CASE REPORT

Central Sleep Apnea due to Positive Airway Pressure Mask Leakage

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Abstract

Complex sleep apnea is the term used for central sleep apnea (CSA) events that arise during positive airway pressure (PAP) titration in a patient undergoing polysomnography (PSG). Multiple mechanisms have been used to explain the same including hypocapnia, loss of central drive, circulatory delay, or arousals. However, PAP mask leakage is an easily correctable cause of CSA during titration studies. We hereby present a case of a middle-aged female who was diagnosed with severe obstructive sleep apnea (OSA), and developed central events during titration, which were easily corrected by readjusting the oronasal mask.

Keywords: Case report, Central sleep apnea, Mask, Obstructive sleep apnea, Positive airway pressure.

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Introduction

Sleep-disordered breathing (SDB) includes a broad spectrum of disorders such as obstructive sleep apnea (OSA), central sleep apnea (CSA), nocturnal hypoventilation, and hypoxemia. Obstructive sleep apnea is among the most commonly diagnosed SDBs characterized by absent airflow despite ongoing respiratory efforts during sleep.[1] Continuous positive airway pressure (CPAP) is the treatment of choice for OSA. However, it has been observed that some patients may develop CSA events after the use of CPAP for OSA. The entity "treatment-emergent central sleep apnea (TECSA)" was coined for this disorder. Subsequently, Gilmartin et al.[2] renamed it as "complex sleep-disordered breathing," and Morgenthaler et al.[3] labeled this as "complex sleep apnea syndrome (CompSAS)." Currently, the use of the terminology "complex sleep apnea syndrome" has increased. Complex sleep apnea syndrome is defined as follows: (A) Having ≥5 obstructive respiratory events seen per hour of sleep during a diagnostic portion of the sleep study; (B) Positive airway pressure (PAP) leading to correction of obstructive events and the occurrence or emergence or persistence of central apnea hypopnea with central index ≥5/hours, and the number of central events being ≥50% of the total; and (C) The occurrence of central events is not better explained by any other disease.[4]

Case Description

A 40-year-old obese female, working in an IT firm (desk job) presented with complaints of excessive daytime sleepiness for the last 6 months. She was recently married, and her partner complained of extremely loud snoring which even does not let her sleep. The complaints of excessive daytime sleepiness were persistent and progressive for 6 months to such an extent that the patient used to fall asleep as soon as she was sitting idle even in official meetings. She noticed that despite attempting to sleep for 8 hours in the night, she required a daytime nap of 2 hours. Recently, for the past 4 months, even her daytime naps were nonrefreshing and she awakens with a headache and persistent sleepiness. She had a near-accident 1 month back due to sleepiness while driving. The patient had got married 5 months back and her spouse complained of snoring by the patient since marriage which had become very loud. She noticed that snoring was less when the patient used to sleep prone and worsened when the patient slept after consuming alcohol.

There was no history of cough, sputum, chest pain, or wheezing. There was no history of tuberculosis. There were no comorbidities. The patient had no night-shift duties or irregular sleep timings. She does not take any sedative medications. The patient had gained 5 kg in the last 6 months and now weighed 110 kg.

On examination, the patient had stable vitals with SO_2 of 96%. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy. A general examination revealed a neck circumference of 49.5 cm and a modified Mallampati class III airway. The respiratory and cardiac evaluation was normal. The patient had an Epworth sleepiness score (ESS) of 19/24, STOP-BANG score of 6/8 suggestive of high risk for OSA, and BMI of 41 kg/m^2.

The patient underwent polysomnography (Fig. 1) which was suggestive of apnea–hypopnea index (AHI) of 45/hour suggestive of severe OSA. During titration with CPAP, the patient

