Case Report

Papilledema and Seizure-like Episodes in a Patient with Obstructive Sleep Apnea Syndrome: A Case Presentation and Literature Review

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Abstract

Background and Objective: Obstructive sleep apnea (OSA) syndrome is a common sleep-related breathing disorder characterized by repeated episodes of the upper airway collapse during sleep. Loud snoring and daytime sleepiness are the main complaints; however, the condition may present with a variety of symptoms and signs.

Case Report: A 65-year-old woman was admitted to neurology ward with the diagnosis of intractable seizures. The physical examination revealed papilledema and huge obesity with body mass index of 41. Brain imaging was unremarkable. The pressure of cerebrospinal fluid was 33 cm H_2O. As respiratory distress was observed, pulmonary evaluations were conducted. Finally, an overnight polysomnography confirmed the diagnosis of severe OSA. Positive airway pressure removed the symptoms.

Conclusion: This presentation suggests that severe OSA should be considered as a differential diagnosis for epilepsy and a probable cause of raised intracranial pressure, especially at the presence of obesity.

Keywords: Epilepsy; Obstructive sleep apnea; Papiledema

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Introduction

Obstructive sleep apnea syndrome (OSAS) is a sleep-related breathing disorder affecting 2-4% of population (1). Patients with OSA have recurrent episodes of the upper airway obstruction during their sleep which often is associated with transient hypoxemia and increasing respiratory effort with subsequent disruption of sleep. The most known risk factors for developing this syndrome include obesity, age, and male gender. The patients with OSAS often present with complaints of breathing pauses during sleep, loud snoring, and excessive daytime sleepiness (EDS) (2).

According to the International Classification of Sleep Disorders-2 published by the American Academy of Sleep Medicine, OSAS is diagnosed with either a respiratory disturbance index (RDI) > 15 irrespective of presence of symptoms or an RDI > 5 accompanied by any of the following features: (1) sleep attacks, EDS, fatigue, or insomnia; (2) awakening with a choking feeling; (3) loud snoring and/or breathing pauses reported by bed partner.

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RDI is defined by a mean number of apneas + hypopneas + respiratory effort-related arousals per hour of sleep. An RDI score above 30 is considered as severe sleep apnea. Overnight polysomnography is the standard method for the diagnosis of sleep apnea during which respiratory flow recordings, pulse oximetry, the chin and legs electromyography, electrooculography, and electroencephalography are simultaneously performed (3).

Undiagnosed OSAS may result in serious consequences including arterial and pulmonary hypertension, cardiovascular diseases (4), impaired cognition, and psychiatric symptoms such as depression and irritability (5, 6). Moreover, neuro-ophthalmic manifestations including optic disc swelling, glaucoma, and ischemic optic neuropathy linked to OSA have been reported (7, 8). Increase intracranial pressure (ICP) may be seen concurrently with OSA (9-11).

While effective and often feasible treatment with continues positive airway pressure is available (12), many of the patients go unrecognized (3, 13). Patients with OSA often do not seek appropriate medical assessment for the diagnosis of disease; therefore, the diagnosis of sleep apnea may be partially delayed due to the nature of the disease (3). In addition, there are conditions that mimic sleep apnea such as seizures, especially in children (14). As a matter of fact, the diagnosis of OSA usually requires a high level of clinical suspicion (3).

Here, we present an OSA patient who had been diagnosed with intractable epilepsy for 2 years when she admitted to hospital with signs and symptoms of increased intracranial pressure.

Case Report

A 65-year-old woman was admitted to the neurology ward of Qaem Hospital in Mashhad, Iran, because of repeated episodes of decreased level of consciousness and persistent headaches. During the previous 2 years, she had been diagnosed with epilepsy based on a history of nocturnal limb jerking and diurnal confusional states which had been aggravated since 1 month ago. Till 2 years ago, there was no history of epileptic events or episodic loss of consciousness. Despite being treated with two antiepileptic drugs (AEDs) (topiramate 200 mg/day and levetiracetam 3 g/day), she had a deteriorating condition. The seizure-like episodes occurred mostly during nocturnal sleep in the form of a limb shaking for few minutes followed by a period of several minutes of unresponsiveness and confusion. The patient suffered from EDS, thus, the events happened in daytime sleep as well.

She had also a 7 years history of hypertension for which was under treatment with captopril 100 mg/day. A history of loud snoring was also detected. Physical examination revealed bilateral papilledema and huge obesity with body mass index of 41. The neck circumference was 47 cm.

Regarding laboratory tests, fasting blood sugar was 109 and 99 mg/dl in two repeated measurements. The blood sugar just after an unconsciousness episode was 167 mg/dl. Urea, creatinine, sodium, and potassium concentration of serum were in normal range. Conventional electroencephalogram showed diffuse slowing predominantly in the range of theta waves, without epileptiform discharges in two repeated records. It was initially interpreted as an interictal finding of epileptic events. Magnetic resonance imaging showed neither hydrocephaly nor

Table 1. Summary of respiratory events

Respiratory event	Total		Supine		Non-supine		REM		NREM	
	Count	Index	Count	Index	Count	Index	Count	Index	Count	Index
Apneas + hypopneas	73	54.8	4	48	69	55.2	0	0	73	54.8
Apneas + hypopneas + RERAs	73	54.8	4	48	69	55.2	0	0	73	54.8
Apneas	62	46.5	4	48	58	46.4	0	0	62	46.5
Obstructive apneas	62	46.5	4	48	58	46.4	0	0	62	46.5
Mixed apneas	0	0	0	0	0	0	0	0	0	0
Central apneas	0	0	0	0	0	0	0	0	0	0
Hypopneas (obstructive)	11	8.2	0	0	11	8.8	0	0	11	8.2
RERAs	0	0	0	0	0	0	0	0	0	0

