RISK FACTORS FOR SLEEP APNEA AND RESULTS OF RESPIRATORY POLYGRAPHY IN PATIENTS WITH ACUTE ISCHEMIC STROKE

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This study investigated the prevalence and risk factors of sleep apnea (SA) in patients with acute ischemic stroke. Among 99 patients assessed using respiratory polygraphy within 7 days of stroke onset, 66.7% had sleep apnea, mostly of the obstructive type. Severe SA was found in 25.3% of cases. Compared to non-apneic patients, those with SA had higher BMI, diabetes, higher Epworth Sleepiness Scale (ESS) and STOP-BANG scores, and a greater likelihood of lacunar stroke. Interestingly, although BMI was higher among patients with SA, it did not remain a significant factor in multivariate analysis. Instead, ESS score (OR = 1.14), early neurological deterioration (OR = 6.81), and diabetes mellitus (OR = 6.53) emerged as independent predictors. The study concludes that sleep apnea is common in acute ischemic stroke and emphasizes the need for objective sleep assessments, as traditional anatomical markers are not reliable indicators in this population.

Keywords: Sleep apnea, acute ischemic stroke, respiratory polygraphy.

I. INTRODUCTION

Sleep apnea, particularly obstructive sleep apnea (OSA), is increasingly recognized as a significant and modifiable risk factor for stroke. Sleep apnea is characterized by recurrent episodes of apnea or hypopnea during sleep, resulting in intermittent hypoxemia, autonomic dysfunction, and systemic inflammation-mechanisms that contribute to endothelial damage, hypercoagulability, and accelerated atherosclerosis, all of which are implicated in cerebrovascular disease pathogenesis. The bidirectional relationship between OSA and stroke further complicates this association: while OSA exacerbates stroke risk through these pathways, acute brain injury may itself impair respiratory control, perpetuating sleep apnea and worsening outcomes.[1]

The prevalence of OSA in acute stroke populations is alarmingly high, with studies reporting rates of 60 – 70%, yet it remains critically underdiagnosed during the acute phase.[2] Barriers to diagnosis include logistical challenges in conducting gold-standard polysomnography (PSG) in hospitalized patients, lack of awareness among clinicians, and competing clinical priorities in stroke management. This oversight is consequential, as untreated OSA is linked to prolonged hospitalization, poorer functional recovery, and increased stroke recurrence.[3] Early identification of OSA could not only elucidate modifiable stroke risk factors but also enable timely interventions such as continuous positive airway pressure (CPAP) therapy, which has shown promise in reducing secondary vascular events.[4]

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To address diagnostic challenges, this study employed respiratory polygraphy (RPG) as a pragmatic alternative to PSG. RPG provides a simplified, bedside-compatible method to assess sleep apnea, validated in stroke settings with high sensitivity for detecting moderate-to-severe OSA.[5] Its feasibility and accessibility make it ideal for integration into routine stroke care protocols, particularly in resource-constrained environments. This study aimed to: 1) Determine the prevalence of sleep apnea in acute ischemic stroke patients using RPG. 2) Investigate associations between sleep apnea and clinical characteristics.

II. MATERIALS AND METHODS

1. Subjects

This prospective, cross-sectional observational study was conducted at the Stroke Center of Bach Mai Hospital between April and October 2024. Consecutive adult patients (≥ 18 years old) admitted with a confirmed diagnosis of acute ischemic stroke were considered for inclusion. Only those who met the predefined eligibility criteria were enrolled. Among eligible patients, selection for polygraphy assessment was performed based on the availability of recording devices, using a randomization sequence generated from https://www.random.org/sequences/.

Inclusion Criteria

- Acute ischemic stroke (symptom onset ≤ 7 days).
- Ability to provide informed consent or availability of a legally authorized representative.

Exclusion Criteria

- Pre-existing diagnosis of sleep apnea or use of positive airway pressure therapy.
- Cognitive impairment (Glasgow Coma Scale score < 12) or communication deficits precluding participation.
- Active pneumonia, heart failure (NYHA Class III/IV) or acute sinusitis, as these conditions may muddle respiratory measurements.

