Medicina Clínica Práctica 4 (2021) 100218

MEDICINA CLINICA PRÁCTICA

www.elsevier.es/medicinaclinicapractica

Original article

Excessive daytime sleepiness due to obstructive sleep apnea☆

Silvia Velika[a], Cahyono Kaelan[b,*], Hasmawaty Basir[a], Audry Devisanty Wuysang[a], Jumraini Tammasse[a]

[a] Department of Neurology, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[b] Department of Anatomic Pathology, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia

ARTICLE INFO

Article history:
Received 24 September 2020
Accepted 15 October 2020

Keywords:
EDS
CPAP
OSA
Respiratory Disturbance Index

ABSTRACT

Excessive Daytime Sleepiness (EDS) is a symptom that arises anytime from a tendency to drowsiness or to fall asleep at the time of intensity and expectation to stay awake. The most common cause associated with EDS is obstructive sleep apnea (OSA). OSA is a sleep disorder characterized by partial or complete obstruction of the airways that causes apnea and hypopnea during sleep. The prevalence of OSA associated with a decrease in sleep quality is *approximately* 3–7% in adult *men* and 2–5% in adult *women in the general population*.

This case presents a 39-year-old man complains of excessive sleepiness during the day for10 years with a duration of 10–15 min, often waking up at night and feeling tired all day. Overnight polysomnography showed Respiratory Disturbance Index 21.6. The patient received 6 cmH_2O of Continues Positive Airways Pressure (CPAP) therapy every night during sleep and modafinil 100 mg once a day.

Introduction

Excessive Daytime Sleepiness (EDS) is defined as a symptom that arises at any time from a tendency to get sleepy or to fall asleep when the intensity and expectation of staying awake.[1] EDS complaints are usually interpreted as feelings of drowsiness, decreased energy, fatigue that cannot be controlled. Kevin R. has reported the prevalence of EDS ranging from 3% to 20% chosen randomly in the elderly and as many as 22.6% in people who have EDS with occupational accidents.[2] Around 65 primary sleep disorders can cause EDS. The most common cause associated with EDS is obstructive sleep apnea (OSA).[3]

Sidney Barwell first discovered OSA in 1956, which is a condition of breathing problems during sleep due to blocked airflow.[4] The prevalence of OSA in the United States is estimated to be around 5–10% of the population. The prevalence of OSA associated with decreased sleep quality is around 3–7% in men and 2–5% in women in the general population.[5] OSA prevalence data in Indonesia are not known with certainty. His previous research reported that OSA occurred as much as 30–40% in the taxi driver population in Indonesia.[6]

Case report

Male 39 years old, from Afghanistan came with complaints always sleepy during the day since ten years ago with a duration of 10–15 min anywhere and anytime, and accompanied by headaches every morning and feeling tired throughout the day. Patients also complain often wake up at night. History of hypertension, diabetes mellitus, epilepsy, head trauma is absent. The patient has a BMI of 22.94, blood pressure 130/80 mmHg, physical, neurological examination within limits normal.

In this patient, PSG was performed twice at the Indian Batra Hospital in 2015. The first PSG was performed for diagnostics and the second for therapy. The first PSG was done with a total recording of 447.4 min with a total sleep time of 412 min and a sleep efficiency of 92.2%. Sleep latency in this patient is 0. Total 15% REM sleep with REM latency: 53 min, and 16.7% stage 1 sleep, 42.8% stage 2 sleep, 16.5% stage 3 sleep, and 8.9% sleep stage 4. The respiratory analysis showed 11 times obstructive apnea and one time mixed apnea with a total of 12 times apnea and apnea index 1.7. There was 136 times hypopnea with RDI 21.6. Patients use 100% of total sleep time in the supine position, with RDI 21.6 supine and non-supine RDI 0.0. RDI in REM sleep 5.8, and RDI in NREM sleep 24.3. The lowest desaturation is 89, with 0.1 min desaturation between 81% and 90%. Periodic leg movement is recorded with a PLM index of 13.0 and a PLM index with the arousal of 0.0. The total arousal index increased by 32.6. Respiratory arousal index 32.6. The second PSG was performed with a complete recording of 461.9 min

☆ Peer-review under responsibility of the scientific committee of the Technology Enhanced Medical Education International Conference (THEME 2019). Full-text and the content of it is under responsibility of authors of the article.

