective headache diaries paired with repeated sleep assessments would better distinguish persistent sleep difficulties from changes surrounding individual attacks. Objective sleep measures could add complementary information, although they would not replace the patient's experience of nonrestorative sleep, which remains relevant to daily functioning and care planning.

The clinical relevance of the observed association between cognitive impairment and poorer functional recovery after ischemic stroke lies in its potential to improve rehabilitation planning. Motor deficits often dominate early assessment, whereas difficulties with attention, executive function, and memory may receive less systematic consideration. Nevertheless, these cognitive domains influence the ability to follow instructions, retain compensatory strategies, and participate consistently in therapy. Cognitive assessment should therefore complement, rather than replace, established measures of neurological severity and physical independence.

These findings support a more individualized approach to rehabilitation, but they do not establish that cognitive screening itself improves outcomes. When impairment is identified, clinicians may consider shorter instructional sequences, repetition, written prompts, and caregiver involvement according to individual needs. Such adaptations are clinically plausible, although their effectiveness requires direct evaluation. Importantly, a low screening score should not be interpreted as evidence that a patient cannot benefit from rehabilitation. It may instead indicate a need to modify how treatment is delivered and how progress is assessed.

Several limitations should guide interpretation. An observational design cannot determine whether cognitive impairment directly contributes to reduced recovery or primarily reflects greater underlying brain injury. Residual confounding may persist despite adjustment for measured clinical characteristics. Lesion location, pre-existing cerebral small-vessel disease, frailty, and the intensity of rehabilitation are potential influences that may not be fully captured by routine assessments. Reverse relationships are also possible, because severe disability, reduced activity, and prolonged hospitalization can adversely affect cognitive performance during recovery.

The timing and method of cognitive evaluation introduce additional uncertainty. Early post-stroke

testing may be influenced by fatigue, sleep disruption, pain, emotional distress, medication effects, or an unfamiliar hospital environment. Language impairment and educational background can also affect screening scores independently of broader cognitive ability. Consequently, a single assessment should not be treated as a definitive characterization of long-term cognitive status. Repeated evaluation after medical stabilization would help distinguish transient difficulties from persistent impairment and clarify whether particular cognitive domains have different relationships with functional outcomes.

Generalizability also requires caution. Patients who can complete cognitive testing may differ systematically from those with severe aphasia, reduced consciousness, or substantial sensory limitations. Underrepresentation of these groups could alter both the estimated frequency of impairment and its apparent relationship with recovery. In addition, rehabilitation access, family support, and discharge practices vary across healthcare settings. These contextual factors may modify the observed association and should be considered before applying the findings to populations with different clinical or social characteristics.

Future research should use prospective, multicenter designs with repeated cognitive and functional assessments and clearer documentation of rehabilitation exposure. Domain-specific testing, where feasible, could identify whether executive dysfunction or attentional deficits offer information beyond global screening scores. Intervention studies are also needed to determine whether cognition-adapted rehabilitation produces meaningful gains in independence, participation, and patient-reported quality of life. Analyses should address missing assessments explicitly, because inability to complete testing is unlikely to occur randomly.

Overall, the findings suggest that cognition is a relevant component of post-stroke recovery assessment, while leaving the direction and modifiability of the association unresolved. Integrating cognitive evaluation into multidisciplinary care may help identify support needs, but treatment decisions should remain individualized and grounded in the patient's broader neurological, functional, and social circumstances.

5. Conclusion

This study suggests that poor sleep quality is associated with greater headache-related disability among adults with migraine, supporting the clinical relevance of sleep assessment in neurological practice. The observed relationship should be interpreted as an association rather than evidence that sleep disturbance directly causes migraine progression. Migraine and sleep problems may influence each other, while psychological distress, medication use, and behavioral factors may contribute to both. Accordingly, the findings favor an integrated clinical perspective rather than a single explanatory mechanism.

From a practical standpoint, assessment of sleep duration, regularity, and perceived quality may help clinicians identify potentially modifiable concerns that are not fully captured by headache frequency alone. Attention to these factors could complement established migraine evaluation and treatment, particularly when patients report persistent disability despite ongoing care. However, the present findings do not establish that improving sleep will necessarily reduce migraine frequency or severity, nor do they justify replacing evidence-based preventive or acute therapies with sleep-focused approaches. Suspected sleep disorders should be evaluated according to their clinical features and relevant diagnostic standards.

Several considerations limit the strength and generalizability of these conclusions. The observational design restricts causal inference, and reliance on self-reported symptoms may introduce recall error and shared measurement bias. Residual confounding remains possible, especially for mood symptoms, analgesic exposure, occupational demands, and other determinants of sleep and pain. Recruitment from a defined clinical setting may also limit applicability to individuals with milder migraine or those who do not seek specialist care. These limitations warrant caution when translating group-level associations into individual treatment decisions.

Future research should prioritize longitudinal designs with repeated headache and sleep assessments to clarify temporal relationships. Incorporating validated disability measures, objective sleep

indicators where feasible, and systematic assessment of comorbidities would improve interpretability. Randomized studies are needed to determine whether targeted sleep interventions provide additional benefit alongside standard migraine management and to identify patients most likely to respond.

Overall, this study highlights sleep quality as a clinically relevant correlate of migraine burden. Its principal implication is to support comprehensive, individualized assessment while maintaining appropriate uncertainty about causation, treatment effects, and the direction of the relationship.

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