2. Methods

Sleep Study

Respiratory Polygraphy (RPG): Sleep apnea was assessed using portable RPG (NOX T3S, NOX MEDICAL, Iceland) within 7 days of stroke onset at the bedside. The device recorded the following parameters overnight at the patient's bedside: Airflow (nasal cannula/pressure transducer), Respiratory effort (thoracic and abdominal belts), Oxygen saturation (SpO_2) via pulse oximetry, Heart rate. For sleep apnea assessment, only studies with ≥ 4 hours of recorded airflow, respiratory effort, and oxygen saturation were analyzed.

Scoring and Definitions:

- Apnea: Cessation of airflow ≥ 90% for ≥ 10 seconds.
- Hypopnea: Reduction in airflow ≥ 30% for ≥ 10 seconds, accompanied by ≥ 3% oxygen desaturation.
- Apnea-Hypopnea Index (AHI): Total number of apneas and hypopneas per hour of recording time.

Patients were categorized into two groups: Non-Sleep apnea: AHI ≤ 10 events/hour, Sleep apnea: AHI > 10 events/hour. This cutoff has been shown in previous studies to provide the best balance of sensitivity and specificity when using respiratory polygraphy (RPG) compared with the gold standard polysomnography (PSG).[6] Furthermore, this threshold is meaningful, as it captures patients at increased risk of adverse cardiovascular and functional outcomes, while reducing the risk of overdiagnosis in patients with mild or incidental events.[7,8]

All studies were manually scored by a board-certified sleep specialist blinded to clinical data,

following the American Academy of Sleep Medicine (AASM) version 3 (2023).

Data Collection

Demographic and clinical data were collected from medical records, including age, gender, body mass index (BMI), history of hypertension or diabetes, stroke severity assessed by the National Institutes of Health Stroke Scale (NIHSS), and stroke etiology based on the TOAST classification. Additional parameters such as LDL-cholesterol levels, STOP-BANG score, Epworth Sleepiness Scale (ESS), and early neurological deterioration (END) were also recorded. Early Neurological Deterioration (END) was defined as an increase in the National Institutes of Health Stroke Scale (NIHSS) score by ≥ 2 points within the first 72 hours of admission, not attributable to hemorrhagic transformation, recurrent stroke, or systemic complications (e.g., infection, metabolic derangements).

Statistical Analysis

Data were analyzed using SPSS statistical software, version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± SD (normally distributed) or median (IQR) (non-normal), and categorical variables as frequency (%). Group comparisons used: Independent t-test or Mann-Whitney U test for continuous variables. Chi-square or Fisher's exact test for categorical variables. Multivariate Logistic Regression: Adjusted for age, sex, BMI, diabetes, LDL-cholesterol, ESS, END, lacunar stroke and stroke severity (NIHSS) to identify independent predictors of sleep apnea (AHI >10). Results were reported as odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

3. Research ethics

The study was conducted in accordance with the Declaration of Helsinki, and informed consent was obtained from all subjects and their legal guardian(s). The protocol and questionnaire were reviewed and approved by the Institutional Review Board at Bach Mai Hospital, Hanoi, Vietnam (IRB-VN01019), decision No. 982/BM-HDDD, dated 08/03/2024. In addition, the study was approved by the Ethics Committee of Hanoi Medical University (IRB-VN01001), approval number NCS2024/GCN-HMUIRB, dated 15/05/2024. To protect privacy, any personal information that could be used to identify a specific individual has been removed. Data were collected anonymously and managed in compliance with data protection legislation.

III. RESULTS

A total of 99 patients were enrolled in the study, with a mean age of 60.3 ± 11 years old. Among them, 69 (69.7%) were male. The median time from stroke onset to the respiratory polygraphy was 3 [IQR 2 – 4] days. Of all participants, 66 patients (66.7%) were diagnosed with sleep apnea (defined as AHI > 10). Among these, 57 patients had predominantly obstructive sleep apnea (86.4%), 7 had predominantly central sleep apnea (10.6%), and 2 had mixed-type sleep apnea. Furthermore, 25 patients (25.3%) had severe sleep apnea with an AHI > 30.

Table 1 compares baseline characteristics between patients with and without sleep apnea. Patients with sleep apnea had significantly higher BMI (24.7 ± 3.4 vs. 23.2 ± 2.5, p = 0.026), higher prevalence of diabetes mellitus (34.8% vs. 9.1%, p = 0.006), higher Epworth Sleepiness Scale (ESS) scores (6.5 [IQR 3 – 12] vs. 4 [IQR 1 – 7.5], p = 0.007), and higher STOP-BANG scores (4 [IQR 3 – 5] vs. 3 [IQR 2 – 4], p = 0.020). Notably, the prevalence of lacunar stroke was significantly higher in the sleep apnea group (59.1% vs. 30.3%, *p* = 0.007).

