



BMH Med. J. 2017;4(2):69-72 **Case Report**

A Case Of Treatment Emergent Central Sleep Apnea

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Abstract

Treatment emergent central sleep apnea (CSA) is defined as the persistence or emergence of central apneas and hypopneas during the CPAP titration for the treatment of OSA. It is a main cause for no improvement with CPAP therapy in an OSA patient. Here we report a case of treatment emergent CSA who presented initially with obstructive sleep apnea and later he developed central apnea with CPAP therapy.

Keywords: treatment emergent central sleep apnea, polysomnography

Case Report

Middle age male presented with increased day time sleepiness and snoring for the past 10 years. There was history of awakening with choking, two episodes during the past 2 months. There was no history of morning headache or lack of concentration. On further enquiry he had history of nocturia and increased fatigue. There was no history of dyspnea, orthopnea, paroxysmal nocturnal dyspnea, chest pain or palpitation.

He had history of sneezing episodes and rhinitis for the past 2 years on exposure to dust for which he was not on regular medication. He was having hypertension for past 3 years and was on anti hypertensives. He had Epworth Sleepiness Scale (ESS) of 14. He was a nonsmoker.

On examination, his weight was 60 Kg and his height was 159 cm. He had a BMI of 28.7. His neck circumference was 15.5 inches (40 cm). He had no craniofacial abnormalities and no thyroid swelling. He had class 3 Mallampatti score. His respiratory and cardiovascular examination findings were normal. His routine blood counts, renal, hepatic and thyroid function tests were within normal limits. His echocardiogram was normal. Polysomnography (PSG) (**Figure 1 and 2**) showed obstructive events followed by desaturation.

Polysomnography report showed:

Obstructive Sleep Apnea (OSA) events – 55/hr
Central Sleep Apnea (CSA) events – 1/hr
Hypopneic events – 8/hr
Apnea Hypopnea Index (AHI) – 65.7/hr

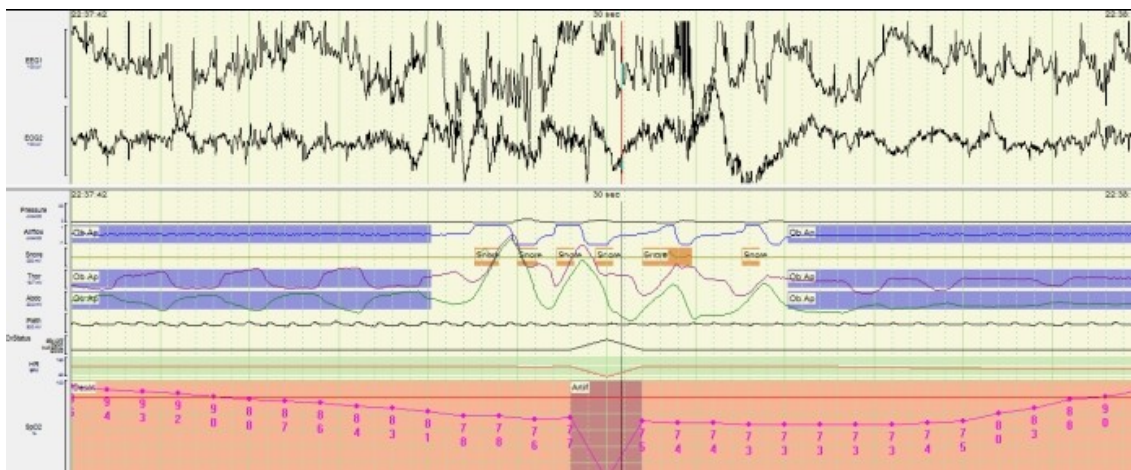


Figure 1: Polysomnography in a 30 sec epoch showing OSA events followed by desaturation.