* Corresponding author.
E-mail addresses: cakaelan@yahoo.com, pmc@agri.unhas.ac.id (C. Kaelan).

https://doi.org/10.1016/j.mcpsp.2021.100218

with a total sleep time of 421.5 min and a sleep efficiency of 91.3%. Sleep latency in this patient is 1.5. 8.5% of total REM sleep with REM latency: 200.5 min, and 15.7% of stage 1 sleep, 51.2% of stage 2 sleep, 16.6% of stage 3 sleep, and 7.9% of stage 4 sleep. Respiration analysis showed 0 obstructive apnea and 0 apnea mix from a total of 1 apnea and apnea index 0.1. There are 11 hypopnea with Respiratory Disturbance Index (RDI) 1.7. Patients used 100% of the total sleep time in the supine position, with supine RDI 1.7 and non-supine RDI 0.0. RDI in REM sleep 15.0 and RDI in NREM sleep 0.5. The lowest desaturation is 0, with 0.0 min desaturation between 81% and 90%. Periodic leg movement is recorded with a PLM index of 12.7 and a PLM index with the arousal of 0.0. The total arousal index increased by 27.0. Respiratory arousal index 27.0. In this patient using The Epworth Sleepiness Scale, a score of 18. Patients were recommended to receive six cmH_2O CPAP therapy every night during sleep. This patient received modafinil 100 mg once a day.

Discussion

OSA is one of the Sleep-Related Breathing Disorders according to the 3rd edition of the International Classification of Sleep Disorders. OSA is a sleep disorder characterized by a partial or complete blockage of the airways that causes apnea and hypopnea during sleep. Apnea and hypopnea occur at least 10 s. Most apnea and hypopnea last 10–30 s but can sometimes continue for up to 1 min or more. Hypopnea is a partial obstruction of the upper respiratory tract accompanied by desaturation of oxygen at least 3% or followed by arousal for at least 10 s. The incidence of apnea and hypopnea usually lasts longer, and oxygen desaturation is more severe at the REM stage and supine position. Symptoms of OSA can include snoring, stopping breathing during sleep, suffocating or choking, not refreshing sleep, excessive sleepiness throughout the day, nocturia, morning headaches, decreased concentration, and memory.[4]

The primary risk factor for OSA is obesity 70% of patients with OSA are obese (BMI > 30).[7] The accumulation of fat in the airway will cause constriction and tend to close when the muscles are loose, especially in the REM sleep phase. Based on previous studies, it was mentioned that neck circumference was more strongly related than BMI, where neck circumference ≥17 inches in men and ≥16 in women correlated highly with OSA.[8]

Before conducting therapy, OSA has to be determined along with the severity of OSA. The criteria for the diagnosis of OSA are based on clinical symptoms found in a comprehensive sleep evaluation. The gold standard for diagnosis of OSA is overnight polysomnography. The severity of the disease is determined by the Respiratory Disturbance Index (RDI). RDI is the amount of apnea, hypopnea, and RERA (Respiratory Event Related Arousal) per hour of sleep. Based on the American Academy of Sleep Medicine criteria, the severity of OSA is divided into[4]:

a. Normal: RDI < 5 times per hour
b. *Mild* OSA: RDI ≥ 5–< 15 times per hour
c. *Moderate* OSA: RDI ≥ 15–≤ 30 times per hour
d. *Severe* OSA: RDI > 30 times per hour

In this patient on the first PSG examination, the RDI 21.6 was obtained. Based on the above criteria, patients can be categorized as moderate OSA. In two PSG measurements, it was found that the patient was 100% sleeping in the supine state, and RDI at supine position 21.6 and while RDI at the time of non-supine position is 0. Several studies reveal the relationship between sleep position and sleep apnea that avoiding supine position during sleep can reduce the number and severity of episodes of obstruction. In the supine caliber position and upper airway resistance is higher, and the upper airway tendency for a collapse is much more significant in the supine position than the lateral position.[9] OSA is seen as a chronic disease that requires long-term multidisciplinary management. In some patients, respiratory disturbances arise when supine position. The use of a wedge pillow can be used to limit sleep in a supine position.[8]

Nasal therapy continuous positive airway pressure (CPAP) was first proposed in 1981. CPAP is almost always effective if the pressure is right. The limitation of using CPAP is the patient's acceptance and tolerance of this tool. This therapy works as a pneumatic splint to prevent airway collapse. The result is a decrease in daytime drowsiness, improved function, and overall daily activities. The desired PAP pressure is determined based on the titration that can be performed during PSG. CPAP titration is carried out according to AASM recommendations. The minimum pressure when starting CPAP is 4 cm H_2O in both children and adults. Maximum CPAP pressure is 15 cm H_2O for patients <12 years and 20 cm H_2O for patients ≥12 years. CPAP pressure must be increased by 1 cm H_2O every 5 min if obtained[10]:

a. One OSA event in a patient <12 years or 2 OSA events in a patient ≥12 years, or
b. One hypopnea event in patients <12 years or 3 hypopnea events in patients ≥12 years, or
c. RERA in patients <12 years or 2 cases of OSA in patients ≥12 years

