

An Overnight Rhythmic Sleep-Wake Pattern in Stroke and Obstructive Sleep Apnea: A Case Report and Literature Review

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Abstract

Background and Objective: Obstructive sleep apnea (OSA) is a common sleep breathing disorder and a comorbid condition in patients with stroke. The prevalence of sleep disorders as a stroke consequence is high. Being independent of vascular risk factors, OSA is associated with increased risk of ischemic stroke. Here, we report a patient with OSA and stroke.

Case Report: This case report presents a patient with a history of stroke and hemiparesis who underwent polysomnography (PSG) for evaluating OSA because of snoring, witnessed apnea, and excessive daytime sleepiness (EDS). Then the subject underwent continuous positive airway pressure (CPAP) titration for treating OSA. In first night of PSG, there was a repetitive cyclic pattern of sleep and arousals. After CPAP titration study and with CPAP pressure of 15 cmH₂O, OSA and rhythmic changes of electroencephalography (EEG) were resolved.

Conclusion: OSA as a risk factor for stroke can be associated with PSG cyclic wake-sleep pattern in these patients, and this pattern can be resolved by CPAP therapy.

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Keywords: Obstructive sleep apnea; Stroke; Continuous positive airway pressure; Arousal

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Introduction

Obstructive sleep apnea (OSA) is a common sleep breathing disorder and a comorbid condition in patients with stroke. OSA and stroke are accompanied with hypertension (HTN), hyperlipidemia, cardiovascular diseases (CVDs), diabetes mellitus type 2 (DM2), and atrial fibrillation (AF) (1). Screening of OSA (as a risk factor) in all patients with stroke is recommended (2). The gold standard treatment for moderate and severe OSA is continuous positive airway pressure (CPAP) therapy (3). Previous studies have shown that treatment of patients with stroke and comorbid OSA with CPAP can lead to better prognosis and improved neurological symptoms (4, 5).

Sleep disordered breathing (SDB) (obstructive or central apnea) is prevalent in patients after

stroke (6). OSA which is airflow reduction (hypopnea) or cessation (apnea) during sleep and central sleep apnea (CSA) (loss in central respiratory drive) can lead to sleep arousals as a defense mechanism (7, 8). Being independent of vascular risk factors, OSA is associated with increased risk of ischemic stroke (9). The most common effects of hemispheric stroke on sleep pattern are sleep fragmentation, decreased sleep efficiency, and increased stage N1 and wake time after sleep onset (10).

This case report presents a patient with a history of stroke and hemiparesis who underwent polysomnography (PSG) for evaluating OSA because of snoring, witnessed apnea, and excessive daytime sleepiness (EDS). Then the subject underwent CPAP titration for treating OSA.

Case Report

Our patient was a 64-year-old man referred to Imam Sleep Clinic affiliated to Tehran University of Medical Sciences, Tehran, Iran. The patient

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was married and had two children. He was a high school graduate. He was not working due to disability. He had following chief complaints: snoring, poor sleep quality, witnessed apnea, EDS, and restless legs syndrome (RLS).

His symptoms gradually started after stroke. He had no symptoms of cataplexy, narcolepsy, and parasomnia. He mentioned mild degrees of depressive and anxiety symptoms [Beck Depression Inventory II (BDI-II) score: 19]. STOP-BANG, Epworth Sleepiness Scale (ESS), and Insomnia Severity Index (ISI) scores were 4, 14, and 19, respectively. According to the International Classification of Sleep Disorders – Third Edition (ICSD-3) criteria, he had RLS.

His past medical history included a single cerebrovascular attack 11 years ago, tonic-clonic seizures, and benign prostatic hyperplasia (BPH). His seizure started shortly after stroke and history was compatible with generalized tonic-clonic seizure, repeating around 2 months after stroke with three months interval. He reported duration of 1-3 minutes for each seizure episode with postictal confusion. Seizure episodes were improved with a daily dose of 300 mg phenytoin and 1500 mg levetiracetam. He had not experienced seizure episode for the past three years. Examinations of carotid arteries and cardiac evaluations were normal and it seems that the type of stroke was cryptogenic.

The patient had no history of smoking. He was under treatment with the following medications: aspirin, clopidogrel, levetiracetam, phenytoin, prazosin, tamsulosin, sertraline, levodopa, and baclofen.