RERA: Respiratory effort related arousals, NREM: Non-rapid eye movements

space-occupying lesions. Magnetic resonance venogram was unremarkable. The pressure of cerebrospinal fluid (CSF) was 33 cm H_2O. Otherwise, CSF examination was normal. Investigation for other causes of raised ICP including infectious diseases and endocrine conditions were not remarkable. As a respiratory distress was observed, consultation with pulmonologist was performed. Then, the patient underwent pulmonary function test and lung perfusion which were normal. Based on otolaryngology consultation, anterior rhinoscopy showed no abnormal findings in terms of septal deviation, nasal polyposis, conchal hypertrophy, and other obstructive lesions. Oropharyngeal examination revealed no tonsillar hypertrophy and following indirect laryngoscopy, laryngeal structures, and vocal fold movement were normal.

Finally, polysomnography was performed which revealed severe OSA with an index of apnea-hypopnea of 54.8 and significant declined blood O_2-saturation. Table 1 presents the respiratory events. As shown in table 2, the blood O_2-saturation decreased frequently (64 times) fewer than 60%. The sleep efficiency was 74%. A summary of sleep structure is illustrated in table 3. During the monitoring, two long periods of apnea was recorded after which, the patient woke up with limb shaking and loud groans, the condition which was described as fits by the patient's daughter. The patient also had a dramatic fall of O_2-saturation to below 20% during the event. Concurrently, electroencephalographic (EEG) monitoring did not demonstrate any epileptiform discharges. Sleep latency in polysomnogram was 0.5 minutes, which was compatible with sleepiness. Bi-level positive airway pressure with an inspiratory pressure of 16 cm H_2O and an expiratory pressure of 12 cm H_2O removed the respiratory events appropriately. After 1 month, the CSF pressure was in normal range. Now, by 6 months administration of this mode of treatment, the patient is symptom-free, the papilledema is resolved and the AEDs are discontinued.

Table 2. Desaturation event counts (by lowest desaturation)

Desaturation event	Supine	Right	Left	Prone	Upright	REM	NREM	Awake	Total
< 95%	5	0	94	0	0	0	98	1	99
< 90%	5	0	94	0	0	0	98	1	99
< 85%	5	0	94	0	0	0	98	1	99
< 80%	5	0	93	0	0	0	97	1	98
< 75%	5	0	92	0	0	0	96	1	97
< 70%	5	0	85	0	0	0	89	1	90
< 65%	5	0	76	0	0	0	80	1	81
< 60%	2	0	62	0	0	0	64	0	64

NREM: Non-rapid eye movements, REM: Rapid eye movements

Table 3. Sleep architecture and sleep stages of the patient

First pre-treatment start	22:54:00	Sleep stages	Time (min.)	TST (%)
Last pre-treatment end	00:41:30	Awake	28	25.9 (TRT%)
Number of periods	1	Stage N1	30.5	38.1
TRT	108 min	Stage N2	47	61.2
TSP	107.5 min	Stage N3	2.5	0.6
TST	80 min	-	-	-
Awake time	28 min	Stage R	0	0
Wake after sleep onset	27.5 min	Movement time	0	0
SE	74%	Technical intervention	0	0 (TRT%)
SOL	0.5 min			
Number of Stage 1 shifts	33			
Awakenings	13			
Stage changes	76			
Number of REM periods	0	REM	0	0
REM latency	- min	NREM	80	100

TRT: Total recording time, TSP: Total sleep period, TST: Total sleep time, SE: Sleep efficiency, SOL: Sleep onset latency, REM: Rapid eye movement

Discussion

OSAS may present with a wide range of symptoms. The most common features are loud snoring, daytime sleepiness, and nocturnal or morning headaches (15). As the diagnosis may be made late (3, 13), other manifestations are not uncommon among patients with this potentially life threatening condition. The exact mechanism of rising ICP, which has been recently noted in literatures (9, 10) is not well known (10). While cerebral vasodilatation due to intermittent nocturnal hypoxemia was hypothesized (7, 9), there may be a coexisting condition in obese individuals (11). Nevertheless, there is some evidence that appropriate treatment of OSA results in lowering ICP (10, 16). Similarly, our patient showed dramatic improvement by treatment.

Interestingly, the patient has relieved from symptoms mimicking epileptic seizures by resolving the sleep disorder. While more prevalent sleep breathing disorders have been shown among epileptic individuals (17, 18), the presented patient was in fact suffering from nocturnal jerking movements and arousals secondary to prolong respiratory events. We also indicated that irresistible sleep attacks secondary to EDS were responsible for episodes of unresponsiveness. A similar status in narcolepsy is considered as a differential diagnosis for epileptic seizures (14). Moreover, diffuse slowing in the routine EEG is a finding seen in drowsiness due to EDS (19). As a comment, we suggest that diffuse slowing in EEG should not be always interpreted as a manifestation of epilepsy. It may be due to several conditions including sleepiness during recording of EEG, and metabolic or hypoxic encephalopathy. All of the above manifestations resolved by appropriate treatment of OSA, in spite of discontinuing AEDs.

Conclusion

Sleep-related breathing disorder should be considered at the presence of EDS, obesity, and a history of loud snoring. Morning headaches raised ICP and nocturnal convulsive like movements may be seen in this condition. An EEG may show slow waves which may be due to sleepiness during the record.

Conflict of Interests

Authors have no conflict of interests.

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