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Obstructive sleep apnea syndrome: The case of residual sleepiness

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ABSTRACT

The case of a 62-year-old male illustrated the medical emergent need for treatment. Severe excessive daytime sleepiness, neurocognitive functioning symptoms were evident from his chronic untreated sleepiness. An all-night polysomnogram confirmed the diagnosis of Obstructive Sleep Apnea (OSA). The implication of this case's chronic untreated experience of OSA is discussed in terms of neurocognitive/brain structure laboratory findings. Results at intake, one and three months of this case's sleep and cognitive functioning are reported.

Keywords: OSA; Excessive Daytime Sleepiness; Cognitive Behavioral Interventions; Treatment Compliance

1. BACKGROUND AND HISTORY

Obstructive sleep apnea (OSA) is a severe respiratory condition. Patients will commonly present with fatigue and excessive daytime sleepiness [1,2]. An all night polysomnographic recording that measures brain waves, muscle tension, heart rate, rate of respiration, and temperature to determine stages of sleep is used to determine the diagnosis; the collapse of pharyngeal muscle closes the throat and blocks oxygen intake (apneic event). This reduction in airflow leads to a drop in oxygen saturation. Following the drop in oxygen saturation, there is an increase in pharyngeal muscle effort. This respiratory effort results in an arousal reaction (measured by EEG) and leads to urgent upper airway opening. This respiratory effort is accompanied by snoring and tachycardia. An apneic event can last from 20 - 40 seconds and most commonly occurs during REM and Stage 1 sleep [3-5]. Mild hypotension with elevation diastolic pressure, brachycardia occurs during the apneic event of sleep apnea. An all-night PSG study is required for diagnosis with the presence of confirming symptoms of sleep apnea at the two-hour interval; a continued positive airflow pressure (CPAP) device is implemented [2].

A 62-year-old male presented to sleep laboratory with chief complaint of excessive daytime sleepiness, dry mouth at waking and complaints from his wife of loud snoring. His excessive daytime sleepiness was present for twenty-eight years. He described two "fender benders" where he fell asleep momentarily at a red light and his foot came off the pedal. He reported waking at night choking and gasping for air for the last 11 years. With this, he did not feel he had a good night of sleep. He snored very loudly and his wife reported that she thought her hearing has been affected—the two children moved their beds in their bedrooms to the furthest wall and used headphones/earplugs regularly. The patient had headaches when he woke up. He denied history of cataplexy, sleep paralysis or sleep related hallucinations. He had no allergies, head or neck injuries. Patient had his tonsils. He denied Parasomnias, restless legs and insomnia.

Patient admitted to heavy cigarette (two packs per day) and alcohol use (nightly 4 - 5 drinks). He had been diagnosed with essential hypertension, lipidemia, gastroesophageal reflux disorder and liver cirrhosis. His medications included naproxen, omeprazole, hydrochlorothiazide, and lipofen. The patient has been obese (BMI = 37) for the last twenty-eight years. The patient works as a car mechanic in a chain automotive store. He has worked in this area consistently but has had seven different positions within this time at different companies with excessive absences and low productivity referenced as causes of the firings/transfers.

The assessment of a patient presenting with symptoms of Sleep Apnea includes a physical examination, history of medical/surgical/sleep events and consideration of ordering laboratory tests such as a complete blood count, oxygen desaturation test, Holter monitor for cardiovascular functioning assessment, mental status examination, and brief cognitive testing. A determination is made at

this point as to whether the symptom criteria are met and if they are, the patient is referred for an all night polysomnographic sleep study.

The physical examination measured an obese male weighing 297 lbs., 68 inches. His blood pressure was 127/84; heart rate was 79 beats per minute. His respiratory rate was 20 with heavy mouth breathing, evidence of smokers cough. The pharynx was crowded, (Mallampati, class 3), he had macroglossia. Neck circumference is 18 inches. The rest of general exam was normal. The neurological exam was positive for short term memory/working memory deficits, attention and concentration difficulties. A screening vocabulary test revealed his cognitive functioning the low average range.

2. POLYSOMNOGRAPHIC TESTING

The patient was given a split night study carried out with 2.0 h baseline data and continuous positive airway pressure (CPAP) titration lasting 4.5 hours. The patient has a CPAP machine with an integrated "smart card". Adherence data on the card reveals that he used the CPAP 58% in month 1, 47% in month 2, and 44% in month 3.