In physical examination, Mallampati class was 3. Body mass index (BMI) was 28 kg/m². Heart rate (HR) and blood pressure (BP) were normal. Cardiac and pulmonary auscultation was normal. No nail clubbing was detected. In neurologic examination, he was alert and had good orientation; he had mild central facial palsy. Left side extremities were paretic (force grade 1/5) and spastic. In sensory examination, he had decreased sensation of pinprick in left side.

In brain imaging, right cortical frontotemporal infarction [right middle cerebral artery (MCA) territory] was detected (Figure 1).

Continuous electroencephalography (EEG) and PSG recording were performed for evaluation of snoring and possible sleep breathing problems. Electrooculography (EOG), submental electromy-

ography (EMG), electrocardiography (EKG), air-flow and respiratory effort, peripheral hemoglobin saturation (SpO₂), and EEG were recorded; sleep stages and arousals were scored according to the American Academy of Sleep Medicine (AASM) criteria (11).



Figure 1. Brain computed tomography (CT)-scan without contrast of the patient

EEG scoring and monitoring in 10- and 30-second epochs was performed and showed a disruption of the sleep-wake cycle, with episodes of sleep and wakefulness recurring regularly during night. N1, N2, arousal, and wakefulness repeated semi rhythmically and led to sleep fragmentation and unusual sleep pattern in hypnogram.

Amplitude of EEG waves in right hemisphere decreased. Not obvious epileptic pattern was detected during study.

Stages N1 and N2 were increased and slow-wave sleep (SWS) and rapid eye movement (REM) sleep were decreased, respectively. In first-night PSG, there was a repetitive rhythmic pattern of sleep and arousals (near 10 epochs of sleep then wakefulness). The patient had 304 limb movements during sleep. Table 1 depicts PSG characteristics of our patient.

After diagnosis of severe OSA [respiratory disturbance index (RDI): 72.3/hour] (11), patient was candidate for CPAP titration study and with CPAP pressure of 15 cmH₂O, sleep apnea and rhythmic changes of EEG were resolved. Positive airway pressure (PAP) titration study was performed one week after PSG study.

Table 1. Polysomnographic (PSG) characteristics

PSG parameter	Value
Total sleep time (minute)	155.3
Time in bed (minute)	248.0
Sleep efficiency (%)	63.6
SOL (minute)	3.5
Wake after sleep onset (minute)	76.5
Stage N1 (%)	25.0
Stage N2 (%)	72.0
Stage N3 (%)	1.0
Stage REM (%)	2.0
RDI (per hour)	72.3
Apnea (n)	48
Hypopnea (n)	139
Mean O ₂ saturation (%)	90.0
Minimum of O ₂ saturation (%)	66.0

PSG: Polysomnography; REM: Rapid eye movement; RDI: Respiratory disturbance index; SOL: Sleep onset latency

In figures 2 and 3, hypnograms of PSG study and CPAP titration study are shown, respectively.

Discussion

Our patient was diagnosed with stroke and severe OSA. In the first night of diagnostic PSG, the patient had increased stage N1, N2, decreased SWS and REM sleep, sleep fragmentation, and an unusual sleep-wake pattern. As diagnosis of severe OSA was made, PAP titration study was performed for the patient and intriguingly, the rhythmic wake-sleep pattern was resolved with CPAP.

More than 50% of patients with stroke have SDB, mostly OSA, and at least 20-40 percent of them show sleep-wake disorder, mostly insomnia, EDS/fatigue, or hypersomnia. Sleep architectural changes can follow acute supratentorial strokes which may persist in poor outcome hemispheric strokes (12).

Our patient had a history of hemispheric stroke; therefore, we assumed that the cyclic sleep-wake pattern of sleep and arousals may be due to brain's ischemia. However, surprisingly, after CPAP titration and with increasing the pressure, unusual sleep pattern became less prominent

and was resolved on hypnogram of titration study.

We had two hypotheses for aforementioned observation:

- 1- Damage to the frontotemporal cortex has created this rhythmic pattern.
- 2- OSA has led to the partially regular occurrence of arousal and rhythmic pattern of wake-sleep.