Table 1. Characteristics of apneic (AHI > 10) and non-apneic patients

Variable	Total (n = 99)	AHI ≤ 10 (n = 33)	AHI > 10 (n = 66)	*p*
Age (years)	60.3 ± 11	58.9 ± 12.3	61.0 ± 10.3	0.374
Male	69 (69.7%)	24 (72.7%)	45 (68.2%)	0.643
BMI (kg/m²)	24.2 ± 3.2	23.2 ± 2.5	24.7 ± 3.4	*0.026*
Neck circumference	37.3 ± 3.8	36.8 ± 3.1	37.6 ± 4.1	0.279
Hypertension	79 (79.8%)	26 (78.8%)	53 (80.3%)	0.860
Diabetes Mellitus	26 (26.3%)	3 (9.1%)	23 (34.8%)	*0.006*
NIHSS	3 (2 – 4)	3 (1 – 4)	3 (2 – 4.25)	0.192
END	16 (16.2%)	2 (6.1%)	14 (21.2%)	0.054
ESS Score	6 (3 – 11)	4 (1 – 7.5)	6.5 (3 – 12)	*0.007*
Stop-Bang Score	4 (3 – 4)	3 (2 – 4)	4 (3 – 5)	*0.02*
Lacunar stroke	23 (23.2%)	10 (30.3%)	39 (59.1%)	*0.007*
Stroke Onset to RPG (days)	3 (2 – 4)	3 (2 – 4)	3 (2 – 4)	0.736

BMI indicates Body Mass Index; NIHSS: National Institutes of Health Stroke Scale; END: Early Neurological Deterioration; ESS: Epworth Sleepiness Scale; RPG: Respiratory Polygraphy

Table 2 presents respiratory polygraphy data. As expected, the apnea-hypopnea index (AHI) was significantly higher in the sleep apnea group (30.4 ± 19.3 vs. 6.2 ± 2.5, p < 0.001). Obstructive apnea index (OAI), central apnea index (CAI), and hypopnea index (HI) were also significantly elevated in this group. The mean oxygen saturation (SpO2) was slightly lower in patients with sleep apnea (93.6% vs. 94%, p < 0.001), while mean heart rate did not differ significantly between groups.

Table 2. Respiratory polygraphy data of patients with and without sleep apnea

Variable	AHI ≤ 10 (n = 33)	AHI > 10 (n = 66)	p
AHI	6.2 ± 2.5	30.4 ± 19.3	< 0.001
OAI	0.8 (0.2 – 2.0)	5.6 (2.7 – 10.4)	< 0.001
CAI	0.2 (0 – 0.9)	0.8 (0.2 – 3.75)	0.003
HI	4.2 ± 2.3	13.6 ± 9.8	< 0.001
Mean HR	67 (60.1 – 71.8)	67 (60.6 – 75.8)	0.667
Mean SpO_2	94 (93.0 – 95.5)	93.6 (92.4 – 94.4)	< 0.001

AHI indicates Apnea Hypopnea Index; OAI: Obstructive Apnea Index; CAI: Central Apnea Index; HI: Hypopnea Index; HR: Heart Rate

A multivariate logistic regression model adjusted for age, gender, BMI, ESS score, neck circumference, END (early neurological deterioration), diabetes mellitus, lacunar stroke and hypertension revealed the following independent predictors of sleep apnea:

ESS Score: Each unit increase in ESS score was associated with elevated odds of sleep apnea (OR = 1.14, 95% CI [1.02 – 1.27], p = 0.018).

END (Early Neurological Deterioration): Patients with END had 6.8-fold higher odds of sleep apnea (OR = 6.81, 95% CI [1.21 – 38.41], p = 0.03).

Diabetes Mellitus: Diabetes was independently associated with a 6.53-fold increase in sleep apnea risk (OR = 6.53, 95% CI [1.45 – 29.34], p = 0.014).