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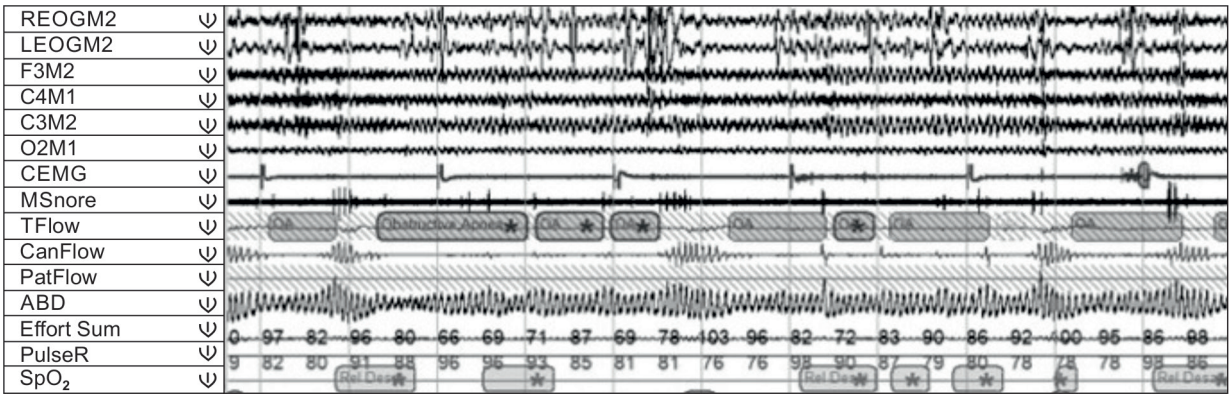

Fig. 1: Polysomnography 10-minute window showing OSA during diagnostic study

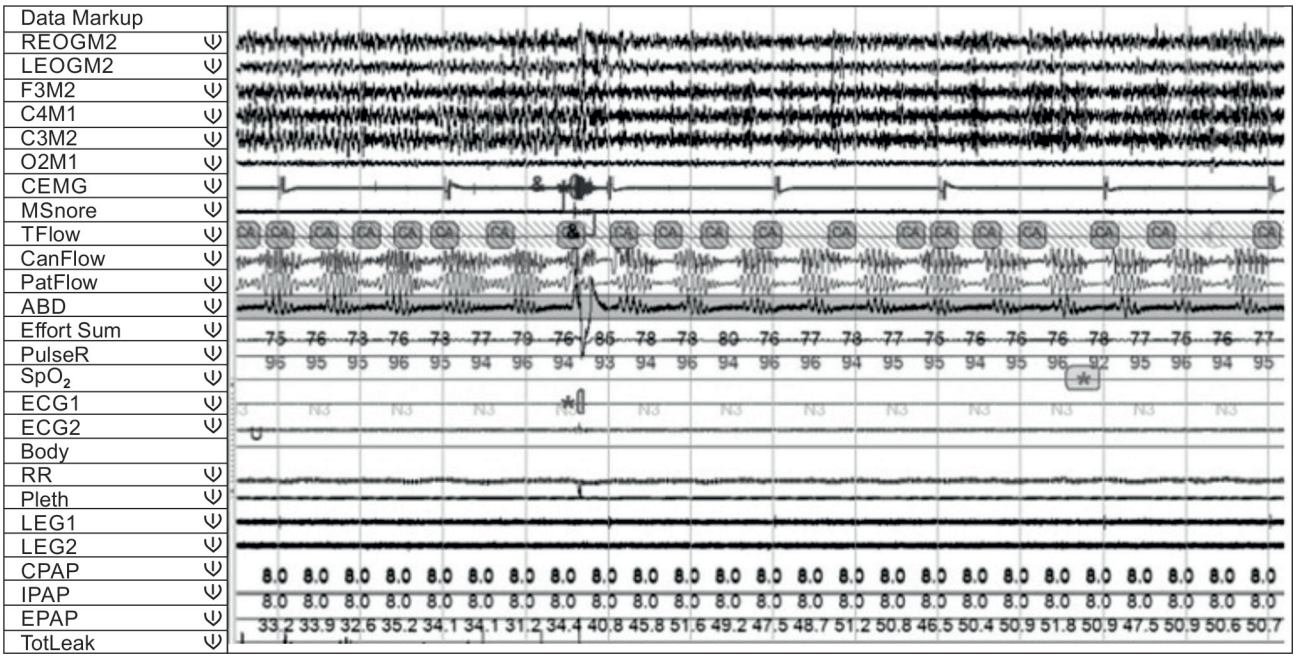

Fig. 2: Polysomnography 10-minute window showing CSA duration titration at 8 cm H_2O pressure