SDB and sleep-wake disorders in patients with stroke happen several times because of brain damage and complications of stroke (12). Cerebrovascular pathologies can be associated with sleep fragmentation, independent of known cardiovascular risk factors and medical comorbidities (13). Sleep fragmentation has a role in risk factors for cerebrovascular pathologies such as HTN and abnormal glucose metabolism; and more sleep fragmentation are associated with atherosclerosis and subcortical brain infarctions (12).

Arousals following resumption of respiration in OSA cause sleep fragmentation, which dramatically is resolved by CPAP therapy (14). It seems that pathogenic mechanism of SDB in patients with hemispheric stroke is related to (decreased upper airway muscle tone) on compromised control of upper airway muscles (15).

As the cyclic pattern of sleep-wake in hypnogram was resolved in the night of titration study, we concluded that OSA led to occurrence of arousals and rhythmic pattern of sleep-wake rather than cerebrovascular pathology in this patient.

OSA predisposes patients to drug resistance, HTN, atherosclerosis, cardiac arrhythmia, hypercoagulation, heart failure (HF), and paradoxical emboli which are decreased after CPAP therapy (9). SDB is an independent risk factor for stroke and CPAP therapy in stroke patients with stroke and OSA improves stroke recovery significantly and prevents vascular events non-significantly (16). Accordingly, we suggest the evaluation and treatment of sleep fragmentation in patients with stroke (for preventing recurring attacks).

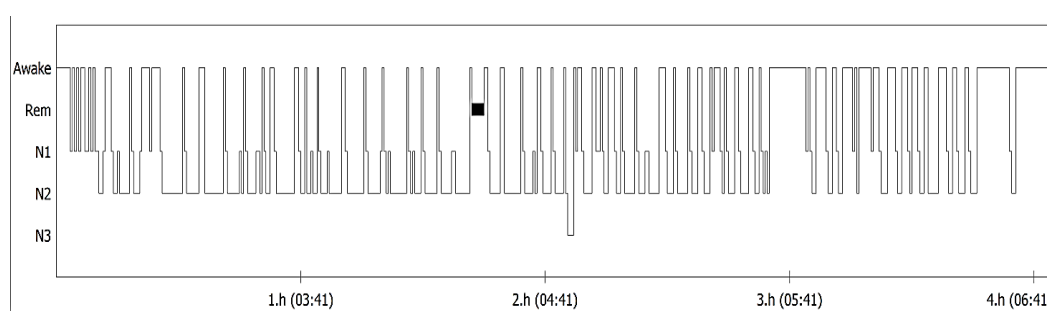


Figure 2. Hypnogram of patient in diagnostic polysomnographic (PSG) study

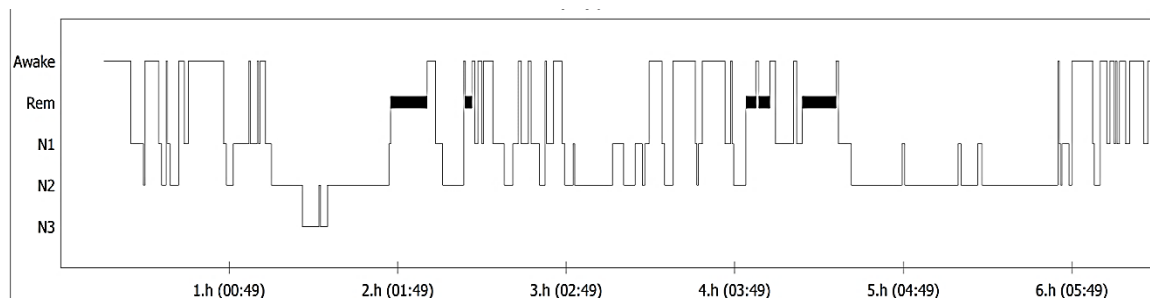


Figure 3. Hyponogram of patient in titration study

Conclusion

OSA as a risk factor for stroke can be associated with cyclic sleep-wake alternating pattern, which can be resolved by CPAP therapy. Neurologists and sleep medicine specialists should be aware that EEG changes during EEG monitoring may be due to sleep breathing problem and not only neurological problem; thus, we recommend performing PSG in patients with stroke and risk factors for OSA or significant sleep problems.

Conflict of Interests

Authors have no conflict of interests.

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