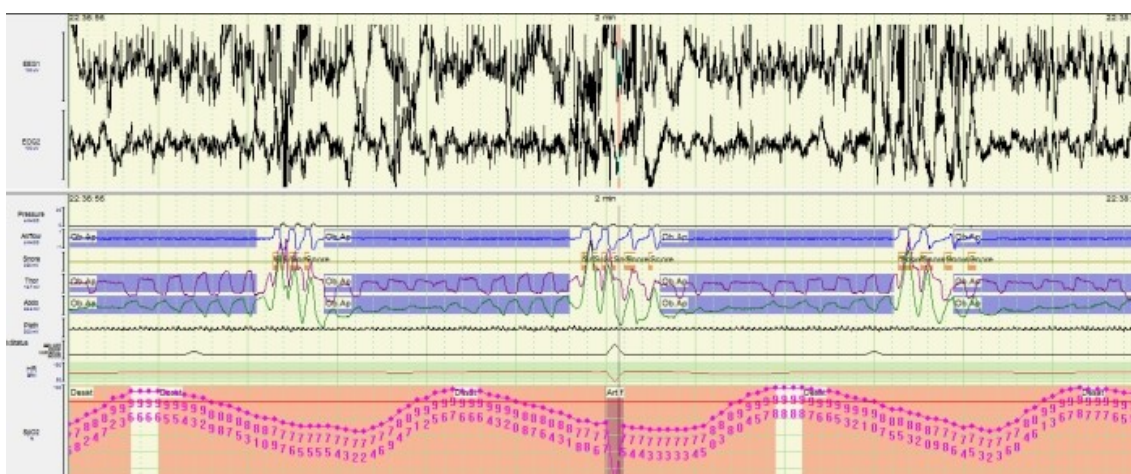


Figure 2: Polysomnography in a 2 min epoch showing multiple OSA events followed by desaturation.

We diagnosed it as severe obstructive sleep apnea syndrome and he was advised auto CPAP therapy during night hours and for follow up after one month.

His symptoms was only mildly relieved after one month. Memory card in his auto CPAP machine showed that he had used it for more than 4 hrs every night and there were desaturation events while on use. We repeated PSG with Continuous Positive Airway Pressure (CPAP) titration to check whether his obstructive symptoms were getting relieved or not with CPAP. We used auto - CPAP at a pressure of 13-15 cm H₂O with a full face mask. (Figure 3 and 4)

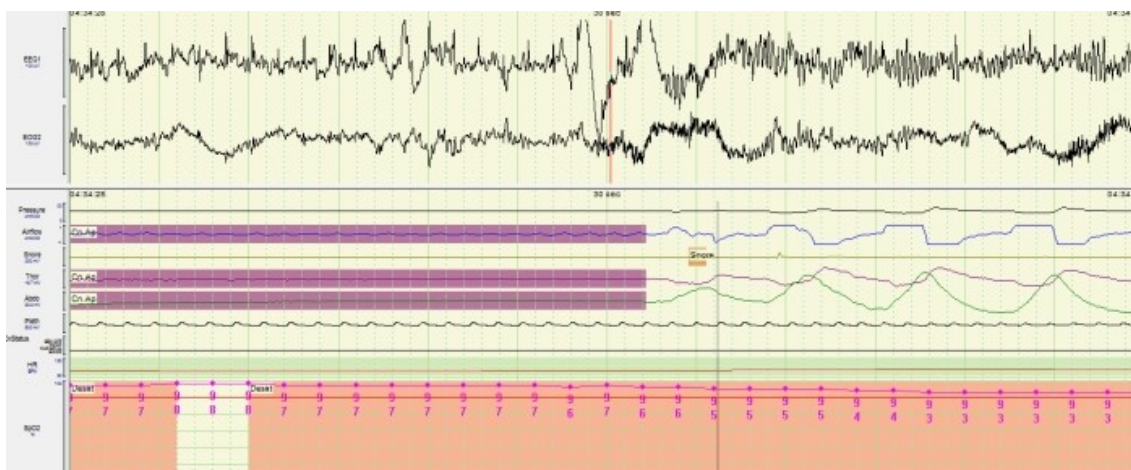


Figure 3: Polysomnography in 30 sec epoch showing central apnea event followed by desaturation while on CPAP therapy.