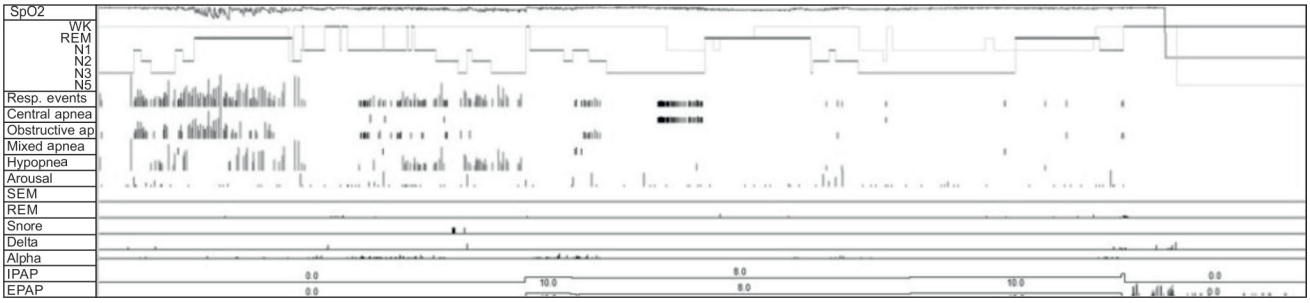

Fig. 3: Hypnogram showing obstructive apnea in the diagnostic part, central events in the titration part, and correction of central events on correcting mask leakage

developed CSA at a pressure of 8 cm H_2O (Fig. 2). The leak from the PAP mask had increased from 20 L to around 50 L. The mask was readjusted and the central events disappeared. The patient was further titrated to 10 cm H_2O pressure for correction of all obstructive respiratory events and prescribed the same. The daytime sleepiness and snoring were corrected within 10 days

of use and the patient felt more alert, energetic, and motivated to follow an exercise schedule. A follow-up at 3 months showed 100% compliance with CPAP use for more than 4 hours at night (Fig. 3).

Discussion

Factors associated with an increased likelihood of CompSAS include male sex, coexisting heart failure, severe OSA, over titration, presence of mixed apneas during initial polysomnography, use of bilevel positive airway pressure (BPAP) without backup rate and with higher pressures, opioid narcotics, high altitude, mouth breathing during sleep, and supine sleep position.[5] Patients with CompSAS are at higher risk of therapy termination and poor compliance.[6] The pathogenesis of CompSAS is poorly understood and may have multiple possible etiologies.

Loop Gain

Loop gain is defined as the ratio of the corrective ventilatory response to the disturbance leading to it during sleep. It occurs as a result of peripheral chemoreceptor response to arterial carbon dioxide tension ($PaCO_2$) in the bloodstream reaching the receptors. A higher pCO_2 level results in a rise in ventilatory drive, causing a decrease $PaCO_2$, which in subsequent cycle causes hypoventilation secondary to reduced ventilatory drive. Normally, a balance is reached whereby ventilation and $PaCO_2$ are at a steady state. In case the ventilatory response exceeds the prior disturbance (i.e., loop gain >1), it results in periodic CSAs. Patients with OSA commonly have high loop gain at baseline which is increased with PAP therapy, resulting in periodic episodes of CSA as described by the above mechanism, in response to slight disturbances such as arousals from sleep. High loop gain is more common in patients with severe OSA and those who receive bilevel (BPAP).[7]

Intermittent Hypoxia

Both acute and chronic intermittent hypoxia cause increased chemoreflex sensitivity leading to increased possibility of central apnea. Thus, patients with OSA are more prone to develop hypocapnic central apnea compared to a healthy population. This is also the explanation of the higher likelihood of developing CompSAS during split night versus full night PAP titration. Optimal treatment with CPAP therapy has been shown to decrease the chemoreflex sensitivity in subsequent months of therapy with resolution of central apneas.[8]

The CO_2 Excretion

Positive airway pressure therapy resolves the upper airway obstruction in OSA, which causes increased CO_2 washout and leads to central apnea when the $PaCO_2$ decreases to a level below the apneic threshold.[9]

Air Leak

Air leak during PAP titration can cause increased CO_2 elimination from the mask along with the anatomic dead space causing acute central apnea.[10]

An easily ignored etiology of the complex leap apnea leak is because of the PAP mask, which is not fitted properly.[11] In our case, correction of the leak resulted in the resolution of the central apnea events. The patient was titrated to an even higher level of PAP for correction of OSA.

Thus, a high index of suspicion needs to be kept for evaluating the leak during PAP titration in a patient of OSA who develops complex sleep apnea.

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