CPAP pressure can be increased if there is a snoring sound for 1 min in patients <12 years or for 3 min in patients ≥12 years. CPAP can be changed to BPAP if the patient feels uncomfortable or if OSA is obtained at maximal CPAP pressure. CPAP titration is said to be optimal if the RDI drops <5 for at least 15 min. Titration is proper if the RDI drops ≤10, or the initial falls 50% from the initial value if found RDI < 15. Intrusion is secure if the titration does not decrease RDI ≤ 10 or down 75% from the initial value of RDI. Titration fails if it does not meet the criteria.[9]

Surgical therapy for OSA includes minimally invasive procedures such as radiofrequency uvular ablation and invasive procedures such as uvulopalatopharyngoplasty (UPP). The success rate of surgery varies depending on the procedure used, but for the UPP, the success rate reaches 44–55%. In some patients with adequate apnea therapy, EDS is still a major problem. In these patients, they received additional modification therapy to improve symptoms.[8] Modafinil is first-line therapy for daytime sleepiness. Modafinil affects the P450 enzyme system, induces 1A2 and 3A, and inhibits 2C19. Therefore there will be drug interactions in patients taking contraceptive drugs, benzodiazepines, and drugs that are metabolized by the P450 system.[11]

Conclusion

This paper reports a case of an OSA sufferer who gives symptoms of excessive sleepiness during the day. OSA was established on PSG, with RDI 21.6 and EDS diagnosis was established using The Epworth Sleepiness Scale questionnaire with a total score of 18.

Conflict of interest

The authors declare no conflict of interest.

References

1. Rosias PPR, Jöbsis Q, Van De Kant K, Robroeks C, Van Schayck CP, Zimmermann LJI, et al. Global condensation: a "climate change" towards better standardisation of exhaled breath condensate measurements. *Eur Respir J* 2008;**31**:684–5, http://dx.doi.org/10.1183/09031936.00157907.
2. Carskadon M. Evaluation of excessive daytime sleepiness. *Neurophysiol Clin/Clin Neurophysiol* 1993;**23**:91–100, http://dx.doi.org/10.1016/S0987-7053(05)80287-4.

3. Lee W, Nagubadi S, Kryger MH, Mokhlesi B. Epidemiology of obstructive sleep apnea: a population-based perspective. *Expert Rev Respir Med* 2008;**2**:349–64, http://dx.doi.org/10.1586/17476348.2.3.349.
4. Bixler EO, Vgontzas AN, Lin HM, Calhoun SL, Vela-Bueno A, Kales A. Excessive daytime sleepiness in a general population sample: the role of sleep apnea, age, obesity, diabetes, and depression. *J Clin Endocrinol Metab* 2005;**90**:4510–5, http://dx.doi.org/10.1210/jc.2005-0035.
5. Morgenthaler TI. Diagnosis of obstructive sleep apnea in adults. *Ann Intern Med* 2015;**162**:455, http://dx.doi.org/10.7326/L15-5062.
6. Wiadnyana IPGP, Susanto AD, Amri Z, Antariksa B. Prevalensi Kemungkinan Obstructive Sleep Apnea dan Faktor-Faktor yang Berhubungan pada Pengemudi Taksi X di Jakarta. *J Respirologi Indones* 2010;**30**:59–64.
7. Faizah N, Ulfiana E, Hadisuyatmana S. The correlation of sleep quality factors in overweight adolescents in a developing country. *Indian J Public Heal Res Dev* 2019;**10**:1416–21.
8. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Susheel P, et al. Clinical guideline for the evaluation management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 2009;**5**(January):263–76.
9. Mador MJ, Kufel TJ, Magalang UJ, Rajesh SK, Watwe V, Grant BJB. Prevalence of positional sleep apnea in patients undergoing polysomnography. *Chest* 2005;**128**:2130–7, http://dx.doi.org/10.1378/chest.128.4.2130.
10. Kushida CA, Chediak A, Berry RB, Brown LK, Gozal D, Iber C, et al. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. *J Clin Sleep Med* 2008;**4**:157–71.
11. Ishizuka T, Murotani T, Yamatodani A. Modanifil activates the histaminergic system through the orexinergic neurons. *Neurosci Lett* 2010;**483**:193–6, http://dx.doi.org/10.1016/j.neulet.2010.08.005.