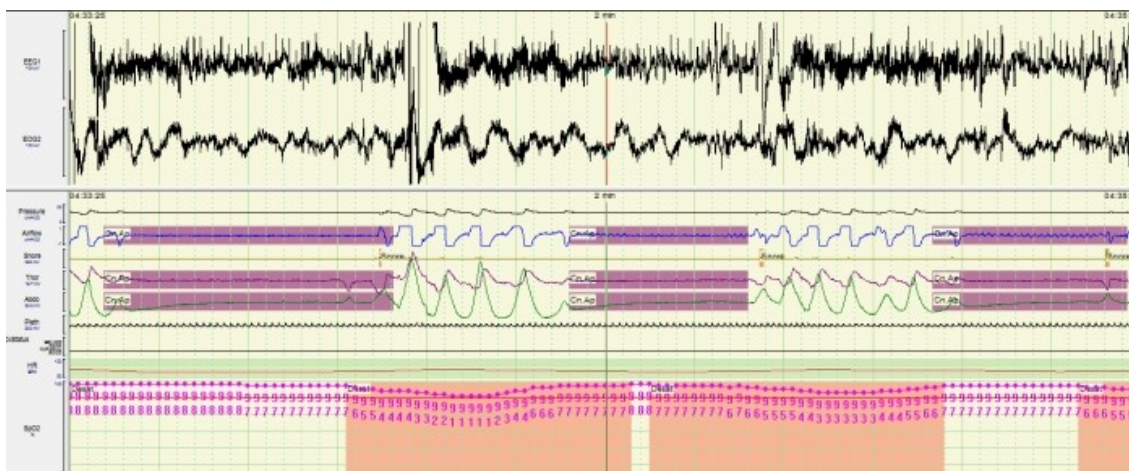


Figure 4: Polysomnography in 2 min epoch showing multiple central apnea events with desaturation.

PSG report came as:

OSA events – 0/hr
 CSA events – 16/hr
 Mixed apneic events -1/hr
 AHI – 17/hr
 Central apnea index -16/hr

Comparing the results of PSG before and after the CPAP titration (Table 1):

Diagnostic PSG	PAP Therapy
OSA – 55/hr	OSA – 0/hr
CSA – 1/hr	CSA – 16/hr
Hypopnea – 8/hr	Mixed apnea -1/hr
AHI -65.7/hr	AHI-17/hr

Table 1

Obstructive events had almost fully disappeared and central apneic events had appeared. Hence we reached a diagnosis of *Treatment Emergent Central Sleep Apnea*.

As the treatment of treatment emergent CSA is CPAP trial for three months, we advised him the same. The patient came for follow up after three months and his symptoms were considerably relieved. Hence we did not repeat PSG. He is on regular follow up.

Discussion

Treatment emergent CSA, also known as complex sleep apnea is defined as the persistence or emergence of central apneas and hypopneas during the CPAP titration for the treatment of OSA [1]. The prevalence of complex sleep apnea has been estimated as 5-15% of OSA patients who undergo positive airway pressure (PAP) therapy [1]. It usually occurs in males or with occult heart failure, severe obstructive sleep apnea, use of over or under titration of CPAP therapy, high altitude, oral breathing, use of respiratory depressant medication such as opioids and benzodiazepines as well as sleep in supine position.

Pathogenesis include intermittent hypoxia which increases chemoreflex sensitivity leading to development of central apnea [2]. Other causes include stretch receptor activation, increased carbon dioxide excretion, and air leak. PAP therapy activates stretch receptors, which inhibits central

respiratory output and causes central apnea. Air leak causes increased CO₂ wash out leading to decrease in PaCO₂ below apneic threshold causing central apnea.

Diagnostic criteria for treatment emergent CSA according to the third edition of the International Classification of Sleep Disorders (ICSD3) include all of the following [1]:

- Diagnostic polysomnography: ≥ 5 obstructive events/hr
- Polysomnography during PAP therapy: Significant resolution of obstructive events and emergence or persistence of central apnea or hypopnea with
 - central apnea index ≥ 5 /hr
 - central apneas and hypopneas is $\geq 50\%$ of total apneas
- CSA is not better explained by another CSA disorder (CSA with Cheyne stokes respiration or CSA due to medication)

Usual treatment is expectant management with CPAP. 2/3rd of patients improved with CPAP within 90 days [3]. Repeat polysomnography recommended after 3 months to check whether symptoms got improved or not. Other treatment modalities include adaptive servo ventilation (ASV) and BPAP with a back up respiratory rate. ASV provides varying amount of inspiratory pressure superimposed on a low level of CPAP with a back up respiratory rate. But ASV devices are more expensive than CPAP.

Conclusion

Reason for no improvement in OSA after CPAP therapy can be treatment emergent CSA. In 50-70% patients it will resolve spontaneously with PAP therapy [4]. Patients who are not responding will require review and other modes of PAP therapy.

References

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