3. TREATMENT OPTIONS

The treatment options in this case of obstructive sleep apnea were to provide a means for the patient to have a patent airway (*i.e.*, CPAP at 14 cm. H_2O setting [1]). The next step was to treat the lifestyle factors that placed him at risk for sleep apnea and to monitor his progress in terms of improved sleep quality. These lifestyle factors are not unique to the patient's age level and treatments are common [2].

The first three months of CPAP use required adjustment on the part of the patient. In the most compliant of patients, variability during this adjustment period occurs [3]. Once a patient patent airway is set up, sleep stages return. The experience of sleeping and obtaining quality sleep (*i.e.*, Stage 2) and perhaps some REM sleep allow the patient to feel more rested [3]. A more complicated neurophysiological worry is prominent in cases like this—of untreated sleep apnea—neurocognitive deficits. Either the sleep fragmentation leading to poor quality sleep and little executive control and/or the hemolytic changes that occur secondary to the lack of oxygen resulting in cognitive executive ability deficits have been found [4]. Neuroimaging techniques have been common the last fifteen years in OSA studies to identify cognitive deficits [5]. In many studies, combined results of neuropsychological testing [6] and imaging [7] have advanced the understanding of the nature of the deficits [8]. Focal reductions in gray matter volume in the left hippocampal cortex, left posterior parietal cortex and right superior frontal gyrus [4]. Significant changes in terms of both gray matter density and metabolic levels at the precuneus, parieto-occipital and prefrontal cortex have been found [7]. These findings, taken together, indicate a lack of brain activity in these regions [5]. Insulin resistance, metabolic syndrome are two serious outcomes of these metabolic effects [7].

Neurobehaviorally, the sleep apneic patient, like the gentleman in this case study have difficulty with tasks requiring attention, vigilance, working memory and visuo-spatial construction. The degree of apnea, its chronicity has been linked to marked changes in these abilities [9]. With the case studied, pre and post testing of these faculties was done to document the impact of his untreated apnea. The Wechsler Memory Scale subtest Forward Digits measures attention and vigilance. The Wechsler Memory Scale subtest Backwards Digits and Letter-Number Sequencing were used to determine working memory. The Purdue Pegboard Assemble subtest was used to measure visuo-spatial construction ability along with executive functioning. With treatment, neurocognitive functioning was improved as documented by the test scores. These findings along with self-reported sleepiness scale are reflected in **Table 1**. With some improvements in sleep, it was found in this case, that improvements in functioning were found [10]. Data illustrated in **Tables 1** and **2** indicate improvement in biobehavioral functioning (e.g., attention, concentration, visual-spatial/motor ability). The patient's self-reported sleepiness, residually, was believed to be related to lifestyle factors.

4. TREATMENT AND CONCLUSION

The bimonthly cognitive behavior therapy sessions focused on integrating exercise and good nutrition (this included cessation of alcohol use). Additionally, a date and means of quitting smoking was determined. The patient was given supportive education about the lifestyle factors that placed him at risk for OSA and after treatment ended, the experience of some of the symptoms.

Table 1. Biobehavioral assessment.

		Follow-up	
	At intake	1 m	3 m
Epworth sleepiness scale	23	20	14
Wechsler memory scale			
Forward digit	4.00	5.00	7.00
Backward digit	2.00	4.00	7.00
Letter-number sequencing	4.00	5.00	5.00
Purdue pegboard, assembly	16	24	26

Table 2. Polysomnograph findings*.

Baseline		CPAP (2 hours later)	
Sleep efficiency	37%	CPAP effective level	18 cm H_2O pressure
Stage nREM 1	83.3%	SpO_2 (on CPAP)	90%
Stage nREM 2	2.7%	Sleep Efficiency	76%
Stage nREM 3	0	Stage nREM 1	78%
Stage REM	0	Stage nREM 2	16%
		Stage nREM 3	1.2%
Apneas	146	Stage REM	4%
		Position: Supine	38%
SpO_2	79%		
Position: Supine	95%		

Note* = 1 and 3 month follow ups were in terms of compliance using the memory chip of CPAP use. At one month (44%) and three month (38%) compliance was determined.

While the patient participated in these sessions, the required logs and records were not complete thus prompting a question of compliance. Follow up sessions and reminder calls for these sessions were set up; the patient attended these sessions intermittently. He has continued to come in at six months and one year assessments of his sleep and CPAP titrations.

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