IV. DISCUSSION

The key findings of this study reveal a high prevalence of sleep apnea in acute ischemic stroke patients, with 66.7% (66/99) exhibiting an AHI > 10 events/hour, predominantly obstructive in nature (86.4%). This aligns with global data emphasizing sleep apnea as a critical comorbidity in stroke populations. Notably, 25.3% (25/99) had severe sleep apnea (AHI > 30), underscoring the need for targeted screening and intervention in this high-risk group.

Early neurological deterioration (END) showed a significant association with sleep apnea (OR = 6.81, 95% CI: 1.21 – 38.41), suggesting a potential bidirectional relationship.[9] While sleep apnea may contribute to secondary brain injury through intermittent hypoxia and sympathetic overactivity, it is also possible that more severe neurological impairment predisposes patients to central or mixed apneic events due to impaired respiratory drive.

Diabetes, particularly Type 2 diabetes, has been identified as a significant risk factor for the development and exacerbation of sleep apnea. This relationship is increasingly notable due to the high prevalence of obstructive sleep apnea (OSA) among individuals with diabetes; studies suggest that approximately 70% of those with Type 2 diabetes also suffer from OSA.[10] The bidirectional nature of this relationship is concerning, as each condition can aggravate the other, complicating management and increasing the risk of severe health complications, including cardiovascular disease and worsening glycemic control. The pathophysiological mechanisms linking diabetes and sleep apnea involve insulin resistance, metabolic dysfunction, and the impact of intermittent hypoxia experienced during apnea episodes. These mechanisms can impair glucose metabolism and contribute to the progression of Type 2 diabetes.

The Epworth Sleepiness Scale (ESS) score demonstrated a statistically significant association with sleep apnea in our stroke population, with each unit increase in ESS linked to a 14% increase in the odds of having sleep apnea (OR = 1.14, 95% CI: 1.02 – 1.27, p = 0.018). This finding is consistent with existing literature where excessive daytime sleepiness, as measured by ESS, has been shown to correlate with the severity of sleep apnea in the general population.[11] However, the role of the ESS in post-stroke patients remains controversial. Stroke-related factors, including cognitive impairment, aphasia, and reduced insight, may limit the accuracy of subjective assessments like the ESS. Moreover, many stroke patients with sleep apnea do not report significant daytime sleepiness, potentially due to reduced physical activity or altered neurological perception of fatigue. Despite these limitations, the persistence of a significant association in our study suggests that, when feasible, the

ESS may still serve as a useful clinical indicator of underlying sleep apnea in selected stroke patients.

Although BMI was associated with sleep apnea in univariate analysis, it did not retain significance in multivariate regression. This suggests that in stroke populations, conventional risk factors for obstructive sleep apnea such as obesity may be less predictive than in the general population. Similarly, neck circumference were not significantly associated with the presence of sleep apnea in patients with acute ischemic stroke. This contrasts with findings in the general population, where increased upper body adiposity is a strong predictor of OSA.[12] The lack of association in our stroke cohort suggests that the pathophysiology of OSA in this context may differ and may be more closely related to stroke-induced neuromuscular impairment, particularly involving the pharyngeal dilator muscles. Stroke-related weakness in upper airway musculature can lead to increased airway collapsibility during sleep, independent of typical anthropometric risk factors.

This study has several limitations. First, it was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Second, the diagnosis of sleep apnea was based on respiratory polygraphy rather than full polysomnography, which may underestimate the severity or type of sleep apnea, especially in detecting arousals or sleep architecture changes. Third, the cross-sectional design does not allow for determination of causality between sleep apnea and stroke-related outcomes. The fourth limitation of this study is that respiratory polygraphy was performed during the acute phase of stroke, which may be associated with technical challenges such as patient instability and an increased likelihood of signal loss or incomplete data acquisition. Finally, although multivariate analysis was performed, residual confounding factors cannot be entirely ruled out.

V. CONCLUSION

Sleep apnea was highly prevalent among patients with acute ischemic stroke, with significant associations observed with diabetes mellitus, early neurological deterioration, and higher Epworth Sleepiness Scale (ESS) scores. While body mass index showed a univariate association with sleep apnea, it was not an independent predictor in multivariate analysis. These findings suggest that in the context of acute stroke, metabolic and neurological factors may play a more central role in the pathogenesis of sleep apnea than traditional risk markers like obesity or neck circumference. Early screening and appropriate management of sleep apnea in stroke patients may be important for improving neurological outcomes.

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The authors declare no conflict of interest relevant to this study.

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